Principles of Orthopedic Practice for Primary Care Providers

Andrew J. Schoenfeld
Cheri A. Blauwet
Jeffrey N. Katz
Editors

Second Edition

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Preface

It is a rare privilege to be able to engage in the creative process of generating a second edition of a text, as we have tried to do here with *Principles of Orthopaedic Practice for Primary Care Providers*. The original title was devised as an homage to the original *Principles of Practice* developed by Sir William Osler and which still today serves as a fundamental cornerstone in general medical care. Our hope was that we could provide similar guidance in the arena of orthopaedics and musculoskeletal medicine and the invitation to compose a second edition may signal that we were able to achieve this goal, even to some small degree. In this second edition, the editors and authorship team have sought to provide a balance of new and classic information relevant to decision-making in the orthopaedic realm, from spinal disorders to sports medicine, joint replacement, and the management of patients with osteoporosis. The second edition also introduces new chapters on pain management, adult spinal deformity, stress and running injuries, and the management of costs that we feel will be of value to primary care clinicians. We hope that the presentation of old and new in this second edition will meet the needs of primary care providers and help to inform the care of patients with orthopaedic and musculoskeletal conditions.

Boston, MA, USA

Andrew J. Schoenfeld
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Acknowledgments

I recognize that there is insufficient space and time to thank all of those who have contributed to, and aided me in, my life's journey and work—of which this text represents but a small part. That said, I want to thank my wife Erin for her ever-present love, dedication, and support—as well as the innumerable sacrifices made as we have pursued this career in academic medicine together. I also want to thank my children: Roman, Alyssa, and Leo, who continue to inspire, entertain, and drive me to do better. All of my accomplishments in life stand in the light and grace of God, who I know has given me more than I can ever hope to repay. I also want to thank the patient faculty and members of the Department of Orthopaedic Surgery at Brigham and Women's, many of whom graciously contributed their time and expertise to the composition of this text. I also could not have asked for a better and more energizing team of co-editors as Jeff and Cheri. As always, I further acknowledge the inspiration and support provided in words and ways seen and unseen by Patricia and David Schoenfeld; Lena and Abraham Schoenfeld; Eneida and Carlos Weber; Laura Ortiz Weber; the Andrew who is always with me; Papito, Madrina, and Madama Isabel Monsanto; Doña Juana Mercader; Josefa de Soler; Amelia Mercader; Judah Loew; Israel Baal Shem Tov; and Nachman, the son of Feiga. I dedicate my portion of this second edition to all of you.

Andrew J. Schoenfeld, MD, MSc

Dedicated to Eli, Stella, and Spencer who bring light to my day and remind me to live with gratitude.

Cheri A. Blauwet, MD

Dedicated to the memory of Dr. Robert Sheon, a brilliant, humble, inspiring pioneer in the evaluation and management of musculoskeletal problems.

Jeffrey N. Katz, MD, MSc

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Part I
The Spine

Chapter 1
Axial Neck and Back Pain

Jay M. Zampini

Introduction

Greater than 80% of all adults will, at one time or another, experience back pain debilitating enough to impair activities of daily living, occupational performance, or quality of life. Although the lumbar spine is affected more frequently than the cervical or thoracic regions, pain that affects any segment of the spine can be termed "axial spinal pain" and should be distinguished from conditions with neurogenic pain, such as neurogenic claudication and radiculitis. The pathophysiology and treatment of axial spinal pain differ from that of the neurogenic conditions, though the two may be present concomitantly. This chapter will review the pathophysiology, evaluation, and treatment of axial pain in the neck and back.

Definition and Epidemiology

Axial pain is defined as pain localized to one or more regions of the spine and/or SI joints without radiation into the lower extremities. It typically is present at all times and not necessarily aggravated by ambulation or activity. Pain may be lessened with rest or lying flat, but this does not have to be the case and is not required for a diagnosis. There are a number of factors that may be responsible for axial pain including joint dysfunction, degenerative changes, trauma, tumor or infection, myofascial structures, and non-organic pain generators.

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With greater than 80% of the adult population experiencing axial spinal pain at some point in life, and many not seeking medical care, it is difficult to make conclusive epidemiologic statements about populations at risk. It can almost be stated that anyone who lives long enough is at risk for back pain. Certain factors are known to associate with a higher risk of chronic axial pain, including obesity, tobacco use, total body vibration as may occur in long-distance truck driving or using a jackhammer, and repetitive hyperextension activities of the lumbar spine.

Clinical Presentation

Pain History

The evaluation of axial spinal pain is no different than any other pain evaluation and should include the time of onset, location of maximal pain, duration, severity, and associated symptoms. An inciting event should be noted if possible. A patient should be asked to consider events in the 2–3 days preceding the onset of pain since the inflammation, which often causes axial spinal pain, will increase over this time period. Body positions or maneuvers that exacerbate or alleviate the pain should be sought as should other associated symptoms. Patients should also be queried as to whether similar symptoms have presented in the past.

A thorough axial pain evaluation is then performed, with consideration given to the structures that may be pain generators. All spinal structures can potentially cause pain. These structures include the vertebral body and disc in the anterior spine; facet joints, other bony processes, interspinous and supraspinous ligaments, and SI joints posteriorly; as well as the myofascial tissue in all spinal regions (Fig. 1.1). As each of these structures performs a unique function, they also possess characteristic patterns of pain that may be elucidated through the history and physical exam. The pain patterns typically associated with dysfunction of each key spinal structure are summarized in Table 1.1.

The history of axial pain should clearly document the presence or absence of any "red flag," signs, and symptoms. A history of acute, high-energy trauma, such as car accidents or falls from greater than standing height, would suggest the need for emergent evaluation. Constitutional symptoms, such as unintended weight loss in excess of 10% of body weight or unexplained fevers or chills, would suggest the need for a neoplastic or infectious work up. Other neurologic "red flags," such as bowel or bladder retention or incontinence, should be sought to identify potential neurologic emergencies.

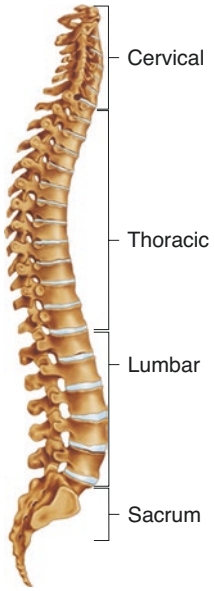

Fig. 1.1 Schematic of the human spine. The spine contains four zones: cervical, thoracic, lumbar, and sacrum

Table 1.1 Pain patterns typically associated with dysfunction of key spinal structures

	Myofascial	Fracture	Discogenic	Facetogenic	Sacroiliac
Injury identified	No	Yes	No	No	No
Tenderness	Trigger point	Focal	No	Focal	Focal
Exacerbating factors	Muscle stretch or activation	Spinal motion	Prolonged sitting or standing	Spinal hyperextension	Forced SI joint motion
Alleviating factors	Muscle rest	Immobilization	Recumbency	Recumbency	Recumbency
Neurologic symptoms	None	Possible	Possible	Possible	None
Referred pain[a]	None	Possible	Possible	Possible	Possible

[a]Cervical spine conditions can cause referred pain between the occiput and the lower scapulae, depending on the spinal level of the condition. Lumbar conditions can cause referred pain to the buttock and posterior thighs

Physical Examination

A specific diagnosis of axial pain can be made most often by the history alone. The physical examination serves to confirm the expected diagnosis. For most patients, it is useful to examine all aspects of the spine not expected to be painful before focusing on the structure anticipated to be the pain generator, since the examination is sure to exacerbate the pain at least temporarily. Any involuntary guarding associated with increased pain can obscure other aspects of the evaluation such as the

neurologic examination. Examination of the sensory, motor, and reflex functions can often be performed first and without any additional discomfort to the patient. This should be followed by a standing examination of the spine. Spinal curvature and posture should be evaluated with attention to shoulder height, pelvic obliquity, and any deviation of spinal balance. Spinal balance generally means that the patient's head is centered over the pelvis in both the sagittal and coronal planes. Gait should be examined from this position as well; attention should be paid to voluntary and involuntary alteration of gait to avoid pain and to any assistance device required for mobility. In the standing position, the spine should be palpated in the midline to determine if any bony tenderness is present. The musculature should be palpated next, again focusing on areas not expected to be tender before palpating potentially painful muscles. Spinal motion should be assessed last as this is often most painful for the patient. Objective measurements of spinal flexion, extension, lateral bending, and rotation, while valuable to document objective responses to treatment, are typically not as helpful for diagnostic purposes.

Next, provocative maneuvers should be performed for diagnostic confirmation if necessary. For axial spinal pain, provocative maneuvers are most useful for confirming the SI joint as the source of pain. A patient should be supine for most of these tests. One sensitive test of the SI joint is performed by passively flexing the hip on the painful side and then abducting and externally rotating the hip while the contralateral leg remains on the examination table. This maneuver—flexion abduction external rotation (FABER) test—compresses the ipsilateral SI joint and reproduces pain as a result. The test is positive if pain near the SI joint is reproduced. The test is nonspecific, however, since several structures are manipulated simultaneously (the hip joint, SI joint, lumbar spine, musculature), and should be followed by other confirmatory tests. If pain at the SI joint can be reproduced by compressing the pelvis either by using bilateral, posteriorly directed pressure on the anterior superior iliac spines (ASIS) in the supine position, the AP pelvic compression test, or by pressure on the greater trochanter with the patient in the lateral decubitus position, the lateral pelvic compression test, then the painful structure can be confirmed to be the SI joint.

Provocative testing of the facet joints or palpation that reproduces pain in this area, or over myofascial structures, can also be helpful in formulating a differential diagnosis. Extension of the neck and lumbosacral region that reproduces axial pain may also indicate the facet joints as a potential source of symptoms. Pain exacerbated on forward flexion at the lumbosacral junction and also reproduced with axial loading of the shoulders may be indicative of discogenic pain. A final aspect of the physical examination includes evaluation of other potentially painful joints in the upper or lower extremities to rule out these structures as additional pain generators or contributors to the overall constellation of symptoms. It is important to realize that there may be more than one clinical entity responsible for symptoms, and there is emerging appreciation for the interplay between the spinal and pelvic structures, as well as the neck and shoulder girdle, in pain syndromes. These clinical conditions are now frequently referred to as "neck-shoulder syndrome" or "hip-spine syndrome."

One further consideration in the examination of a patient with axial pain is the impact of psychological somatization and symptom magnification. These patients will perceive pain that is either present without any physical disruption of a spinal structure or out of proportion to what would be expected by the physical condition. To make this determination requires a nuanced approach to patient evaluation; several classic findings, termed Waddell's findings, have been reported to correlate with somatization and symptom magnification. Gentle downward compression of a patient's head does not cause any motion of the lumbar spine and should, therefore, cause no low back pain. Similarly, if spinal motion is simulated—with rotation of the shoulders, back, and pelvis at the same time—the spine itself is not affected, and no pain should be experienced. Finally, light touch of the skin overlying the spine should not produce pain. Observation of pain with any of these maneuvers should alert the clinician that non-organic factors are contributing to the patient's pain and should be taken into account when planning further evaluation and treatment.

Differential Diagnosis and Diagnostic Testing

Myofascial Pain

Muscles are the structures most susceptible to fatigue and overuse injury as well as to injuries resulting from acute demand exceeding muscle capacity. These injuries collectively comprise the most common cause of spinal pain and are generically called strains. Activation or passive stretch of the injured muscle will exacerbate the pain. Palpation will reveal focal, typically unilateral tenderness at the site of muscle injury. Multiple painful triggers may be encountered in the paraspinal musculature of patients with myofascial pain syndromes, such as fibromyalgia. Imaging does not help confirm a diagnosis but does rule out other potential etiologies as a cause of pain.

Pain Associated with Fractures and Ligamentous Injuries

In both young and old patients, referred pain can be felt in a pattern characteristic of the level of injury. Injuries close to the upper cervical spine will have referred pain to the occiput; injuries of the lower cervical spine will have referred pain even as far distally as the lower aspect of the scapulae. Similarly, lumbar fracture patients can complain of referred pain to the buttocks or upper thighs. Dermatomal symptoms to the hands or feet do not represent referred pain and suggest that a full neurologic exam should be included. Palpation reveals focal tenderness at the sight of injury. Plain film and computed tomography (CT) imaging are used to diagnose or confirm a fracture. Magnetic resonance imaging (MRI) may be required if these initial studies are negative to evaluate for concomitant disc or ligamentous injury or to assess the acuity of a particular fracture.

Discogenic Pain

Several painful conditions have been shown to localize to the disc: tears of the annulus, herniated discs, and degenerative disc disease (Fig. 1.2). With an annular tear, patients complain of axial pain deep inside the spine and focally at or near the injury site. Pain is typically increased with lumbar flexion or sitting and relieved with lumbar extension or lying flat. Plain film images may be read as negative depending on the extent of degenerative changes involving the disc space (Fig. 1.3). MRI is the diagnostic test of choice and will accurately display the amount of disc degeneration at various levels within the spine (Fig. 1.4). As a result, this imaging modality is nonspecific and cannot identify which, if any of the degenerative discs identified, is the cause of a patient's axial pain.

Facetogenic Pain

Patients with painful, degenerative facet joints will complain of morning pain and stiffness of the back. Spinal extension increases the load borne by the facet joints, and patients will complain that this maneuver exacerbates the pain. Referred pain is often present with painful facets: upper cervical facet referred pain may be perceived along the occiput with lower cervical referred pain felt in the shoulders or scapulae. Lumbar referred pain is perceived within the buttocks, pelvis, or posterior thighs. Spinal extension may increase the sensation of referred pain. It should be noted that the discs

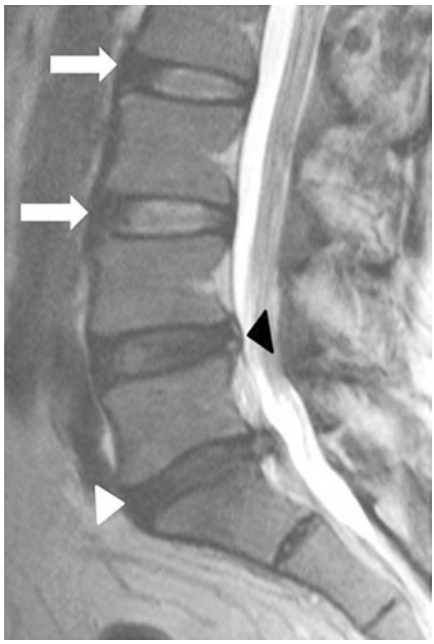

Fig. 1.2 This sagittal, T2-weighted MRI of the lumbar spine shows normal (*white arrow*) and degenerative discs. The degenerative discs show decreased disc height and low disc signal from loss of disc hydration (*white arrow head*) and annular tearing (*black arrow head*)

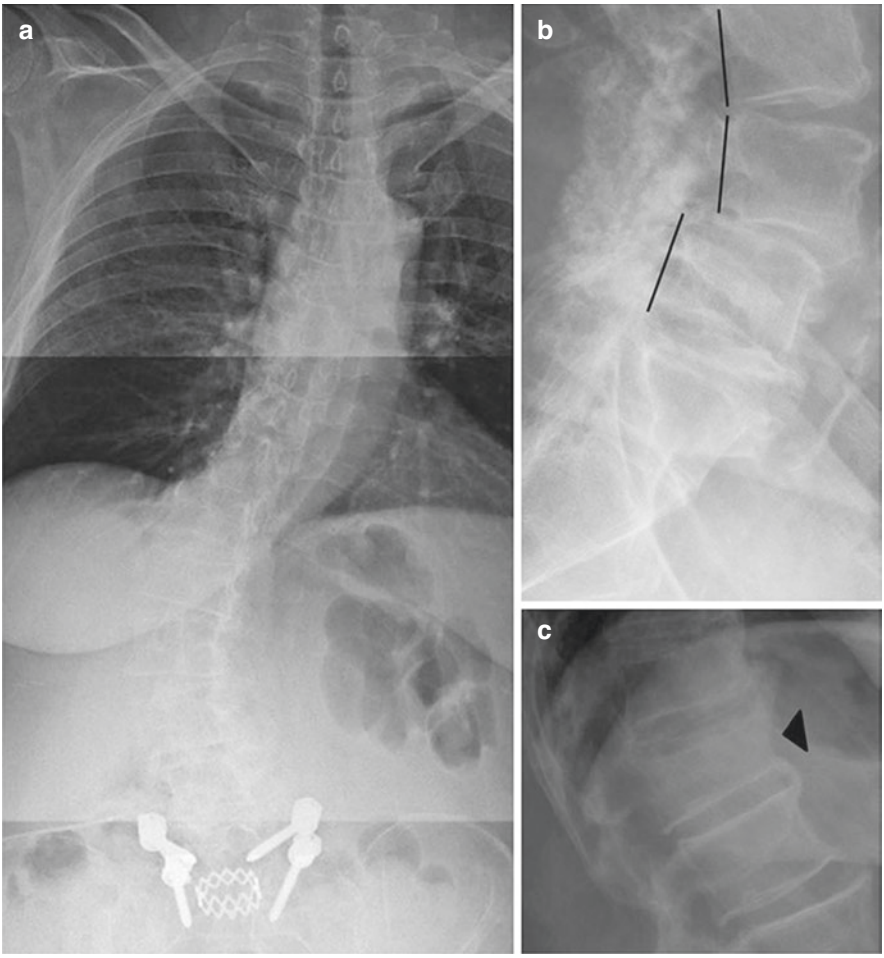

Fig. 1.3 Planar radiographs of the lumbar spine are ideal to identify and monitor scoliosis (**a**), spondylolisthesis (**b**), and compression fractures (**c**)

and facet joints age or degenerate concomitantly and may be symptomatic simultaneously. These patients will note that prolonged sitting and standing both exacerbate pain. Plain film, CT, and MR imaging can all demonstrate evidence of facet arthrosis, although none of these imaging modalities is considered a specific test.

Sacroiliac Pain

The SI joints form the link between the spine and pelvis. The joints are extremely stable as a result of strong ligaments on both the posterior and anterior aspects of the joint. Patients with painful sacroiliac joints complain of pain just medial to the posterior superior iliac spines, the bony prominences at the top of the buttocks. Patients

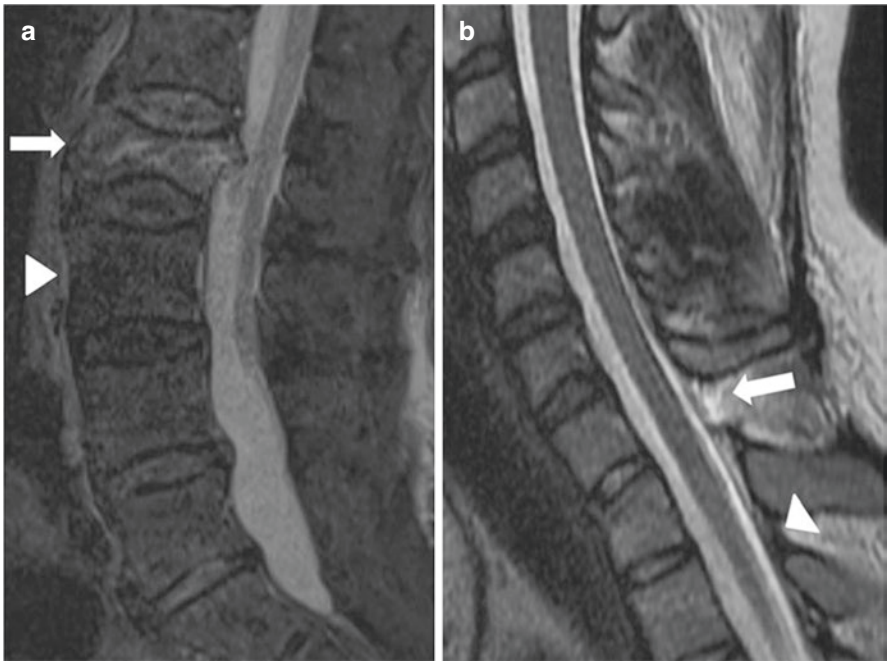

Fig. 1.4 MRI is useful for identifying the source of axial spinal pain including occult fractures (**a**) and ligament sprains (**b**). The occult fracture (**a**) is identified by the high STIR signal in the vertebral body (*arrow*) compared to low signal in an uninjured vertebra (*arrow head*). The ligament injury (**b**) is shown at the arrow compared to a normal-appearing ligamentum flavum seen at the level below (*arrow head*)

may experience pain with lumbosacral range of motion, ambulation, or single-leg stance. The unique location and function of the SI joints allows for a somewhat more focused examination than for other degenerative spinal conditions. At least three other provocative maneuvers (FABER test, thigh thrust, Gaenslen's test, and/or pelvic compression) should be positive to confirm SI pain with relative certainty. Plain film images and CT scans may show joint degeneration, while active inflammation or synovitis can be appreciated on MRI. The extent of findings localized to the SI joint does not necessarily correlate with the degree of a patient's SI-related pain.

Conditions Causing Referred Pain to the Spine

All evaluations of axial spinal pain should consider non-spinal sources as well. Visceral, vascular, autoimmune, neoplastic, and infectious conditions are responsible for 2–3% of all axial spine pain. These conditions often cause non-mechanical pain, or pain that does not change with spinal motion. Patients will report that they "Just can't get comfortable in any position." Red flag signs and symptoms should be sought in these patients with a concomitant vascular examination as deemed necessary.

Nonoperative Management

A large majority of patients with newly diagnosed axial pain will return to their baseline state of spinal health within 4–6 weeks, oftentimes with little to no treatment. For this reason, noninvasive, nonoperative modalities are the preferred choice for the treatment of axial spinal pain.

For patients with acute spinal pain—whatever the underlying origin—a short period of rest from aggravating maneuvers is indicated. A patient should not be placed on complete bed rest for more than 1–2 days. After even a few days of bed rest, the musculature of the entire body, including the paraspinal muscles, will begin to atrophy, making effective rehabilitation a challenge. The patient should be advised to return to activity as soon as possible with avoidance of the most painful activities. Additionally, nonsteroidal anti-inflammatory drugs should be prescribed at an appropriate dose for the purposes of pain relief. An oral steroid taper can also be used but should be used with caution, as several reports have suggested that oral steroids may reduce the efficacy of later, more invasive treatments such as injections.

By 2–4 weeks following symptom onset, most patients will have recovered sufficiently to resume most activities of daily living and even more strenuous activities such as exercise. It is at this point that physical therapy (PT) can be helpful to further reduce pain and to begin rehabilitation and prevention of future exacerbations. Therapists can perform pain-relieving treatments including massage, stretch, and spinal manipulation to accelerate pain reduction. This phase of treatment may also include chiropractic care and acupuncture. The long-term goals of PT should focus on improving muscle strength. Patients with muscle strains require strengthening of the injured muscle and all muscles that support the spine (known as the "core" musculature) to become better able to participate in the activities that initially precipitated the pain. Even patients with annular tears, herniated discs, and degenerative conditions can benefit from the trunk stability provided by strengthening the paraspinal musculature. Using one or more of these three noninvasive treatments, greater than 90% of patients should experience relief of acute axial pain, and many should experience long-term maintenance of spinal health.

Patients who fail to achieve relief of axial spinal pain through activity modification, oral agents, and therapy often can be treated with spinal injections. Injection techniques vary and are chosen for the specific pathology to be treated. Chronic muscle strains or muscle spasm may benefit from trigger point injections at the point(s) of maximal muscle tenderness. Recalcitrant cases of muscle spasm, particularly with cervical torticollis, are sometimes treated with injection of botulinum toxin (Botox, Allergan, Dublin, Ireland).

Axial pain thought to result from the disc or facet joints can be treated with epidural and perifacet injections, respectively. Epidural injections typically involve localization of the affected spinal level on fluoroscopy followed by injection of lidocaine and a corticosteroid. Immediate reduction of the pain with the effect of the topical anesthetic agent confirms the target as a pain generator. Epidural injections are best reserved for pathology within the spinal canal—disc herniations and occasionally annular tears. Patients with facet pathology benefit from perifacet

injections. These injections can be placed directly into the facet capsule; however, most pain specialists now inject anesthetic cranially and caudally to the facet to block the medial branch of the dorsal primary ramus of the nerve root, the main innervation of the joint. These medial branch blocks have been found to be safer and more effective for reduction in pain emanating from the facets. Additionally, medial branch blocks can be used to plan radiofrequency denervation of the facet joint, a technique that offers longer-term relief of facet-based pain in well-selected patients.

Aside from pain relief, two other benefits are provided through spinal injections. First, if a patient experiences partial relief with the injection, he or she may be better able to participate in therapy. The two modalities can then work synergistically to accelerate recovery and prevent future recurrence. Second, the application of a topical anesthetic agent or corticosteroid can help to predict if a patient will respond favorably to surgery. Temporary but substantial relief of symptoms implies that a more permanent treatment option, namely, surgery, could be considered in select clinical scenarios.

Indications for Surgery

Surgery is not indicated for the vast majority of patients with axial neck and back pain for several reasons: the condition is often not amenable to surgery (e.g., muscle strain, ligament sprain), the condition is stable and self-limited (e.g., most compression fractures and nearly all spinous process and transverse process fractures), or imaging findings are too diffuse to determine which process represents the main pain generator (e.g., multilevel degeneration with axial pain). Surgical treatment of axial pain is currently well indicated for patients with scoliosis and kyphosis, spondylolisthesis, and spinal instability resulting from fractures and dislocations. Surgical intervention for degenerative disease with axial pain in the absence of neurogenic symptoms is rarely indicated, and only if the degeneration is localized, patients have failed to achieve sustained pain relief with nonoperative modalities, and significant clinical information can confirm that the degenerative conditions identified are the sole pain generators. The clinical information best able to predict a positive outcome following surgery is the observation of complete (or near complete) resolution of axial pain with focal spinal injections coupled with consistent, reproducible physical examination findings pointing to the degenerative structures as pain generators. Additionally, the patient's history should be free of other psychosocial factors that could confound treatment. These factors include psychiatric conditions with predominant somatization symptoms, presence of active litigation related to an injury associated with the pain (e.g., car accidents, work-related injuries), and the presence of an active workers' compensation claim.

Operative Management

One of the most compelling reasons to avoid surgery for axial pain, if at all possible, is that fusion-based procedures are the primary treatment for these conditions. The main rationale for fusion follows the logic that pain from a moving structure can be controlled by eliminating motion at the structure. In all segments of the spine and SI joints, fusion involves preparing the environment surrounding two bones to be conducive for the growth of a new bone. The bridging bone will then join the two initially independent segments into a single structure.

Anterior Spinal Fusion

Spinal fusion can be performed from an anterior approach to the disc space between the vertebral bodies. These operations are termed "interbody" or "intervertebral" fusions for this reason. The technique is most often used for anterior cervical spine surgery and in the lumbar spine for discogenic back pain. Anterior fusion enjoys the advantage of a large space for the placement of bone graft for fusion between the well-vascularized vertebral bodies. Cervical spine surgery is readily accomplished in this manner with a relatively minimally invasive approach that exploits natural anatomic planes between the trachea, esophagus, and major neurovascular structures in the neck. Thoracolumbar surgery, however, has the disadvantage of requiring exposure through the thoracic and abdominal cavities with attendant risk of injury to the visceral and vascular structures contained therein. Bone graft, either from a cadaveric donor or from the anterior iliac crest, is impacted into the space previously occupied by the intervertebral disc to achieve the fusion. This is typically stabilized using a metal plate affixed to the anterior aspect of the vertebrae with bone screws, as such instrumentation has been shown to provide more immediate stability and enhance the likelihood of fusion.

Postoperatively, patients often use a cervical collar or brace to protect the spine until pain begins to resolve. The fusion site will heal over the course of several months and is monitored using periodic radiographs. Visualization of bone bridging between the intended vertebrae signifies complete healing of the fusion.

Posterior Spinal Fusion

Thoracolumbar fusion is most commonly performed using a posterior approach. The advantage of the posterior approach in the thoracic and lumbar regions is that long segments of the spine can be accessed without violating the thoracic

and abdominal cavities and complication rates are reduced as a result. Fusion can be achieved by placing an interbody graft using carbon fiber or titanium cages, cadaver bone, or autograft from the iliac crest or elsewhere. Stabilization is achieved via bone screws anchored to the vertebrae through channels created in the pedicles and connected by rods. Patients may be given a back brace to assist mobilization after thoracolumbar posterior fusion. The brace is typically used only until a patient's pain resolves and the muscles once again become able to assist stability. In patients with osteopenia or osteoporosis, a rigid brace may be prescribed for use until the fusion site shows signs of consolidation on radiographs.

SI Joint Fusion

Fusion of the SI joint requires debridement of the cartilage of the joint with replacement of the cartilage with bone graft. The SI joint can be accessed anteriorly or posteriorly with bone graft taken directly from the ilium. Stabilization is achieved using a plate bridging from the sacrum to the ilium or via percutaneously placed screws that span the joint space.

After SI fusion, patients are instructed to use crutches or a walker to assist in mobilization. Weight bearing on the operative limb is restricted to the so-called "toe-touch" or "touchdown" weight bearing for several weeks following surgery.

Expected Outcomes

The vast majority of patients (up to 90%) with acute axial pain can be expected to experience pain relief within 6 weeks of symptom onset. Patients with initial episodes of pain can, therefore, be reassured that the pain will resolve and not result in a chronic condition. In general, the longer a patient experiences activity-limiting axial pain, the longer treatment will take to relieve the pain, and the less likely he or she will be to experience complete pain relief. This observation was recently confirmed in an analysis of the multicenter Spine Patient Outcomes Research Trials (SPORT). Patients with lumbar disc herniations who experienced functional limitations for greater than 6 months were found to have inferior results, irrespective of treatment, as compared to patients in pain for less than 6 months. It is unclear if this finding suggests that patients developed chronic pain syndromes independent of the initial pain generator or if permanent structural damage to the spine was responsible.

If a patient is unable to achieve satisfactory relief through nonoperative measures, fusion-based procedures have been shown to result in long-term reductions in pain and improvement in function for only 60–70% of well-selected patients

with axial neck and back pain. Reports of randomized trials and observational studies have shown that some well-selected patients could achieve pain relief and functional improvement following surgery. The selection process must be rigorous, however, in order to assure the best outcome possible. Ideally, patients should be free from nicotine products and should not be involved in litigation over the cause of pain to assure optimal outcomes. Patients must additionally be prepared to expect that no treatment will completely eliminate back pain. They should be counseled that pain reduction will approximate what was achieved with spinal injections and should be willing to accept that a 50% reduction in pain may be the best that can be achieved. Patients expecting full alleviation of pain following surgery should have their expectations appropriately adjusted through counseling from primary care physicians and surgeons prior to agreeing to any procedure (Table 1.2).

Table 1.2 Summary of axial neck and back pain disorders with synopsis of presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Myofascial pain	Trigger point tenderness Limited or no focal pain	Primarily clinical	Rest, ice, NSAIDS PT Trigger point injection	N/A
Fracture/ ligamentous injury	History of trauma Focal tenderness to palpation over injured region	Plain films/CT MRI—if there is concern for ligamentous injury	Rest, ice, NSAIDS PT Spinal bracing	Spinal instability or failure of nonoperative management with persistent pain Spinal stabilization procedures often require instrumented fusion
Discogenic back pain	Pain worse with sitting or standing Forward flexion exacerbates the pain	MRI-degenerative changes involving the discs (may not be diagnostic)	NSAIDS PT Spinal injections	Reserved for select cases where nonoperative treatment fails Fusion-based procedure
Facetogenic pain	Pain worse with standing and ambulation Extension exacerbates the pain	MRI-degenerative changes involving the facet joints (may not be diagnostic)	NSAIDS PT Facet injections, radiofrequency lesioning, rhizotomy	Reserved for select cases where nonoperative treatment fails Fusion-based procedure

PT physical therapy, *CT* computed tomography, *MRI* magnetic resonance imaging, *NSAIDs* nonsteroidal anti-inflammatory drugs

Suggested Reading

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Chapter 2
Sacroiliac Joint Dysfunction and Piriformis Syndrome

Erika T. Yih and Danielle L. Sarno

Part I: Sacroiliac Joint Dysfunction

SI Joint Anatomy, Innervation, and Function

Anatomy

The sacroiliac (SI) joint is a large axial joint connecting the spine to the pelvis. Lying at the junction of the sacrum and the ilium, the average surface area of the joint is 17.5 cm^2, and the average volume is 0.6–2.5 mL [1]. However, the SI joint varies widely in size, shape, and contour between individuals and sometimes even between sides within a single individual [2]. Although the SI joint is typically characterized as a large synovial joint, only the anterior third is a true synovial joint, while the rest of the junction has an absent or only rudimentary posterior capsule and is instead supported by an intricate ligamentous system [3]. The SI joint is also supported by a large network of pelvic and lower extremity muscles, some of which (e.g., gluteus maximus, piriformis, biceps femoris) are functionally connected to the SI joint ligaments and therefore affect joint mobility [4].

The surface of the SI joint is flat until puberty, when the iliac surface starts to become rougher and duller and develop some fibrous plaques [4, 5]. In the third and

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fourth decades of life, elevations and depressions develop and enhance stability of the joint. As the articular surfaces erode over time, the synovial cleft narrows to 1–2 mm in individuals aged 50–70 years and to–1 mm in those over 70 years old [5].

Innervation

Like the size, shape, and contour, the innervation of the SI joint can be highly variable, even between sides in the same individual [6]. Cadaveric studies have demonstrated that the posterior SI joint is innervated by a fine nerve plexus formed by the lateral branches of the dorsal rami from the lumbosacral spine, but the exact levels remain debated [6, 7]. In one study of 25 cadavers, the lateral branches of S1 and S2 contributed to the plexus in 100% of specimens, S3 in 88%, L5 in 8%, and S4 in 4% [6]. Radiofrequency ablation of the L5 dorsal ramus and S1–S3 lateral branches have been shown in randomized, placebo-controlled studies to have significant and prolonged effectiveness in improving chronic SI joint pain, supporting the idea that the dorsal rami from these levels are involved in SI joint innervation [8, 9]. The dorsal ligaments surrounding the SI joint are also innervated by at least the L5 primary dorsal ramus and the lateral branches of S1–S3 dorsal rami [10, 11]. The innervation of the anterior aspect of the SI joint is similarly ambiguous, with some studies suggesting there is no nervous innervation to this part of the joint and others suggesting L2–S2, L4–S2, and L5–S2 ventral rami innervation [4].

Function

The main function of the SI joint is to transmit and dissipate truncal loads to the lower extremities while maintaining stability [4]. In order to achieve this, the joint is supported by a large network of strong ligaments. The extensive dorsal ligamentous structure stabilizes the joint by serving as a strong connecting band between the sacrum and ilium to limit motion in all planes of movement [3, 4]. Indeed, the typical range of motion at the SI joint has been measured to be very small, with less than 4 degrees of rotation and up to 1.6 mm of translation [12, 13]. It has been postulated in the past that hypermobility in the SI joint leads to SI joint-mediated pain, but this relationship is unclear, as comparison between symptomatic and asymptomatic joints did not show any difference in range of motion [12]. Additionally, the prevalence of abnormal SI joint movements has been estimated at 20% in healthy, fit college students and 8–16% in asymptomatic individuals [14, 15].

Etiology and Pathology of SI Joint Pain

SI joint pain occurs as a combination of axial loading and abrupt rotation [4], which can result from traumatic causes such as falls, motor vehicle accidents, lifting, and pregnancy or atraumatic causes including arthritis, scoliosis, inflammatory arthropathy (sacroiliitis), and infection [16]. The pathological source of pain can be intra-articular, extra-articular, or a combination of both. Arthritis, arthropathies, and infection are examples of etiologies that could cause intra-articular pathology, whereas ligamentous injury, myofascial pain, fractures, enthesopathy, and pregnancy are etiologies that could cause extra-articular pathology, such as SI joint posterior ligament complex pain. Extra-articular sources of pain tend to be more common [4].

Epidemiology

Numerous studies have examined the prevalence of SI joint pain in patients presenting with a complaint of low back pain, and most have reported a prevalence of 15–30% in this population [17–19]. In a retrospective study assessing the inciting events in 54 patients with injection-confirmed SI joint pain, 44% of cases were due to traumatic etiology, 21% were from cumulative effects of repeated stress, and 35% were idiopathic [20]. There is a higher prevalence of SI joint pain in females, which is thought to be due to gender-based anatomical differences in sacrum position (more horizontal in females) and ligament laxity (to allow for parturition) [21]. Pregnancy in particular predisposes individuals to SI joint pain through a combination of weight gain, exaggerated lordotic posture, hormone-induced ligament laxity, and mechanical trauma during childbirth. There are numerous other factors that also increase stress on the SI joints and thus predispose a person to develop SI joint pain gradually. These risk factors include obesity, true or apparent leg length discrepancy, gait abnormalities, repetitive strain or low-grade trauma (e.g., from prolonged exercise), scoliosis, and spinal surgery (particularly spinal fusion to the sacrum) [4, 16]. In addition to altering force transmission across the SI joint, lumbar spine surgery can also trigger SI joint pain due to ligament weakening, violation of the SI joint cavity, and/or postsurgical hypermobility [4].

Diagnostic Evaluation

History

Pathology in the sacroiliac joint causes unilateral pain (unless both joints are affected) to an area just inferior to the ipsilateral posterior superior iliac spine [22]. However, this area is common to other sources of pain, and many studies have made attempts to clarify pain referral patterns from the SI joint. Although the frequency of involvement in each region varies between studies, overall the pain referral map most commonly includes the buttocks, lower lumbar region below the L5 spinal process, and lower extremity and also sometimes includes the groin, upper lumbar region, and abdomen [18, 23–26].

Physical Examination

Dozens of physical exam maneuvers have been developed to aid in the diagnosis of SI joint-mediated pain, but diagnosis through history and physical examination remains difficult due to the highly variable and nonspecific presentation of patients with SI joint pain, as well as the low validity and reliability of SI joint-focused physical exam maneuvers [27–30]. Because spinal and hip pathology are extremely common and can present similarly to SI joint pain, the physical examination should include a thorough neurologic examination along with the evaluation of straight leg raise and assessment of pain and range of motion in the lower thoracic and lumbar spine. Patients with only SI joint pain are typically neurologically intact, but they may demonstrate pain-inhibited weakness, subjective non-dermatomal extremity sensory loss, and other distal sensory complaints [5]. The hip joint should also be thoroughly tested to look for intra-articular hip pathology.

Localized tenderness with palpation of the sacral sulcus (Fortin's point/at the insertion of the long dorsal ligament inferior to the posterior superior iliac spine) may indicate SI joint-mediated pain. The prevalence of positive provocation SI joint tests in patients with low back pain is greater than the accepted prevalence of SI joint pain, suggesting that the exam maneuvers are nonspecific and have a significant false-positive rate [31]. Indeed, despite the myriad diagnostic exam maneuvers, previous clinical studies have struggled to identify any piece of the medical history or physical exam maneuver that can accurately and consistently identify dysfunctional SI joints as pain generators [18, 24, 32]. However, performing multiple SI joint maneuvers together may have better clinical utility in assessing SI joint pain, as the positive predictive value increases with multiple positive tests. Several studies have reported that a combination of tests with at least three positive SI provocation tests (e.g., Patrick's/FABER test, thigh thrust, compression) is better able to identify SI joint pain than a single test [26, 32–36]. Laslett et al. determined that using a battery of six SI joint tests – distraction (Fig. 2.1), thigh thrust (Fig. 2.2), Gaenslen's with posterior rotation (Fig. 2.3), Gaenslen's with anterior rotation (Fig. 2.3),

Fig. 2.1 Distraction test (testing bilateral SI joints simultaneously). The patient lies supine, and the examiner applies a vertically oriented, posteriorly directed force to both the anterior superior iliac spines (ASIS). Many examiners choose to do this exam maneuver by standing next to the patient with arms crossed, placing the heels of both hands on the patient's anterior superior iliac spines, and applying downward and outward pressure, causing distraction of the SI joints

Fig. 2.2 Thigh thrust test (testing left SI joint while stabilizing sacrum). The patient lies supine with the ipsilateral hip and knee flexed at 90 degrees. The examiner places one hand beneath the sacrum to fix its position and uses the other hand to apply a downward force through the line of the ipsilateral femur. By applying axial pressure along the length of the femur, the femur is used as a lever to push the ilium posteriorly, producing a posterior shearing force at the SI joint

Fig. 2.3 Gaenslen's test (testing right SI joint in posterior rotation and left SI joint in anterior rotation). The patient lies supine with the leg of the symptomatic side hanging off the examination table. The examiner flexes the contralateral hip and puts the contralateral knee in 90 degrees of flexion. The examiner then applies a downward force to the lower leg to hyperextend the hip, while also applying a superior and posterior (flexion-based) counterforce to the flexed leg, pushing it in the cephalad direction. This stresses the SI joints with a torsion force. The Gaenslen's test can be done on both sides for evaluation of both anterior and posterior rotation at both SI joints. *This image was taken to help the reader visualize the maneuver more clearly. For the patient's safety, it is advised to perform this maneuver standing next to the patient

compression (Fig. 2.4), and sacral thrust (Fig. 2.5) – three or more positive tests yielded a sensitivity of 91% and a specificity of 78% [31]. Furthermore, the Gaenslen's test was least valuable, and two positive tests out of the other remaining four tests still yielded a sensitivity of 88% and specificity of 78% [34]. When all six provocation tests were negative for index pain, the SI joint could be ruled out as a source of low back pain [34]. Patrick's test, sometimes referred to as the FABER test (flexion, abduction, external rotation), is another provocative maneuver often described to assess SI joint pain. This test is helpful in detecting limited hip motion and distinguishing hip pain from SI joint pain [37]. The maneuver is performed with the patient supine with hip and knee flexed to 90 degrees and the foot placed on top of the opposite knee in a "figure four" position. The thigh is abducted fully and externally rotated toward the exam table. The examiner stabilizes the contralateral ASIS and exerts a downward pressure on the abducted knee. When performing the maneuver, anterior or groin pain is more suggestive of hip pathology, whereas low back or buttock pain may indicate SI joint-mediated pain [37]. It is important to note that SI provocation tests, even when performed in a cluster, should not be treated as standalone clinical tests but rather used and interpreted in a larger clinical context only when other diagnoses are ruled out or deemed unlikely [38].

Fig. 2.4 Compression test (testing bilateral SI joints simultaneously). The patient is side-lying, and the examiner places their hands over the upper part of the iliac crest and applies a vertically directed force toward the floor. This force is translated across the pelvis and therefore compresses both SI joints

One particularly helpful approach to identifying patients in whom SI joint-directed physical exam maneuvers would *not* be particularly helpful was described by Laslett [31]. Provocation SI joint tests are often positive in those with nerve root pain from herniated lumbar disc and in those whose symptoms fit a pattern called the centralization phenomenon [31]. The centralization phenomenon is a common clinical observation where referred pain moves from a distal to a more proximal (midline back) location when patients with low back pain are examined using standardized test movements and sustained postures focusing on repeated range of motion to end-range [39]. In this standardized examination initially described by McKenzie, the examiner begins by recording baseline symptom locations with emphasis on the most distal symptoms. The patient is then asked to move from standing position to end-range lumbar flexion before returning to the starting position. Any loss or abnormal quality of the movement is noted, and any change in the patient's symptoms after performing maximal lumbar flexion is recorded. The patient then repeats maximal forward flexion 10–12 times and reports any lasting change in location or intensity of symptoms. Standing extension is assessed in a similar manner, and the flexion and extension exam maneuvers are repeated in a recumbent position (flexion performed by bending knees up and hugging to the chest and extension performed by pushing shoulders up off the bed from prone position) [39].

The centralization phenomenon has been evaluated for reliability and validity in many studies [40–46]. It has been found to be highly specific to discogenic pain and

Fig. 2.5 Sacral thrust test (testing bilateral SI joints simultaneously). The patient lies prone, and the examiner places one hand directly on the midline of the sacrum at the apex of the curve of the sacrum, reinforcing it with the other hand. The examiner then applies a vertically directed force to the midline of the sacrum at the apex of the curve of the sacrum. This force is directed to the patient's anterior and produces an anterior shearing force at both SI joints since the ilia are fixed by the examination bench

is not observed in patients with confirmed SI joint pain or facet joint pain [47–51]. Additionally, studies looking at dual SI joint blocks and provocation discography have revealed that SI joint pain and discogenic pain rarely co-exist [47, 52]. Therefore, SI joint provocation tests that are positive in the presence of the centralization phenomenon are likely falsely positive since the centralization phenomenon indicates discogenic pain, which rarely co-exists with SI joint pain. Thus, as a rule of thumb, there is low clinical utility in performing SI joint maneuvers in patients whose symptoms can be made to centralize during a McKenzie-type physical exam.

Differential Diagnosis

SI joint pain is a difficult pathology to diagnose, particularly in distinguishing it from the pathology in the lumbosacral spine. Pain that originates in the lower lumbar spine may refer to the SI joint and vice versa. Lumbar disc disease, nerve root compression, zygapophyseal joint pain, myofascial syndromes, and symptoms from non-spinal structures (musculoskeletal, gastrointestinal, genitourinary, gynecologic) may all mimic SI joint pain because of their overlapping pain referral patterns. Additionally, several rheumatological disorders may cause inflammation at

the SI joint. These include ankylosing spondylitis, reactive arthritis, psoriatic arthritis, Behçet's disease, and hyperparathyroidism. Appropriate radiographic studies and laboratory findings, including HLA-B27 and inflammatory markers, should be obtained when these are suspected.

Imaging

Unfortunately, no imaging studies are consistently helpful in the diagnosis of primary sacroiliac joint pain. Plain radiographs are relatively nonspecific, as 24.5% of asymptomatic patients over the age of 50 have abnormal SI joint findings on plain radiographs [53]. Low sensitivity of CT (57.5% sensitive) and bone scan (12–46% sensitive) make these imaging modalities poor screening tests for SI joint pain [54–56]. Imaging is typically only done to help assess for other causes of pain or to rule out red flags such as fractures, infections, and malignancy. For instance, MRI is useful in detecting soft tissue pathology such as tumors or early inflammatory changes in spondyloarthropathies.

Diagnostic Injections

SI joint blocks have widely been considered the most reliable, "gold standard" method to diagnose SI joint pain for many years, but they remain problematic and should mainly be pursued when other sources of pain have been ruled out. Extravasation of anesthetic to surrounding pain-generating structures is a common occurrence and can lead to false-positive blocks [57]. Conversely, inadequate anesthetic spread to the anterior and cephalad parts of the SI joint can result in false-negative blocks. The validity of SI joint blocks remains unproven and highly disputed [58]. However, due to the limitations of the history, physical examination, and imaging modalities, diagnostic SI joint blocks remain the only means of establishing a diagnosis of intra-articular SI joint pain [11, 58].

The diagnostic SI joint block is performed through fluoroscopic-guided, intra-articular injection of local anesthetic. Injections should not be performed without imaging guidance, as successful intra-articular injection was accomplished in only 22% of patients in a study on SI joint injections performed with a blind approach [59]. An ideal positive response to SI joint block is complete or near complete relief of pain, although ≥75% pain relief is often accepted as diagnostic of SI joint-mediated pain, and in cases of 50–75% pain reduction, SI joint may still be a major contributor to pain [16]. There is a risk of false-positive response to a single diagnostic block, and it is recommended to perform dual blocks with injection of different anesthetics with different durations of action on two different occasions (often lidocaine for the first block and bupivacaine for the second block) [58]. However, clinically, this is rarely done because clinicians often opt to proceed to therapeutic block with local anesthetic and a steroid, as this approach is more time- and cost-effective, and the block itself is a definitive treatment. Of note, intra-articular SI

joint injections may not identify patients with pain from the SI dorsal ligaments. Multi-site, multi-depth sacral dorsal rami lateral branch local anesthetic blocks can reduce pain from the dorsal and interosseous ligaments and, therefore, help to diagnose SI joint posterior ligament complex pain [60].

Treatment Options

Many options are available for the treatment of SI joint pain, ranging from non-interventional management to injection and denervation procedures to surgical interventions. As with other types of chronic pain, psychopathology and other psychosocial factors can greatly affect patients' pain experiences and treatment responses. Therefore, identifying and treating concomitant psychiatric pathology through a multidisciplinary approach is paramount to optimal management of chronic SI joint pain. Lifestyle modifications should also be considered.

Physical Therapy

Physical therapy should aim to address underlying pathology. Deficits in strength and flexibility should be identified and corrected. Patients should be trained in proper body mechanics and posture, as electromyographic activity has shown differences in the timing of muscle firing patterns in symptomatic SI joints compared to age-matched asymptomatic controls [61]. Specific attention should be paid to strengthening certain muscles including the hamstrings, gluteus maximus and medius, piriformis, erector spinae, latissimus dorsi, and iliacus muscles, which have ligamentous and fascial attachments to the SI joint complex [5]. Early mobilization is important in preventing harmful effects of immobilization, such as muscle atrophy and ligament weakening.

Manual Therapy and Manipulation

Manual therapeutic techniques use direct manipulation, direct mobilization, or indirect techniques (e.g., muscle energy) and are often performed by osteopathic physicians and chiropractors [5]. These techniques can be trialed for 3–4 weeks, but if the patient does not respond, other treatment options should be considered. Ideally, if pain begins to improve with manual therapy, the patient should begin a structured exercise program to promote restoration of soft tissue flexibility, strength, and balance [5].

Orthotics

In patients with true or apparent leg length discrepancy, shoe inserts can be used to more evenly distribute the load borne by the SI joints. However, it may be beneficial to start out conservatively, only correcting half the leg length incongruity because many patients already compensate by altering their gait or posture [5].

Pelvic or SI belts worn just superior to the greater trochanters may also be helpful in pain relief and/or proprioceptive feedback by decreasing sacroiliac joint motion by about 30% [62]. However, these should not be worn long term and should instead be rapidly weaned to avoid psychological dependence, muscle weakness, and loss of flexibility from overreliance and core muscle atrophy [63].

Medications

Oral medications can be helpful in SI joint pain relief, especially when used to augment physically-based therapeutic modalities. Short-term use of non-opioid analgesics such as nonsteroidal anti-inflammatory drugs, acetaminophen, serotonin and norepinephrine reuptake inhibitors (duloxetine), or topical analgesics (e.g., lidocaine ointment or patches, diclofenac gel, menthol, capsaicin) may be considered. For patients with inflammatory rheumatological disorders with SI joint involvement/sacroiliitis, many pharmacological agents exist, and management of rheumatological disease should be overseen by a trained rheumatologist.

Intra-Articular Injection

As described earlier, intra-articular SI joint injections with corticosteroid and local anesthetic are often both diagnostic and therapeutic. The most common nonsurgical procedure performed for SI joint pain, the injection of corticosteroid into the SI joint, offers anti-inflammatory mechanisms for pain control. It can be helpful for both intra-articular and extra-articular etiologies of pain, as there will commonly be some extravasation into nearby structures [57]. There is differing input about whether ultrasound or fluoroscopy offers better accuracy for intra-articular injection [59, 64, 65], but either is an option. Many physicians prefer the fluoroscopic-guided approach to confirm intra-articular placement with contrast. As with corticosteroid injections to other areas, the frequency of injection should be limited due to concerns about damage to cartilage, tendons, and ligaments with repeated steroid injections. As a rule of thumb, intra-articular corticosteroid joint injection should be limited to three injections in a 6-month period or four in a 1-year period [66]. Caution also is advised in patients with uncontrolled diabetes, surrounding joint osteoporosis, and coagulopathies or on anticoagulation.

Radiofrequency (RF) Ablation

After positive diagnostic multi-site multi-depth sacral lateral branch blocks, RF ablation of lateral branches may be considered. RF ablation aims to provide longer-lasting pain relief by applying an electrical current generated by radio waves to heat nerve fibers and cause denervation. The lateral branches of the lumbosacral dorsal rami are targeted due to their role in innervating the posterior portion of the SI joint, specifically the dorsal and interosseous ligaments. The efficacy of conventional RF ablation has been the subject of numerous studies with varying results showing sustained relief at 6 months in anywhere from 40% to 60% of patients [9, 67–70]. The major drawbacks to RF ablation are the complex and highly variable innervation of the posterior SI joint, as well as the inability to alleviate pain emanating from the ventral SI joint. In addition to conventional RF ablation, modified techniques are being studied, including cooled RF ablation, which has shown some promise in producing superior results compared to conventional RF ablation [69, 70].

Prolotherapy and Platelet-Rich Plasma (PRP) Injection

For SI joint pain of extra-articular etiology, prolotherapy and PRP may be considered. The theory behind prolotherapy is that the injection of a dextrose solution creates an inflammatory response, which may cause fibroblastic migration and collagen proliferation, ultimately resulting in ligamentous widening and strengthening. Similarly, PRP therapy uses injections of a concentration of a patient's own platelets to harness the body's natural healing system to accelerate healing of injured tendons, ligaments, muscles, and joints. Although these therapies are not widely accepted yet, prolotherapy and PRP have been reported in some studies to have favorable outcomes (more pain reduction, longer duration) for relief of SI joint pain compared to intra-articular steroid injection [71–73].

SI Joint Fusion

Surgical intervention may be considered in cases of continued or recurrent SI joint pain refractory to conservative treatment. Surgical candidates should be carefully selected and should only include patients with>75% relief from SI joint block and who have been evaluated and ruled out for lumbar spine and pelvic ring pain sources [5, 63]. The procedure of choice is SI joint fusion or arthrodesis, in which spanning plates and/or trans-iliosacral screws are placed to stabilize the SI joint. This removes movement within the SI joint that may be contributing to pain. Open arthrodesis, while standard in the past, is now generally reserved for cases of revision surgery, nonunion, and aberrant anatomy [63]. In recent years, minimally invasive percutaneous techniques have been developed, and these are now generally recommended as first-line surgical treatment due to decreased blood loss,

hospital stay, and mean surgical time compared to open arthrodesis [74, 75]. Following surgery, patients are prescribed limited weight bearing followed by a gradual return to weight bearing with sequential physical therapy. Overall, long-term success rate for SI joint fusion is quite favorable, estimated to be about 70–80% [76–80].

Conclusion

The diagnosis and management of patients with SI joint pain is very challenging due to the lack of specific historical features, examination maneuvers, or radiological findings to provide a definitive diagnosis. Pain originating from other sources should be ruled out first. Performing a battery of specific SI provocation tests may help guide clinical decision-making in the right context. Although its validity remains disputed, SI joint block with an anesthetic agent remains the most reliable means to diagnosing SI joint pain. Treatment should be multimodal with lifestyle modifications, physical therapy, manual therapy, orthotics, and non-opioid analgesics being first-line. Interventional pain management options include intra-articular corticosteroid injection, RF ablation, and prolotherapy. If SI joint pain persists despite these interventions and other causes have been ruled out, percutaneous SI joint fusion may be considered.

Part II: Piriformis Syndrome

Anatomy

Piriformis syndrome results when the sciatic nerve is compressed by the piriformis muscle (e.g., due to anatomical variations, trauma, muscle hypertrophy, or muscle spasm).The piriformis muscle is one of the deep muscles of the hip, functioning as an external rotator when the hip is extended and as a hip abductor when the hip is flexed [81]. It originates on the anterior surface of the sacrum and the sacrotuberous ligament, runs laterally through the greater sciatic foramen, and inserts on the inner surface of the superior greater trochanter [82]. The piriformis muscle is innervated by branches of the posterior division of the S1 and S2 ventral rami. In a cadaveric study, 90% of cadavers had traditional anatomy with an undivided sciatic nerve emerging below the piriformis muscle [83]. However, six different anatomical variations were also found [83]. Additionally, variations in the piriformis muscle body and tendon have also been observed, with 43% of cadavers in one study demonstrating fusion of the piriformis tendon with the obturator internus tendon prior to insertion [84]. It has been hypothesized that anatomical variations in the sciatic nerve and piriformis muscle could predispose a patient to piriformis syndrome [85, 86].

Etiology

When the piriformis muscle is overused, irritated, or inflamed, it can lead to irritation of the adjacent sciatic nerve. Given the common anatomical variations, there are multiple potential anatomical areas of sciatic nerve compression as the nerve passes through, above, or below the piriformis. Additionally, since the piriformis tendon often fuses with the tendons of other deep hip external rotators (most commonly the obturator internus) prior to insertion [84], pathology in other muscles may play a role as well. The piriformis, along with the other deep external rotators of the hip, can be stressed due to poor body posture chronically or an acute injury that results in sudden, strong internal rotation of the hip [87].

Epidemiology

Piriformis syndrome is a relatively rare cause of low back pain and/or sciatica, estimated to account for roughly 6% of all cases [81, 87]. With an estimated incidence of new cases of low back pain and sciatica at 40 million annually, the incidence of piriformis syndrome would be roughly 2.4 million per year [87]. In the majority of cases, piriformis syndrome occurs in middle-aged patients with a reported ratio of male to female patients being affected 1:6 [87].

Diagnostic Evaluation

History

Patients with piriformis syndrome typically present with hip pain, buttock pain, dyspareunia in female patients, and sciatica [88]. Symptoms are often made worse by prolonged periods of sitting (e.g., driving) or by rising from a seated position [84]. Systematic reviews have shown that the most common presenting symptoms are buttock pain, external tenderness over the greater sciatic notch, and aggravation of pain with sitting [89, 90]. Many patients will have a history of trauma to the piriformis muscle, ranging from falls to abnormal stretching of the muscle during athletic events [91].

Physical Examination

Although no physical examination maneuver is diagnostic for piriformis syndrome, examination findings can help inform clinical suspicion. Several tests passively stretch the piriformis in order to cause compression of the sciatic nerve and reproduce sciatica symptoms. One example is the FADIR test, where the patient's hip is

placed in flexion, adduction, and internal rotation while in a supine position. A reproduction of the patient's typical posterior pelvic pain or paresthesia represents a positive finding. Other maneuvers reproduce symptoms by causing active piriformis muscle contraction and subsequent compression of the sciatic nerve. In the active piriformis test, the patient is placed in the lateral side-lying position, and the patient actively abducts and externally rotates the hip, while the examiner resists these movements [92]. Additional maneuvers are thought to create tension along the irritated sciatic nerve and recreate the patient's symptoms. In the seated piriformis stretch test, the patient is seated on the edge of the exam table with the hip flexed to 90° and the knee extended. The examiner palpates the sciatic notch and provides hip adduction and internal rotation [92]. The combination of the seated piriformis stretch test and the active piriformis test has shown a sensitivity of 0.91 and specificity of 0.80 for the endoscopic finding of sciatic nerve entrapment [93].

Imaging and Diagnostic Testing

Electrodiagnostic testing and imaging are typically used to exclude other causes of lower extremity nerve pain. Electromyography in the setting of piriformis syndrome is often normal and is most useful to exclude other conditions such as lumbosacral radiculopathy. MRI of the spine is critical to assess the spinal canal and nerve roots when excluding radiculopathy or spinal stenosis as a cause of sciatica or buttock pain. MRI of the pelvis may show anatomical abnormalities or asymmetry of the piriformis muscles, but these findings are not pathognomonic and can occur in asymptomatic individuals [94–96].

Treatment

Initial management of piriformis syndrome should focus on nonsurgical multidisciplinary care. Medications (nonsteroidal anti-inflammatory drugs, muscle relaxants, and neuropathic agents) and physical therapy remain mainstays for the treatment of piriformis syndrome [81]. The initial goal of physical therapy is to restore proper length to the muscle and release myofascial trigger points, thereby reducing the compressive force on the sciatic nerve [97, 98]. Therapy should focus on lumbosacral stabilization, hip strengthening, and correction of biomechanical errors across the hip, pelvis, and spine that could predispose to gluteal pain [99].Taut painful bands noted on palpation of the piriformis can be treated manually through trigger point injections, dry needling, acupuncture, manual pressure, and massage [81].

Injections of the piriformis muscle with either local anesthetic alone or anesthetic in combination with corticosteroid can be therapeutic. However, some studies have shown no benefit from injecting a corticosteroid in addition to local anesthetic [100]. Given the muscle atrophy and other side effects, intramuscular glucocorticoid injection should be considered in only very limited cases [81]. Botulinum toxin

injections have also shown promise in the treatment of piriformis syndrome, but its high cost is a barrier [101, 102].

When all conservative management fails, surgical intervention involving tenotomy of the piriformis muscle tendon and sciatic nerve decompression may be considered [103]. However, results after surgery are not always predictable, and no large, prospective, randomized, controlled trials have been performed [81, 104].

Conclusion

The diagnosis of piriformis syndrome is complicated, with many diagnoses having overlapping symptoms. There is currently no definitive diagnostic test, and piriformis syndrome remains a clinical diagnosis of exclusion. However, history, physical examination, electrodiagnostic testing, and imaging modalities can narrow the differential diagnosis and help inform clinical suspicion of piriformis syndrome. Nonsurgical treatment remains the mainstay of piriformis syndrome treatment, with a focus of optimizing biomechanics.

Summary Tables 2.1 and 2.2

Table 2.1 SI joint pain

History	Pain in low back below L5 level, PSIS, buttocks
Physical exam	Evaluate for other sources of pain first with full neurological exam, spine exam, hip exam If other sources of pain are unlikely, perform battery of SI provocation tests (distraction, thigh thrust, Gaenslen's, compression, sacral thrust). If none are positive, SI joint pain can be ruled out. With three or more positive tests, there is 91% sensitivity and 78% specificity for SI joint pain If patient's symptoms can be centralized through McKenzie-type examination (repeated range of motion to end-range), pain is likely discogenic, and there is low clinical utility for performing SI joint tests
Differential diagnosis	Important to rule out lumbosacral spine pathology (disc disease, nerve root compression, facet joint pain) Consider myofascial syndromes and gastrointestinal, genitourinary, gynecologic causes Consider rheumatological diseases (ankylosing spondylitis, reactive arthritis, psoriatic arthritis)
Workup	Imaging is not very helpful except to rule out other sources of pain MRI can help with evaluating soft tissue for tumors and bony edema that may indicate sacroiliitis from rheumatological disorders If suspect rheumatological etiology, send inflammatory labs and HLA-B27
Diagnosis	Once other sources of pain are ruled out, diagnostic SI joint block with local anesthetic
Treatment	First-line: lifestyle modifications, physical therapy, manual therapy, orthotics, non-opioid analgesics Interventional pain management: intra-articular corticosteroid injection, multi-site multi-depth sacral lateral branch blocks, RF ablation, prolotherapy, PRP Surgical option: percutaneous SI joint arthrodesis for refractory pain

Table 2.2 Piriformis syndrome

History	Pain in buttocks and hip region, sciatica Worse with sitting History of trauma or stretch injury to piriformis
Physical exam	Evaluate for other sources of pain first with full neurological exam, spine exam, hip exam, lower extremity exam If other sources of pain are unlikely, perform FADIR, active piriformis test, and/or seated piriformis stretch test to guide clinical suspicion
Differential diagnosis	Important to rule out lumbosacral spine pathology Consider myofascial syndromes and gastrointestinal, genitourinary, gynecologic causes
Workup	Imaging and electrodiagnostics to rule out other sources of pain MRI can help with identifying anatomical abnormalities or asymmetry
Diagnosis	Clinical diagnosis
Treatment	First-line: lifestyle modifications, physical therapy, non-opioid analgesics Interventional pain management: injection of local anesthetic, injection of anesthetic with corticosteroid, injection of Botox Surgical option: tenotomy of the piriformis muscle tendon and sciatic nerve decompression

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Chapter 3
Pain Management for Chronic Musculoskeletal Disorders

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Myofascial Pain Syndrome

Myofascial pain syndrome (MPS) is a common pain condition characterized by aberrantly taut bands of skeletal muscle and fascia called myofascial trigger points (MTrPs).The prevalence of MPS is uncertain due to conflicting diagnostic criteria. Previous studies have estimated the lifetime prevalence of MPS to be as high as 85% [1]. The prevalence of MPS is reported to vary according to patient population. MPS seems to be higher among patients with chronic spondylotic low back pain and among women. In a cross-sectional cohort study of 224 patients diagnosed with nonspecific neck pain by their primary care physician, all (n = 224) were found to have MPS [2]. In a cross-sectional cohort study of patients undergoing outpatient treatment for back pain, 90% of patients were found to have MPS [3].Within a Danish cohort of 1504 patients between 30 and 60 years of age, the prevalence of MPS was almost twice as high among women compared to men (65% and 37%, respectively) [4].

The pathogenesis of MTrPs is poorly understood but commonly believed to stem from muscle injury (e.g., muscle trauma, cumulative and repetitive strain, postural dysfunction, poor ergonomics, fatigue, etc.), ultimately resulting in focal dystonia with elements of peripheral and central sensitization. Increased myocyte tone, a characteristic of MTrPs, is hypothesized to occur due to disruptions in myocyte sarcoplasmic reticulum and spillage of calcium into the sarcoplasm, increased production and release of acetylcholine at the neuromuscular junction, or sympathetically mediated increases in myocyte excitability [5–7]. Increased myocyte tone may

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then lead to chronic impairments in blood flow, decreased availability of cellular nutrients, and increased generation of noxious stimuli [5].

On exam, MTrPs are often palpable, firm, and tender and may be the foci of localized pain. Spontaneously painful MTrPs are called active. MTrPs that are tender to palpation but not inherently painful when unprovoked are termed latent. The muscles of the head, neck, shoulders, upper back, and lower back are the most common sites for active and latent MTrPs.

A comprehensive multimodal strategy involving a combination of patient education, physical therapy, pharmacologic treatment, and interventional treatment is optimal in the treatment of MPS. Treatment should ultimately be guided by a thoughtful history and physical exam aimed at identifying not only the regions affected by MPS but also the predisposing or exacerbating conditions (e.g., history of trauma, depression, anxiety, spondylotic degeneration, arthritis, etc.). A 20-year-old patient with a history of depression and MPS affecting the muscles of the neck and shoulders may benefit from treatment with a serotonin-norepinephrine reuptake inhibitor (SNRI) antidepressant. A 70-year-old patient with MPS affecting the lower back muscles and a severely arthritic hip may maximally benefit from total hip arthroplasty, leading to amelioration of both the hip and back pain.

Pharmacologic Therapy

The classes of medications most commonly used in the treatment of MPS include nonsteroidal anti-inflammatory medications (NSAIDs), antidepressants, muscle relaxants, opioids, and local anesthetics.

For many patients with MPS, NSAIDs represent first-line therapy for acute and short-term treatment. Prolonged use may be limited due to gastrointestinal, renal, and hematological side effects. In a randomized controlled trial (RCT) of 153 patients with myofascial pain syndrome affecting the trapezius, Hsieh et al. found pain significantly better controlled with topical diclofenac compared to topical menthol placebo [8]. In a RCT of patients undergoing PT for nonspecific neck pain, the addition of ibuprofen to low continuous heat therapy was found to significantly reduce pain scores compared to low continuous heat therapy alone [9]. In addition, these patients were more likely to be compliant with home exercise programs [9].

Tricyclic antidepressants (TCAs) and SNRIs are commonly used for the management of a variety of pain conditions, including MPS. They have been found to exert analgesic effects independent of antidepressant effects through various mechanisms, including inhibition of norepinephrine and serotonin reuptake. Multiple studies have found TCAs to be effective in treating temporomandibular pain and tension-type headaches attributable to myofascial pain. In a double-blind, placebo-controlled, three-way cross-over study of non-depressed patients with chronic tension-type headache, Bendtsen et al. found amitriptyline significantly associated with reductions in headache pain and myofascial pain elicited from palpation of the frontal, sternocleidomastoid, masseter, temporal, and trapezius muscles [10].

Skeletal muscle relaxants are a structurally diverse group of medications including cyclobenzaprine (Flexeril), carisoprodol (Soma), methocarbamol (Robaxin), tizanidine (Zanaflex), baclofen (Lioresal), and benzodiazepines. Muscle relaxants act centrally within the brain and spinal cord to decrease skeletal muscle tone, thereby mitigating the increased muscle tone characteristic of MPS. Skeletal muscle relaxants are commonly used adjuncts employed in the treatment of MPS. Evidence supporting their use in the treatment of acute and chronic MPS has been mixed. In a meta-analysis in patients with acute low back pain, cyclobenzaprine was associated with only modest improvements in pain compared with placebo and significant increases in the risk of side effects [11]. In a RCT evaluating patients presenting to the emergency room with acute myofascial pain, the addition of cyclobenzaprine to ibuprofen was not found to confer a significant improvement in pain but was, again, associated with central nervous system side effects [12]. In a RCT of patients being treated for myofascial pain affecting the lower back and pelvis, methocarbamol was regarded as an effective treatment by roughly two-thirds of patients [13]. In a prospective cohort study of patients with chronic MPS, tizanidine was found to improve pain and sleep in 89% and 79% of patients, respectively, without the occurrence of serious adverse events [14].

Local anesthetics (e.g., lidocaine, bupivacaine, ropivacaine, etc.) used topically or via local infiltration are commonly used as a first-line treatment for MPS. Local anesthetics reversibly antagonize voltage-gated sodium channels, thereby increasing the threshold for electrical excitability. Multiple studies have reported improvement in MPS pain with the application of lidocaine patches. Rauck et al. reported an average pain improvement of 33% following heated lidocaine/tetracaine patch application in patients with >1 month of pain related to MTrPs [15]. Lin et al. reported significant improvements in pain and function after 2 weeks of treatment among patients treated with lidocaine patches compared to placebo [16].However, no significant differences were noted at 1 week or 4 weeks [16].

Minimally Invasive Therapy

Multiple treatment options have been employed for the treatment of MPS including, but not limited to, acupuncture, myofascial release, and dry needling. The current text will discuss the utilization of trigger point injections and botulinum toxin injection.

Trigger Point Injection

Trigger point injections (TPI) are the most common procedural technique employed for MPS.

A small-gauge (typically 25–27G) needle is used to inject local anesthetic (e.g., 0.5%–2% lidocaine, 0.25%–0.5% bupivacaine, 0.5% ropivacaine) with or without steroid (e.g., dexamethasone, methylprednisolone) into active and latent MTrPs identified by physical exam. Ideally, a local twitch response is elicited upon needle entry into the MTrP. Ultrasound may be used for identification of MTrPs and visualization of local twitch responses elicited from deeper and smaller muscles. On ultrasound, MTrPs can be visualized as hypoechoic bands with increased fiber alignment heterogeneity [17, 18]. Possible complications of TPIs are site-specific but almost universally include a risk of bleeding, hematoma, infection, nerve injury, nerve blockade, and/or worsening pain. Special care must be given in performing TPIs within the neck, shoulder, and chest wall due to increased risk of vascular injury, intravascular injection, and pneumothorax. Benefits of TPIs may be sustained for weeks to months. The need for repeated interventions is common. Factors most predictive of success include a history of localized symptoms, successful MTrP identification on examination and injection, and balanced multimodal care including ongoing physical therapy. Patients with a history of widespread pain and comorbid psychiatric disease are less likely to experience significant benefit. TPIs have been shown as an effective treatment modality for MPS, with uncertainty regarding the ideal injectate due to a lack of high-quality evidence [19].

Botulinum Toxin Injection

Botulinum toxin type A (BoNT-A) is a potent neurotoxin endogenously produced by the bacterium *Clostridium botulinum*. BoNT-A is used in the treatment of MPS for its ability to inhibit neuromuscular conduction, thereby reducing the dystonia characteristic of MPS. BoNT-A cleaves the t-SNARE protein SNAP-25, thereby inhibiting the docking and release of acetylcholine at the motor endplate. For pain that is localized, affecting the lower back, shoulders, and neck, injections are carried out into the painful dystonic muscles identified on exam. A small-gauge needle (e.g., 25 g–30 g) is used to inject between 5 IU and 20 IU BoNT-A per site into MTrPs identified on clinical exam. Side effects are rare and most commonly involve excessive untoward paralysis, worsening pain, or infection [20]. BoNT-A is most often utilized for MPS pain associated with cervical neck pain, headache syndromes, or spastic torticollis. Evidence describing the utility of BoNT-A in treating MPS in the extremities, pelvis, and lower back shows significant benefit compared to placebo but mostly equivocal differences when compared with local anesthetic [21, 22]. BoNT-A may also be beneficial in MPS associated with pelvic floor dysfunction. In a meta-analysis evaluating the efficacy of BoNT-A injection among women with symptomatic pelvic floor myofascial pain, Meister et al. described significant improvements in pain, dyspareunia, dyschezia, and quality of life sustained for 12 weeks post-injection [23]. A multi-center RCT evaluating pelvic floor myofascial pain found similar symptom improvement following BoNT-A with local anesthetic (0.2% ropivacaine) versus local anesthetic (0.2% ropivacaine) alone [21].

Lumbar Spine

There are a number of pain-generating pathologies of the lumbar spine that may contribute to axial lower back pain, with or without radiation of pain into the buttocks and lower extremities. The various etiologies of pain may include facetogenic pain, spinal stenosis of either the central canal or neural foramen, radiculitis due to disc herniation, discogenic pain, vertebral endplate pain, or pain related to compression fractures.

Depending on the constellation of symptoms, history and physical exam, as well as correlation with pathology on plain film and advanced imaging modalities of the lumbar spine, the pain specialist may consider various interventions to treat the patient.

Lumbar Epidural Steroid Injections

Lumbar spinal stenosis of the central canal and neural foramen and potentially resultant lumbar radiculitis have numerous potential etiologies. These commonly include disc height loss, facet joint hypertrophy, ligamentum flavum hypertrophy, or other consequences of lumbar spondylosis, with potential nerve root ischemia due to peri-radicular fibrosis [24]. Another important cause of lumbar radicular symptoms is herniation of the nucleus pulposus from the intervertebral discs. Despite these variable etiologies, they generally share symptomatology of radicular neuropathic pain within the lumbosacral dermatomes, with or without associated weakness, changes in sensorium, and loss or diminishment of deep tendon reflexes. Physical exam maneuvers, including the straight leg raise or Laségue test, and advanced imaging such as computed tomography (CT) or magnetic resonance imaging (MRI) of the lumbar spine may help with the diagnosis and delineation of the segmental level that is affected.

Epidural steroid injections are the most commonly performed procedure in pain clinics in the United States [25].They are most commonly performed at the lumbar levels, for symptoms of radiculitis and radiculopathy. The goal of the procedure is to inject medication at a pre-specified level into the epidural space. The medication mixture most often includes a corticosteroid, utilized for its inhibitory effect on inflammatory pathways, and is often accompanied by a local anesthetic or saline [26]. The local anesthetic or saline volume may contribute to increased medication spread or inflammatory mediator washout, and the local anesthetic may provide more rapid diagnostic value and additional therapeutic efficacy via vasodilation adjacent to ischemic nerve roots [24, 26, 27].The level to be injected is typically chosen after a detailed history and physical examination to delineate the involved nerve roots, with the procedure being most effective when these findings are concordant with pathology on advanced spine imaging [28]. The safety of epidural access is also considered when determining the level of access.

Approaches to the epidural space utilized by pain providers may be caudal, interlaminar, or transforaminal, with fluoroscopic guidance as the standard of care in current practice, although ultrasound and CT guidance have been used [29]. Though approaches vary by provider comfort and preference, generally the transforaminal approach is utilized when the radiculitis is found to be localized to specific nerve roots or when performed for a more specific diagnostic indication, such as presurgical planning. The interlaminar approach is often used when multiple segmental levels of pathology exist and can be directed paramedian if one side has worse pathology. A caudal approach may be utilized if the other two approaches have been proven to be especially difficult or when prior surgical intervention has made the epidural space less predictable to access.

Due to a large number of studies with differing patient cohorts, indications, procedure technique, and utilized medications, there has been some discordance for overall effectiveness of epidural steroid injections, as well as durability of pain relief and improvement in function. Cohen and colleagues published one of the more extensive reviews on the subject. Overall, the most consistent positive evidence has been for treatment of lumbar disc herniation, resulting in radiculitis, with some positive evidence for treatment of lumbar stenosis [30–32]. Another particular outcome of interest is the delay or avoidance of lumbar spine surgery, of which there is some evidence to show that lumbar epidural steroid injections may reduce progression to spine surgery [33, 34].

Adverse effects and complications range from relatively benign and short-lived to devastating, which are, fortunately, exceedingly rare. Minor adverse effects and events may include transient hyperglycemia in diabetic patients, adrenal suppression, accidental dural puncture potentially leading to a post-dural puncture headache, and transient lower extremity weakness due to the spinal effect of the local anesthetic [35, 36]. Serious adverse events include epidural hematoma, intravascular injection (especially of particulate steroid formulations) leading to spinal cord infarct, and epidural abscess, which may result in paralysis [37–39].

Lumbar Facet Interventions

The facet or zygapophyseal joints, along with the intervertebral discs, stabilize the spine against excessive motion. They are formed by articulation of the inferior articular processes of the vertebra above and the superior articular processes of the vertebra or sacrum below and are thus named accordingly (e.g., L4–L5 facet joint). Sensory innervation of the facet joints is specifically provided by the medial branch of the dorsal ramus of the lumbar nerve roots. At the lumbar level, these nerves course in a predictable manner over the junction of the transverse process and superior articular process. Each lumbar facet joint is innervated by two dorsal rami medial branch nerves, from the lumbar nerve root above, as well as from the lumbar nerve root at the same level.

Injury to the facet joints is generally a result of repetitive trauma and translated stress from degenerative disc disease and has a strong association with increasing age. The most commonly affected lumbar facet joints are L3–L4, L4–L5, and L5–S1 [40, 41]. Less common etiologies of facet joint arthropathy are the group of inflammatory arthritis conditions, such as rheumatoid arthritis and ankylosing spondylitis, synovial cysts, and pseudogout [42]. Reports of prevalence as a generator of low back pain vary widely, from as low as 15% to as high as 61%, when using certain thresholds of pain relief (from 50% to 100%) after a diagnostic nerve block of the facet joint [43]. Lumbar facet joint arthropathy may present as axial low back pain, may be unilateral or bilateral, and may have pain radiation laterally toward the flank or into the buttock area. Physical exam maneuvers including eliciting tenderness to palpation and facet loading (lumbar spine extension with rotation) may be performed, though no pathognomonic signs have been identified [44]. Advanced spine imaging can also be considered, with CT being potentially better for evaluation consistency than MRI, though overall evidence for use of these modalities is weak for identifying painful lumbar facet joints [44, 45].

Utilizing known facet joint innervation and anatomic location, dorsal rami medial branch nerves can be selectively blocked to diagnose and treat axial low back pain generated by lumbar facet arthropathy and arthritis. Interventions performed on the facet joints are the second most common procedure performed by pain specialists, second only to epidural steroid injections [25]. Fluoroscopic guidance is used to accurately and safely inject medications, typically local anesthetics with or without glucocorticoids, at the location of the medial branch nerves. Ultrasound-guided lumbar medial branch blocks have also been described [46]. An additional technique is intra-articular lumbar facet joint injection, which may be considered for facet joints with active inflammation.

Lumbar medial branch blocks or facet joint injections are commonly performed for diagnostic purposes or therapeutic relief or both. For purely diagnostic nerve blocks, a small volume of local anesthetic (<0.5 milliliters) is injected around the medial branches innervating the facet joint. Significant, but short-lasting relief is indicative of a positive diagnostic block. After a positive diagnostic block, a follow-up procedure that can be considered is a radiofrequency neurotomy of the medial branch nerve to provide more long-lasting relief. In this procedure, small cannulas are placed in parallel with the medial branches, and an electrical current is produced by radio waves to heat nervous tissue to a pre-specified temperature for a pre-specified amount of time to ablate nerve transmission. Prior to ablating the medial branch nerve, sensory and motor testing is performed to confirm that the needle tip is adjacent to the medial branch nerve and away from the nerve root at the neural foramen.

Similar to studies regarding efficacy of lumbar epidural steroid injections, there is a large body of literature regarding interventions targeting the lumbar facet joints. Again, with differences in patient cohorts, selection and diagnostic criteria, and procedural technique, a wide range of reports of efficacy exist. Relief of lower back pain after radiofrequency neurotomy in well-selected patients has been shown to be

significant up to the 1-year time point [47]. Another large study reported no clinically important improvement of chronic low back pain after radiofrequency neurotomy compared with a standardized exercise program alone [48].

With regard to the safety and adverse effect profile of medial branch blocks and ablation, there is a potential for intravascular needle placement, which may be mitigated by syringe aspiration prior to injection and use of contrast media. After ablation procedures, there is a small risk of pain, dysesthesia and numbness, which generally resolve in days to weeks [49, 50]. During ablation procedures, it is important to use multiplanar fluoroscopic images and sensory and motor testing to confirm needle tip placement to avoid inadvertent ablation of unwanted targets, such as the lumbar nerve roots.

Sacroiliac Joint Interventions

The sacroiliac joints are the synovial joints between the sacrum and ilia, with many ligamentous and muscular connections, especially on the posterior aspect. The sacroiliac joints serve to restrict motion in all planes and to dissipate truncal loads to the lower extremities. The posterior aspect of the sacroiliac joint receives sensory innervation via the lateral branches of the L4–S4 dorsal rami. The sensory innervation of the anterior aspect of the sacroiliac joint is more ambiguous, with potential contribution from the ventral rami of L4–S2.

Sacroiliac joint dysfunction describes pain generated at the sacroiliac joint, which commonly occurs due to increased stress on the joint. Age-related degenerative changes occur within the sacroiliac joint, including surface irregularities, crevice formation, and fibrillation of the iliac and sacral surfaces [51]. There may be a history of trauma translated to the joint or translated stress from adjacent stress-bearing structures, such as the lower lumbar facet joints and intervertebral discs. In addition, sacroiliitis is often a prominent characteristic of the adult spondyloarthropathies. Symptoms may include superior buttock pain, which may include radiation of the pain into the lateral or posterior thigh or groin and, uncommonly, inferior to the knee. Bending and sitting are often exacerbating factors, while standing generally relieves the pain. On physical exam, there may be reproduction of pain with manual palpation over the sacral sulcus and reproduction of pain with a number of provocative maneuvers, such as the Patrick's test, Gaenslen's test, distraction, sacral thrust, or compression maneuvers. Imaging studies, such as radionuclide bone scans and CT, have been shown to have inadequate sensitivity (<60%) for identification of sacroiliac joint pain [52, 53]. Diagnosis of sacroiliac joint pain is often made after diagnostic local anesthetic injections, with improved accuracy with higher thresholds of pain relief with dual injections [54]. Although widely acknowledged as a significant etiology of low back pain, especially below L5–S1, reports of prevalence vary between 15% and 25% as these reports have differed on method of diagnosis, such as by physical exam, imaging studies, or diagnostic injections with varying criteria [40, 51].

Interventions performed by pain management specialists may include intra-articular and peri-articular sacroiliac joint injections, lumbosacral lateral branch nerve blocks, and radiofrequency neurotomy of these same nerves. Injection into the sacroiliac joint is performed by directing a needle with fluoroscopic guidance through the posterior capsular tissue into the joint space formed between the sacrum and ilium. A mixture of corticosteroid with local anesthetic is often injected. An alternative intervention for sacroiliac joint dysfunction is fluoroscopically guided nerve block of the sensory innervation of the posterior sacroiliac joint, as described by Dreyfuss and colleagues [55]. Similar to the previously described radiofrequency neurotomy of the lumbar medial branches for facetogenic pain, the lumbosacral lateral branches are amenable to neurotomy (conventional and cooled) to treat sacroiliac joint-mediated pain.

The evidence for intra-articular and peri-articular sacroiliac joint injections with corticosteroid and local anesthetic is generally positive with regard to pain relief and improved function, typically for a duration of 3 months [56–58]. Studies regarding radiofrequency neurotomy showed approximately 50% improvement at 6 months, with slightly more improvement when cooled radiofrequency neurotomy was performed compared with traditional radiofrequency neurotomy [59, 60].

The risk of adverse events after intra-articular sacroiliac joint injections is minimal, with minor reported events of injection site soreness or pain exacerbation [61]. For sacral lateral branch neurotomy procedures, rates of procedural adverse effects are also very low and may include transient post-procedural neuropathic pain [62].

Thoracic Spine

The prevalence of thoracic spine pain is lower compared to low back and neck pain, as the thoracic region of the spine is relatively protected against postural pain and mechanical dysfunction compared to the more mobile cervical and lumbar regions [63]. However, patients with pain originating from the thoracic spine still make up a significant proportion of patients seen in the interventional pain management setting and make up a comparatively younger cohort of patients [64, 65].

Thoracic Facet Interventions

The thoracic facet joints are also formed by articulation of the inferior articular processes of the vertebra above and the superior articular processes of the vertebra below. Sensory innervation of the thoracic facet joints is similar to that of the lumbar facet joints, such that the sensory innervation is provided by the medial branch of the dorsal ramus of the nerve root from the same level and the level above. In cadaveric studies, the course of the nerve is more consistently targeted at the superolateral corner of the transverse process [66].

Using diagnostic thoracic medial branch blocks with local anesthetic, a 48% prevalence of thoracic facetogenic pain was determined in patients with midback and upper back pain, albeit with a high false-positive rate [67]. Pain referral patterns of thoracic facetogenic pain may range from the suprascapular region for the upper thoracic facet joints and scapular region, midback, and laterally toward the iliac crests for the middle and lower thoracic facet joints [68, 69].

For axial upper back and midback pain, the pain specialist may consider thoracic medial branch blocks for diagnostic and therapeutic purposes. They are most often fluoroscopically guided, may be unilateral or bilateral, and typically include an injection of corticosteroid and local anesthetic. Similarly, radiofrequency neurotomy may be considered if the nerve blocks are diagnostic but not long-lasting.

As interventions for thoracic facetogenic pain are performed less commonly than for cervical and lumbar facetogenic pain, there is accordingly less literature devoted to these interventions. However, there is fair evidence to support the performance of therapeutic thoracic medial branch blocks to treat chronic facetogenic upper back and midback pain [64]. Regarding radiofrequency neurotomy of the thoracic medial branches for thoracic facetogenic pain, the evidence is positive but limited, and it was recently reported that cooled radiofrequency neurotomy may be effective for thoracic facetogenic pain [70, 71].

In addition to similar risks related to lumbar medial branch blocks, such as inadvertent intravascular injection, there is the additional theoretical risk of lung injury, potentially resulting in pneumothorax [72]. For radiofrequency neurotomy, there are similar risks of post-procedural pain and, rarely, superficial skin burns [71].

Thoracic Epidural Steroid Injections

Thoracic spinal stenosis may be due to compression from ligamentum flavum ossification, posterior longitudinal ligamentum ossification, and thoracic disc herniation [73]. Degeneration of the thoracic disc and endplate irregularities and osteophyte formation are common findings, but thoracic disc herniations are much less common than what occurs at the cervical and lumbar regions [74]. They are estimated to make up just 0.25%–1% of all disc herniations. When they do occur, it is most commonly in the mid-thoracic and lower thoracic spine, at T8–T11 [75].The lower incidence of herniations is ascribed primarily to the reduced allowable flexion at the thoracic level compared with the lumbar and cervical levels [74]. Thoracic disc herniation or spinal stenosis may present with localized thoracic back pain, axial pain down the spine, or radicular pain around the flank and chest wall. Additional symptoms may include bladder dysfunction (typically urgency), motor deficits, and sensory impairment, such as paresthesia or dysesthesia, in the lower extremities [76]. Advanced imaging of the thoracic spine, including MRI or CT, is helpful in determining the nature and location of pathology. Overall description of the evaluation of thoracic discogenic pain is limited [77].

Thoracic epidural steroid injections may be performed to treat upper back and midback pain due to disc herniation, discogenic pain, spinal stenosis, and/or post-surgery pain. The epidural approach may be interlaminar or transforaminal, depending on the location and specificity of pathology, with the transforaminal approach providing a more specific and localized target. The injectate typically includes corticosteroid with or without local anesthetic.

There is limited literature regarding thoracic epidural steroid injections, but there is overall fair evidence for thoracic epidural steroid injection to treat pain from thoracic disc herniation and disc degeneration [78, 79].There is even less evidence regarding the transforaminal approach for thoracic epidural steroid injections, though a high rate of short-term pain relief has been reported [80].

The majority of adverse events related to thoracic epidural injections are minor and may include local numbness, muscle spasm, headache, vasovagal symptoms, and minor bleeding. Major complications are pneumothorax and paraplegia resulting from inadvertent injection of particulate steroid into a collateral of the artery of Adamkiewicz, both of which have an extremely rare incidence [80, 81].

Additional interventions performed at the thoracic spine, such as thoracic paravertebral injections and intercostal nerve blocks, may address a myriad of thoracic pain syndromes but are beyond the scope of this chapter regarding spinal pain etiologies.

Cervical Spine

The cervical spine segments are oriented specifically to allow complex movements of the neck, including flexion, extension, rotation, and lateral bending. After degenerative changes or injury, the cervical spine may be involved in various pain syndromes, such as facetogenic axial neck pain, cervical discogenic pain, cervicogenic headaches, and cervical stenosis or disc herniation, resulting in upper extremity radiculitis and radiculopathy.

Based on history and physical exam, with correlation with pathology on plain film and advanced imaging modalities of the cervical spine, a number of interventions may be considered and performed by pain specialists.

Cervical Epidural Steroid Injections

Cervical spinal stenosis has a positive correlation with advancing age. Various etiologies resulting in cervical spinal stenosis may include ossification of the posterior longitudinal ligament, ossification of the ligamentum flavum, and spondylosis. With cervical spondylosis, degenerated intervertebral discs and loss of disc height may increase the transfer of stress to the uncovertebral joints and facet joints, resulting in hypertrophy and osteophyte formation, and potentially ligamentum flavum

buckling, all of which may decrease the diameter of the cervical spinal canal, as well as the neural foramen [42]. The most commonly affected segmental levels include C3–C4, C4–C5, and C5–C6.

Regarding disc herniation resulting in cervical radiculopathy, its occurrence is less common than at the lumbar region. Compared to lumbar intervertebral discs, cervical discs are thicker at the anterior aspect compared with the posterior aspect and have a less well-defined nucleus and annulus structure. Their function is less important for disbursing axial load. Cervical disc herniation resulting in cervical radiculopathy is most common for the C6 and C7 nerves [82].

The symptoms of cervical stenosis and radiculopathy may include dermatomal neuropathic pain, upper extremity weakness, paresthesia or other sensory disturbance, and loss or diminishing of reflexes, which can be assessed with history and physical exam. Advanced imaging, such as MRI of the cervical spine, may help corroborate physical exam findings with precise description of the location and severity of pathology. While chronic neck pain is an extremely common complaint, cervical radicular pain has been reported to have an annual incidence of 83 per 100,000 [82].

Cervical epidural steroid injections may be performed to treat symptoms of cervical stenosis, cervical disc herniation, and cervical discogenic pain. At the cervical level, they may be most safely performed via an interlaminar approach. The segmental level at which the epidural space is accessed is chosen based on the diagnosed level of pathology, as well as the safety and feasibility of epidural access, though it has been recommended to be performed at the C7–T1 level and preferably no higher than the C6–C7 level [83]. Typically, a combination of a corticosteroid and a local anesthetic or saline is injected into the epidural space. The transforaminal approach has become less favorable due to the increased acknowledgment of risk, weighed against the potential therapeutic benefit of the injection. This is due to the variable vessel courses and anastomoses between the vertebral and cervical arteries in the anatomic location traversed during the transforaminal approach. Inadvertent intravascular injection, especially of a particulate steroid, can lead to catastrophic injury to the patient. In consideration of this, avoidance of particulate steroid, local anesthetic test doses, real-time contrast visualization, and digital subtraction are recommended.

Cervical epidural steroid injections have been shown to be most beneficial in the treatment of radiculopathy due to herniated discs, with fair evidence for the treatment of spinal stenosis, discogenic pain, and pain following cervical spine surgery [84, 85]. However, the benefit may be most apparent in the short term and should be used as a therapy after more conservative treatments are employed [86].

As alluded to previously with the presence of important vasculature in the vicinity of the procedural anatomic area, several reports of significant adverse events related to cervical epidural steroid injections have been made. Vascular injection of particulate steroid and resultant embolism to the brain or spinal cord and vasospasm or dissection due to needle trauma may occur, with higher risk associated with the transforaminal approach. In addition to vascular insult, bleeding diathesis, direct

neural trauma, pneumocephalus, and high spinal anesthesia are possible major adverse events, which can result in death [86].

Cervical Facet Nerve Blocks and Ablation

Similar to the lumbar facet (or zygapophyseal) joints, the cervical facet joints are diarthrodial joints, with articulation of the inferior articular process of the upper vertebrae with the superior articular process of the vertebrae immediately below. At the cervical levels, the superior and inferior articular processes arise from the vertebral lateral masses (or articular pillars), which are at the junction of the pedicle and lamina. Innervation of the cervical facet joints is more variable than that of the lumbar and thoracic facet joints. From C3–C4 to C7–T1, they similarly have dual innervation from medial branches of the dorsal rami of the nerve root from the level above, as well as the same level. The medial branches at these levels course in a predictable path across the waist of the corresponding articular pillars, which can be identified with fluoroscopic imaging. For the C2–C3 facet joint, the major innervation is derived from the C3 dorsal ramus. The C3 dorsal ramus divides into two medial branches: one termed the third occipital nerve and the other termed the deep medial branch. The C3 deep medial branch, similar to the lower cervical medial branches, courses around the waist of the C3 articular pillar. The third occipital nerve courses lateral to the C2–C3 facet joint. The C2 dorsal ramus provides some innervation to the C2–C3 joint, and the medial branch becomes the greater occipital nerve.

Pathology of the cervical facet joints may be related to gradual degeneration and osteoarthritis of the joints, or from traumatic injury such as a whiplash etiology, or other flexion/extension injuries. Cervical facet joint pathology may result in axial neck pain, proximal upper extremity pain, and referred pain in the head or cervicogenic headaches. Cervicogenic headaches are often unilateral but may be bilateral, with painful areas including the tendinous insertions of the occipital area and along the areas innervated by the greater and lesser occipital nerves [87]. By definition, cervicogenic headaches arise from pathology in the neck that can include cervical facet arthropathy and are otherwise based on criteria from the International Headache Society [88]. There are no clinically validated physical exam maneuvers that are pathognomonic for cervical facet joint dysfunction [89]. Plain radiographs may screen for instability or gross fractures or lesions, and advanced imaging, such as CT or MRI, may further evaluate cervical facet joint dysfunction. For patients with head, neck, and upper extremity pain, the cervical facet joints are found to be the responsible pain generator, based on diagnostic cervical medial branch nerve blocks, in up to 36–55% of patients [90, 91]. However, there was a high false positivity rate.

Cervical medial branch nerve blocks are not often performed higher than those for the C2–C3 facet joint (which is the first joint of the spine possessing a true joint capsule and synovium), although atlantoaxial joint block techniques are described [42].

For C2–C3 and lower cervical facet joints, cervical medial branch nerve block injections can be considered to treat axial neck pain, referred pain to the proximal upper extremities and head, and cervicogenic headaches [92]. Cervical facet nerve blocks are often performed at two to six levels per procedure, unilaterally or bilaterally, and may include a combination of a local anesthetic and corticosteroid. If symptomatology includes upper axial neck pain, and potentially radiation of pain into the occiput, the procedure may often include blocks of the C2 medial branch or greater occipital nerve, the C3 and C4 medial branches, and potentially the third occipital nerve. If the axial neck pain is lower, and potentially with radiation into the upper back and proximal upper extremities, the pain specialist may consider medial branch nerve blocks at all or some of the levels of the C4–C5–C6–C7 segments. Cervical facet nerve blocks are most commonly performed with fluoroscopic guidance, and a posterior or lateral approach is utilized. As previously described, radiofrequency neurotomy at the same segmental levels may be considered if diagnostic validity is established with the nerve blocks, especially if only short-term therapeutic value occurs. Intra-articular cervical facet joint injections may also be performed, though they are performed much less commonly.

Cervical medial branch blocks have been shown to provide therapeutic benefit with both pain relief and improved functional status in patients with chronic neck pain [93]. In one study, single injections provided pain relief for approximately 14–16 weeks, and patients typically received three to four injections per year [94]. Radiofrequency neurotomy of the cervical facet joints has been shown to provide pain reduction for up to 8 months in patients with cervical facet joint dysfunction [89, 95].There is less evidence for cervical medial branch blocks and radiofrequency neurotomy to treat cervicogenic headaches, though a trend for therapeutic benefit at 3 months has been demonstrated [92, 96].

Adverse events related to the performance of cervical facet joint injections and neurotomy are infrequent but may include inadvertent intrathecal injection if the nerve root sleeve is entered, inadvertent vascular injection, and hematoma [89, 97, 98].

Post-Laminectomy Syndrome

Surgical decompression with and without fusion is a mainstay for the treatment of symptomatic spinal stenosis refractory to conservative management [99, 100]. Laminectomy and spinal fusion have remained among the top ten most commonly performed procedures in the United States requiring an inpatient stay from 2008 to 2015 [101]. Post-laminectomy syndrome (PLS) or failed back surgery syndrome (FBSS) is defined by persistent regional or radicular pain refractory to previous spinous surgery. The prevalence of PLS is difficult to ascertain, due to its broad definition and heterogeneous etiologies, but has been estimated to affect between 20% and 40% of patients undergoing spine surgery [102, 103]. Spinal cord stimulation (SCS), dorsal root ganglion (DRG) stimulation, and intrathecal pump (ITP) drug

delivery are three treatment modalities utilized by pain management specialists in the treatment of patients with PLS.

Spinal Cord Stimulation

Inspired by the gate-control theory of pain proposed by Melzak and Wall in 1965, Spinal Cord Stimulation (SCS) was first performed by Norman Shealy in 1967 [104, 105]. The gate-control theory hypothesized that within the spinal cord, nociceptive signals (delivered by Aδ fibers and C fibers) compete with non-nociceptive signals (delivered by Aβ fibers) for transmission to the brain. A shift in this balance, induced by SCS, is hypothesized to decrease the perception of pain. SCS is used in the treatment of a wide variety of chronically painful conditions (e.g., PLS, complex regional pain syndrome, radiculopathy, neurogenic pain, claudication, visceral pain). The mechanism of pain relief provided by SCS has since been expanded since the proposal of gate-control theory and is additionally hypothesized to be dependent on the nature of the disease being treated and parameters of stimulation employed.

SCS implantation is a staged procedure, whereby electrodes are placed within the epidural space and used to deliver an electrical current. SCS leads are of two types: (1) flat paddle leads and (2) cylindrical percutaneous leads. Paddle leads are implanted via surgical laminotomy and will not be discussed in this text. Cylindrical percutaneous leads are placed percutaneously via an epidural introducer needle. The first stage of SCS implantation is the trial, during which temporary leads are placed into the epidural space with the distal ends of the SCS leads remaining outside of the body and connected to an external generator. A typical trial is an outpatient procedure and lasts between 5 and 14 days, during which time the patient determines if the pain relief provided by the spinal cord stimulation is adequate. At the conclusion of the trial, the percutaneous leads are removed. Trial success is typically defined as greater than 50% improvement in pain and/or function. In the case of a successful SCS trial, the patient will then move onto permanent implantation, which requires implantation in an operating room via one to two small incisions for lead and generator placement.

Stimulation patterns most commonly employed by current SCS devices typically fall within one of the three categories: (1) traditional or tonic, (2) burst, and (3) high frequency. The goal of traditional SCS is to generate a paresthesia, overlapping the area of the patient's pain, thereby overshadowing nociceptive signaling from the area. Burst SCS currents mimic the firing of thalamic cells and are hypothesized to stimulate both medial and lateral pain pathways. The medial pathway is thought to process the affective-motivational component of pain [106]. High-frequency stimulation is paresthesia-independent and utilizes a current with low amplitude and high frequency. High-frequency stimulation is hypothesized to attenuate nociceptive signaling via modulation of wide dynamic range neurons of the dorsal horn. It is common for patients to cycle between different modes of stimulation in the control of their pain.

High-quality evidence describing the utility of SCS for PLS is limited. North et al. randomized 50 patients with a history of radicular pain refractory to previous decompressive surgery to undergo either repeat surgical intervention or SCS [107]. Success was defined as at least 50% improvement in pain and patient satisfaction with treatment [107]. Spinal cord stimulation was significantly more successful than reoperation (9/19 patients versus 3/26 patients, $p < 0.01$) [107]. Kumar et al. randomized 100 patients with PLS to undergo either SCS plus conventional medical management (CMM) or CMM alone [108]. Spinal cord stimulation patients were found to have better pain relief, improved functional capacity and health-related quality of life, and satisfaction with treatment [108].

Complications following SCS range from continued or worsened pain to paralysis and even death. Up to 40% of patients are estimated to experience one or more complications postoperatively [109]. Lead migration, lead fracture, and surgical site infection are among the most common postoperative complications. Lead migration has been reported to occur at rates between 2.5% and 23.1%, and lead fracture has been reported to affect up to 10% of SCS patients [109]. Infection rates up to 10% have been described among SCS patients postoperatively [109].

Dorsal Root Ganglion Stimulation

Dorsal root ganglion (DRG) stimulation is an expansion of traditional SCS that can be employed for treatment of localized pain in a specific dermatome that is refractory to other more conservative treatments. DRG stimulation is indicated for the management of complex regional pain syndrome but has been used in other pain syndromes. There are 29 paired dorsal root ganglia (8 cervical, 12 thoracic, 5 lumbar, and 4 sacral) located on the distal ends of the dorsal roots in the anterolateral epidural space. The dorsal root ganglia serve as relay stations, housing the cell bodies of primary sensory neurons and modulatory spinal glial cells. Platinum electrodes are placed in the neuroforaminal stenosis and used to deliver an electric current to selected nerve ganglia.

DRG stimulator placement is similar to SCS placement. Both devices are implanted in a staged fashion, whereby leads are percutaneously placed through the epidural space as a trial for 5–14 days, followed by permanent device implantation after a successful trial. A notable difference is that DRG leads are placed into the desired neural foramina versus the dorsomedial epidural space as in SCS.

The evidence describing the utility of DRG stimulation for PLS is weak (level II–III) and preliminary. In a case series of 12 patients with PLS, Huygen et al., described ≥50% reduction in pain for most patients undergoing DRG lead placement at level L2 or L3. In a prospective cohort study of 51 patients with chronic neuropathic pain, DRG was found to be less effective for PLS than other diagnoses [110]. Presently, DRG is not considered as a first-line treatment for PLS but may be considered in cases refractory to other treatments such as SCS [111]. Comparative

studies between DRG and SCS are limited. Complications following DRG implants are likely similar in nature and frequency to SCS [112].

Intrathecal Pump

Intrathecal drug delivery (IDD) dates back to the works of Dr. James Corning and Dr. August Bier. Dr. Corning is credited with the first use of neuraxial anesthesia in 1885 [113]. However, controversy exists whether his injectate was placed into the subarachnoid space or the epidural space. In 1898, Dr. Bier injected cocaine into the subarachnoid space, providing sufficient anesthesia for surgery [114]. This was the first procedure done under spinal anesthesia. Today, intrathecal pumps are used to continuously deliver analgesic medication to the subarachnoid space at doses much lower than oral or intravenous therapy. For example, intrathecal opioids can produce analgesia at doses 1/300th the dose required by oral administration. Lower systemic concentrations are often desired to avoid harmful side effects associated with analgesic medication. Medication classes commonly used for continuous intrathecal administration include opioids, calcium channel blockers, gamma-aminobutyric acid agonists, and alpha-2 adrenergic agonists. Of these, only morphine, baclofen, and ziconotide are approved for this purpose in the United States. Candidates for ITP therapy should have pain refractory to prior treatments or suffer side effects prohibitive of oral medication administration. Contraindications to neuraxial intervention (e.g., coagulopathy, systemic infection) are contraindications to ITP implantation. Confounding psychological conditions and shortened life expectancy also serve as important factors that may preclude the use of ITP therapy.

ITP implantation is a staged procedure involving a trial followed by permanent implantation. The trial is performed to determine if neuraxial IDD provides sufficient clinical improvement. Trials are performed in either inpatient or outpatient settings. During a trial, medication is administered via single-shot intrathecal administration, an epidural catheter, or an intrathecal catheter. If a catheter is used for dose titration, inpatient monitoring is usually preferred due to the possibility of rostral spread and respiratory depression. Permanent ITP implantation is performed in the operating room and can be done under sedation, spinal anesthesia, or general anesthesia. Permanent ITP implant involves threading a catheter to the desired level within the intrathecal space and then connecting that catheter via a subcutaneous tunnel to an implanted pump reservoir.

Intrathecal pumps come in multiple sizes and hold either 20 or 40 ml of medication. Medication dosing can be changed easily via a wireless communication system. The medication itself can also be changed via the ITP access port that is available by percutaneous puncture through the skin and into the pump reservoir. The typical battery life of an ITP is approximately 5 years, after which time the patient requires a pump exchange done in the operating room. Medication refill requirements vary greatly depending on the reservoir volume of the ITP and the medication dosage, but on average, ITP medications are refilled every 2–4 months.

Acute and subacute complications related to the procedure include postdural puncture headache, incomplete hemostasis, seroma formation, and infection. Epidural hematoma formation may present with acute neurological change and may require emergent surgical evacuation. Similarly, deep surgical infection may also require emergent incision and debridement and device removal.

Extreme caution and vigilance are required in programming and refilling the ITP. Overdose or underdose may occur with inappropriate programming. Subcutaneous medication injection outside of the ITP may result in a "pocket fill" and toxic overdose. Medication delivery can become impeded by catheter kinking or fracture or granuloma formation at the catheter tip. Granuloma formation may also cause neurologic deficits by local mass effect [115]. Kinking or fracture may require catheter replacement by surgical access. Granuloma formation may be reversed by changing the medication but may require surgical excision.

Intrathecal pump medication choice and dosing is often the product of an effort to balance desired analgesia and avoid unwanted side effects. Morphine and ziconotide are the only medications approved for intrathecal analgesia. Fentanyl, hydromorphone, bupivacaine, and clonidine are also used in an off-label fashion for their analgesic effects. An ITP injectate may be a mixed solution containing two or more mechanistically distinct medications (e.g., hydromorphone and bupivacaine). High-quality comparative studies evaluating different ITP medications are lacking. According to a previous multinational survey, most physicians significantly reduce or complete discontinue systemic opiate therapy before or during the ITP trial [116]. Grider et al., described the strategy of microdosing in 2011, whereby all patients were weaned of systemic opioids for a 6-week period [117]. After ITP implantation, intrathecal morphine was titrated in the microgram dose range until satisfactory analgesia was achieved [117].

Historically, ITP use has been reserved as a salvage procedure for patients with noncancer chronic pain refractory to more conservative treatments. High-quality evidence supporting the use ITPs is limited but suggests utility in select patients. In a randomized, double-blind, placebo-controlled study of 220 patients with chronic pain, Rauck et al. found pain control significantly improved after the institution of ziconotide IDD. In this study, PLS was the most common source of pain [118]. The visual analogue score among patients with PLS improved by an average of approximately 30% [118]. Most (92.9%) of patients receiving ziconotide experienced an adverse event, most commonly dizziness (47.3%) and nausea (41.1%). Occasionally, dual-modality management using a combination of SCS and ITP therapy is utilized for pain control refractory to single-modality management. In a case series of 11 patients with PLS who underwent nonsimultaneous implantation of both SCS and ITP devices, all patients reported both devices imparted significant improvements in quality of life [119]. However, extremely judicious use of dual-modality management seems necessary before employing this strategy, as 7 of 11 patients (64%) experienced a hardware-related complication necessitating revision surgery [119] (Table 3.1).

Table 3.1 Summary of chronic musculoskeletal pain with a synopsis of presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Chronic musculoskeletal pain	Chronic pain involving one or more joints, the neck, back and/or myofascial structures Stiffness and limited range of motion Poor activity tolerance	Plain film radiographs of affected musculoskeletal region Computed tomography and MRI, as well as other advanced imaging studies as indicated to rule out occult pathology or injury	Nonsteroidal pain medication, muscle relaxers, antidepressants Physical therapy and non-weight bearing exercise Injections including trigger point, facet joint, epidural, sacroiliac joint, and peripheral joint procedures	Spinal cord stimulator or intrathecal pain pump patients with noncancer chronic pain refractory to more conservative treatments

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Chapter 4
Adult Spinal Deformity

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Definition and Epidemiology

Adult spinal deformity (ASD) is defined as a deviation of the spine in either the coronal or sagittal planes. ASD is typically a degenerative process originating from the intervertebral disc with destruction of the disc resulting from enzymatic destruction of disc tissue and loss of proteoglycans in the disc [1]. Ultimately, these biochemical changes and destruction of the disc lead to a change in load bearing and instability in the intervertebral and facet joints between the vertebral bodies. Compensatory paraspinal muscle strain further aggravates this cycle of ongoing muscle weakness, spinal compression, and deformity. Deformity can occur in a multitude of ways: scoliosis, kyphosis, lordosis, loss of kyphosis or lordosis (flatback), rotation (of one vertebral body in relation to another), and listhesis (translation of one vertebral body on another). Notably, osteoporosis contributes to and may accelerate spinal deformity [2]. As such, medical treatments to combat osteoporosis can potentially slow the progression of spinal deformity, highlighting the importance of recognizing and addressing decreased bone mineral density early on.

Cobb Angle Measurement

Spinal deformity is evaluated by angular measurements of spinal curvature using Cobb angles. These angles correlate with the severity of the condition and are important to consider in both non-operative and surgical management of patients

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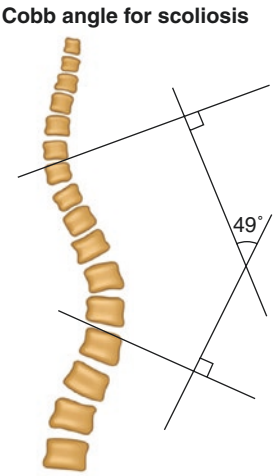

Fig. 4.1 Depiction of measurements used in developing a Cobb angle

with spinal deformity. Specifically, Cobb angles are measured by first identifying the upper and lower vertebral bodies that define the deformity. From there, tangent lines are extended from the upper border of the most cranial vertebral body and from the lower border of the most caudal vertebral body. The Cobb angle itself is defined as the angle formed by the intersection of lines that are perpendicular to the above tangent lines (Fig. 4.1).

An imbalance in the coronal plane is defined as a deviation greater than 10° as measured by the Cobb angle. In the sagittal plane, kyphosis refers to the forward curvature of the spine. The thoracic spine normally has kyphosis between 30 and 50° as measured by the Cobb angle. Normally, the cervical and lumbar spine have lordosis; lordosis describes the posterior curvature of the spine, with normal lumbar lordosis being between 35° and 80° [3]. Overall, ASD is the result of progressive and asymmetric degeneration of multiple spinal elements including the discs and facet joints, with sufficient degeneration to compress and potentially compromise the spinal cord or neural components [4].

Scoliosis

Adult spinal deformity includes a variety of different presentations. With regard to scoliosis, de novo scoliosis develops after completion of skeletal maturity. De novo scoliosis encompasses degenerative causes, iatrogenic causes, and post-traumatic causes. Progressive idiopathic scoliosis is a result of untreated scoliosis from childhood. In contrast to progressive idiopathic scoliosis, which often primarily affects the thoracic spine, de novo scoliosis usually affects the lumbar spine and lacks classic curve patterns typically seen in adolescent idiopathic scoliosis. Other differences include decreased magnitude of curvature and a faster rate of progression in de novo

scoliosis compared to progressive idiopathic scoliosis. De novo scoliosis is estimated to progress at a rate of 1.64°/year compared to 0.82°/year in progressive idiopathic scoliosis [5].

Kyphosis/Lordosis

With regard to deformity in the sagittal plane, there are several presentations which are commonly seen in the primary care setting. Flatback syndrome is a presentation of adult spinal deformity consisting of loss of lumbar lordosis due to straightening of the normal lumbar sagittal curve [6]. Flatback syndrome is an iatrogenic condition which results from loss of sagittal spinal alignment due to prior surgical intervention on the lumbar spine [6, 7]. Degenerative hyperkyphosis is a result of excessive anterior curvature of the spine; while the exact cutoff for a Cobb angle measurement varies, an angle of greater than 40° of regional kyphosis is typically used to define hyperkyphosis [8]. A significant portion of hyperkyphosis is thought to be a result of vertebral fractures in osteoporosis [9]. Notably, degenerative hyperkyphosis can contribute to not only pain and spinal deformity but also worsening pulmonary function and increased risk of further vertebral fractures [9].

Epidemiology

The increasing prevalence of musculoskeletal diseases parallels the aging global population. Numerous factors including improving healthcare and improved standards of living have extended average life spans [10]. Predictions show that the total aged population is expected to increase, with a quarter of the population of the United States expected to be older than 65 years old by 2060 [10]. Within this population, the prevalence of spinal deformity is high, with estimates of 32–68% of people over the age of 65 having a spinal deformity [4]. As spinal deformity is typically due to age-related degenerative changes, it is not surprising to note very high prevalence particularly in the highly aged population. One estimate of lumbar scoliosis in the United States noted a prevalence of >50% in patients ≥90 years old [11]. There was also no gender difference in adult spine deformity prevalence [11].

De novo scoliosis has a mean age of presentation of 70.5 years and is estimated to have a population prevalence of 6% in adults over the age of 50 [12, 13]. Degenerative hyperkyphosis is present in about 20–40% of adults, with a mean age of 78.3 years [9]. While the incidence of flatback syndrome as reported in the literature varies and depends on the spinal levels of the prior surgeries [14], figures range from 5% to 49% of symptomatic loss of lumbar lordosis after lumbar spine surgery [15, 16].

The rate of spinal deformity surgery has also risen dramatically. One estimate based on the National Inpatient Sample (NIS) database found the case rate increased from 4.16 per 100,000 adults in 2001 to 13.9 cases per 100,000 adults in 2013 [17].

In particular, hyperkyphosis is associated with osteoporotic vertebral fractures [4]. There is evidence that treatment of osteopenia and osteoporosis through increased screening and improved pharmacotherapeutic options is leading to reduced numbers of vertebral compression fractures, countering the trend of increased hyperkyphosis with age [19, 20]. Treatment of osteoporosis via bisphosphonates, parathyroid hormone, selective estrogen receptor modulator, raloxifene, and monoclonal antibody inhibitor to RANK ligand, denosumab, and other treatments have been shown to reduce the relative risk of vertebral fractures [21].

Weakening of paraspinal muscles with age also leads to reduced compensatory support for the progressively deforming spine. Other age-related conditions that contribute to an increased prevalence of adult spinal deformity in older populations include neurodegenerative disorders [18].

The morbidity from adult spinal disorders is high. Survey-based studies comparing physical and mental well-being of adult patients with spinal deformity to scores of patients with other chronic diseases found similar or worse scores when compared to patients with conditions such as hypertension, diabetes, heart disease, or lung disease [22, 23]. This trend has also been corroborated internationally in a study across 8 industrialized countries which found that patients with ASD had worse 36-item short-form survey (SF-36) scores than any chronic condition reported, including chronic lung disease, diabetes, and congestive heart failure [24]. Notably, within the ASD patients in this study, surgically indicated ASD candidates had the worst scores, while postsurgically corrected ASD patients had the best scores [24].

The type of spinal deformity also correlates with health-related quality of life (HRQL) metrics, with thoracic scoliosis patients having improved HRQL compared to lumbar scoliosis patients [23]. Furthermore, there is also a strong correlation between spinal deformity and mental health disorders. Among patients with musculoskeletal disorders, patients with ASD had the highest psychological burden, including depression, sleep disturbance, and anxiety, based on a review of the National Inpatient Sample [25].

Clinical Presentation and Diagnosis

Patients with symptomatic ASD may present with several notable symptoms, including back pain, sensation of imbalance or changes in posture (e.g., forward-pitched posture, truncal shift), or neurological symptoms such as radiating pain or paresthesia, or weakness [26]. Some patients present only with radiographic abnormalities noted incidentally on imaging. The decision to refer patients to spinal surgical providers is often dependent upon patient history and examination. The assessment of patients suspected to have ASD based on presentation or on imaging

should first entail a complete history and physical of both the neurological and musculoskeletal systems in addition to a radiographic assessment [27]. The history should include a thorough review of pain symptoms, neurological symptoms, and mobility. Pain symptoms should be documented by location, change over time, and severity, along with aggravating and alleviating factors. The history should be extended to include HRQL assessments to understand how debilitating a patient's ASD symptoms are in terms of mobility, activities of daily living, and psychological symptoms. HRQL assessments provide a more holistic understanding of the impact of ASD on a patient's life beyond what radiographic assessments can provide [28]. This is particularly important as multiple radiographic parameters have been found previously to not directly correlate with HRQL assessments except for global tilt or lumbar lordosis index, which did correlate with the Oswestry Disability Index (ODI) [29]. In general, sagittal deformity has been shown to correlate more with clinical symptoms and worse HRQL scores than coronal deformity [30, 31].

A variety of validated tools exist for assessing HRQL in ASD including the Scoliosis Research Society 22-question Questionnaire (SRS-22), the Numeric Rating Scale (NRS) Back/Leg Pain, and the Oswestry Disability Index (ODI) [29, 32]. Assessing frailty via the ASD-frailty index provides another benchmark for both baseline functional status, surgical candidacy, and surgical risk stratification [32]. A history of relevant comorbidities should also be taken including a history of osteoporosis and osteopenia; cardiac, diabetes, and pulmonary history which may affect surgical candidacy; and a history of prior surgery [21, 33, 34].

The exam of an ASD patient should include assessing for deformity in supine, sitting, and standing positions, making note of any changes in alignment during transitions between these positions. Sagittal balance is assessed by facing the side of the patient and noting overall alignment while assessing for maintenance of normal cervical lordosis, thoracic kyphosis, and lumbar lordosis. Truncal shift or coronal imbalance should be assessed with patients standing upright with knees in full extension. Note should be made of any curvature of the spine, asymmetry of the waist or hips, or asymmetric prominence of the scapulae. Patients can also be examined for scoliosis while bending forward at the hip to 90° in what is known as the Adams Forward Bend Test (Fig. 4.2) to observe any asymmetric prominences of the back [35].

A variety of compensatory mechanisms may be identified on physical exam which reveal sagittal spinal imbalance. Positive sagittal imbalance is evident with a forward-pitched posture and possible knee flexion in an attempt to keep the head over the pelvis [36]. As a result, patients with long-standing positive sagittal imbalance may have hip flexion contractures. In patients with myopathy or neuromuscular disorders, gait testing may reveal compensatory postures such as camptocormia (involuntary flexion of the thoracic and lumbar spine when standing or sitting that disappears when supine). Neurological symptoms and signs should be always assessed including sensation, weakness, claudication, myelopathy, reflexes, tone, gait, clonus, and bowel/bladder dysfunction [37]. It is also important to assess the upper extremities for signs and symptoms to suggest concomitant cervical disease which may affect treatment planning [37].

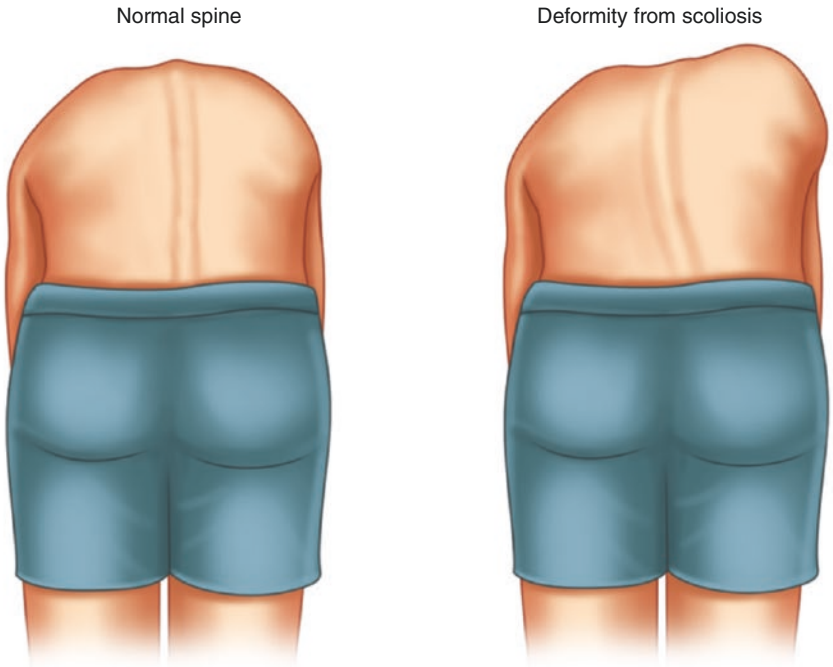

Fig. 4.2 Depiction of the Adams Forward Bend Test demonstrating a positive finding of scoliotic deformity

A thorough radiographic evaluation is important in ASD to evaluate the nature and degree of deformity. An initial imaging approach includes radiographs with posteroanterior (PA) and lateral standing 36-inch views to properly assess the coronal and sagittal planes, respectively. Patients with pelvic obliquity may also benefit from radiographic studies done with a shoe lift or standing blocks [35]. The Scoliosis Research Society has developed a radiographic classification system for ASD called the Schwab Adult Spinal Deformity Classification [38]. It previously correlated HRQLs with radiographic parameters [38] and was recently updated to include pelvic tilt parameters [39]. The addition of these parameters is important as sagittal vertical axis (SVA) and pelvic tilt (PT) are correlated with HRQL measures [40]. Additionally, a positive sagittal balance is linearly correlated with worsening disability and symptoms [41].

The Schwab classification system can be broken down into curve type which describes the coronal deformity and modifiers which describe the sagittal component [39]. The curve type is determined by the maximal Cobb angle in the coronal plane. There are four curve types: *T*, a thoracic major curve greater than 30°; *L*, a thoracolumbar (lower than T10) or lumbar major curve greater than 30°; *D*, both a thoracic and thoracolumbar/lumbar curve greater than 30°; and *N*, no major coronal deformity. There are three sagittal modifiers: pelvic incidence minus lumbar lordosis (PI-LL), global alignment, and pelvic tilt. The PI is the angle between the

perpendicular line to the sacral end plate midpoint and the line between the sacral end plate and the midpoint of the bilateral femoral heads. The LL is measured as the Cobb angle between the L1 and S1 superior end plates. The difference between these angles is the PI-LL and is classified as 0 (<10°), + (10–20°), or + + (>20°). The pelvic tilt refers to the degree of pelvic retroversion which is a measure of compensation for sagittal malalignment. Pelvic tilt relates to compensation and has been correlated with the degree of correction required [42]. PT is classified as 0 (<20°), + (20–30°), and + + (>30°). The global alignment modifier refers to the sagittal vertical axis (SVA) which is the distance between the sagittal C7 plumb line and the posterior, superior sacrum. SVA is classified as 0 (<40 mm), + (40–95 mm), and + + (>95 mm) [39].

Advanced imaging options should also be considered, particularly in patients with abnormal neurologic findings on exam or numbness, weakness, bowel or bladder issues, or problems with balance or coordination in their presenting history. This typically entails magnetic resonance imaging (MRI) without contrast of the suspected symptomatic portion of the spine. MRI provides excellent soft tissue resolution to better understand the neural elements of a patient. Other imaging modalities may include CT myelography (typically used in patients who cannot have an MRI to evaluate for spinal stenosis), CT scans of the spine to evaluate bony anatomy if surgery is being planned, bending views on plain film radiographs to assess range of spinal motion, and rarely MR angiography (MRA) to evaluate cervical vasculature as part of surgical planning [35]. These advanced imaging modalities serve as maps to help guide diagnosis and treatment options and for pain management procedures and surgical planning, if such treatments are indicated and if the patient is interested and medically suitable for such intervention.

Non-operative Management

The goal of treatment of adult spinal deformity is to maintain or improve pain and functional status. Few clear guidelines exist regarding the prioritization and selection of non-operative treatment modalities [4, 43]. Several studies, however, have identified predictors of patients who might benefit from non-operative management. Patients with lower baseline SRS scores and lower baseline disability were more likely to have clinical improvement with non-operative management [44, 45]. Various radiographic parameters have also been shown to be predictive of benefit with non-operative management including smaller thoracolumbar Cobb angle, sacral slope, and lumbar lordosis [44]. However, radiographic predictors vary across studies and more research on this topic is needed [45].

There are many non-operative strategies including physical therapy, pain management, and psychological therapy. Evidence regarding optimal non-operative treatment strategy is often lacking, with other such non-operative interventions including acupuncture, yoga, chiropractic treatments, massages, ice/heat treatments, and other exercise and activity modification regimens. One of the challenges

for implementing physical therapy is the lack of clear and reproducible protocols even in small studies which showed possible improvement [46]. Therapy consisting of core exercise, aquatic training, and strength training can help strengthen the core and paraspinal muscles to support the spine [4, 44]. While bracing is the standard of care for non-operative cases of adolescent scoliosis [47], bracing has only been shown to provide temporary pain relief in adult scoliosis and can weaken surrounding musculature during this process [48]. Pain management interventions primarily with nerve root blocks and epidural injections have shown some relief in radicular pain though the overall data to support benefit is minimal [49, 50]. Ultimately, patient satisfaction is an important factor in deciding to continue with non-operative management alone as the greatest benefit is seen in patients with high current satisfaction [51].

Indications for Surgery, Operative Management, Postoperative Management

Selecting patients for surgery involves consideration of the patient's degree of pain and disability, radiographic parameters, age, and comorbidities to estimate surgical risk versus benefit (Table 4.1). Encouragingly, well-indicated patients often derive benefit from surgical management. In a combined randomized and observational cohort study of adult lumbar scoliosis, there was a high crossover of 64% from the non-operative to the operative group, and the operative group had significantly improved SRS-22 and ODI scores at 2-year follow-up [51]. This discussion can be initiated in the primary care setting and made through a multidisciplinary discussion with the spinal surgeon especially when patients have complex medical comorbidities.

Several risk assessment and planning tools have been developed. The Adult Deformity Surgery Complexity Index (ADSCI) was developed to estimate risk based on anticipated surgical complexity [52]. Scheer and colleagues developed a preoperative predictive model to evaluate the likelihood that patients will reach a minimal clinical improvement difference (MCID) in their ODI score. Predictors of improvement with surgery included the radiographic parameters PI-LL, SVA, and the T1 spinopelvic inclination, the HRQL scores of SRS activity and NRS back pain, and female sex [53]. Additionally, higher grades in the SRS-Schwab radiographic classification previously described correlate with poorer HRQL measures and the need for surgical correction [54].

Radiographic parameters can also be used to identify target deformities for surgical correction. For example, the T1 pelvic angle, a measure of sagittal deformity, corresponds to HRQL measures [55]. Additionally, pelvic incidence-lumbar lordosis mismatch and nonnormative sagittal vertical axis and pelvic tilt measurements also correlate with worse HRQL measures [56].

Table 4.1 Summary of adult spinal deformity with a synopsis of presentation, diagnostic testing, and suggested management strategies

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Adult spinal deformity (scoliosis, kyphosis)	Focal pain within the thoracic or lumbar spine Visible deformity with standing or range of motion of the spine	Plain film radiographs CT MRI	NSAIDS PT Bracing Trigger point or other spinal injections to help with axial or myofascial pain and allow maximal participation in therapy and paraspinal stabilization	Instrumented spinal fusion, usually with osteotomy for correction of deformity Clinical severe deformity with neurologic or other physiologic compromise Failure of other non-operative modalities with clinically significant impact on quality of life and activities of daily living

CT computed tomography scan, *MRI* magnetic resonance imaging, *PT* physical therapy, *NSAIDs* nonsteroidal anti-inflammatory drugs

Multiple comorbidities, including mental health disorders, should be considered to determine surgical candidacy. Patients undergoing surgical ASD correction with mental health disorders such as depression, sleep, and anxiety disorders had increased rates of surgical complications and revision compared to controls [25, 57]. Appropriate mental healthcare should thus be considered in surgical candidates. While older patients are generally poorer surgical candidates [58], age can be accounted for in defining appropriate spinopelvic alignment goals which are less aggressive for older patients [59].

Despite higher complication rates, older patients also experience greater improvements in HRQL measures and frailty indices [60, 61]. Frailty can be assessed using the ASD-frailty index (ASD-FI) and is important to assess given the higher risk of complications in patients with higher frailty scores [62]. However, a high frailty score does not preclude surgical intervention, as patients with a higher ASD-FI do report higher improved outcomes by the SRS-22 and NRS Back/Leg Pain postoperatively compared to patients with lower ASD-FI scores [32]. Higher body mass index (BMI), smoking, and osteoporosis are also associated with increased rates of complication and worse surgical and clinical outcomes [63, 64]. Finally, the presence of neurological symptoms and signs such as radiculopathy, weakness, and claudication are correlated with indication for surgery [37]. When considering these comorbidities, particularly modifiable risk factors such as BMI, smoking, and osteoporosis, the role of the primary care physician in helping to counsel patients and treat conditions that may predispose to surgical complication is of critical importance.

There are many surgical approaches to managing ASD [65–67]. A combined anterior/posterior fusion is the most common approach for severe ASD [68]. A

posterior-only approach is also possible and has been shown to reduce the complications from an anterior thoracoabdominal approach while achieving similar results [69]. Specific surgical classifications have been described. A six-level operative treatment plan was designed by Lenke and Silva to determine optimal operative approach based on patient symptoms and radiographs [70]. The levels are decompression alone, decompression with limited posterior spinal fusion, decompression and lumbar curve instrumented fusion, decompression with anterior and posterior spinal fusion, thoracic fusion, and osteotomies based on curvature deformity [70]. There is also a classification system for grades of osteotomy to treat ASD developed by Schwab et al. [71]. Finally, minimally invasive surgery (MIS) is emerging as an option for ASD correction, and the minimally invasive spinal deformity surgery (MISDEF) algorithm can be used to guide decision-making around MIS management [72, 73].

Biologic agents have gained traction for their use in potentially augmenting spine fusion. rhBMP-2 is an osteoinductive growth factor with approval for use in anterior lumbar interbody fusions [3]. The use of rhBMP-2 in long spine fusion and scoliosis surgery has been shown to have similar outcomes to structural bone graft harvested from the iliac crest, with the advantage of sparing patients the morbidity of graft harvesting [74]. Important to consider however are the complications of bone morphogenetic protein use, which include heterotopic bone formation, seroma, or postoperative radiculitis, and rare but potentially increased risk of cancer [3].

Various radiographic and surgical factors can be used to anticipate complications. Radiographic modifiers, specifically degree of lordosis and sagittal balance, predicted higher rates of surgical complication in 2-year follow-up [75]. Surgical approaches that included fusions extending to the sacrum compared to fusions above the sacrum and circumferential compared to anterior or posterior approaches had higher rates of complications [75]. Common postoperative complications to monitor for include infection, wound complications, neurologic deficits, instrumentation malposition or failure including rod breakage, development of deformity or proximal junctional kyphosis above the instrumented levels, fractures, and pseudoarthrosis with lack of bone healing [3].

Conclusions

Adult spinal deformity is rising in prevalence alongside an aging population. It can present in the coronal or sagittal plane and have devastating effects on patient quality of life. As such, increasing collaboration between orthopedic spinal surgeons and primary care physicians can help in optimally managing this growing patient population. A thorough history and examination, including assessment of HRQL measures, is important to assess the degree of disability and overall morbidity caused by a patient's ASD and can help inform patient discussions and guide the decision to pursue non-operative or operative management strategies.

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Chapter 5
Cervical Radiculopathy and Myelopathy

Kevin M. Hwang, Amandeep Bhalla, and James D. Kang

Cervical Radiculopathy

Definition and Epidemiology

Cervical radiculopathy represents dysfunction of one or more cervical nerve roots that typically presents with radiating pain in the upper extremity and varying degrees of sensory loss, motor weakness, and reflex changes. Population-based studies have shown an annual incidence of 107/100,000 men and 64/100,000 women, with a peak incidence in the sixth decade of life. About 15% of patients report an antecedent episode of physical exertion or trauma that precedes symptom onset. Identified risk factors for cervical radiculopathy include white race, smoking history, and prior lumbar radiculopathy. The majority of the cases stem from compression of nerve roots in the lower cervical spine, most commonly at C6–7, likely due to greater segmental mobility and smaller neuroforamina in this region.

Clinical Presentation

Cervical radiculopathy is usually the result of neuroforaminal stenosis due to a herniated disc, overgrowth of the uncovertebral joints anteriorly, or facet joint hypertrophy posteriorly. This stenosis can manifest with pain, sensory disturbances,

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diminished reflexes, and muscle weakness that correspond to the affected nerve root. A general understanding of the myotomes and dermatomes of the cervical spine aids in diagnosis (Fig. 5.1). However, radicular symptoms do not always follow a predictable pattern of the affected root, and the type and intensity of symptoms vary widely. Some patients complain of less specific upper trapezial and interscapular pain, or discomfort about the shoulder girdle. There may also be more than a single nerve root involved, or anatomic variations in innervation, such that symptoms seem to cross over dermatomes and/or myotomes. Radiculopathy may also be present in the bilateral upper extremities and can exist concurrently in patients with myelopathy or peripheral nerve compression syndromes.

The physical exam performed in a systematic, root-specific manner, can elucidate sensory disturbances, motor deficits, and diminished reflexes. Pain and sensory changes in the affected root distribution are more commonly seen, while motor weakness and reflex changes are encountered less often. The examiner can sometimes reproduce radicular pain by performing the Spurling test, where the patient extends the neck and bends it toward the affected side. This maneuver narrows the neuroforamina and causes root impingement. As a corollary, patients often endorse relief of radicular symptoms when they sleep with their arm overhead, which enlarges the neuroforamina and decreases root compression. One must examine the shoulder with various maneuvers (refer to shoulder chapter) to rule out intrinsic shoulder pathology which can mimic or coexist with cervical radiculopathy. Shoulder pain that seems to localize anteriorly is generally intrinsic to that joint, but shoulder pain that localizes to the posterior scapular region or radiates past the midarm to the elbow or hand is typically referred from the cervical spine.

For any patient presenting with cervical radiculopathy, care must be taken to screen for concurrent myelopathy. Part of the history should include inquiry about

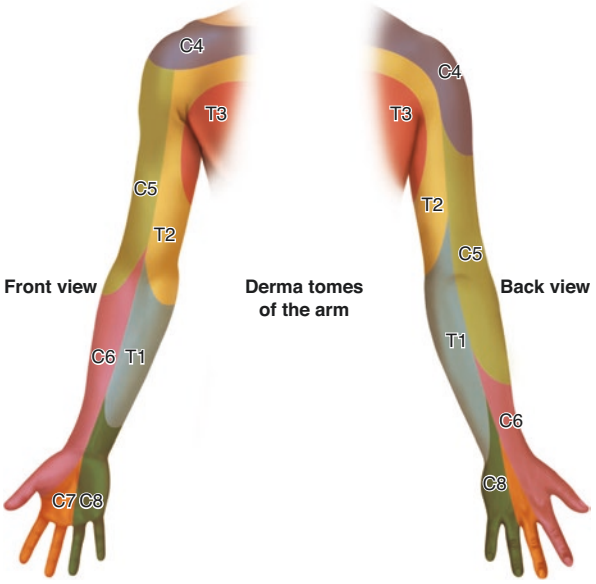

Fig. 5.1 Map of the most common anatomic distribution of cervical dermatomes and myotomes in the arm. The C5 through C8 levels are most frequently affected by radiculopathy

changes in gait, manual dexterity while performing fine motor tasks, and bowel and bladder incontinence. Screening for myelopathy should also include an examination for the presence of long tract signs, including tests for positive Hoffman or Babinski signs, as well as clonus or an inverted brachioradialis reflex. A more detailed discussion of the evaluation for myelopathy will be discussed in a later section.

Differential Diagnosis and Diagnostic Testing

The diagnosis of cervical radiculopathy is typically made using clinical history and physical exam alone, without the need for imaging or special tests. The differential diagnosis for radicular symptomatology includes peripheral nerve entrapment, brachial plexus injury, and tendonopathies (shoulder and elbow) of the upper extremity. Less commonly, infectious (herpetic zoster) or post-infectious (Parsonage-Turner) etiologies may produce similar symptoms. Some patients will present with a neck-shoulder syndrome where pathology coexists at both anatomic locations. Hence they will have both radicular features as well as intrinsic shoulder pain (rotator cuff pathology) with certain maneuvers and therefore can often be confusing to the clinician. Patients should also be screened for "red flags," such as unexplained weight loss, fever, intravenous drug abuse, and history of previous cancer, which may suggest the possibility of infection or tumor.

Cervical radiculopathy can exist concurrently with peripheral neuropathy, a so-called "double crush syndrome", where there is pathologic compression at more than one location along the course of a peripheral nerve. This may present a diagnostic challenge. For example, a patient with carpal tunnel syndrome may also have a C6 radiculopathy, which may result in an overlapping distribution of numbness and sensory deficits. In patients with carpal tunnel syndrome, it is helpful to inquire if symptoms radiate from the neck and to perform a provocative Spurling test to assess for radiculopathy. Proximal muscles supplied by the C6 nerve, such as the biceps or common wrist extensors, will be spared by carpal tunnel syndrome but may be affected in radiculopathy. By screening patients in this manner, fewer cases of double crush syndrome would go undiagnosed, and patients would benefit from timely treatment of both the cervical radiculopathy and peripheral nerve compression. Interestingly, the diagnosis of double crush syndrome is often made when patients are dissatisfied with the outcomes of a carpal tunnel release, presumably because of coexisting C6 radiculopathy.

Because the diagnosis is reliably made clinically and the natural history is usually self-limiting, it is reasonable to limit the use of diagnostic imaging until patients have been symptomatic for 4–6 weeks. The imaging helps to confirm the diagnosis and to facilitate treatment. Of course, if there is a concern for infection, tumor, or progressive motor deficits, diagnostic imaging should be obtained expeditiously. Plain anterior-posterior and lateral cervical radiographs are of limited diagnostic value, but they do demonstrate overall cervical alignment, and the extent of degeneration as evidenced by intervertebral disc height loss and osteophyte formation.

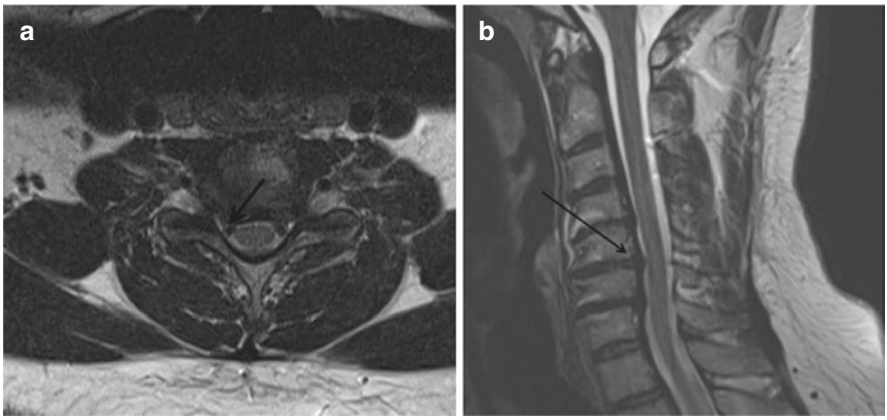

Fig. 5.2 Axial (**a**) and sagittal (**b**) magnetic resonance images (MRI) of a disc process at C5–C6 causing a right-sided radiculopathy (black arrows)

Magnetic resonance imaging (MRI) is the study of choice for cervical radiculopathy. MRI provides detail of the neural elements and surrounding soft tissue structures (Fig. 5.2). When an MRI is contraindicated, a computed tomography (CT)-myelogram can be useful to show focal areas of compression.

Results from MRI should be interpreted cautiously given the high sensitivity for detecting abnormalities. It is well established that asymptomatic patients have a high incidence of positive MRI findings, so areas of nerve root compression must be correlated with clinical findings. From a surgeon's perspective, it is ideal when there is correlation between anatomic abnormalities on neuroradiographic studies, patients' symptoms, and physical exam findings. In cases where imaging studies are equivocal, selective nerve root injections at the suspected level of involvement can be both diagnostic and therapeutic. Furthermore, electromyography studies and nerve conduction tests can be used adjunctively when patient's history and physical exam are inadequate to differentiate cervical radiculopathy from other neurologic causes of pain. For example, the presence of abnormal insertional activity in the paraspinal musculature can differentiate cervical radiculopathy from brachial plexopathy. These studies should be interpreted in the context of the clinical exam and radiographic findings and can effectively rule out other sites of compression. When there is concomitant shoulder pain that coexists and the clinical exam would suggest an intrinsic shoulder problem, an MRI of the shoulder may also be considered to clarify the diagnosis.

Nonoperative Management

Nonsurgical management is the mainstay of treatment for cervical radiculopathy. There is a lack of well-established nonsurgical treatment guidelines based on high-quality scientific evidence, and much of conservative treatment is centered

on level 4 and 5 evidence. In the setting of herniated disc material, chemical inflammatory mediators significantly contribute to radicular pain. These properties make oral anti-inflammatory medications an efficacious first-line treatment. Narcotics should rarely be prescribed for routine analgesia but can be considered on occasion for breakthrough pain or in patients who cannot tolerate NSAIDs. Some patients benefit from a multimodal analgesic regimen, which may include muscle relaxants, antidepressants, and gabapentin in conjunction with oral NSAIDs. For symptoms that are unresponsive to anti-inflammatories, in patients without medical contraindications, an oral tapered steroid regimen may also be prescribed.

Postural education, improved ergonomics, and lifestyle modification help to improve functional capacity. Patients are encouraged to mobilize early and to participate in physical therapy once pain has subsided. There is no proven role for immobilization or bed rest. Nonimpact aerobic exercises such as stationary biking can help relieve symptoms and maintain fitness. Some patients also derive temporary relief from intermittent home traction, which temporarily enlarges the neuroforamina and decompresses the exiting roots. Traction is not advised in patients with myelopathy, since lengthening the spinal column across an area of cord compression can be dangerous.

For persistent symptoms that have not been adequately relieved by oral analgesics, and functional rehabilitation, corticosteroid injections can be considered. Epidural corticosteroid injections offer a powerful, locally concentrated anti-inflammatory effect. Selective nerve root injections target the perineural space surrounding the affected root and avoid the spinal canal. Although relatively safe, epidural injections are invasive and come with risks, which include but are not limited to dural puncture, epidural hematoma, and epidural abscess. Conservative management should be continued for at least 6–8 weeks since the natural history of most cervical radiculopathy is for spontaneous pain resolution within 75–90% of patients. Patients may continue to see symptomatic improvement over more than 6 months.

Indications for Surgery

While conservative management is the predominant treatment for this typically self-limiting condition, there are cases where surgery is warranted and largely beneficial. Ideal surgical candidates have neuroradiographic evidence of root impingement, with corresponding root dysfunction, and persistence of symptoms despite several months of conservative care. Functionally significant motor deficits and debilitating radicular symptoms not responsive to conservative measures are indications for earlier surgical intervention. Subtle motor weakness which can be seen in early acute radiculopathy is often due to inflammation and pain and should spontaneously resolve with conservative management. However, if the weakness persists or progresses and leads to early muscle atrophy, the patient should be referred to a spine specialist for closer surveillance.

Operative Management and Expected Outcomes

Anteriorly based pathologies such as soft and hard disc herniations are the most common causes of cervical radiculopathy. The majority of patients are treated with an anterior cervical discectomy and fusion (ACDF). The anterior approach allows excellent exposure of the cervical spine and involves removal of the offending disc. It is muscle sparing and involves minimal blood loss. Once the discectomy is performed, the posterior longitudinal ligament can be resected, offering directly visualization of the dura and exiting nerve roots. Fashioned iliac crest autograft, allograft, or an interbody device is placed in the decompressed interspace to impart stability and to promote bony fusion across the motion segments. The graft restores intervertebral height and indirectly expands the neuroforaminal space. Advantages of the anterior approach include access to both central and lateral disc herniations, low infection and wound complication rates, and relatively minimal postoperative pain. The major disadvantages of ACDF are the risks for nonunion at the fusion site and persistent speech and swallowing difficulties due to retraction of the esophagus and laryngeal nerves.

A subset of cervical radiculopathy patients are eligible for cervical disc arthroplasty instead of an ACDF. The approach and manner of decompression are essentially similar to that for a fusion, except an artificial disc is placed in the interspace. The theoretical advantage of cervical disc arthroplasty (CDA) is preservation of motion at the surgical level, potentially mitigating the risk of adjacent segment disease and subsequent need for reoperation. It also eliminates the risk for pseudarthrosis. ACDF and CDA have been shown to have essentially equivalent patient-reported outcomes in medium-term clinical trials (2–10 years); however, debate persists regarding CDA's effectiveness in decreasing adjacent segment disease and need for reoperation. Cervical adjacent segment disease is believed to occur at an annual incidence of about 3%, regardless of the surgery performed, and it is unclear if this is consequence of fusion or due to the natural history of disc degeneration. The long-term mechanical durability and clinical outcomes data for cervical disc arthroplasty have also not yet been realized as long-term prospective trials are only starting to report 10-year data.

A posterior approach involving a laminoforaminotomy can be used to address anterolateral disc herniations or foraminal stenosis. The posterior approach to the spine involves dissection through the muscular raphe in the midline of the neck. Direct access to the compressed nerve root is achieved with removal of bone from the overlying facet and lamina, without destabilizing the motion segment. Proponents of the posterior laminoforaminotomy value the direct visualization of the nerve root, and avoidance of fusion and its attendant complications. Drawbacks of this procedure include inability to restore foraminal height with an interbody graft, as well as risk for recurrence as degenerative changes ensue.

A high rate of clinical success is to be expected for surgical decompression of the cervical nerve roots for cervical radiculopathy, regardless of approach. Patients commonly experience lasting relief of arm pain and improvements in motor and sensory function. Up to 10 years after surgery, patient satisfaction is reported at more than 90%.

Cervical Myelopathy and Myeloradiculopathy

Definition and Epidemiology

Cervical spondylotic myelopathy is the most common cause of spinal cord dysfunction in adults, and its incidence is likely underreported. Cervical spondylotic myelopathy results from age-associated degenerative changes to structures about the spinal cord, including disc degeneration, ligamentous hypertrophy, and osseous changes. These anatomic changes encroach upon the spinal canal and can lead to direct compression of the cord. Congenital spinal stenosis anatomically predisposes the development of cervical myelopathy. Patients with cervical spondylotic myelopathy have a much greater risk for spinal cord injury. Primary care physicians play an important role in the management of cervical myelopathy, as early detection and prompt referral for surgical evaluation can greatly improve patient outcomes.

Clinical Presentation

The pathophysiologic effects of spinal cord compression are thought to be a combination of direct mechanical effects on the neural tissue and related alterations in vascular supply. Presenting symptoms can include gait instability, diminished manual dexterity, motor weakness, sensory loss, incontinence, and permanent functional disability. The spectrum of disease severity and variation in symptomatology are commensurate with the many different manners in which the spinal cord can be functionally compromised by compression. For example, pathology that affects the dorsal column may predominantly manifest with proprioceptive loss in the extremities. The clinical course of cervical spondylotic myelopathy is marked by periods of neurologic stability with stepwise deterioration of neurologic function. Approximately 20–62% of patients will deteriorate neurologically within 3–6 years of diagnosis, and patients with even mild cervical myelopathy may have increasing difficulties with managing activities of daily living as years pass.

A thorough history and physical exam help to illicit subtle cues of spinal cord dysfunction. Patients may endorse subacute changes in their gait, demonstrate instability on exam, and have difficulty with tandem heel-to-toe walking more than a few steps. Patients may also report difficulty performing fine motor tasks, like buttoning a shirt or using chopsticks. The examiner can test hand dexterity with the grip and release test, where patients rapidly open and close their hands while being timed. Patients are normally able to do this about 20 times in 10 seconds. This test of manual dexterity can be used to survey stability of neurologic function over time. Additional evidence of spinal cord dysfunction occurs with extension of the neck causing an electrical shock-like sensation to shoot down the spine, the so-called Lhermitte's sign. This maneuver dynamically decreases the space available for the spinal cord and exacerbates symptoms.

Patients may also exhibit long tract signs, which are indicative of damage to the corticospinal tracts. The Hoffman's reflex, for example, should raise concern for cervical myelopathy when positive. To test this the examiner flicks the distal phalanx of the index or middle finger, and a positive finding is seen with flexion of the distal phalanx of the thumb. Other clinical findings of upper motor neuron dysfunction include an extensor plantar response known as the Babinski sign, where firmly stroking the lateral border of the foot results in extension of the great toe, or the inverted radial reflex, where a strike by a reflex hammer to the brachioradialis tendon elicits not only wrist extension but also finger flexion.

It is important to note that the absence of upper motor neuron signs (i.e., hyperreflexia, Hoffman sign, inverted brachioradialis reflex, clonus, and Babinski sign) does not preclude the diagnosis of myelopathy. The presence of long tract signs is not highly sensitive, and patients with unequivocal cervical myelopathy may in fact manifest no such signs. Up to one-fifth of patients who otherwise are myelopathic on the basis of history, correlative advanced imaging, and subjective improvement after decompression do not have long tract signs on presentation. Certain coexisting conditions can diminish the reliability of long tract signs in detecting spinal cord dysfunction. For example, in patients with myeloradiculopathy, concurrent radiculopathy can diminish the transmission of long tract signs. Diabetes, through its effect on peripheral nerves, is also thought to have a dampening effect on the transmission of neurologic reflexes. A higher index of suspicion for myelopathy should be had for patients with diabetic peripheral neuropathy. Even in the absence of long tract signs, concerning clinical symptoms combined with correlative imaging studies should guide treatment decisions.

Primary care physicians should remain vigilant for cervical myelopathy even in patients presenting with lumbar spine symptoms, such as neurogenic claudication and radiculopathy. A red flag symptom such as gait instability should immediately stoke concern for concomitant cervical myelopathy. Interestingly, it is not an uncommon presenting clinical scenario for patients with primarily low back symptomatology to have an underlying cervical disease. In fact, the coexistence of lumbar and cervical spinal stenosis has been reported in up to 15% of patients. A focused lower extremity exam may not illicit positive long tract signs, since concomitant lumbar spinal stenosis may dampen CNS signal transmission. It is therefore appropriate to screen patients presenting with lumbar spinal stenosis for concomitant cervical myelopathy by thoroughly examining both the upper and lower extremities.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis for cervical myelopathy includes other central nervous system disorders as well as neuropathy and the long-term effects of alcohol abuse or certain vitamin deficiencies. When cervical myelopathy is suspected, upright plain radiographs are used to assess for alignment, segmental stability, and degree of degeneration. The condition of the spinal cord and influence of surrounding

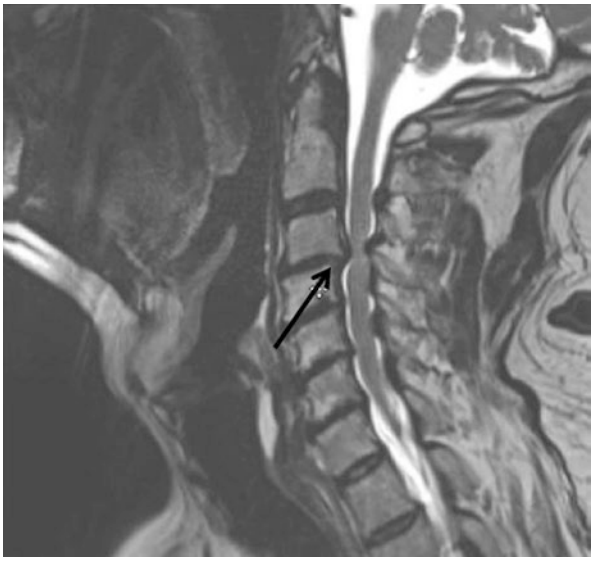

Fig. 5.3 Sagittal MR image demonstrating severe spinal stenosis and myelomalacia (black arrow) at the level of C3–4 in a patient with cervical spondylotic myelopathy

structures is evaluated with an MRI, or CT-myelogram in cases where MRI is contraindicated (Fig. 5.3). The patient should be referred to a spine surgeon to discuss treatment options and establish care for routine surveillance.

Nonoperative Management

Although surgical decompression of cervical myelopathy is the only manner in which the natural history of the disease can be altered, not all patients desire to undergo surgery. Many patients function well with mild forms of myelopathy and remain neurologically stable for years. However, there is always a risk for functional decline, which patients should reasonably be made aware of. A treatment plan is formulated between the care team and the patient after discussing the risks and benefits of surgery versus expectant management. Medical comorbidities such as diabetes, significant cardiac or renal disease, and advanced age may sway the balance of surgical risks and benefits toward nonoperative care.

There is no role for injections in patients with cervical spondylotic myelopathy. Physical therapy may improve the functional capacity of the patient, but will not alter the natural history of the disease. Anti-inflammatory medications and neuromodulators may help to alleviate radicular symptoms when simultaneously present. Rigid cervical orthoses have not been shown to be beneficial. Nonoperative interventions such as cervical traction and manipulation are not supported by high-quality evidence and associated with case reports of catastrophic complications. In general, when patients present with myelopathy, it is advised that the patient be referred to a spine specialist for consideration of surgery.

Indications for Surgery

The goal of surgery is to decompress the spinal cord and arrest further neurological decline. The thought process surrounding decompression is that the patient is far less likely to worsen in the absence of ongoing cord compression, and this is overwhelmingly the case. Indeed, some patients experience improvement of neurologic symptoms postoperatively. Others may experience further deterioration, even after a successful decompression, but these patients are in the minority. The most common etiology for neurological decline after an adequate decompression is the development of a new, adjacent focus of cord compression.

Patients may elect to defer surgery when there is mild evidence of spinal cord dysfunction, though this is not without some risk. It is difficult to predict which patients will have stable disease without decompression and which are at risk for further progression. These patients can be screened at regular intervals for evidence of neurological decline. Such deterioration may be subtle, and it is advantageous for patients to be followed by the same physician over time. Evidence of decline should be indicative of the capacity for progression and once again prompt a discussion regarding surgery. Patients with more pronounced or progressive clinical findings, and/or evidence of severe cord compression, should consider surgical intervention as soon as is reasonably possible. These patients are likely to be at greater risk for further functional decline, with the possibility of a devastating spinal cord injury in the event of a traumatic event that stresses a spinal cord that is already compromised.

Operative Management and Expected Outcomes

Anterior, posterior, and combined surgical approaches may be utilized, depending on a variety of factors, including anatomic location of the compression, alignment of the spine, and consideration of distinct complications associated with each approach. Decompression is the chief goal of surgery, and selection of the approach is performed with this priority in mind. In the lordotic cervical spine, in the setting of ventral compression, a posterior laminectomy can effectively allow the cord to freely float away dorsally. The most commonly used posterior surgical technique is a laminectomy and instrumented fusion. This involves removal of the posterior lamina and segmental instrumented fusion. Advantages of this include the potential for wide decompression, stabilization to prevent subsequent post-laminectomy kyphosis, and fusion to improve pain related to spondylosis. Laminoplasty, an alternative technique, expands the diameter of the spinal canal by expanding the lamina only on one side. Laminoplasty directly decompresses posterior impinging structures and indirectly decompresses the ventral cord. Advantages of this procedure include maintained segmental distribution of axial and rotational forces and preservation of motion. This is a reasonable option in patients with poor biologic potential for bony fusion.

An anterior approach can directly address anterior pathology, such as a central disc herniation. This approach involves discectomy or corpectomy (Fig. 5.4),

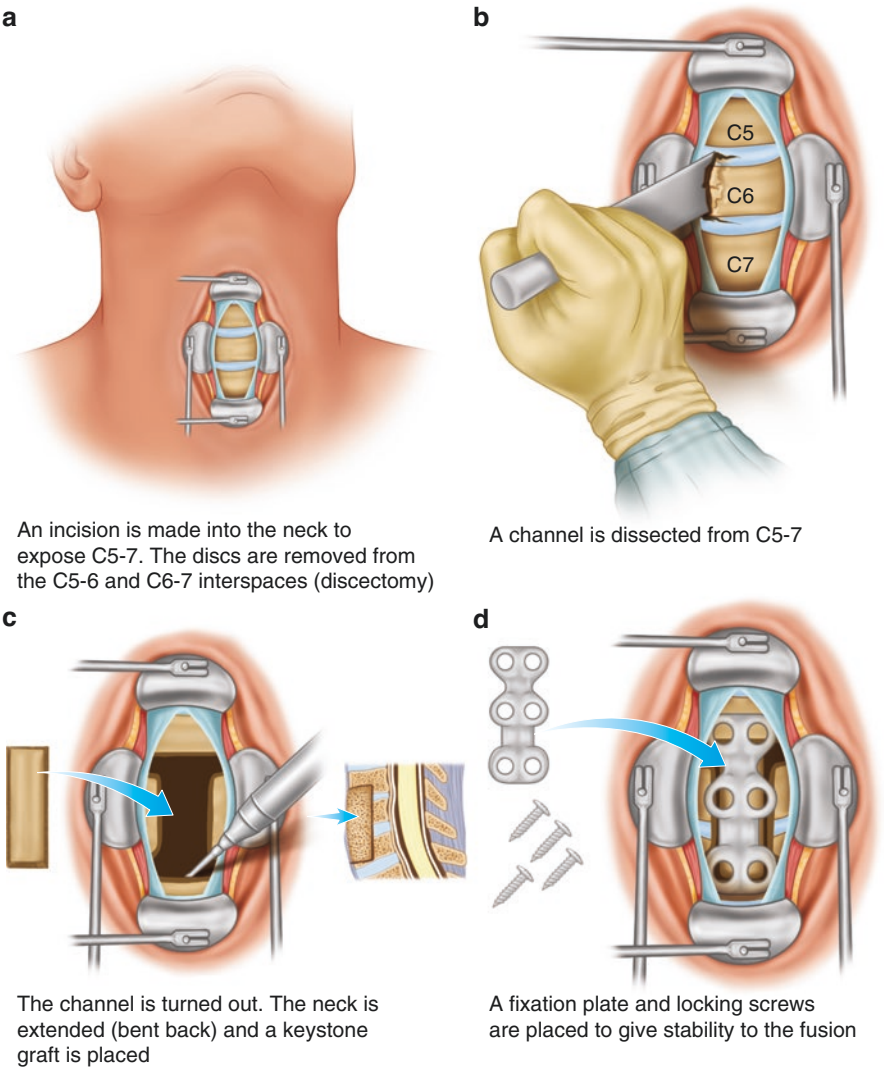

Fig. 5.4 Depiction of an anterior cervical approach with corpectomy and reconstruction using a strut graft, anterior plate, and instrumentation

depending in part on the location and extent of anterior pathology. It is particularly useful when ventral compression exists in the setting of neutral or kyphotic cervical spine alignment, precluding the possibility of indirect decompression with a posterior procedure. Both anterior and posterior approaches are effective in improving patient's quality of life and have comparable outcomes. Posterior approaches have a higher rate of complications, particularly infection or wound breakdown. Ultimately, the success of the surgery is most closely linked to the adequacy of the

Synopsis of presentation, diagnostic testing, and treatment options for patients with cervical radiculopathy or myelopathy

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Cervical radiculopathy	Radiating pain, with possible sensory deficits, and motor weakness in the distribution of the affected nerve root	MRI or CT-myelogram	Physical therapy Anti-inflammatory medications	Radicular symptoms refractory to conservative management Significant motor weakness	Anterior cervical discectomy and fusion Cervical disc arthroplasty Posterior laminoforaminotomy
Cervical myelopathy	Gait instability, diminished fine motor dexterity, sensory deficits and motor weakness, hyperreflexia, bowel and/or bladder incontinence	MRI or CT-myelogram	Generally not advocated Counseling about risks of disease progression Routine surveillance of neurologic function	Myelopathy in the setting of static or dynamic spinal cord compression	Anterior cervical discectomy/ corpectomy and fusion Posterior cervical decompression and instrumented fusion Posterior cervical laminoplasty

MRI magnetic resonance imaging, *CT* computed tomography

spinal cord decompression. Surgical intervention has a better prognosis if patients with myelopathy are treated at an earlier clinical stage before severe spasticity or loss of ambulatory function occurs. Once the spinal cord undergoes irreversible chronic changes, the surgical goal is to prevent further neurologic deterioration since full recovery is often a challenge. Classically, patients were told to expect surgical decompression would arrest the progressive decline in their neurologic function. Recent studies have shown that patients, especially those with a shorter duration of neurologic deficit, can expect to regain a portion of their lost function after surgery.

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Chapter 6
Lumbar Disc Herniation and Radiculopathy

Christopher M. Bono and Andrew K. Simpson

Abbreviations

CSF	Cerebrospinal fluid
EMG	Electromyograph
MMPI	Minnesota Multiphasic Personality Inventory

Definition and Epidemiology

Lumbar disc herniations with radiculopathy are very common; most believe them to be a point along the degenerative cascade, which spans from mild internal disorganization of the disc to more advanced spondylosis. Nonetheless, disc herniations can also occur in very young individuals, indicating that there may be more acute or subacute disc injuries that culminate into herniations. Considering the body of literature devoted to disc herniations in the pediatric population, it would be hard to imagine that degenerative changes have occurred in such young patients. Whether these changes occur through degenerative mechanisms and microtrauma over time or more acutely, the situational and anatomic components needed for herniation are

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a loss of structural integrity of the outer layer, termed the annulus fibrosus (or annulus for short), and an abrupt increase in intradiscal pressure that exceeds the threshold at which the inner portion of the disc (nucleus pulposus) is contained. This can be the result of a high-impact mechanism, such as a sports injury, or something as mundane as a sneeze. In many, if not most, cases, there is no clear identifiable point of "injury," with symptoms developing without clear incident.

The prevalence of symptomatic herniated discs has been estimated to be about 1–3%, and they are more common in persons aged 30–50 years old. The age prevalence has much to do with the natural history of disc degeneration. Specifically, the loss of disc hydration and disc material over time with aging means that there is less disc material available to herniate in the later stages of life. Additionally, there are some anatomic location differences that have been appreciated, with younger patients more frequently presenting with lower lumbar disc herniations (L4–L5, L5–S1), while upper lumbar disc herniations (L2–L3, L3–L4) are more common in older individuals. This phenomenon is likely resultant from the fact that the degenerative cascade proceeds much earlier in the lower lumbar segments, as demonstrated in epidemiologic studies. There is also a gender predilection for males, likely coincident with a disproportionately higher prevalence of more physically demanding jobs in men.

Clinical Presentation

The hallmark of clinical presentation of a lumbar disc herniation is radicular lower extremity pain. In its most classic and straightforward form, a patient's pain would closely follow the distribution of a single-lower lumbar nerve root. For example, a patient with an L5–S1 right-sided paracentral (i.e., not immediately in the midline, but slightly off to one side) herniation should complain of pain in the S1 distribution, which can extend from the buttock, down the posterior calf, and into the lateral plantar surface of the foot. In reality, patient's complaints vary considerably and do not always follow such discrete dermatomal patterns.

Patients can also complain of varying degrees of numbness and weakness, while neither is requisite for the clinical diagnosis. Again, these complaints ideally follow a specific nerve root distribution. Using the example from above, the patient might complain of push-off weakness on the right side with ambulation, a manifestation of diminished strength of plantar flexion, controlled by the S1 nerve root. Similarly, there may be a complaint of numbness along the lateral and plantar surface of the foot on the affected side.

When evaluating a patient with a suspected lumbar disc herniation, it is important to note any events leading up to the current endorsement of symptoms as well as the patient's prior musculoskeletal health status. It is not uncommon for patients to have a history of a short period of prodromal back pain immediately preceding the complaint of leg pain. It is presumed that this period of back pain may reflect the acute injury to the disc and annular injury that precedes the herniation of nuclear

disc material. Leg symptoms are not immediately noted in many cases, even in the presence of large disc herniations with substantial nerve root compression. This phenomenon may be resultant from a temporal delay between the time of annular injury and subsequent herniation of disc material or may be explained by the fact that acute compression of a nerve root may not immediately result in pain. Radicular pain may be a function of enduring compression and the onset of inflammation. It is also important to note if the patient has had a history of recurrent or chronic back pain episodes. It is critical to delineate these prior events from the current episode of radicular pain, as it is the latter that will be the focus of current treatment and will have the highest likelihood resolving.

Patients with a lumbar disc herniation and radiculopathy should be differentiated from those with spinal stenosis and neurogenic claudication, as the natural history of these two processes is quite different. While radiculopathy from a disc herniation is often self-limiting, spinal stenosis from circumferential extrinsic neural compression is likely to progress anatomically and symptomatically over time. Symptomatically, disc herniation patients will have unremitting, radiating lower extremity pain. This is not relieved by sitting and is more often aggravated by it. Lying supine and sleeping can be equally troubling. In the exam room, lumbar disc herniation patients usually prefer to stand and often report that upright activities, like walking, are more comfortable for them. In contrast, patients with lumbar stenosis and neurogenic claudication are most comfortable at rest, either sitting or lying down. They experience the most pain when they ambulate distances, feeling relief by flexing forward or sitting for a short period of time. Patients should be asked about bowel and bladder incontinence and perianal anesthesia. Though exceedingly rare, these complaints can indicate the presence of a cauda equina syndrome, which should be urgently evaluated and treated if indeed present.

The physical examination of an affected patient should consist of a detailed motor and sensory assessment of the lower extremities. The patella tendon (L3/L4) and Achilles (S1) tendon reflexes should also be evaluated. Isolated, unilateral loss of one of the reflexes can be a sign of nerve root compression from a disc herniation. There are some provocative maneuvers that can also be helpful in establishing the clinical diagnosis. With the patient lying supine, a straight leg raise test can be performed by elevating the extremity with the knee extended. The test is considered positive if radicular leg pain is reproduced with 30–70° of hip flexion on the affected side. The same test can be performed on the contralateral side and, if positive, will reproduce pain on the ipsilateral lower extremity. This is a very specific, but not sensitive, physical exam finding for a lower lumbar disc herniation. Straight leg raises may also be performed with the patient seated on an exam table. Upper lumbar disc herniations may be associated with a positive femoral stretch test, which is performed with the patient prone and the examiner slowly raising the lower leg with knee flexion. Radicular pain in the thigh is considered positive.

The patient's gait should be observed during evaluation as well. While gait can be affected simply by pain, a Trendelenburg sign may also be present, noted as the hip sagging on the affected side, a result of hip abductor weakness. A foot drop may

also be noted in certain scenarios. Patients often compensate for this by "high stepping" to accommodate for the lack of ability to dorsiflex the foot.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis of radiating lower extremity pain is wide. It can be related to any process that causes nerve root or cauda equina compression, such as spinal stenosis, tumor, abscess, or epidural lipomatosis (an accumulation of fat inside the spinal canal). Processes such as infection or neoplasm can be associated with so-called red flag signs and constitutional symptoms (e.g., fever, night sweats, weight loss, malaise). Peripheral nerve entrapment can also present similarly. This can occur in the pelvis within the piriformis fossa, about the knee (most commonly near the fibular head where the common peroneal nerve is most vulnerable), or even in the lower leg. Nonneural causes should also be considered, such as vascular insufficiency, which can present with similar radicular-like leg pain. Vascular claudication from peripheral vascular disease is not uncommon and should be considered in patients with vascular history, abnormal or asymmetric pulse examination, or claudicatory leg pain that does not immediately resolve with rest.

In the primary care setting, obtaining plain films of the lumbar spine in a patient with a suspected lumbar disc herniation is generally of low yield. Disc herniation cannot be visualized on plain films. In addition, they are not highly sensitive for ruling out other more concerning pathology, such as tumor or infection. Plain films have a potential role in preoperative planning, as spinal alignment and other morphological variations may influence surgical decision-making. However, these can be deferred until a decision for surgery has been reached.

A non-contrast-enhanced MRI is the imaging modality of choice for the detection of lumbar disc herniation (Fig. 6.1). It provides superior visualization of the soft tissues, ligaments, discs, neural elements, and spinal fluid. Furthermore, it is highly sensitive for the presence of tumor or infections. It must be appreciated, however, that the prevalence of disc herniations is not insubstantial in asymptomatic individuals. One classic study found a 20% prevalence of disc herniations in an asymptomatic population less than 60 years old. The use of contrast should be reserved for those with particular findings on a non-contrast-enhanced MRI, such as the presence of a suspected neoplastic lesion. Some feel that contrast-enhanced studies are necessary in patients who have had previous lumbar surgery; however, this has been contested. A CT scan will not be able to differentiate the neural elements and is of low utility. CT myelography is a reasonable alternative advanced imaging study but should be reserved for patients who have a clear contraindication to MRI. CT provides suboptimal soft tissue differentiation and requires invasive myelogram, which has both logistic and cost implications as well as a higher risk profile. When referring patients for surgical or interventional consideration, it is important to note that these procedures cannot be planned without advanced imaging such as an MRI.

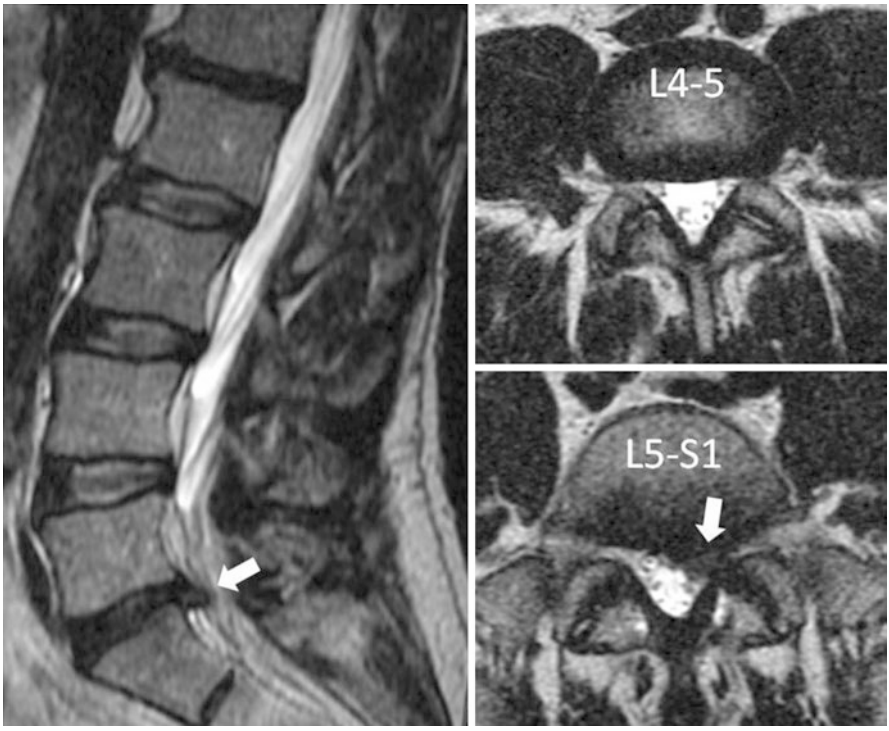

Fig. 6.1 Sagittal and axial MR images of a patient with a left-sided L5–S1 paracentral disc herniation(*white arrows*). MRI is the diagnostic modality of choice as it clearly demonstrates the distinction between neural elements, disc, and ligamentous structures

Electrodiagnostic studies, such as electromyographs (EMGs) and nerve conduction studies, are rarely needed in the routine work-up of a lumbar disc herniation. Such tests should be reserved for patients in whom the diagnosis is unclear or concomitant non-spinal pathology is suspected. It may also have a role delineating the most symptomatic level in a patient with multiple sites of nerve root compression.

Nonoperative Management

Nonoperative management of radiculopathy should begin with nonnarcotic analgesic medications for pain control. Nonsteroidal anti-inflammatory medications and acetaminophen should be considered first-line agents. Patients should be encouraged to be as mobile as possible, as there is strong evidence that more than 3 days of bed rest can perpetuate back pain. Neuroleptic medications, such as gabapentin or pregabalin, can also be used to specifically target neuropathic pain. Oral steroids should be reserved for patients in excruciating pain that is severely limiting their ability to ambulate and function. While steroid medications are often rather effective, they carry a greater side effect profile.

Education is perhaps the most overlooked component of nonoperative care. Affected individuals should be informed of the generally favorable natural history of this condition. There is an approximate 90% chance of resolution or improvement of symptoms (to the point of avoiding surgery) within 3 months of onset following nonoperative care. Many individuals, in fact, likely have disc herniations with radicular pain, ascribed to a bout of "sciatica," that resolves within a couple of weeks without any formal nonoperative care or physician evaluation. In the absence of neurological deficits or cauda equine syndrome, nonoperative care appears to be safe.

If a patient has had persistent radicular symptoms that have not improved within a couple of weeks of pharmacological management, physical therapy can be initiated. While most physical therapists will perform an initial evaluation, review imaging reports, and develop a specific treatment plan, the prescription ideally should request range of motion exercises and stretching. Core strengthening is a common component of physical therapy as well. Therapy is usually performed two to three times per week for 4–6 weeks.

Epidural steroid injections deliver a focal anti-inflammatory to the site of the disc herniation and affected nerve and serve both diagnostic and therapeutic roles in the treatment of lumbar disc herniation. These are usually not utilized as a first-line treatment as many patients will have a brief disease course. Injections should be reserved for those patients who have not responded to pharmacological and physical therapy. Spinal injections can be performed by anesthesia pain physicians, physiatrists, interventional radiologists, or surgeons. There are a variety of specific injections that can be performed, with the injection specialist deciding among them based on the type of herniation. A reasonable protocol for injections is as follows. After an initial injection is performed, it may take 1–2 weeks for the steroid to take effect. If leg pain resolves, there is little indication for another injection. If there is little or fair response to the first injection, a second may be attempted. Again, if the pain resolves, there is scant justification for a third injection. More so, if two injections failed to be beneficial, there is really no indication for a third attempt. Injections may also be utilized by specialists for diagnostic purposes, to elucidate the specific causal site of symptoms in patients with multiple areas of nerve compression on imaging studies.

Indications for Surgery

Surgery can be electively performed in a patient who has failed a 6–12-week course of nonoperative care. An implied prerequisite for surgery is, of course, signs and symptoms that are concordant with imaging findings of a lumbar disc herniation. An indication for surgery earlier than 6 weeks would be if a patient has a progressive deficit or a functionally limiting deficit, such as a foot drop that is impeding ambulation. Substantial canal compromise due to the size of the herniation (Fig. 6.2), which may manifest as cauda equina syndrome, is also an indication for more urgent surgical intervention.

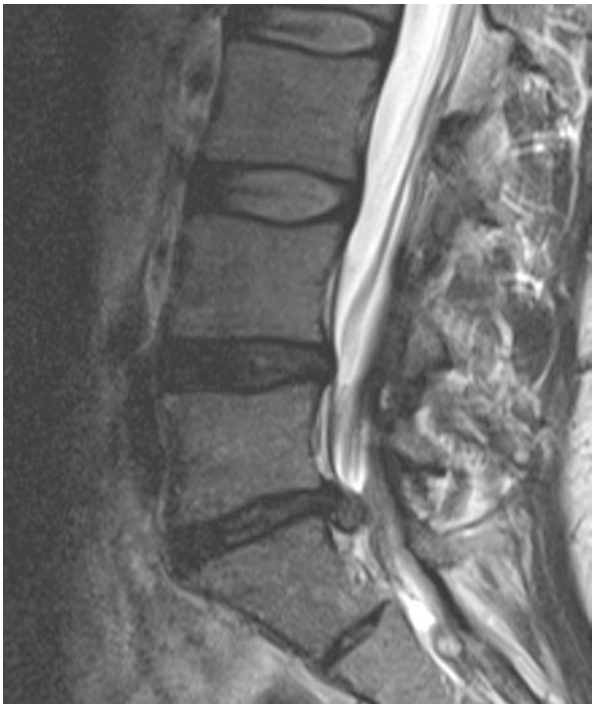

Fig. 6.2 Sagittal MR image of a patient with a large disc herniation at L5–S1 causing near complete occlusion of the spinal canal. Such a scenario may manifest as cauda equina syndrome

While there is a general sense that, if possible, patients should avoid spinal surgery as long as possible, this can potentially be detrimental to those with lumbar disc herniations. There is evidence that those undergoing surgery within 6–9 months of symptom onset have superior outcomes as compared to those who wait longer. The exact mechanism for this relationship is not well understood, though it is logical that long-standing compression and vascular compromise may increase the likelihood of permanent neuropathic pain.

Operative Management

A lumbar discectomy and nerve decompression can be performed using a variety of techniques. The traditional approach is an open procedure, sometimes termed a microlumbar discectomy, microscopic discectomy, or simply a discectomy. By definition, a microscopic procedure is performed using an operating microscope. This does not imply that it is performed in a more minimally invasive manner or with a smaller incision. In reality, a standard open lumbar discectomy can usually be performed through a relatively small incision (4–5 cm). Less invasive techniques are becoming increasingly utilized and accepted, with surgeons performing the same neurologic decompression and discectomy utilizing smaller tubular channels to access the spinal canal. Additionally, endoscopic procedures using small portals and

cameras, similar to arthroscopy for knees, are demonstrating similar efficacy to traditional open discectomies with reduced recovery times. These more modern surgical techniques have brought the treatment of lumbar disc herniation, for the most part, into the outpatient setting. As these techniques continue to progress, and the collateral damage and risk profiles of these interventions decrease, it is likely that both surgeons and patients will opt for surgical solutions earlier in the disease course of lumbar disc herniation.

While there are a multitude of evolving ways to access the spinal canal, the anatomic goals and key components of the procedure are consistent. A small portion of the lamina above and below and part of the medial facet joint at the operative level are removed. The ligamentum flavum is then excised to allow access to the spinal canal. The descending nerve root is retracted toward the midline, which usually reveals the disc herniation (Fig. 6.3). The herniated fragment is mobilized from the surrounding soft tissues using a variety of blunt instruments. The fragment can then be removed using a grasping instrument.

Expected Outcomes

Surgical treatment of lumbar disc herniations is among the most satisfying for both surgeon and patient. In general, the chance of a successful outcome is about 80–90%. It is very important, however, to accurately characterize what "success" means to the patient prior to surgery. Based on the best available evidence, a patient can expect an 80–90% chance that leg pain (which includes buttock pain) and everyday function will substantially improve. Axial back pain has a number of potential etiologies that may or may not be related to the site of the disc herniation, and, as such, it is clear from available data that back pain *may* improve but that it does so much less reliably than leg pain. In general, many spine surgeons are reluctant to perform surgery on patients with only axial back pain complaints, as the identification of a focal pain generator and successful treatment of these patients is far more unreliable than those with clear radicular complaints.

Patients often present with complaints of paresthesias, numbness, and weakness. It is equally important to explain that these are not as reliably improved with surgery as pain and function. Nerve compression can result in intrinsic damage to spinal nerves, and, while surgery can effectively remove extrinsic nerve compression, we do not yet have reliable interventions to affect change on intrinsic nerve recovery. These intrinsic factors and associated recovery potential likely account for some of the heterogeneity we see in nerve recovery after surgery. There is certainly contrasting literature on recovery of strength deficits following discectomy. Neurological deficits with disc herniation often improve with or without surgery, though there is literature to suggest that the degree of recovery and the pace of recovery may be improved with surgical treatment. Regarding numbness, it has been the authors' experience that intermittent, subjective numbness has a better chance of resolving than persistent anesthesia. Discussion of these tendencies is critically important

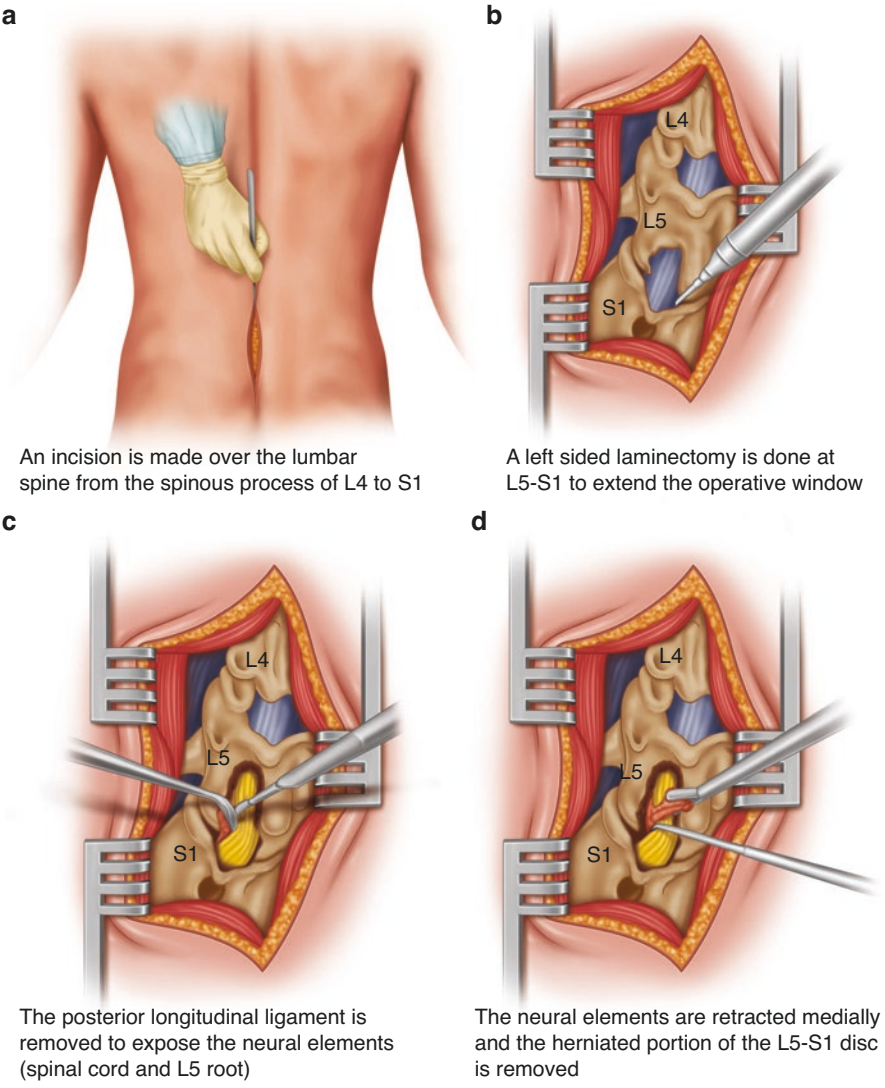

Fig. 6.3 After the ligamentum flavum has been removed (**a**, **b**), the descending nerve root and cauda equina are identified and retracted toward the midline (**c**). This usually reveals the annulus of the disc and the herniation. The herniated fragment is mobilized from the surrounding soft tissues and removed with a grasping instrument such as a pituitary rongeur (**d**)

prior to surgery as patients often intuitively expect that any neurological deficits will be immediately reversed postoperatively.

It is important to recognize a number of factors that can influence surgical outcomes. It has been long thought that larger disc herniations are associated with better outcomes. More recent work has not supported this but instead has reported that

the volume of herniated disc removed at surgery is more important. There is conflicting data regarding the relationship between the anatomical level or location of the herniation and surgical outcomes.

The effect of psychosocial factors on the outcomes of disc herniations cannot be overstated. Among a variety of factors assessed, one study found psychological status as measured by the Minnesota Multiphasic Personality Inventory (MMPI) to be the most predictive of surgical outcomes. Other studies have corroborated these results, with additional factors such as self-confidence and optimism to be associated with superior surgical results. Conversely, patients receiving workers' compensation are less likely to report favorable outcomes following discectomy.

There are a number of complications that can occur during or after lumbar discectomy, with infection among the most common. The risk for wound infection is influenced primarily by patient factors, including diabetes, obesity, smoking, and immunosuppression. By nature of the procedure itself, the nerve roots are being mobilized and can result in a new postoperative neurological deficit, albeit this complication is rare. More commonly, however, patients may experience a transient self-limiting radiculitis (leg pain) postoperatively as a result of nerve mobilization or inflammatory mediators.

Dural tear and cerebrospinal fluid (CSF) leak can also occur during the procedure. If this occurs, suture repair or patching of the tear may be necessary. Postoperatively, the patient may require 1–3 days of bed rest in order to decrease the intrathecal pressure on the area of repair. Dural tears have been reported to occur in up to 4% of primary lumbar discectomies. Fortunately, their occurrence does not portend a poor outcome.

The risk of recurrent disc herniation varies widely and is influenced by patient age, type of herniation, and size of the associated annular defect. It can also be influenced by surgical technique. Recurrent herniations can occur in up to 5–10% of patients following a primary lumbar discectomy. While many of these recurrent herniations will improve with nonoperative measures like the index condition, some may require surgical intervention. Fortunately, the results of operative treatment for a recurrence are reportedly comparable to those achieved after index discectomy (Table 6.1).

Table 6.1 A summary of lumbar disc herniation and radiculopathy presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Nonoperative management	Indications for surgery	Operative management
Lumbar disc herniation with radiculopathy	Unilateral, radiating leg pain Variable degrees of mild sensory and motor deficit	MRI to detect the site of disc herniation, level involved, and degree of compression CT myelogram only if MRI is contraindicated	PT: stretching, range of motion of the low back and lower extremities Nonnarcotic analgesic medications Interventional modalities like epidural steroid injection	Progressive neurological deficit or cauda equine syndrome (rare) Persistence of substantial symptoms despite 6–12 weeks of structured nonoperative treatment	Lumbar discectomy (also known as microdiscectomy)

Suggested Reading

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Chapter 7
Degenerative Lumbar Spinal Stenosis and Spondylolisthesis

Daniel G. Tobert and Mitchel B. Harris

Abbreviations

CT	Computed tomography
DLS	Degenerative lumbar spondylolisthesis
EMG	Electromyography
ESI	Epidural steroid injections
LSS	Lumbar spinal stenosis
MRI	Magnetic resonance imaging
NASS	North American Spine Society
NCS	Nerve conduction studies
NSAIDs	Nonsteroidal anti-inflammatory drugs
PT	Physical therapy
PVD	Peripheral vascular disease
SPORT	Spine Patient Outcomes Research Trial

Introduction

Degenerative changes in the lumbar spine primarily manifest as low back pain; less commonly the clinical presentation is claudicant or radicular in nature. Lumbar spinal stenosis (LSS) and degenerative lumbar spondylolisthesis (DLS) are two prevalent degenerative conditions that can range from mild axial pain to debilitating symptoms with signs of neurologic compromise.

The term *stenosis* derives from the Latin prefix "steno," meaning "narrowing." The term *spondylolisthesis* derives from the Latin "spondylo," meaning "spine," and "listhesis," meaning "slip." Both terms refer to radiographic observations and do not

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necessarily correlate with patient symptoms. As such, clinical care decisions are made based on the nature and severity of the symptoms arising from stenosis and/or spondylolisthesis. The purpose of this chapter is to further clarify the varied clinical presentation of these conditions and detail the diagnostic workup, treatment options, and expected outcomes for degenerative lumbar spinal stenosis and spondylolisthesis.

Lumbar Spinal Stenosis

Definition and Epidemiology

The prevalence of LSS has been reported in the range of 5–10% of the population, regardless of age. However, up to 40% of patients over the age of 60 meet the radiographic criteria for LSS. Fortunately, the prevalence of symptomatic LSS is lower than that observed radiographically, with 7–10% of patients over 65 experiencing radiculopathy or neurogenic claudication. LSS presents equally in males and females. It should be noted that a subset of patients develop LSS earlier in life based on congenitally narrowed spinal canals. In addition, patients with achondroplasia or osteopetrosis may develop symptomatic LSS in the third or fourth decade of life.

Clinical Presentation

The manifesting symptom of LSS is leg pain, and the type of leg pain depends not only on the level of stenosis (e.g., L4–L5) but also the location within the spinal canal (e.g., central vs. lateral recess or foraminal). If the thecal sac is compressed centrally, *neurogenic claudication* often results. This condition is described by patients as a dull ache or burning sensation that originates in the low back or gluteal region and travels down the lower extremities without following a specific nerve root distribution. Pain radiating distal to the knee is not required for the diagnosis, as patients can present with pain solely in the gluteal region. This sensation is typically worse when walking or with activities involving extension of the lumbar spine, such as sitting or standing upright for a prolonged period of time. Anatomical studies have demonstrated further narrowing of the lumbar spinal canal in extension, increasing the severity of compression on an already compromised thecal sac. Flexion in the lumbar region results in an expansion of the contours of the spinal canal. This accounts for observations that symptoms are partially or fully alleviated when leaning forward, pushing a grocery cart or using an ambulatory aid.

Aside from neurogenic claudication, LSS can also cause lumbar radiculopathy. This symptom arises from compression of a nerve root as it exits the thecal sac. Unlike neurogenic claudication, patients with nerve root compression describe leg

pain and paresthesias in a specific nerve root distribution. The L5 nerve root is most commonly affected in the setting of LSS. Classic symptoms manifest as pain radiating down the outside aspect of the thigh, wrapping around the front of the leg and ending in the webspace of the first and second toes, with associated paresthesias. However, due to the natural variation in dermatomal mapping, a L5 radiculopathy can have features of that classically thought to be L4 or S1.

Physical examination findings are not consistent among patients with LSS, and typically patients are neurologically intact. Provocative nerve root tension signs, such as the straight leg raise test, are frequently negative in LSS. Careful extension of the lumbar spine with examiner assistance or asking to the patient to stand for a period of time during the exam may provoke neurogenic claudication symptoms. In large population studies, frank motor weakness is present only 25% of the time, and sensory changes are seen in approximately 20–50% of patients. Decreased reflexes in the patella and Achilles tendon can also be seen in LSS and may be asymmetric. Any presence of an upper motor neuron sign, such as hyperreflexia, dense lower extremity paralysis, sustained ankle clonus, or an upgoing great toe excursion during the Babinski maneuver, should alert the clinician to cephalad pathology along the neuroaxis (e.g., cervical or thoracic regions) or involving the brain.

Differential Diagnosis and Suggested Clinical Testing

The demographic predisposed to LSS is also at risk of other degenerative musculoskeletal conditions including peripheral joint arthritis, peripheral vascular disease, and neuropathy. Hip arthritis most commonly presents with groin pain exacerbated by weight-bearing or movements involving the hip. On physical examination, patients will exhibit a painful loss of hip flexion and internal rotation. If there is concern for hip joint pathology, a standing AP view of the pelvis should be obtained to assess that area. Often, radiographic evidence of degenerative pathology in both the lumbar spine and hip is present. This entity, termed the "hip-spine syndrome," is increasingly being recognized as a common condition in the aging population. Musculoskeletal specialists often use diagnostic injections to help characterize the contributions of each pathology to a patient's pain.

Peripheral vascular disease (PVD) can result in *vascular claudication*, and symptoms can overlap significantly with neurogenic claudication. Clues that aid the clinician during history and physical examination are summarized in Table 7.1. Patients with PVD will generally describe a set distance or amount of exertion before symptoms develop, regardless of position. Inquiring about symptoms during bicycling (stationary or traditional) can help distinguish claudication by history. Whereas the neurogenic claudicator will report being able to bicycle without issue due to the flexed forward truncal position, the vascular claudicator will develop symptoms due to the increased metabolic demand in the vascularly compromised lower extremity musculature. Typically, the pain begins distally near the ankle and moves proximally during vascular claudication. The well-known predisposing factors for PVD,

Table 7.1 History and physical exam findings that help differentiate between neurogenic claudication, radiculopathy, and vascular claudication

	Neurogenic claudication	Radiculopathy	Vascular claudication
Location	Proximal to distal, non-dermatomal	Proximal to distal, dermatomal	Distal to proximal, non-dermatomal
Quality	Dull, achy	Sharp	Dull, achy
Severity	Variable	Variable	Variable
Timing	Related to posture, can be constant	Generally constant but can be related to posture	Only with activity unless end stage
Aggravating factors	Standing, sitting, walking upright	Certain movements and extension reliably worsen	Walking uphill, increased physical exertion
Alleviating factors	Leaning forward, walking uphill	Certain movements and flexion improve	Cessation of activity unrelated to position
Physical exam	Palpable pulses, no distal skin changes	Palpable pulses, no distal skin changes	Hairless, shiny legs, diminished or absent pulses

including diabetes mellitus, smoking, and hyperlipidemia, are often present. Physical exam findings for patients with PVD include diminished distal pulses, hairless extremities, lipodermatosclerosis, or frank ulceration. Concern for PVD should prompt measurement of ankle-brachial indices and noninvasive arterial/venous flow studies.

Diabetic neuropathy also results in symptoms that can mimic LSS. However, diabetic neuropathy is not activity related and usually arises in a "stocking and glove" distribution. Less common mimickers of LSS include multiple sclerosis, transverse myelitis, or compressive lesions of the lumbosacral plexus.

When LSS is suspected after history and physical examination, imaging is required to confirm the diagnosis and evaluate the extent of disease. Plain films of the lumbar spine should be obtained with upright AP, lateral, and flexion/extension views. These views often show diffuse degenerative changes but are helpful insofar as they portray the sagittal and coronal alignment of the lumbar spine while under physiologic loading. Flexion and extension views aid in the evaluation of spondylolisthesis and its potential for spinal hypermobility or instability. Magnetic resonance imaging (MRI) is the most useful modality when evaluating for LSS. The images obtained from MRI are able to delineate relationships between osseous, soft tissue, and neural structures of the lumbar spine. Therefore, MRI helps define the degree of stenosis (narrowing) and the specific neural structures involved (Figs. 7.1 and 7.2). This information, in combination with a history and physical exam, can definitively establish the diagnosis of LSS.

If MRI is contraindicated because of implanted ferromagnetic devices, computed tomography (CT) with myelography can be used. The soft tissue resolution of CT with myelography is much lower in comparison to MRI, and the study is invasive. Nonetheless, a CT myelogram can determine the level(s) of stenosis and differentiate soft tissue pathology that is anterior, posterior, or lateral to the thecal sac.

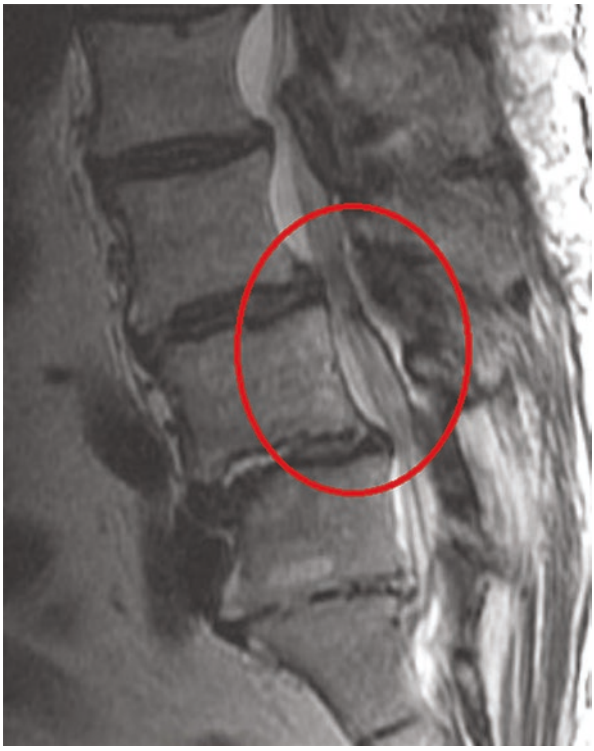

Fig. 7.1 Sagittal MRI image demonstrating loss of disk space height, disk protrusions, and buckling of the ligamentum flavum at L3–L4 and L4–L5 in the setting of two-level spondylolisthesis (*red circle*)

Electromyography (EMG) and nerve conduction studies (NCS) are not a standard part of the LSS workup and should not be ordered unless evaluation of a secondary diagnosis is warranted.

Nonoperative Management

The natural history of LSS has not been clearly defined, but existing research can help guide the clinician when counseling patients about treatment strategies. A well-done natural history study has demonstrated that 70% of patients with LSS have similar symptoms after an average follow-up of 4 years, with 15% worsening clinically and another 15% reporting symptomatic improvement. Oral medications can be helpful in the management of LSS. Nonsteroidal anti-inflammatory drugs (NSAIDs) can provide partial symptom relief but have predictable renal, gastrointestinal, and cardiovascular side effects with prolonged use. Opioid medications should be avoided due to the risk of dependence, gastrointestinal effects, and cognitive alterations.

Physical therapy (PT) focused on core strengthening and range of motion should be tried as an initial therapy if not otherwise contraindicated by other medical

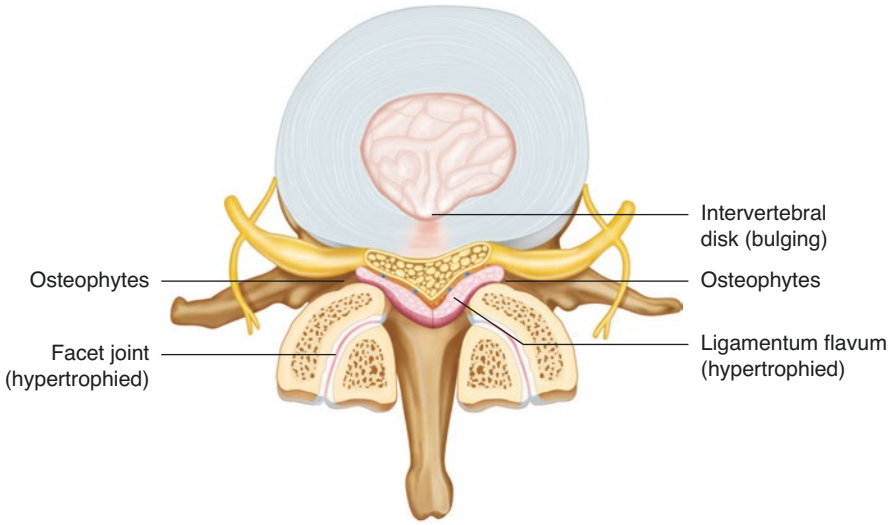

Fig. 7.2 Pictorial demonstrating stenosis resulting from a combination of central disk bulging, facet joint osteophytes, and ligamentum flavum hypertrophy

conditions. Patients should be counseled to attempt at least 6 weeks of therapy with daily completion of a home exercise program before reassessing symptoms. In a randomized controlled trial comparing PT to surgical treatment in patients with LSS, nearly half of the patients who were treated with PT alone noted some degree of improvement at 2-year follow-up.

Epidural steroid injections (ESI) under fluoroscopic guidance are commonly used to treat symptoms from LSS. The premise behind an accurately placed ESI is that a reduction of local inflammatory mediators will help improve pain. However, a randomized controlled trial of 400 patients with moderate to severe symptomatic LSS found ESI had no benefit at 6-week follow-up. ESI may be more beneficial for patients with LSS who present with radicular symptoms as opposed to those manifesting neurogenic claudication. Physicians treating patients with LSS and considering ESI should take into account the patient's predominant complaint, as well as their candidacy for other nonoperative and surgical interventions, before proceeding with a referral for injections.

Indications for Surgical Management

An absolute indication for surgical management is a progressive neurologic deficit or bowel and bladder dysfunction due to compression of the lumbosacral nerve roots. Fortunately, this scenario is rare in LSS, presenting in less than 1% of patients. In most instances, surgical management is typically recommended following a

patient's failure to satisfactorily derive benefit from a nonoperative treatment regimen.

A shared decision-making approach between patient and provider should be used when discussing surgical management. The clinician should understand the patient's level of function prior to the onset of LSS in contrast to their level of function at the time of presentation. Walking distance is a good metric for assessing functional status. A patient who would otherwise be able to walk long distances but is limited to one or two city blocks because of LSS symptoms experiences significant quality of life impairment. Ultimately, however, it is the patient who must conclude that their quality of life has deteriorated to the point where surgical treatment should be considered.

A discussion of surgery begins by assessing the patient's expectation for treatment. This information helps guide the discussion of surgical management and will often prevent a less favorable patient-reported outcome. Careful scrutiny of the medical history should be performed to help manage perioperative risk. For example, patients with significant cardiovascular comorbidities not evaluated within a year should be referred to a cardiologist for evaluation and perioperative risk assessment. If a patient is anticoagulated, a clear plan for cessation and resumption of anticoagulant therapies should be devised with the patient's other physicians. While this care coordination can be cumbersome in the increasingly fragmented medical care system, it is essential to prevent surgical complications.

Research efforts are underway at many institutions to predict which patients will achieve the greatest benefit from surgical treatment. These ongoing efforts utilize patient-reported outcome measures, such as PROMIS, and advanced statistical techniques. One common finding is the importance of the patient's disability prior to undergoing surgical treatment. Although perhaps intuitive, it suggests that the patient who is more functionally impacted by the symptoms of stenosis is more likely to report a benefit from surgical decompression.

Operative Management

Operative treatment of LSS is increasingly common. Between 1994 and 2006, surgery performed for LSS increased by over 900%. Technological advances have provided a multitude of options for the spine surgeon. Nonetheless, the succinct goal of operative treatment is decompression of the neural elements that are believed to be the cause of the claudicant or radicular symptoms. This is categorically accomplished by removal of the offending surrounding structures while maintaining spinal stability.

Depending on the extent of stenosis, a *laminotomy* or *laminectomy* can be performed, which entails removal of a portion or the entire lamina, ligamentum flavum, and hypertrophied medial facets at a given level.

A laminectomy is performed in conjunction with fusion, or *arthrodesis*, if there is concern for concomitant instability. Instability can exist preoperatively (such as

spondylolisthesis discussed later) or as a result of surgical decompression. Preoperative instability is assessed with flexion/extension radiographs but can often be inferred on MRI or CT imaging. Typically, if more than 50% of the facet joints are removed during decompression, a surgeon will perform a fusion at the time of surgery.

Many patients inquire about the use of micro-endoscopic techniques for lumbar spine surgery. This is usually performed when single-level stenosis is present lateral to the thecal sac, or the *lateral recess* of the spinal canal. The advantages of this technique are a less extensive soft tissue dissection and a smaller surgical scar. However, this method risks incomplete decompression, and some studies have shown a higher rate of dural tear. At the time of this writing, decompression with open laminectomy or laminotomy remains the gold standard for operative management of LSS.

Expected Outcomes

Achieving a successful outcome following treatment of LSS begins with a transparent discussion between the patient and clinician about available treatment options and the patient's expectations. Numerous studies have reported patient satisfaction rates close to 90% when carefully indicated for surgical decompression. Other research has shown that the patients least satisfied with surgical treatment are those that have back pain as their predominant symptom. This highlights the crucial role of listening to the patient's history when evaluating a patient for LSS. Other factors that portend a less favorable surgical outcome are increased medical comorbidities and increased baseline functional disability. Patients with diabetes, obesity, and rheumatoid arthritis are at increased risk of a surgical site infection postoperatively.

Degenerative Lumbar Spondylolisthesis

Definition and Epidemiology

Broadly, spondylolisthesis refers to translation of a vertebral body in relation to adjacent vertebral structures. Spondylolisthesis can be further distinguished by whether a developmental anomaly is present, if the neural arch is intact (i.e., the posterior osseous structures are in continuity with the anterior vertebral body), or if spondylolisthesis occurs at a level adjacent to a fused segment. This section focuses on DLS, which refers to translation with an intact neural arch in the setting of predisposing arthritic changes.

L4–L5 is the most common level where DLS occurs, and the patient is typically in the fifth decade of life or later. DLS is present four times more often in females than in males. This skew in prevalence is thought to be a result of increased ligamentous laxity observed in females secondary to hormonal differences, but definitive causality has not been established. In addition, genetic factors are thought to play a role in the development of DLS, and the condition may be more prevalent in African-American women. A population study found radiographic evidence of DLS in approximately 8% of females and 3% of males, and these numbers vary slightly depending on the ethnicity studied.

Clinical Presentation

The most common presenting complaint in the setting of DLS is neurogenic claudication that results from concomitant spinal stenosis at the level of the listhesis. As the cephalad vertebral body translates anteriorly, the space available for the thecal sac becomes dynamically stenotic. Patients describe a limit to their ability to maintain a standing position or walking before needing to lean forward or sit down. A patient's symptoms may be described as less severe when cycling or pushing a grocery cart, which is attributed to flexion of the lumbar spine during these activities and decreased compression on the thecal sac.

Less commonly in DLS, patients report radicular symptoms or purely axial back pain. The most common level nerve root involved in DLS is L5. A concomitant motor weakness of great toe extension and ankle dorsiflexion can occur if the L5 nerve root is involved but is not observed in the majority of cases.

Differential Diagnosis and Suggested Clinical Testing

Similar to the differential diagnoses for LSS, degenerative changes in the hip and knee must be considered in the event of DLS. Osteoarthritis of the hip can refer pain to the medial aspect of the knee through the anterior branch of the obturator nerve, and a patient's description of this can mimic radicular pain. Degenerative hip pain can be exacerbated immediately on standing and with internal rotation of the femur, and a weight-bearing plain film of the pelvis is recommended to look for the presence of degenerative hip pathology.

Peripheral vascular disease should always be considered in a patient with activity-related leg pain. As noted in Table 7.2, the factors that distinguish vascular claudication from neurogenic claudication are a fixed physical activity limit that does not vary with position, improvement of symptoms by standing upright, diminished or absent distal lower extremity pulses, and skin changes from chronic hypoperfusion.

Table 7.2 A summary of degenerative lumbar spinal stenosis and spondylolisthesis with a synopsis of presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Lumbar spinal stenosis	Neurogenic claudication Radiculopathy	MRI/ CT—myelogram if MRI contraindicated	PT Non-opioid oral medications Lifestyle modification	Rapidly progressive neurologic deficit Failure of nonoperative regimen	Laminotomy Laminectomy Laminectomy with fusion
Degenerative lumbar spondylolisthesis	Neurogenic claudication Radiculopathy	Upright lateral lumbar plain film MRI to evaluate extent of stenosis	PT Non-opioid oral medications Lifestyle modifications	Rapidly progressive neurologic deficit Failure of nonoperative regimen	Laminectomy without fusion (elderly, frail) Laminectomy with fusion

There is often radiographic evidence of DLS as a patient is being worked up for a complaint of low back, leg, gluteal, or hip pain. The challenge for the clinician is to distinguish DLS as the primary etiology of a patient's symptoms or merely a secondary radiographic finding. The most useful radiographic study in the evaluation of DLS is a weight-bearing lateral lumbar plain film. The weight-bearing film is important because it evaluates the alignment of the lumbar spine during physiologic loading, and spondylolisthesis can be missed if a lateral radiograph is taken in the supine position and the translated vertebral body reduces to its anatomic position. Flexion/extension views are useful if the initial upright lateral film is equivocal or to evaluate for a mobile spondylolisthesis.

Because the symptoms of LSS and DLS overlap almost completely, a lumbar spine MRI helps evaluate for stenosis in the setting of DLS. However, similar to plain films taken in the supine position, the lack of anterior translation on MRI does not rule out DLS. Subtle clues on MRI can alert the clinician to the presence of spondylolisthesis. On the axial sequences, a facet joint effusion greater than 1.5 mm is suggestive of DLS and should prompt weight-bearing plain films if they have not been already obtained.

Nonoperative Management

All patients with DLS should receive nonoperative therapies as first-line treatment. The majority of patients will not require operative management in the absence of a progressive neurologic deficit. Natural history studies regarding DLS are limited. One study followed a group of 40 patients with DLS for an average of approximately 8 years, and only 10% developed worsening symptoms during this time

period. Interestingly, none of the patients whose symptoms worsened developed radiographic progression of the spondylolisthesis. There is not a correlation between radiographic severity of DLS and clinical symptoms.

As with LSS, nonoperative treatment modalities include physical therapy and non-opioid oral medications. The North American Spine Society (NASS) published an updated evidence-based clinical guideline in 2014 on the treatment of DLS and concluded there is insufficient evidence to make a recommendation for or against injections, although this modality is frequently employed, especially in those patients adamantly against consideration of surgery. A more recent study by Gerling et al. reported no benefit with injections for patients with DLS over a 4-year period. Additionally, the rates of surgery were the same (approximately 60%) for patients regardless of the use of injections.

Indications for Surgical Management

Absent of a progressive neurologic deficit attributable to DLS, the indications for surgical management are based on the severity of a patient's symptoms and the extent of their response to conservative care. The Spine Patient Outcomes Research Trial (SPORT) prospectively randomized over 600 patients with DLS to nonoperative or operative treatment. As a result of substantial crossover between the two cohorts, the publication of this trial included both intention-to-treat and as-treated analyses. Using as-treated analysis, a statistically significant improvement in pain and function was found at 4-year follow-up in the operative group.

The opportunity for symptom alleviation and functional improvement with surgical treatment must be weighed against the risks. Typically, operative treatment can be considered if symptoms related to DLS have led to a loss of independence in daily activities. Likewise, if symptoms have led to an unacceptable degradation in the quality of life for a patient, then surgical treatment can be offered.

Operative Management

The typical surgical treatment for symptomatic DLS is decompression of the stenotic areas and arthrodesis (Fig. 7.3). As described above, a laminectomy is typically performed to decompress the thecal sac dorsally. There are retrospective studies in the literature that maintain positive results can be achieved following decompression alone (without arthrodesis) in the setting of low-grade spondylolisthesis. For an elderly patient with multiple comorbidities and low functional activity, decompression alone may be a viable option.

However, higher-quality data demonstrates superior and more durable results which can be achieved when arthrodesis is performed in addition to decompression in the setting of DLS. Specifically, there is level 1 evidence showing a lower

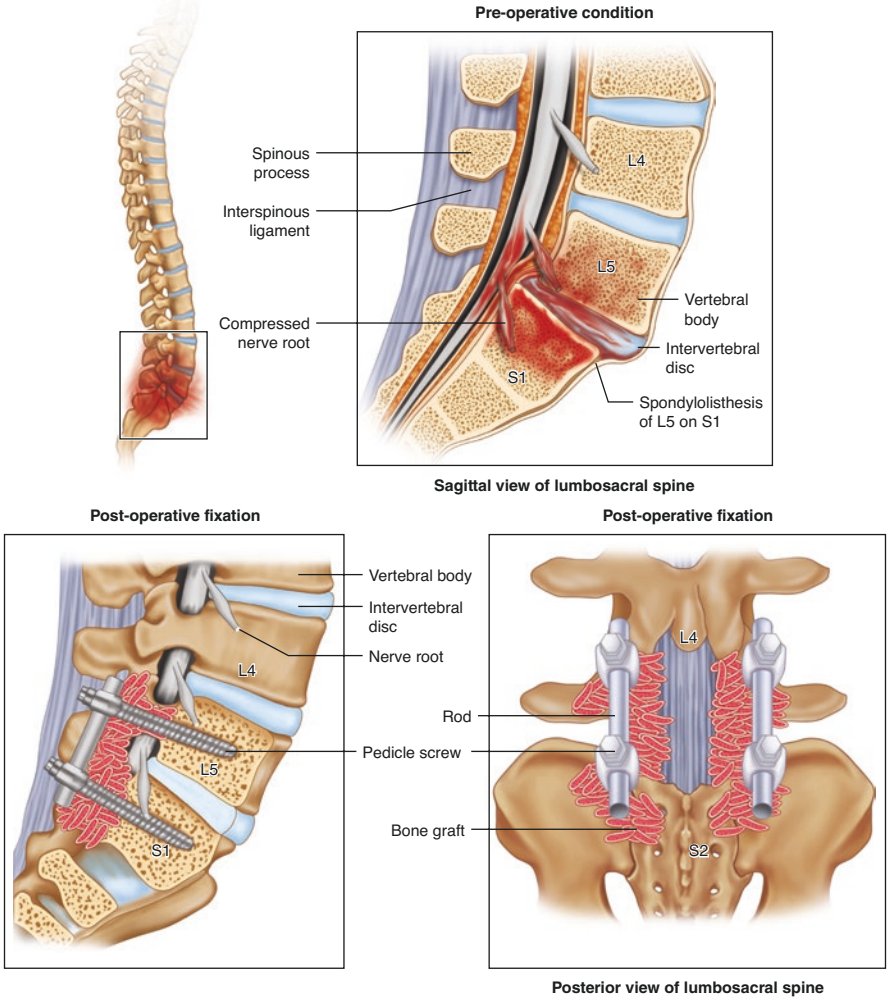

Fig. 7.3 Schematic demonstrating a lumbar spondylolisthesis treated with an instrumented fusion-based procedure. Screws and connector rods are used to stabilize the fusion site and allow for osseous integration

reoperation rate in patients that undergo fusion. This is reflected in the NASS clinical guideline for DLS, where a stronger recommendation is made for both decompression and arthrodesis as compared to decompression alone. Arthrodesis is often performed with instrumentation based on data suggesting it can improve fusion rates. However, there is no evidence to support a contention that the use of instrumentation improves clinical outcomes. Given the lack of objective high-quality

data, the decision for instrumentation is made by the treating surgeon based on their clinical experience, patient factors, and surgical goals.

Expected Outcomes

The natural history of symptomatic DLS favors nonsurgical management, and yet of those who undergo operative treatment, the majority can expect a positive outcome. The SPORT reported that 86% of patients were satisfied with the results of surgical intervention.

Numerous studies have evaluated factors that influence outcomes in the surgical treatment of DLS, but few provide high-quality data. There are suggestions in the literature that patients who achieve fusion, regardless of instrumentation, experience superior clinical outcomes. As such, factors that predispose a patient to pseudarthrosis after an attempted fusion, such as smoking, chronic steroid use, end-stage renal disease, and diabetes, may influence the ultimate surgical result. The duration of symptoms prior to surgical treatment does not appear to influence outcomes. Obesity as defined by a body mass index over 30 kg/m^2 is associated with an increased surgical site infection rate and need for reoperation.

Suggested Reading

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Part II
Osteoporosis

Chapter 8
Osteoporosis, Vertebral Compression Fractures, and Vertebral Cement Augmentation

Marco L. Ferrone and Andrew J. Schoenfeld

Osteoporosis

Definition and Epidemiology

Bone density-related issues exist on a spectrum, with osteoporosis representing the most advanced stage of bone density loss, while osteopenia is a milder manifestation. Osteoporosis is defined as loss of bone mass and disruption of internal architecture, which can culminate in the development of fragility fractures, even with little or no precipitating trauma. Osteoporosis may also be present in the absence of fractures. As the population ages, the incidence and prevalence of osteoporosis is anticipated to exponentially increase over the next few decades. At present, it is estimated that osteoporosis affects 10 million older adults in the United States, 80% of whom are women. In addition, there are a further 18 million individuals living with osteopenia.

Osteoporosis variably affects different populations across the globe and is less prevalent in the developing world and far more common in Europe and among those with European ancestry. In the United States, known risk factors include female biologic sex, Caucasian or Asian race, a family history of osteoporosis, chronic consumption of caffeine, nicotine and alcohol, low body weight, deficiencies of dietary calcium and vitamin D, insufficient physical activity, and advancing age. Obesity, or higher BMI, has previously been shown to be protective against the development of osteoporosis.

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Clinical Presentation

Osteopenia and osteoporosis are clinically silent until fractures develop. This fact underpins the critical necessity for testing individuals who may otherwise be at risk. Patients who present with a fragility fracture should be evaluated for osteoporosis during, or after, treatment of the injury. The goal here is the prevention of future fragility fractures, especially in light of the prospect for future fractures to develop after a sentinel injury. Cumulative fractures, or a major injury such as a hip fracture, can have immediate adverse effects on the prospect of functional independence, quality of life, and overall survival.

Differential Diagnosis

Osteoporosis is characterized as a primary or secondary condition. Primary osteoporosis can occur in both genders but is characteristically encountered in postmenopausal women. Secondary osteoporosis is caused by medications and other medical conditions or diseases, as seen in patients using glucocorticoids and with hypogonadism or celiac disease.

Although osteoporosis is most commonly the result of bone loss, it can also derive from a failure to achieve optimal bone mass as a young adult. In this setting osteoporosis is not due to accelerated bone loss, but rather sub-optimal development. This sub-optimal development of bone mass in younger individuals may be the result of, or exacerbated by, malnutrition, malabsorption, eating disorders, a variety of chronic diseases, or severe inactivity.

Diagnostic Testing

Screening laboratory tests are carried out in all patients being evaluated for primary osteoporosis and should include a basic metabolic panel, complete blood cell count, liver function tests, thyroid function tests, gonadal hormone levels, and a serum 25-hydroxyvitamin D level. Vitamin D deficiency is one of the most common causes of reduced bone mineral density, especially in men. Laboratory tests for secondary osteoporosis should be directed at the suspected underlying cause of the condition.

While plain radiographs and advanced imaging studies such as computed tomography (CT) are often used in the evaluation of patients with osteoporosis and especially in those with suspicion of fracture, radiolucency associated with the condition will not readily be present on imaging until the loss of more than 30% of bone mineral density has already occurred. As a result, these imaging modalities are not reliable screening tools for osteoporosis in the absence of a suspected fracture.

Dual energy X-ray absorptiometry (DEXA) is the most widely utilized approach to measuring bone mineral density (BMD). DEXA scans measure BMD within the lumbar spine and proximal femur. DEXA scores are reported as a T-score, with the value in standard deviations (SD) as compared to a healthy young adult white woman, and a Z-score, where the value is given in SD as compared to age-, race-, and sex-matched controls. T-scores of −1.0 or greater are considered normal. Those in the range of −1.0 to −2.5 are reflective of osteopenia. Patients with scores that are lower than −2.5 are considered to have frank osteoporosis. An osteopenic score in the presence of a fragility fracture is also considered reflective of osteoporosis and represents an indication for treatment.

Nonoperative Management

Osteoporosis in and of itself is not a surgical condition and can be treated with a variety of medical interventions ranging from calcium and vitamin D supplementation to disease-modulating agents. The opportunity to address all modifiable risk factors for impaired mobility and increased risk of falls should also be recognized, including smoking, alcohol, exercise, living situation, use of walking aids, and visual disturbances.

While correcting vitamin D and calcium intake are the most common interventions and those with the lowest side effect profile, a number of medications to preserve bone stock should also be considered. Bisphosphonate medications are typically considered a first-line treatment for patients with osteoporosis. There are two classes of bisphosphonates, based on whether the medication contains nitrogen. The nitrogen-containing bisphosphonates (alendronate, pamidronate, ibandronate, risedronate) inhibit farnesyl pyrophosphate synthetase, the enzyme required for osteoclasts to resorb bone. The non-nitrogen-containing bisphosphonates (clodronate, etidronate, tiludronate) precipitate the apoptosis of osteoclasts by creating a toxic analogue of adenosine triphosphate.

Bisphosphonates have been shown to reduce the risk of fragility fractures by approximately 50% following 1 year of use. Side effects from the oral formulations include esophageal erosions and stomach inflammation, while intravenous formulations can cause flu-like symptoms. All bisphosphonates carry a risk of osteonecrosis of the jaw, as well as atypical fractures with long-term use. The most characteristic manifestations of these atypical fractures mainly present in the subtrochanteric region of the femur. The risk to benefit ratio for bisphosphonate use in women at risk of insufficiency fracture is thought to be exceedingly low for the period of the first 5 years following treatment initiation. Strategic use of drug holidays may also balance the efficacy of these medications against the potential for side effects, osteonecrosis, and atypical femur fractures.

Calcitonin is another medication that can be used in the treatment of osteoporosis. This medication is administered intranasally and binds to osteoclasts, thus inhibiting bone resorption. It is not generally considered first-line therapy, but may

be used in patients who cannot tolerate bisphosphonates. While BMD has been shown to increase with the use of calcitonin, specific studies have reported these to be only modest for the most part. Common side effects are related to the means of intranasal administration, including topical irritation, rhinitis, and bleeding. There is some concern regarding a link between calcitonin use and the risk of malignancy, but evidence for this remains inconclusive at this time.

Denosumab is a monoclonal antibody administered monthly as a subcutaneous injection. Denosumab works through the biochemical pathway that allows for the differentiation of osteoclasts. Denosumab has been shown to increase BMD in postmenopausal women and leads to concomitant reductions in fragility fractures. Denosumab is typically well tolerated by most patients but carries similar risks of osteonecrosis of the jaw as the bisphosphonates. The main obstacle to use of denosumab tends to be its high cost and insurance approval.

Teriparatide is a recombinant version of parathyroid hormone, administered subcutaneously daily, and is the only known anabolic agent used to treat osteoporosis. It has been approved for use in the United States since 2002 and has been found to increase BMD by 8% and reduce the risk of fragility fractures after 1 year of use. Overall, teriparatide leads to greater increases in BMD than the other anti-osteoporotic medications. It has also been shown in recent randomized studies to enhance bone fusion in patients undergoing spine surgery in the context of osteoporosis.

Obstacles to the use of teriparatide include cost, the requirement for daily injection, and concerns for osteosarcoma. Due to this warning, teriparatide is contraindicated in patients with a history of some cancers and Paget's disease. Other side effects are typically well tolerated, including mild nausea, dizziness, and headaches. A newer parathyroid hormone analogue, abaloparatide, has been reported to reduce the risk of insufficiency fractures in women, with a lower risk of side effects as compared to teriparatide. Abaloparatide's mechanism of action is a selective activation of the parathyroid hormone type I receptor.

Expected Outcomes

Patients who are found to be osteoporotic or develop fragility fractures should be considered for some type of medication to decrease the risk of future fracture. Sizable reductions in fracture risk can be achieved even after only a short period of appropriate medication use. If a patient fails to respond satisfactorily to first-line medications based on DEXA, or develops another fragility fracture while on appropriate medications, consideration should be given to the use of denosumab or teriparatide.

Vertebral Compression Fractures

Definition and Epidemiology

It is estimated that, worldwide, approximately 9 million fractures occur as a direct result of osteoporosis. The most common locations of these fragility fractures include the hip, distal radius, and vertebral body in the thoracic and lumbar spine. The lifetime risk of an osteoporotic fracture is between 30% and 40% in the developed world for women and about 10–15% for men. Osteoporotic fractures account for significant disability-adjusted life years (DALYs) lost. For perspective, when considering DALYs lost, osteoporotic fractures are more impactful than most cancers, rheumatoid arthritis, and hypertension.

Approximately 1.5 million vertebral compression fractures (VCFs) occur annually in the United States. The yearly incidence is 10.7 per 1000 women and 5.7 per 1000 men. The estimated annual cost of treating these injuries is $746 million. The most influential risk factor for a VCF is preexisting osteoporosis, but other issues contribute as well. These include conditions that increase the likelihood of a patient's fall, such as poor eyesight, dementia, and frailty.

Clinical Presentation

VCFs occur as a result of a flexion compression force. The clinical presentation is most often related to a fall, and patients typically complain of axial back pain with mechanical and positional components. A clear mechanism of injury is not always reported, however. These fractures present as anterior wedging with preservation of the posterior wall of the vertebral body and without retropulsion into the spinal canal. For these reasons neurologic function is generally preserved. The injury may be localized to a single level or multiple levels can fracture simultaneously (Fig. 8.1). In severe cases, patients may present with acute fractures in the setting of multiple old compression injuries or fractures in various stages of healing. In other settings, individuals may develop a new compression fracture within a previously injured vertebral body (a so-called acute on chronic compression fracture) or sustain a nonunion of previously unrecognized fracture. These types of injuries may be more difficult to differentiate and necessitate advanced imaging modalities such as CT or magnetic resonance imaging (MRI).

Presenting complaints are generally consistent with axial back at the injured level and reproducible tenderness to palpation on physical exam. Over time, multiple VCF may be acquired, leading to kyphotic posture, loss of height, and chronic back pain as well as limited mobility.

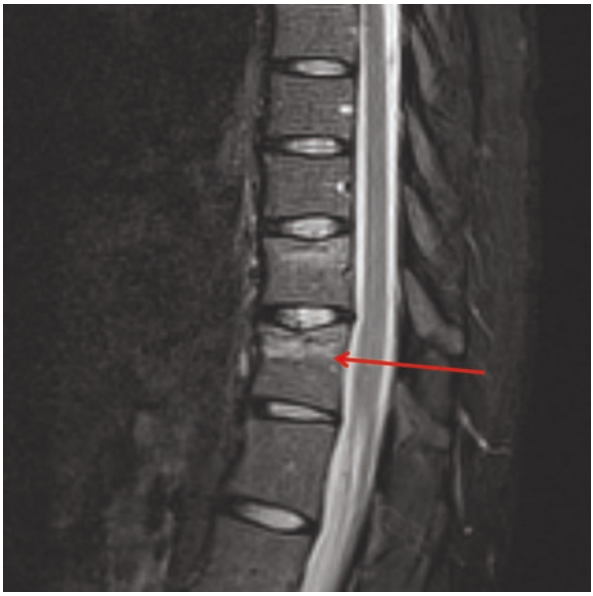

Fig. 8.1 Sagittal reconstruction MRI image demonstrating an acute compression fracture at T12 (red arrow) in a patient following a ground-level fall. An adjacent segment compression injury is also present at T11

Differential Diagnosis

The differential diagnosis for VCFs may include advanced degenerative changes or spinal deformity, elder abuse, infection, and malignancy.

Diagnostic Testing

Plain radiographs, including high-quality anteroposterior and lateral views, often can not only demonstrate the fracture but also give a general sense of the bone quality. CT scans can show more detail and bony architecture, but both CT and plain films are unreliable when it comes to determining the acuity of the VCF. MRI is most helpful in this regard and can also provide useful information on the status of the neural elements and integrity of the spinal canal. MRI may be used to detect the presence of new acute injury within a previously existing compression deformity. In the setting where multiple vertebral bodies are fractured and one is being considered for an intervention, determining the acuity of the symptomatic level can be critical for appropriate care. CT and MR imaging are also helpful when ruling out underlying malignancy or infection.

Nuclear medicine scans can be useful in determining the acuity of a fracture and in ruling out underlying malignancy. The specificity can be vastly improved with use of single photon emission computed tomography (SPECT) technology. Nuclear medicine studies are not considered part of the standard evaluation of patients who

are otherwise not at risk for a malignant process and where CT or MRI studies are conclusive regarding a diagnosis. Establishing an underlying diagnosis of osteoporosis in the setting of previously identified VCF can be achieved using DEXA imaging, as outlined above.

Nonoperative Management

Nonoperative treatment is the preferred course of care for most VCFs. Immediate treatment centers around analgesia and activity modification. Immobility and bed rest should be minimized as they predispose patients to the development of urinary tract infections, bedsores, deep vein thrombosis, pneumonia, and deconditioning. When considering analgesics, care must be taken to balance the need for pain control with the risk of delirium, impaired sensorium, and further falls. Nonsteroidal medications should be the first line of treatment unless they are contraindicated. A short course of narcotic medications can be considered in those who fail management with nonsteroidals. If appropriate, bisphosphonates and calcitonin that are begun in the acute post-injury period have been found to have an analgesic effect.

Bracing can be considered as an adjunct for pain control. The use of a brace may provide some stability to the fractured segment and off-load painful paraspinal musculature. The use of a brace has not been shown to prevent the development of kyphosis or accelerate the healing process. If utilized, bracing is typically trialed for a period of 4–6 weeks, or until the patient does not have substantial fracture-related pain while not wearing the brace. The patient should be gradually weaned from the brace as they slowly return to full activities. In this time period, physical therapy can also serve as a useful adjunct to maintain mobility, improve core strength, and provide additional pain relief modalities.

Indications for Operative Management

Operative intervention is used sparingly for VCFs. Open surgical procedures are reserved for cases of neurologic compromise, frank instability in the setting of a VCF, or severe deformity. Poor bone quality which contributed to the development of the VCFs is a complicating factor for open spinal reconstruction with instrumentation. There is, however, a more widely accepted role for cement augmentation (Fig. 8.2) which is typically performed percutaneously with image guidance. Patients with radiographically confirmed acute compression fractures (generally within 90 days of symptom onset) may be considered for a cement augmentation procedure if they have severe pain that has proven refractory to conservative measures, including the use of a back brace or in patients for whom bracing is contraindicated. Several studies have reported enhanced pain relief, improved quality of life, and reduced mortality in patients receiving cement augmentation as compared to

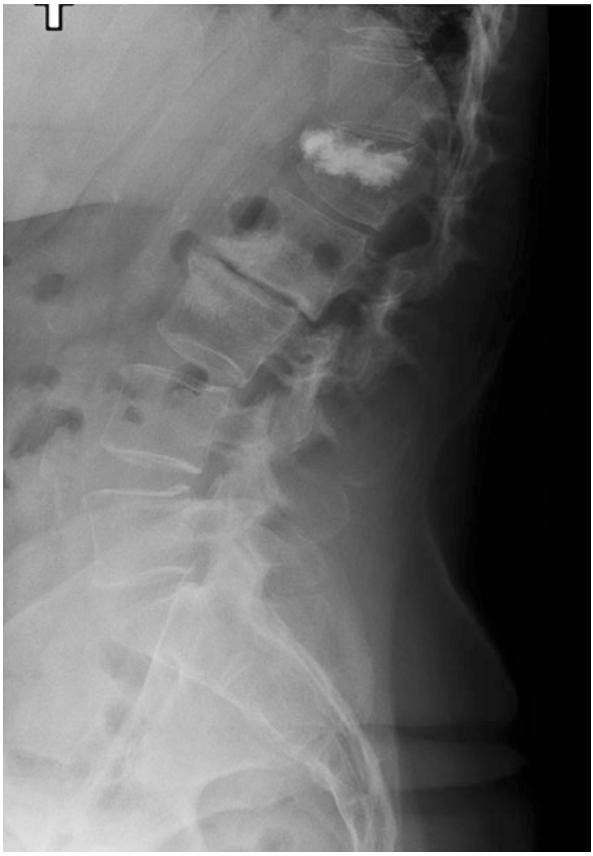

Fig. 8.2 Lateral plain film radiograph of a patient with a compression fracture at L1 that was treated using a cement augmentation procedure

those managed completely nonoperatively. One retrospective study in particular reported that the life expectancy of patients treated with cement augmentation was 85% longer as compared to those who received no such intervention. The results of this investigation have not been substantively replicated in other contexts.

Vertebroplasty

Vertebroplasty was originally developed in 1984 for the treatment of painful spinal hemangiomas and spinal fractures secondary to malignancy. The technique uses bone cement, [polymethylmethacrylate (PMMA)], which is injected through cannulae into the vertebral body defect. Image guidance is used to place the cannulae within the fractured vertebral body, as well as to monitor for cement extrusion. The imaging used can be fluoroscopic or CT guided. The cement interdigitates with the trabeculae of the bone and is allowed to cure, thus stabilizing the fracture.

Kyphoplasty

Kyphoplasty, a variation of the vertebroplasty procedure using inflatable bone tamps, was developed in 1996. The concept is similar to vertebroplasty, as both rely on percutaneous working cannulae placed into the fracture site. Kyphoplasty, however, relies on the use of a balloon that is inflated creating a void into which the cement is placed under low pressure (Fig. 8.3).

Both procedures have very similar safety profiles, and many of the perceived advantages of kyphoplasty, including restoration of vertebral body height and

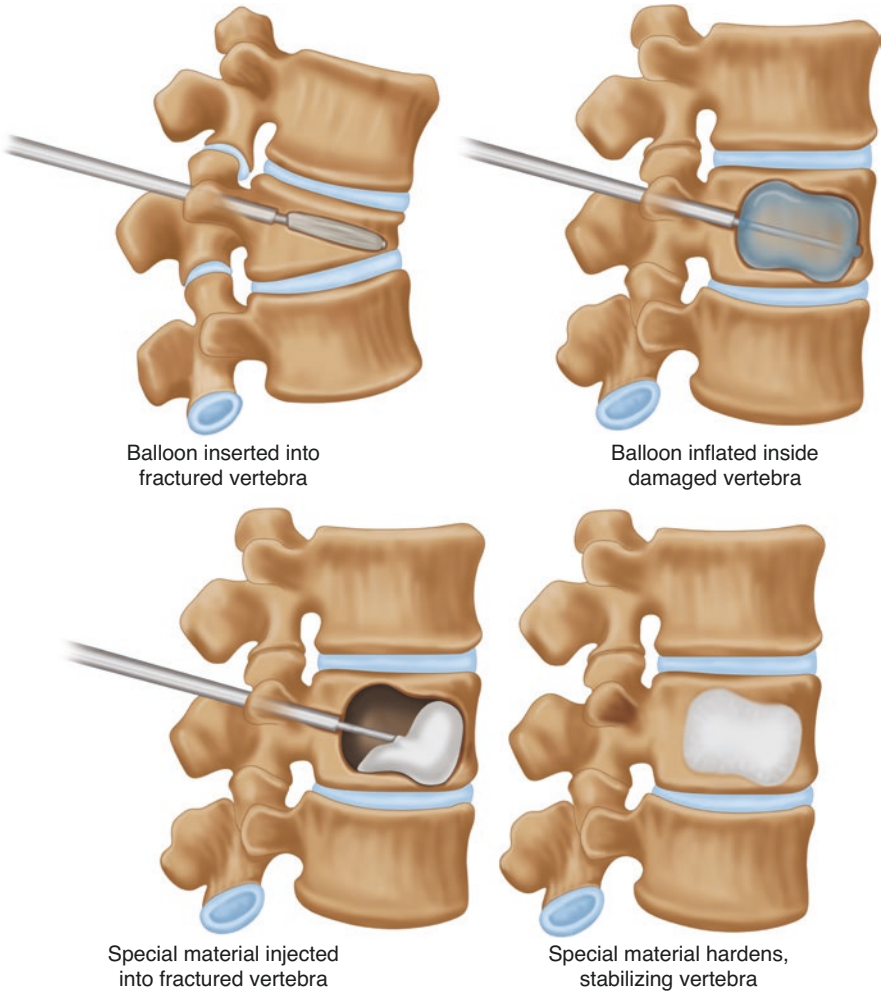

Fig. 8.3 Schematic of the kyphoplasty procedure. A balloon tamp is introduced into the fractured vertebral body and inflated. The balloon is then removed and the resultant cavity is backfilled with bone cement, inserted under low pressure

correction of focal kyphosis at the fracture site, have not been reliably demonstrated in larger studies. Kyphoplasty is the more expensive procedure (as much as ten times the cost of vertebroplasty) but also the only one of the two to have level I evidence supporting its use. Risks are mainly from cement extravasation and include neurologic compromise as well as cement or fat emboli traveling to the lungs. Historically, vertebroplasty was performed by interventional radiologists in imaging suites using local anesthetics, while kyphoplasty was performed by spine surgeons in operating rooms under general anesthesia.

Expected Outcomes

Cement augmentation has a role in patients with intractable pain. Good to excellent results, measured by degree of pain relief, can be expected in the range of 75–100% for both vertebroplasty and kyphoplasty, as well as nonoperative management. Cement augmentation procedures have not been shown to expedite the rehabilitation process, decrease the likelihood of kyphosis, or reduce the potential for future fractures. Regardless of the choice of management, most patients are able to return to pre-injury levels of function by 12 weeks following fracture. Patients with such injuries are at elevated risk for future fractures, and the most effective means of minimizing the potential for subsequent VCFs is through the use of appropriate osteoporosis medication and maintenance of physical activity. Even after healing a VCF, patients are more likely to have residual issues with back pain. Patients also may develop postfracture sequelae, including chronic pain and/or post-traumatic kyphosis (PTK) at the fracture site (Fig. 8.4). VCFs at the thoracolumbar junction have the highest likelihood of progressing to PTK. PTK can be very disabling, and some individuals may require open spinal osteotomy and instrumented reconstruction to correct a severe kyphosis (Table 8.1).

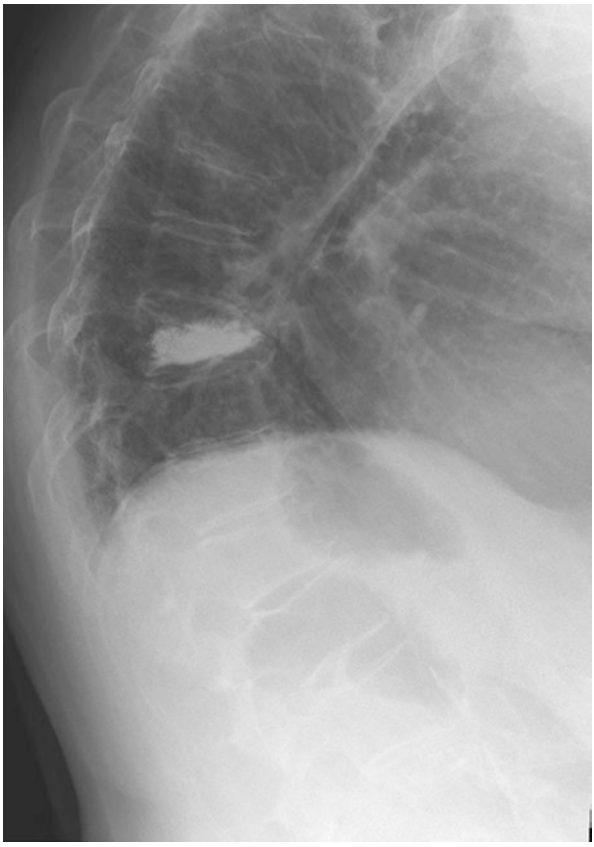

Fig. 8.4 Lateral plain film radiograph of a patient who developed post-traumatic kyphosis in a patient with a compression fracture at the thoracolumbar junction. The post-traumatic deformity developed, despite the patient having been treated with a cement augmentation procedure

Table 8.1 Summary of osteoporosis and vertebral compression fractures with a synopsis of their presentation, diagnostic testing, and suggested management strategies

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Osteoporosis	Clinically silent unless screened for the condition or insufficiency/ fragility fractures develop May be diagnosed following a sentinel fragility fracture (e.g., hip, wrist, or spine)	DEXA	Bisphosphonates, calcitonin, denosumab, teriparatide PT to improve core strengthening, posture, activity tolerance, joint mobility and function "Fall proofing" living space	Not applicable
Vertebral compression fracture	Focal pain within the thoracic or lumbar spine Does not always present in the context of antecedent trauma or injury, especially in those with more severe osteoporosis	Plain film radiographs CT MRI – determine fracture acuity	Rest, NSAIDS PT Bracing	Cement augmentation if pain is refractory to conservative care Open reconstruction in event of neurologic compromise, instability, or post-traumatic kyphosis

DEXA dual energy X-ray absorptiometry, *PT* physical therapy, *CT* computed tomography, *MRI* magnetic resonance imaging, *NSAIDs* nonsteroidal anti-inflammatory drugs

Suggested Reading

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Part III
The Hip

Chapter 9
Hip Soft Tissue Injuries

Cheri A. Blauwet and David M. Robinson

Abbreviations

AP	Anteroposterior
FABER	Flexion, abduction, and external rotation
GTPS	Greater trochanteric pain syndrome
MRI	Magnetic resonance imaging
NSAIDs	Nonsteroidal anti-inflammatory drugs
PT	Physical therapy
U/S	Ultrasound

Introduction

Soft tissue injuries involving extra-articular regions of the hip have the potential to cause significant functional morbidity and reduction in quality of life. Additionally, given the complexity of anatomy in the hip and lumbopelvic region, these entities may be difficult to differentiate and diagnose due to commonly overlapping pain patterns. Here we describe the most common soft tissue disorders involving the hip, categorizing each diagnosis as those that present as anterior, lateral, and posterior hip pain. By doing so, we aim to aid clinicians in considering a differential and expeditiously determining the correct diagnosis and treatment approach. It is important to note that the majority of hip soft tissue injuries can improve, or resolve, with appropriate rehabilitation and nonoperative treatment strategies.

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A. J. Schoenfeld et al. (eds.), *Principles of Orthopedic Practice for Primary Care Providers*, https://doi.org/10.1007/978-3-030-74625-4_9

Anterior Hip/Groin Disorders

Osteitis Pubis

Definition and Epidemiology

Osteitis pubis is a noninfectious inflammatory process involving the symphysis pubis, a nonsynovial, nonvascular joint composed of fibrocartilage. Muscle imbalances between the rectus abdominis and adductors are thought to disrupt the force distribution across the symphysis, thereby altering the joint's biomechanics and predisposing it to injury. Primary osteitis pubis is caused by repetitive microtrauma alone or in conjunction with opposing shearing forces across the pubic symphysis, especially with repetitive movements associated with sports that involve kicking (e.g., soccer) or repetitive hip abduction/adduction activities. Concurrent femoroacetabular impingement may be a risk factor through increased compensatory movements across the symphysis pubis. Many cases of osteitis pubis are secondary, however, and can occur during or after pregnancy or as a sequela of infection or trauma.

Clinical Presentation

Osteitis pubis typically presents as midline groin pain with or without radiation to the medial or anterior thigh or abdomen. Primary osteitis may be aggravated with activity and relieved by rest. Similar to other causes of hip or pelvic pain, osteitis pubis can be accompanied by a waddling gait or limp. Patients may describe pain provocation with rapid hip flexion from an extended position (e.g., kicking or hurdles). On physical examination, patients will have point tenderness to palpation directly over the symphysis pubis. Pain may also be elicited with passive hip internal rotation and/or active hip adduction. A comprehensive hip examination is recommended.

Differential Diagnosis

Other causes of groin pain that can present similarly to osteitis pubis include inguinal hernia, pubic rami stress fracture, intra-articular hip disease, genitourinary disease, osteomyelitis, and athletic pubalgia.

Diagnostic Testing

The diagnosis of osteitis pubis is often determined by history and physical examination alone. Gradual onset of midline anterior pelvic pain and pubic symphysis tenderness or pain with resisted adductor testing are characteristic of osteitis pubis.

Plain radiographs are often obtained when the etiology of pain is unclear or if symptoms persist despite conservative treatment. The preferred image is an anteroposterior (AP) pelvis film. Positive findings are usually not apparent until at least 4 weeks after the onset of symptoms and include subchondral erosive change, joint irregularity, and sclerosis. Of note, these findings may also be present among asymptomatic individuals. If pelvic instability is suspected (e.g., difficulty walking, waddling gait), flamingo stress views should be obtained which can reveal pathognomonic findings (>2 mm of vertical displacement or >7 mm of symphysis widening). Ultrasound may also be used to rule out inguinal hernia as well as to visualize dynamic widening at the pubic symphysis.

If the diagnosis is still in question after examination and plain film imaging, an MRI can be obtained and may reveal high signal intensity within the pubic symphysis or periarticular subchondral edema. With chronic disease, subchondral sclerosis and osteophytes may be seen. Radionuclide scanning (e.g., bone scan) should be reserved for patients in whom MRI and/or ultrasound are equivocal.

Nonoperative Management

Management is initially conservative, consisting of relative rest from provocative activities, ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and PT. Core and lumbopelvic strengthening, adductor stretching, and balance control are key components of the rehabilitation program. In cases of concomitant pelvic floor dysfunction, pelvic floor therapy may also be considered. In recalcitrant cases, pubic symphyseal corticosteroid injection or an oral prednisone taper may be trialed.

Indications for Operative Management

Depending on the degree of instability and dysfunction, various surgical procedures have been described, ranging from simple debridement to symphyseal joint fusion. The majority of surgical interventions are considered salvage procedures with limited proven efficacy and are solely reserved for the most recalcitrant cases.

Expected Outcomes

Recovery is generally expected with conservative treatment. Several weeks to months may elapse, however, before symptoms completely resolve. Sports participation is permissible as long as symptoms are tolerable and there is no evidence of instability (Table 9.1).

Table 9.1 Summary of soft tissue disorders of the hip with synopsis of presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Osteitis pubis	Insidious midline groin pain Tender to palpation over pubic symphysis	Primarily clinical MRI—periarticular edema at symphysis pubis	Rest, ice, NSAIDs PT Image-guided injection	Rarely indicated Pubic symphysis debridement; symphyseal joint fusion
Athletic pubalgia	Groin pain over pubic tubercle Negative tests for true hernia	Primarily clinical MRI—increased signal intensity at anteroinferior pubic ramus	Rest, ice, NSAIDs PT Image-guided injection	Hernia repair if indicated Repair of abdominal wall in cases of breach of rectus abdominis aponeurosis
Adductor strain	Acute onset groin pain Tenderness to palpation of injured myotendinous region	Primarily clinical U/S—defect of muscle or tendon, associated hematoma MRI—obtain if tendon avulsion or complex injury is suspected	Protected weight-bearing, rest, ice, NSAIDs PT	Reserved for complete avulsions of adductor insertion
Iliopsoas disorders	Insidious onset anterior hip or groin pain Pain with activities requiring hip flexion or adduction	X-ray (AP pelvis and hip) U/S	PT U/S-guided steroid injection	Endoscopic release or lengthening of iliopsoas tendon
Greater trochanteric pain syndrome (GTPS)	Slow-onset dull pain at lateral hip Point tenderness over greater trochanter GT	Primarily clinical MRI or U/S—gluteus minimus/medius tendinopathy or tear, associated bursitis	Activity modification, ITB stretching PT U/S-guided steroid injection	Endoscopic repair ITB release or lengthening

Table 9.1 (continued)

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Hip pointer	Acute, traumatic onset of pain at iliac crest Localized tenderness over iliac crest	X-ray (AP pelvis)—rule out fracture MRI—evaluate for bone or soft tissue edema	Protected weight-bearing, rest, ice, NSAIDs PT	None
Ischiofemoral impingement	Slow-onset of deep buttock pain +/− radiation distal posterior leg Pain typically provoked with hip extension/adduction/external rotation *or* hip flexion/internal rotation	MRI—edema within quadrates femoris muscle U/S—dynamic evaluation of quadrates femoris impingement with hip ER/IR	PT U/S-guided injection into ischiofemoral space	None

PT physical therapy, *U/S* ultrasound, *MRI* magnetic resonance imaging, *NSAIDs* nonsteroidal anti-inflammatory drugs

Athletic Pubalgia

Definition and Epidemiology

Athletic pubalgia is a clinical syndrome of groin pain without demonstrable inguinal hernia that is usually encountered in high-level athletes. It is more common in men than women and typically seen in sports that require repetitive cutting such as ice hockey, soccer, football, and rugby.

Athletic pubalgia results from repetitive trauma and/or loading of the pelvic stabilizers, commonly involving the confluence of the rectus abdominis insertion and origin of the adductor longus on the pubic tubercle (Fig. 9.1). Muscle imbalance between strong proximal thigh muscles and relatively weaker lower abdominal muscles creates a shearing force across the hemipelvis and is thought to play a pivotal role in the development of this overuse injury. Athletic pubalgia is an overarching term describing a range of pathology in this region, often including distal rectus abdominis strain, partial tear or avulsion, adductor longus tendinopathy, enthesopathy, or partial tear, or a combination of these entities.

Clinical Presentation

Symptoms of athletic pubalgia typically develop in an insidious fashion without a sudden or traumatic onset of pain. Groin pain just lateral to midline is the

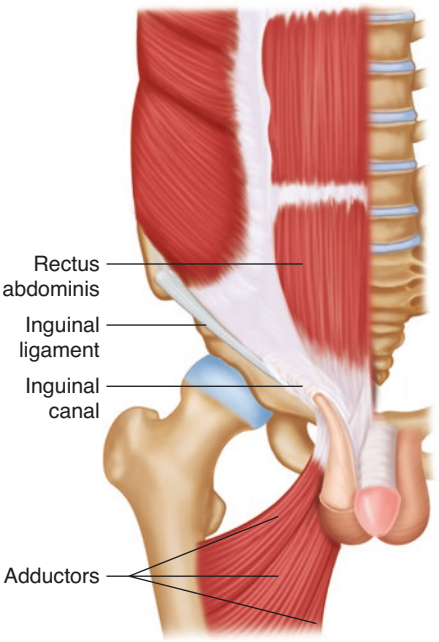

Fig. 9.1 The pertinent anatomy involved in cases of athletic pubalgia. Pathology typically occurs in the region of the rectus abdominis and proximal adductor longus aponeurosis. Although the inguinal canal is located just adjacent to this, athletic pubalgia is a musculoskeletal condition that typically occurs in the absence of a true inguinal hernia

predominant symptom. Pain may radiate toward the inner thigh, perineum, rectus musculature, or the testicles in approximately 30% of men due to entrapment of the ilioinguinal, iliohypogastric, or genitofemoral nerves. Patients may become symptomatic with sports activity, coughing, or Valsalva maneuvers.

In contrast to cases of true inguinal hernia, physical examination of the groin typically fails to detect a bulge or the sensation of an impulse with coughing or straining. There may be tenderness to palpation of the pubic tubercle just lateral to midline over the rectus/adductor aponeurosis. With acute injuries, there may be associated swelling. Patients should be tested for pain with resisted hip flexion, hip adduction at varying degrees of hip flexion, and following abdominal muscle contraction.

Differential Diagnosis

The differential diagnosis for athletic pubalgia is similar to that of osteitis pubis, as noted above. Additionally, it important to note that athletic pubalgia may coexist with intra-articular hip pathology in athletes, such as femoroacetabular impingement or labral tear.

Diagnostic Testing

The diagnosis of athletic pubalgia is largely clinical. Plain radiographs may be helpful for differentiating between a true osteitis pubis (with radiographic findings as

noted above) versus isolated soft tissue injury, as seen in athletic pubalgia. MRI can be helpful in the diagnosis of athletic pubalgia; however, findings are often quite subtle and require a specific athletic pubalgia protocol to improve sensitivity. In positive cases, findings may include increased signal intensity on T2-weighted imaging involving the anteroinferior aspect of the pubic symphysis (also known as a "secondary cleft sign"). Other findings may include osteitis pubis, tenoperiosteal disruption or frank tear of the adductor aponeurosis, and marrow edema at the pubic tubercle.

Ultrasound offers the best method for diagnosing abnormalities within the superficial inguinal canal including visualization of occult hernias. Ultrasound is also useful in evaluation of pathology at the rectus abdominis/adductor aponeurosis. Sonopalpation can help to localize the pathology.

Nonoperative Management

Generally, the acute management of groin pain suspected to be athletic pubalgia consists of conservative care, rest from offending activities, ice, trial of anti-inflammatory medications, and PT. Gradual restoration of flexibility and core strengthening and stability is integral to functional recovery. In recalcitrant cases unresponsive to conservative management, diagnostic/therapeutic injections (typically guided by ultrasound imaging) can be helpful.

Indications for Operative Management

When pain continues despite appropriate conservative care, and the athlete cannot return to their previous activity, surgery may be considered. Surgical treatment of athletic pubalgia consists of both open and laparoscopic approaches and depends upon the underlying etiology. True hernias are repaired with or without the use of mesh. Other surgical procedures include repair of the adductor/rectus abdominis aponeurotic plate, adductor longus tenotomy or release, and decompression of the genital branch of the genitofemoral nerve. When concurrent femoroacetabular impingement exists, this may be addressed concomitantly.

Expected Outcomes

Prognosis is highly favorable, although some may experience recalcitrant symptoms. If the symptoms are not severe, continued sports participation may be allowed. Surgical repair, when indicated, has been shown to be successful in a majority of cases. Generally, the athlete is allowed to return to play in 2–6 weeks with a laparoscopic repair and 1–6 months following open repair.

Acute Adductor Strains

Definition and Epidemiology

Adductor strains are a common cause of acute groin pain, particularly in athletes and active individuals. These injuries often occur in association with sports that involve kicking or rapid changes of direction with pivoting, such as ice hockey, soccer, tennis, basketball, and squash, wherein the adductor group sustains repetitive and rapid eccentric contraction.

Adductor injuries are classified according to anatomy and severity. The adductor longus is most commonly involved, secondary to its relative length, greater tendon to muscle ratio, and weaker attachment at the pubic crest. Grade 1 injuries involve a low-grade tear of a small number of muscle and/or tendon fibers, causing pain but minimal loss of strength or motion. Grade 2 injuries constitute high-grade partial tears, causing pain, swelling, and decreased motion and strength, but not complete loss of function. Grade 3 injuries involve complete disruption of the muscle-tendon unit with loss of muscle function.

Clinical Presentation

The patient should be asked to describe the onset of pain (acute or chronic), its severity, and any radiating features. Features may include tenderness to palpation of the injured myotendinous region, bruising, and, in the case of grade 3 injuries, a palpable defect. Acute injuries are more often myotendinous, whereas chronic injuries localize to tendinous insertions. The squeeze test produces pain with resisted adduction at 45° of hip flexion, often with associated loss of muscle power.

Differential Diagnosis

Alternative diagnoses to be considered include athletic pubalgia, osteitis pubis, ilioinguinal/obturator nerve entrapment, stress fractures of the pelvis and femoral neck, intrinsic hip pathology (femoroacetabular impingement, labral tear, chondral lesion, osteoarthritis), and referred pain from the lumbar spine. Many of these pathologies may refer pain to the adductor region; however, palpation and provocative testing of the adductors are less likely to reproduce symptoms.

Diagnostic Testing

In many cases, the diagnosis of adductor strain is straightforward and based solely on history and physical examination. In such cases, diagnostic imaging is not necessary. When diagnosis is less clear or there are findings concerning for a grade 3 injury, imaging studies are obtained. Pelvis and hip radiographs can be

used to exclude other conditions such as osteitis pubis or femoroacetabular impingement. Anteroposterior (AP) views of the pelvis are recommended at a minimum. Musculoskeletal ultrasound can further visualize the adductor myotendinous structure, bony attachment sites, and associated nerves. Ultrasound is useful for determining the exact location and extent of injury as well as for monitoring recovery.

Obtaining an MRI is generally not necessary for evaluating most adductor injuries, especially given the high sensitivity and low cost of ultrasound. Indications for MRI include suspected tendon avulsion, complex injuries involving more than one structure, injuries that fail to improve despite compliance with an appropriate rehabilitation program, and patients with chronic or recurrent groin pain in which the diagnosis remains in question.

Nonoperative Management

Although the recommended treatment course for adductor strain is dictated by the severity of symptoms, this generally consists of a period of protected weight-bearing and rest from provocative activities. In the acute phase, ice and compression may be utilized as needed to reduce associated hemorrhage and edema. Complete immobilization should be avoided as this promotes muscle stiffness and scarring. The use of anti-inflammatory analgesic medications is controversial but is typically helpful in the acute phase. It is also appropriate to begin early mobilization with PT. Rehabilitation should be focused on balancing muscle length and strength. Isometric exercises are utilized initially for pain management, progressing to eccentric exercises to strengthen the myotendinous region and facilitate tissue healing. Core and lumbopelvic strengthening involving the hip abductors, lateral hip rotators, and hamstrings should also be performed. The modified Hölmich protocol has been described with success. The patient may resume activity when both range of motion and muscle strength are fully restored.

Indications for Operative Management

There is no high-level evidence that surgical repair of grade 3 adductor strains yields superior outcomes to nonsurgical management. Surgery is typically reserved for complete avulsions at the adductor longus origin. Open repair with suture anchors is the surgical treatment of choice.

Expected Outcomes

Prognosis varies depending upon the extent of injury and patient activity. In general, grade 1 adductor strains require between 10 and 21 days before the patient can return to sports. Grade 2 injuries require 4–6 weeks, and grade 3 tears or avulsions may require 2–3 months before complete recovery.

Iliopsoas Muscle-Tendon Complex Disorders

Definition and Epidemiology

The iliopsoas muscle-tendon complex is directly anterior to the hip joint and consists of three muscles—psoas major, psoas minor, and iliacus. Iliopsoas pathology is often overlooked as a cause of hip pain but includes a number of clinically significant syndromes including iliopsoas tendinitis or tendinopathy, iliopsoas bursitis, and internal snapping hip syndrome (coxa saltans).

Iliopsoas tendinitis and/or tendinopathy affects young adults more commonly, with a slight female predominance. Acute injuries typically involve an eccentric contraction of the iliopsoas muscle but also may be due to direct trauma. Overuse injury is more likely to lead to iliopsoas tendinopathy and may occur in activities involving repeated hip flexion or external rotation of the thigh, including dancing, rowing, running (particularly uphill), track and field, soccer, and gymnastics.

Internal snapping hip is most commonly caused by the iliopsoas tendon sliding over the femoral head, the iliopectineal eminence, or internally over the iliacus muscle when the hip is ranged from flexion/external rotation to extension/internal rotation, resulting in a palpable and often audible snap in the region of the groin. Like iliopsoas tendinopathy, it is common in sports that require repetitive hip movements.

Clinical Presentation

Patients often present with complaints of an insidious onset of anterior hip or groin pain. Initially, the patient may note pain with specific sports-related activities that require forceful hip flexion or adduction, such as jogging, running, or kicking. Pain with simple activities such as putting on socks and shoes, rising from a seated position, and walking upstairs or on inclines may also be reported. Runners often describe anterior groin pain when trying to lengthen their stride during speed training or with uphill running.

On physical examination, there may be tenderness to palpation along the course of the iliopsoas myotendinous junction just anterior to the hip joint. With the patient supine and with their heels raised off the table to approximately 15°, tenderness can be assessed by palpating the psoas muscle below the lateral inguinal ligament at the femoral triangle. Pain is exacerbated by iliopsoas activation. Hip range of motion may also be painful as will a flexion, abduction, and external rotation (FABER) test. When the patient's hip is ranged from the FABER position to extension and neutral, a snap may be felt over the groin, identifying coxa saltans. Secondary dysfunction of the hip flexor muscle-tendon complex due to an underlying intra-articular disorder commonly occurs.

Differential Diagnosis

Other causes of anterior hip or groin pain include intra-articular pathology (e.g., labral tear, osteoarthritis), rectus femoris injury, adductor injury, athletic pubalgia, osteitis pubis, and occult hernia.

Diagnostic Testing

Plain radiographs of the pelvis and hip are initially helpful to evaluate for underlying intra-articular hip pathology or other osseous abnormalities. Ultrasonography has been used more frequently as a noninvasive diagnostic adjunct in the diagnosis of iliopsoas muscle-tendon injuries. Demonstration of a thickened or irregular iliopsoas tendon with or without distension of the iliopsoas bursa is a typical associated finding. As the iliopsoas bursa is frequently contiguous with the hip joint, the presence of bursitis may be indicative of underlying intra-articular pathology. In cases of internal snapping hip, dynamic ultrasound can assess for visible and palpable snapping of the iliopsoas tendon.

In refractory cases, MRI can prove useful by allowing for concomitant evaluation of the iliopsoas complex as well as the hip joint itself. Given overlapping clinical presentations, advanced imaging may be helpful in determining the more significant pain generator. In cases of iliopsoas tendinitis or tendinopathy, T2-weighted images may demonstrate increased signal intensity either in a peritendinous distribution (tendinitis) or within the tendon itself (tendinopathy). In cases of acute myotendinous injury, both T1- and T2-weighted images may depict a region of high signal intensity.

Nonoperative Management

Iliopsoas injuries are typically managed conservatively. Rehabilitation should involve progressive iliopsoas loading complemented by core and lumbopelvic stabilization. Soft tissue mobilization and hip flexor stretching to restore the muscle-tendon unit to its full length should also be emphasized to promote biomechanical optimization. Joint mobilization of the hip and lumbosacral region may be considered. For recalcitrant cases, judicious use of ultrasound-guided corticosteroid injection to the iliopsoas tendon sheath and/or iliopsoas bursa may be of both therapeutic and diagnostic value.

Indications for Operative Management

In refractory cases, endoscopic release or lengthening of the iliopsoas tendon may be performed.

Expected Outcomes

Prognosis for recovery is excellent for iliopsoas muscle-tendon complex disorders. The presence of tendinopathy may prolong recovery; however, even this condition generally responds to conservative care. For cases requiring surgery, return to sports can be anticipated in 3–4 months postoperatively.

Lateral Hip Disorders

Greater Trochanteric Pain Syndrome

Definition and Epidemiology

Greater trochanteric pain syndrome (GTPS) is a clinical entity of lateral peritrochanteric pain involving several conditions such as tendinopathy of the gluteus medius and minimus at their insertion on the greater trochanter and external snapping hip. Involvement of regional bursae is variable and usually secondary to the tendinopathy. Typically, there are three main bursae in the trochanteric region: subgluteus medius, subgluteus minimus, and subgluteus maximus bursae (Fig. 9.2). The subgluteus maximus bursa is often implicated as the source of pain in greater trochanteric bursitis, which often accompanies the tendon pathology described above.

The gluteus medius and minimus muscles play a primary role in hip abduction and pelvic stabilization in walking, running, and single-leg stance. Weak hip abductors increase adduction during gait, leading to increased compression of the gluteus medius and minimus tendinous insertions. Similar to rotator cuff tendon pathology in the shoulder, involvement of the gluteus minimus and medius can range from

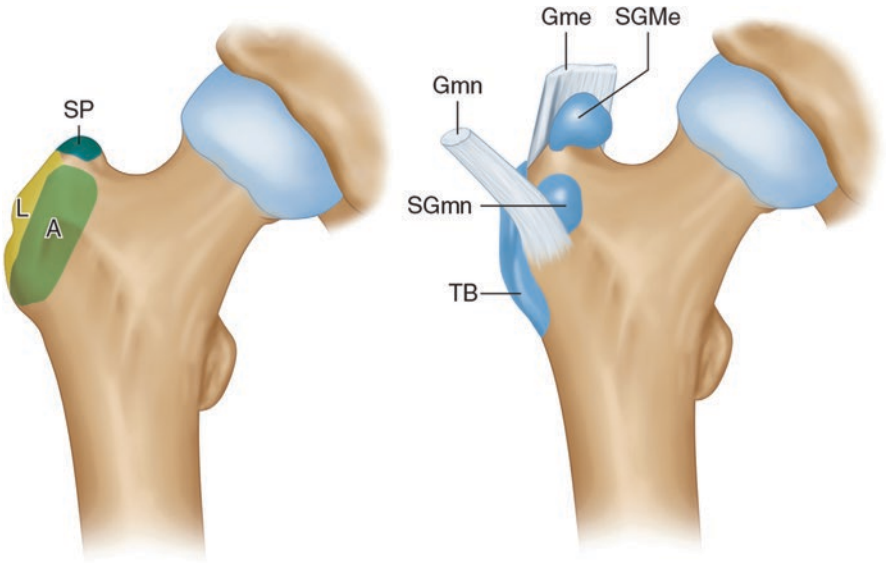

Fig. 9.2 The pertinent anatomy involved in cases of greater trochanteric pain syndrome. Note the complex anatomy of the greater trochanteric region of the lateral hip. The figure on the left depicts the anterior, lateral, and superoposterior facets of the greater trochanter. The figure on the right depicts the insertion of the gluteus minimus on the anterior facet and gluteus medius on the lateral and superoposterior facet of the greater trochanter, with the associated subgluteus minimus, subgluteus medius, and greater trochanteric bursa. (With permission from *Radsource—ProtonPACS.* http://radsource.us/gluteus-minimus-tear-trochanteric-bursitis/)

peritendinitis to full-thickness tears. Mechanically induced tissue failure is a key feature of GTPS.

External snapping hip refers to the posterior iliotibial band (ITB) snapping over the greater trochanter during activity. While sometimes asymptomatic, repetitive snapping can lead to a thickened ITB, bursal pathology, and pain. Tightness of the ITB is often implicated, and this may be intrinsic or secondary to overactivation of the tensor fascia latae compensating for weak gluteal muscles.

GTPS is common. Female-to-male incidence of GTPS is approximately 4:1. Age at presentation is most common in the fourth through sixth decades of life. Risk factors include female gender, hip abduction weakness, obesity, knee pain, iliotibial band tenderness, and low back pain. Other conditions associated with GTPS include scoliosis; leg-length discrepancy; intra-articular conditions of the hip, knee, and foot; and painful foot disorders such as plantar fasciitis.

Clinical Presentation

The key complaint in patients with GTPS is dull pain in the lateral hip and point tenderness over the greater trochanter. Pain may increase with running, ambulation, prolonged standing, climbing stairs, and direct pressure when lying on the painful side such as when sleeping. A useful clinical question asks patients to "point where the pain is." Patients with GTPS point to the lateral hip, whereas those with intra-articular hip disease generally point to the groin and the anteromedial thigh.

On physical examination, the patient should be tested for pain with single-leg stance held for 30 seconds, single-leg squat, and isolated hip resisted abduction from a side-lying position. ITB tightness can be accessed via the Ober test. Patients with more severe symptoms will also have weakness with a positive Trendelenburg sign during single-leg squat (Fig. 9.3). Tenderness to palpation of the greater trochanteric prominence is the key physical examination finding of GTPS.

Differential Diagnosis

A variety of other conditions can result in lateral hip pain and include intra-articular hip disorders, sacroiliac joint disease, referred pain from lumbar spine disorders, meralgia paresthetica, and piriformis syndrome.

Diagnostic Testing

Initial imaging should include plain radiographs to assess for bony involvement (e.g., hip degenerative changes) that may be an underlying exacerbant of symptoms. In chronic cases of GTPS secondary to gluteus minimus or gluteus medius tendinopathy, one may see enthesopathy and/or calcifications adjacent to the greater

Fig. 9.3 Patient with findings of mild Trendelenburg (contralateral pelvic tilt) upon left single-leg squat. This finding is common in cases of hip abductor (gluteus medius) weakness or tear

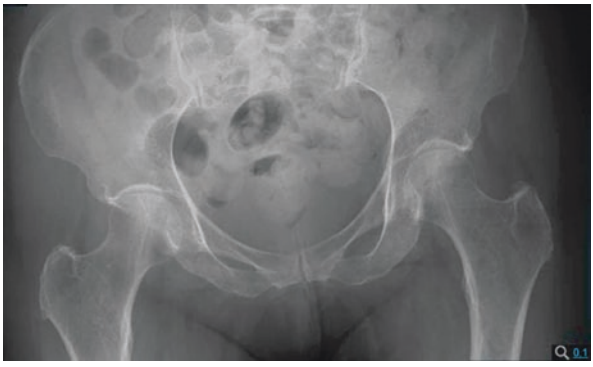

Fig. 9.4 Radiographic findings consistent with bilateral chronic gluteus minimus/medius tendinopathy with associated enthesopathy at the greater trochanter

trochanter (Fig. 9.4). MRI or ultrasound can be used to detect soft tissue pathology such as bursitis, tendinopathy, enthesopathy, and partial- or full-thickness tendon tears. Ultrasound can dynamically evaluate for external snapping hip. MRI can additionally be helpful in the evaluation of intra-articular hip disorders or bone stress injury involving the pelvis or femoral neck.

Nonoperative Management

GTPS is a self-limited condition in the majority of patients. Therefore, the goal of treatment is to relieve symptoms and prevent functional impairment. Supportive management includes activity modification, massage, stretching and tissue lengthening of the IT band, and strengthening exercises for the hip abductors, core, and lumbopelvic stabilizers. For patients whose symptoms do not improve, a single corticosteroid injection to the bursa (if identified) or peritendinous region of the gluteus minimus or medius can be trialed. It is recommended that this be performed under image guidance (typically ultrasound) in order to avoid intra-tendinous injection of steroid, which can result in further tendon degeneration and worsening of symptoms. More recently, shock wave therapy and platelet-rich plasma have been used with some success.

Indications for Operative Management

Surgery can be considered for patients with severe pain and functional impairment after 12 months of conservative management or those who have full-thickness tears of the gluteus medius or minimus resulting in lumbopelvic instability and severe Trendelenburg gait. If the gluteus medius tendon has a full-thickness tear, it can be repaired, most commonly via an endoscopic approach with debridement of degenerative tissue, curettage of the bone surface, reattachment of the tendon using bone anchors, and direct repair of the tendon. Occasionally, subgluteus maximus bursectomy or ITB lengthening/release is offered.

Expected Outcomes

GTPS, as stated earlier, is largely a self-limited condition with the majority of patients reporting 100% relief of symptoms with watchful waiting or completion of a high-quality rehabilitation program. Surgical intervention is rarely necessary. However, when performed, it has been found to be successful in recalcitrant cases of GTPS.

Hip Pointer (Iliac Crest Contusion)

Definition and Epidemiology

A hip pointer is a contusion to the iliac crest, often resulting from a direct blow sustained during sports activity or other trauma such as falls. Minimal overlying adipose tissue predisposes the iliac crest to contact injuries. Subsequent hematoma development may affect nearby musculature. This condition is most commonly seen in football, soccer, and ice hockey players.

Clinical Presentation

The patient will report a history of trauma with acute onset of pain. They may have severe pain directly over the iliac crest that worsens with movements that involve activation of the deep core and abdominal musculature such as with coughing, sneezing, or running. Hip abductor strength may be impaired. The condition may cause severe disability for a brief period of time, with associated swelling or ecchymosis. On physical examination, the patient should be assessed for associated regional muscle spasm. Lumbar spine and hip range of motion should be evaluated.

Differential Diagnosis

Other possible etiologies include iliac crest fracture or apophysitis, avulsion injuries, abdominal wall injury, or acute strain of the muscles overlying the iliac crest, namely, the gluteus medius, the gluteus minimus, and the tensor fasciae latae. Other non-musculoskeletal causes of abdominal pain or flank pain, such as renal calculi, should be considered.

Diagnostic Testing

An anteroposterior radiograph of the pelvis is helpful to evaluate for fracture or other bony abnormality. In more severe cases, ultrasound may be utilized to evaluate for a focal muscle defect and hematoma. MRI can be helpful in the assessment of either bony or soft tissue edema.

Nonoperative Management

Initial treatment should attempt to minimize swelling or hematoma formation with the application of ice and compression. Hematomas that are not adequately treated may later develop into myositis ossificans. In the acute phase, the affected lower extremity should be partially immobilized with crutches and protected weight-bearing. As symptoms improve, a gentle and gradual PT program should be implemented, initially focusing on range of motion followed by isometric strengthening. Ultimately the patient can be progressed to aggressive multidirectional strengthening and neuromuscular rehabilitation.

Expected Outcomes

Prognosis is generally excellent, with return to full activity dictated by sufficient resolution of pain. Often this occurs in 2–4 weeks. In the case of athletes returning

to play, repeated injury can be avoided via the application of protective padding to the region of the iliac crest.

Posterior Hip Disorders

Ischiofemoral Impingement

Definition and Epidemiology

Ischiofemoral impingement syndrome is defined by posterior hip pain related to impingement of soft tissues between the ischial tuberosity and lesser trochanter of the femur, primarily involving the quadratus femoris muscle. Narrowing of the ischiofemoral space may be positional, congenital, or acquired, such as in cases of prior hip arthroplasty, hypertrophy due to osteoarthritis, or fractures involving the lesser trochanter. The quadratus femoris muscle originates at the anterior portion of the ischial tuberosity and inserts on the posteromedial aspect of the proximal femur, and its main role is to assist in external rotation and adduction of the hip. Ischiofemoral impingement is more common in women and affects individuals of all ages.

Clinical Presentation

Patients with ischiofemoral impingement present with chronic load-dependent pain deep in the buttock, usually without a precipitating injury. Mechanical symptoms may be described during long-stride walking as the lesser trochanter forcefully bypasses the ischial tuberosity in more severe cases. Given the proximity of the quadrates femoris muscle to the sciatic nerve, the pain may radiate distally to the posterior thigh and leg. Bilateral involvement has been observed in 25–40% of patients. On physical examination, patients may report pain during hip range of motion, and symptoms may be reproduced by a combination of hip extension, adduction, and external rotation or with hip flexion and internal rotation.

Differential Diagnosis

The differential diagnosis for ischiofemoral impingement includes proximal hamstring tendinopathy or hamstring strain, lumbosacral radiculopathy, piriformis syndrome, or intra-articular hip pathology with posterior radiation to the buttock.

Diagnostic Testing

Plain radiographs of the pelvis and hip may be helpful to evaluate for other entities such as intra-articular hip pathology which may mimic ischiofemoral impingement. Thereafter, both ultrasound and MRI have utility in evaluating this difficult clinical entity. MRI may reveal edema within the quadratus femoris muscle, indicative of repetitive impingement and focal inflammation. Ischiofemoral space distances <15 mm have been suggested as diagnostic. Additionally, ultrasound may be useful in offering the ability for dynamic assessment, noting soft tissue impingement between the ischial tuberosity and lesser trochanter when the hip is brought into external rotation. With ultrasound assessment, side-to-side comparison is critical for determining whether any noted impingement might be pathologic versus a normal variant dependent on the patient's anatomy.

Nonoperative Management

Treatment of ischiofemoral impingement is nearly entirely conservative, and there is little indication for surgical intervention unless a true bony defect or structural abnormality is noted. Rehabilitation should be focused on optimizing hip biomechanics through a progressive core and lumbopelvic stabilization program emphasizing hip external rotator strengthening. Educate the patient on how to avoid movement patterns which may exacerbate symptoms such as long-stride walking. In refractory cases, ultrasound-guided injection into the ischiofemoral space (with care to avoid the sciatic nerve) may be helpful for both diagnostic and therapeutic purposes. Recalcitrant pain with obvious structural abnormalities may warrant surgical consultation for possible lesser trochanter resection.

Expected Outcomes

Treatment outcomes are typically quite favorable with the use of a multifaceted and comprehensive rehabilitation program, as outlined above.

Suggested Reading

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Chapter 10
Femoroacetabular Impingement, Labral Tears, and Hip Arthroscopy

Matthew J. Best and Scott D. Martin

Femoroacetabular Impingement and Labral Tears

Summary of Epidemiology

Femoroacetabular impingement (FAI) and resultant labral tears are increasingly recognized as a cause of pre-arthritic hip pain, particularly in younger athletes. Indeed, 22–55% of patients presenting with hip pain have a labral tear. Tearing of the labrum is most commonly attributed to FAI, as 95% of patients with FAI will have a labral tear. Conversely, 49–87% of patients with a labral tear have FAI. Rarely, labral tears are observed in the setting of acute trauma as they can occur with hip dislocation.

FAI is comprised of two different types of bony abnormalities which may be present in isolation, or in combination of one another: cam and pincer lesions (Fig. 10.1). Cam lesions refer to an asphericity of the femoral head and loss of the normal head-neck junction, while pincer lesions involve either overcoverage of the femoral head by the acetabulum or retroversion of the acetabulum. These deformities result in increased contact between the femoral head-neck junction and acetabular rim during flexion and internal rotation of the hip. Cam lesions cause shearing at the chondrolabral junction, and pincer lesions lead to direct contact between the rim and femoral neck compressing the labrum. This repetitive trauma to the labrum and loss of the labral seal is a proposed precursor to subsequent development of hip osteoarthritis; however, further studies are needed to definitively identify a causative relationship.

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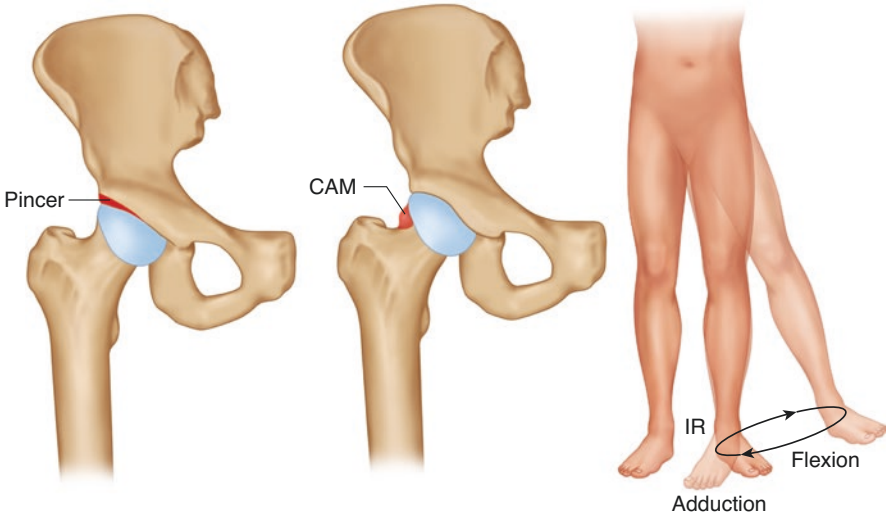

Fig. 10.1 Illustrations of the two bony abnormalities comprising FAI: cam and pincer lesions. Pincer lesions refer to overcoverage or acetabular retroversion. Cam lesions represent a prominence at the femoral head-neck junction

Labral tears and FAI are more commonly recognized in an athletic population as these patients experience symptoms when testing the extremes of motion and impact loading the hip. The underlying cause of FAI is largely genetic; however, activity level and increased hip loading during skeletal development may also contribute. The incidence of labral tears significantly increases with age, as even in an asymptomatic population, 69% of patients will have a labral tear on MRI by age 38. Additionally, in an older population, underlying arthritis may be the more contributory source of hip pain compared to labral tear. Therefore, correlating symptoms with physical exam and imaging is crucial in providing appropriate care.

Gender-specific differences do exist. Females present with worse functioning, while males often have larger tears and chondral damage. Additionally, males are more likely to have cam lesions than females. Patients often present in the fourth and fifth decade of life; however, the diagnosis is increasingly being recognized in a younger active population with severe impingement. Given the concern for progression to arthritis, these patients may be treated more aggressively than an older population. The diagnosis can be elusive, as patients may report symptoms for almost 2 years prior to diagnosis while seeing multiple providers. An awareness and understanding of the disease is essential for all providers who see patients with hip pain.

Clinical Presentation

Patients with labral tears often present with anterior hip or groin pain which can be exacerbated by activities, especially those involving hip flexion and internal rotation. Pain is often described as deep, and in a "C" distribution around the anterolateral aspect of the hip. Sporadic mechanical symptoms such as catching, popping, or locking are frequently reported. Symptoms often occur with pivoting activities or deep flexion, such as rising out of a low chair, and resolve quickly after offending activity is ceased. Complaints of continued achiness and sensitivity of the hip, however, are more suggestive of underlying arthritis or chondral damage. When obtaining the history, it is important to elicit symptoms that could be due to an alternate etiology. Radicular symptoms, such as numbness, tingling, and pain radiating distal to the knee, are far more suggestive of spine pathology.

On physical exam, the most sensitive test is pain with flexion, adduction, and internal rotation (FADIR test) while the patient is lying supine. This movement is most likely to cause anterior impingement, with compression of the labrum. Hip flexion may be limited to less than 90 degrees and internal rotation may be reduced to less than 10 degrees. While most tears are located anterosuperior on the acetabulum, posteroinferior labral tears can rarely occur. Symptoms specific to this can be elicited by abducting the patient's leg off the exam table while applying slight flexion and external rotation (FABER test).

Symptoms may be recreated by having the patient squat deeply. Observing the patient's gait can also be helpful, as labral tears rarely lead to an antalgic gait from pain in the gluteal or trochanteric region from abnormal mechanics as seen with lower spine and gluteal pathology. Gluteal tendinopathy can often be detected by a Trendelenburg sign where during the stance phase of gait, the contralateral hip will droop. In addition, the patient will have difficulty maintaining balance with one-legged stance on the affected leg.

Full assessment of the patient's range of motion, strength, and neurovascular status is important in ruling out alternate diagnoses.

Differential Diagnosis and Suggested Diagnostic Testing

Symptomatic labral tears can be difficult to diagnose and require careful consideration of the history, physical exam, and imaging. Other common diagnoses important to exclude are osteoarthritis, spine and lower back pathology, sciatica, abductor tendon tears, internal coxa saltans, and external coxa saltans. In females, symptoms can occasionally mimic gynecologic issues.

Imaging should begin with x-rays including AP pelvis and dedicated AP and lateral views of the affected hip. A Dunn lateral is easiest to view the femoral head-neck junction. A false profile view can also be obtained to view femoral head coverage for dysplasia. An AP of the pelvis is important to ensure the patient is not rotated

when the image is taken, as this can affect interpretation of acetabular coverage or retroversion. A pistol grip deformity or asphericity of the femoral head indicates a cam deformity (Fig. 10.2). A crossover sign, in which the anterior wall is observed crossing over more lateral to the posterior wall, is indicative of acetabular retroversion (Fig. 10.3). The lateral center-edge angle should be estimated, as an angle under 20° is considered dysplasia and under 25° is borderline that may require further work-up. The lateral center-edge angle is measured between a line superior from the center of the femoral head and a line from the center of the femoral head to the most lateral aspect of the sourcil. Coxa protrusio, in which the deepest aspect of the acetabulum crosses the ischioilial line, or coxa profunda, when the femoral head crosses this line, is also indicative of patients with overcoverage. The alpha angle is used to assess cam lesions. On the lateral view, a line is drawn through the center of the femoral head directly down the center of the femoral neck, and another line is drawn out lateral to where the femoral head-neck junction no longer follows the expected spherical contour. An alpha angle over 56° in males and 46° in females is consistent with a cam lesion. Radiographs should also be closely evaluated for signs of arthritis such as decreased joint space, osteophytes, subchondral cysts, and sclerosis.

Given the high prevalence of labral tears even in an asymptomatic population, MRI/MRA should be done with caution and only when there is high suspicion that a labral tear is the source of symptoms. While MRI and MRA are both highly sensitive for labral tears, in our opinion, MRA allows easier visualization (Fig. 10.4). Most labral tears are located anterosuperiorly, and the axial oblique may provide the

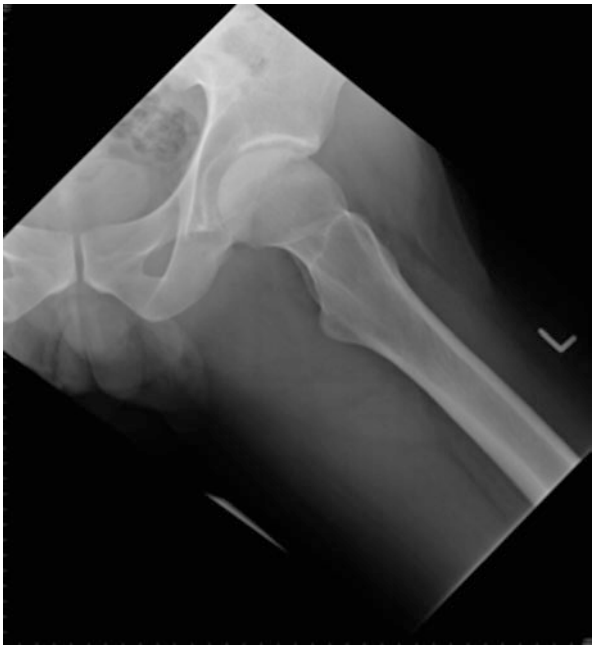

Fig. 10.2 Frog-leg lateral image of the left hip demonstrating a cam lesion

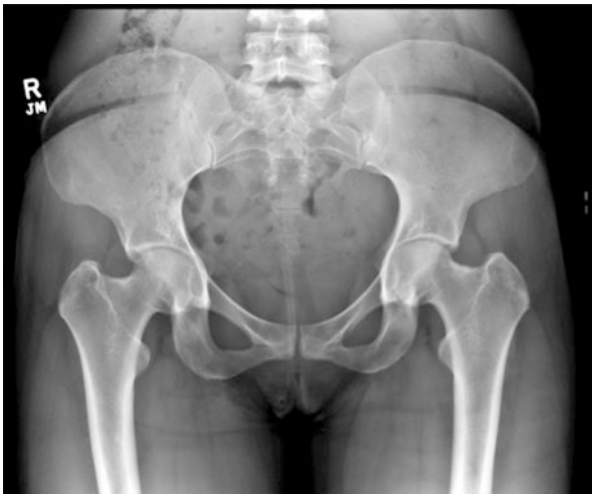

Fig. 10.3 AP pelvis in a patient with bilateral hip pain. Bilateral pincer lesions as identified by a crossover sign. Coxa profunda is also present

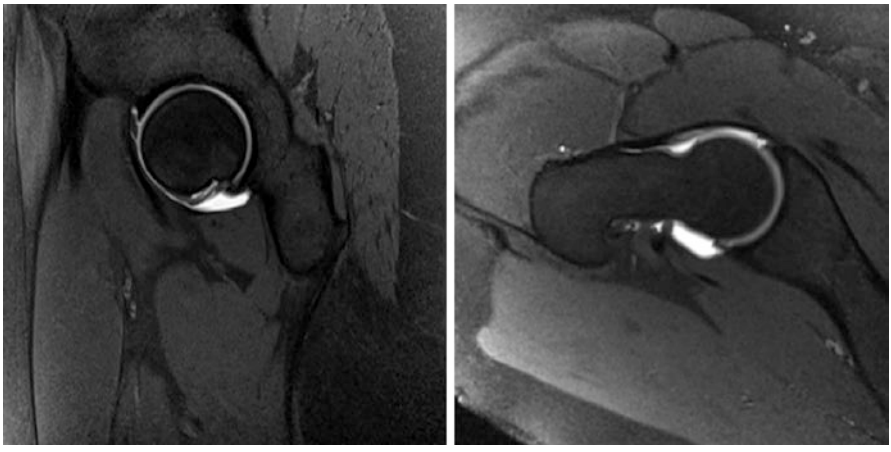

Fig. 10.4 An anterosuperior labral tear can be seen on the MRA sagittal (*left*) and axial oblique T1 FS (*right*) views. Dye has filled in the area between the labrum and acetabular rim, indicating a tear

best viewing plane. Careful attention should be paid to the chondrolabral junction, as tearing beyond this region may be irreparable. In addition to assessing the labrum, the cartilage within the joint should be evaluated, and the subchondral bone should be assessed. Subchondral cysts and edema have been associated with worse outcomes following arthroscopy.

An intra-articular anesthetic arthrogram with concomitant injection of steroid can serve both diagnostic and therapeutic purposes and is often done simultaneously with an MRA. An improvement in pain shortly after the injection is thought to be more suggestive of intra-articular pathology such as a labral tear. A negative

response is concerning that further evaluation is needed. However, false negatives are seen with this test and are thought to be due to overexpansion of the joint capsule due to excessive fluid within the joint. Therefore, a false test should not definitively rule out the diagnosis. The steroid aspect of the injection is for pain relief and reduction of inflammation.

Non-operative Management (PT, Medications, Injections, Other)

Non-operative management is typically the first-line treatment for all patients with FAI and labral tears, except in rare cases of severe FAI in a young patient when there is significant concern for rapid joint destruction. Non-operative management consists of physical therapy to improve mechanics and correct any kinetic chain abnormalities. An intra-articular steroid injection may be used for patients with acute exacerbation of pain or as a diagnostic tool to localize the source of pain when an anesthetic is included. Oral nonsteroidal anti-inflammatories and acetaminophen are recommended for pain relief and to reduce inflammation until the steroid injection takes effect.

Patients with hip pain often compensate by loading other areas of the pelvis, sacroiliac joint, spine, and knee. Therefore, physical therapy should focus on all these areas with an emphasis on regaining range of motion, strength, and dynamic stability. Patient should be allowed to slowly layer in activities as tolerated, with the goal of returning to most activities by 6 months. All patients are advised to temporarily limit activities involving impact loading, frequent pivoting, and extremes of motion, especially hip flexion and internal rotation. Specifically, squats, lunges, deadlifts, and distance running are strongly discouraged as the patient recovers from an acute episode of hip pain.

While many patients respond well to therapy, some will report continued symptoms and dissatisfaction with their activity limitations. Injections can be administered approximately 4 months apart in the event of a recurrent flare, if significant pathology is not observed on imaging. Non-operative management is used for the majority of patients with symptomatic labral tears as they often improve without need for further intervention. At this time, prophylactic surgery for asymptomatic FAI or labral tears is not indicated.

Indications for Surgery

One of the key indicators to pursue surgical intervention is failure of non-operative management. A significant proportion of patients treated with intra-articular injections, PT, and activity modification report improvement in their pain and symptoms. However, if symptoms persist, surgery may be warranted.

There is a strong association between FAI and the development of subsequent arthritis, and therefore, surgical intervention for FAI can be considered a joint preservation procedure. More aggressive surgical treatment can be considered in younger patients, and those with more severe signs of impingement on x-ray. Ideal candidates for surgery are those with minimal arthritis, age less than 40 years, intermittent symptoms, clear impingement, and labral tear on imaging and those who have persistent symptoms despite non-operative management.

Operative Management

Surgery for FAI can be performed through an open or arthroscopic approach. Arthroscopic techniques for FAI have evolved over the past decade to allow for faster recovery and quicker rate of return to sports and activities. However, open approaches are still utilized in cases of very complex deformity or hip dysplasia. Hip arthroscopy for FAI and labral pathology entails arthroscopic labral repair with acetabular and/or femoral osteoplasty (shaving of bone to normal contour) as indicated based on imaging. Upon entry into the joint, the labrum, chondrolabral junction, and cartilage are evaluated. The degree of arthritis can be more accurately assessed with direct visualization during hip arthroscopy than with imaging such as x-ray or MRI.

The quality of the labrum is evaluated, and repaired if possible. Given the key functions of the labrum in sealing the joint and providing joint stability, it is our opinion that as much of the native labrum should be preserved as possible. Anchors are placed into the acetabulum and the labral tear is repaired with suture (Fig. 10.5). Various augmentation procedures have also been described. If ultimately irreparable, the labrum may be debrided to prevent continued symptoms. However, it is thought that these patients will proceed to arthritis and total joint replacement more rapidly. In cases of substantial labral damage, reconstruction of the labrum is with autograft capsular tissue or remote autograft or allograft using iliotibial band or hamstring tendons.

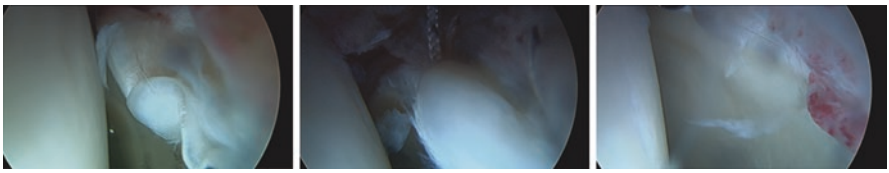

Fig. 10.5 These images demonstrate a labral tear as viewed during arthroscopic repair. For orientation, the femoral head is to the *left* and the acetabulum is to the *right*. *From left*: the labral tear can be identified by the wavy and frayed tissue. The capsular side of the labrum is elevated off the acetabulum, and an anchor is placed behind the labrum. Suture is then passed through the area of the tear. Once secured, the tissue returns to its anatomical location, and the knots are hidden as they lie on the capsular side

The acetabular rim and femoral head-neck junction are both inspected. Bony abnormalities are removed with a burr to obtain a normal bone contour, and fluoroscopy is used to ensure adequate resection. Additionally, hip motion is observed during surgery to ensure that all sources of impingement have been addressed.

Orthobiologics such as bone marrow aspirate concentrate (BMAC) contain numerous growth factors and anti-inflammatory molecules. In addition, BMAC contains concentrated mesenchymal stem cells derived from the bone marrow and has been studied in cartilage defects of the knee. In patients undergoing hip arthroscopy for FAI and labral tears, BMAC is currently being studied as an adjuvant used along with labral repair with the ultimate goal of improving outcomes and possibly slowing the progression of arthritis. BMAC can be harvested and applied concomitantly during the hip arthroscopy procedure. More research is needed to determine the efficacy of BMAC and other orthobiologics.

Hip arthroscopy is most commonly performed as an outpatient procedure, and the patient is allowed immediate weight bearing with a foot-flat gait on crutches. Patients use crutches for a minimum of 6 weeks to protect the repair by preventing the pelvis from tilting or lurching. A graded protocol is initiated with the patient first regaining motion on a stationary bike with low resistance, followed by swimming and use of an elliptical machine. Physical therapy may not be necessary, as aggressive treatment can irritate the hip and stress the labral repair. The objective is to return all patients to full activity by 6 months after surgery.

Expected Outcome (Benefits, Complications) and Predictors of Outcome

Patients commonly report an improvement in pain and symptoms after surgery, with most returning to pre-injury activity level. Competitive athletes are often able to return to sport and are satisfied with the results. Surgery primarily impacts the symptoms of acute pain and locking; however, achiness of the hip attributed to osteoarthritis is difficult to alleviate as there is currently no approved treatment to regenerate cartilage. While requiring further study, this is thought to be a joint-preserving procedure that can prolong the development of arthritis and subsequent hip replacement. Factors that have been associated with worse outcomes include age over 40 years and signs of advanced arthritis such as preoperative hip joint space narrowing. At this point in time, the only surgical treatment for advanced arthritis is total hip replacement. Complications after hip arthroscopy are rare, but can include infection, bleeding, and numbness or paresthesias, especially in the area of the perineum, and are rarely permanent. Additionally, not all patients may feel that their symptoms have improved with surgery, and some may still develop arthritis and require conversion to total hip replacement later in life.

Abductor Tendon Tears

Summary of Epidemiology

The tendons of the gluteus medius and minimus muscles of the hip are commonly referred to as the abductor tendons and can be considered analogous to the rotator cuff of the shoulder. These large muscles travel laterally from the ilium to attach at specific sites on the greater trochanter. Tears of the abductor tendons increase in prevalence with age, and females are much more commonly affected than males. Partial and complete tears of the abductor tendons can be seen in up to 20% of patients over 50 years old and are also common in patients with hip arthritis. Historically, many patients with greater trochanteric pain were presumed to have greater trochanteric bursitis, but recently, it has been understood that many of these cases were actually due to abductor tendon tears.

Abductor tears most often develop insidiously without history of trauma and in the setting of chronic tendon degeneration or tendinopathy. Less commonly, the tear may be attributed to an acute injury. In patients with tendinopathy of the gluteal tendons, there is progressive loss of tendon organization and structure, which eventually leads to tear. The lateral position of these tendons and the unique load mechanics of the hip joint may predispose the abductor tendons to stress. In addition, diabetes and hyperlipidemia have also been associated with tendinopathy, possibly from chronic inflammation or microvascular disease. Patients will often report symptoms that have failed to resolve, or slowly worsened, over an extended period of time despite rest or therapy regimens.

Clinical Presentation

Patients with tears of the abductor tendons most commonly report pain located laterally over the greater trochanter. Pain in the groin is more indicative of an intra-articular process. The area over the greater trochanter may be tender to palpation, as this is the attachment site for these tendons. Pain may also be exacerbated by activity, as patients may notice slight alterations in gait or weakness with activities requiring hip abduction.

While patients often report pain and dysfunction, physical exam is important in determining the competency of the musculotendinous unit. Observing the patient's gait, the physician should pay close attention for a contralateral pelvis drop as the patient balances on the injured leg while walking (Trendelenburg gait). Similarly, the physician should stand behind the patient with one hand on each iliac crest as the patient flexes their leg to 90° with balancing on the other. If the contralateral pelvis drops while standing on the injured leg, this is considered a positive Trendelenburg sign suggesting impairment of the gluteal tendons. A worse indicator is if the patient cannot balance at all. If untreated, these deficits may be slowly

progressive and less likely to resolve with surgical intervention, as tendon degeneration and retraction along with muscle atrophy may occur with time.

Further examination to evaluate the remaining function of the tendon should be conducted. With the patient lying on the uninjured leg in the lateral decubitus position, place the injured leg in slight knee flexion and hip extension so that it may rest on the table. The patient should then actively abduct against resistance to determine strength. Additionally, the physician should passively abduct the leg from this position so that it is parallel to the table. The patient should then attempt to hold this position as the examiner provides a downward force. Patients with abductor tendon tears may have weakness with this maneuver and in some cases, may have difficulty even holding the leg in abduction against gravity. Comparison to the uninjured side can identify deficits.

Additional physical exam maneuvers of the hip, including range of motion, strength, and provocative maneuvers, should be done to verify that the source of pain is not intra-articular. Intra-articular abnormalities can present as lateral pain. Additionally, the lower spine and neurovascular status should be evaluated.

Differential Diagnosis and Suggested Diagnostic Testing

On presentation, all patients should undergo standard radiographs of the hip including anteroposterior (AP) and lateral, in addition to an AP pelvis. The integrity of the joint, lumbosacral spine, and sacroiliac joint should be assessed for pathology as they may all contribute to lateral hip pain. Arthritis of these areas may produce pain radiating around the outside of the hip. Iliotibial band syndrome may also cause lateral-based hip pain, but would not result in the same functional deficits seen with gluteal tendon tears. Additionally, this diagnosis is more typically attributed to overuse, not an insidious onset without any identifiable exacerbating factors. Iliotibial band syndrome typically improves with activity modification.

Ultrasound can be used assess the abductor tendon and can be placed directly over specific sites of pain. The torn tendons can appear hypoechoic and often are enlarged in the setting of chronic tendinopathy. Ultrasound can also detect calcifications and bony abnormalities. Ultrasound may also provide therapeutic benefit, as it can be used to help guide injections into the site of pain.

Magnetic resonance imaging (MRI) is the definitive test to identify abductor tendon tears (Fig. 10.6) with specificity of 95% and sensitivity of 73%. On MRI, the tendon can be seen separated from the greater trochanter as the muscles tend to retract superiorly. Inflammation within the tendon, and in the bony attachment site, may be observed. The size and thickness of the tear can also be appreciated. An angiogram is not needed given the extra-articular nature of the injury. If there is concern for an intra-articular cause, an intra-articular anesthetic arthrogram may be conducted. As pain relief with the injection signifies intra-articular sources, patients with gluteal tears typically do not report improvement with injection.

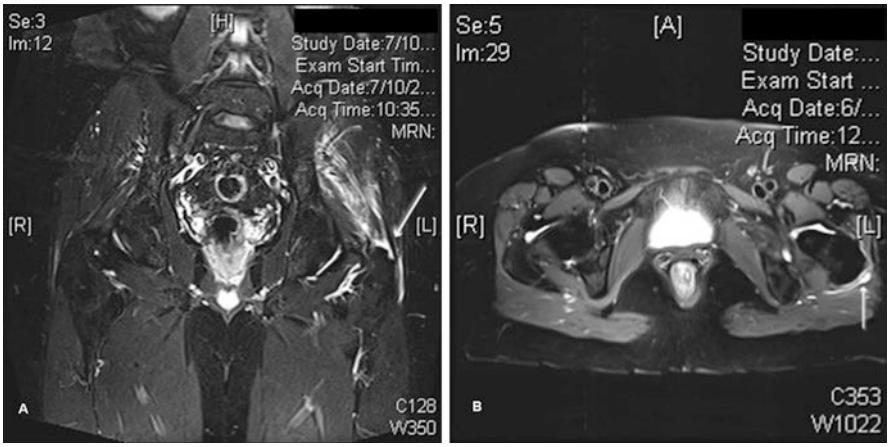

Fig. 10.6 Gluteal tendon tears are best evaluated on MRI. Here is a T2 coronal (**a**) and axial (**b**) view. When comparing the *left* to the *right* side, a hyperintense space can be seen between the greater trochanter and gluteal tendons indicating a tear (*white arrow*). The tendon tends to retract superiorly

Anesthetic and steroid injections into the tendon attachment site should be done with caution. Pain relief with an injection suggests the gluteal tendon is the source of pain.

Non-operative Management (PT, Medications, Injections, Other)

Gluteal tendon tears should be treated with an initial trial of non-operative management that may entail physical therapy, ice, insertion site steroid injections, and non-steroidal anti-inflammatories as needed. Activity modification that involves avoiding repetitive hip abduction activities and avoiding direct pressure over the painful area (such as sleeping on the affected side) may also be helpful. Corticosteroid injections in the site of pain have been shown to be effective for some patients. More recently, platelet-rich plasma (PRP) injections have been studied for abductor tendon tears and tendinopathy. Some studies have shown moderate to complete improvement in pain up to 6 months after PRP injection. One randomized controlled trial showed that in patients with chronic gluteal tendinopathy, a single ultrasound-guided PRP injection resulted in greater improvement in pain and function than a single corticosteroid injection, and these improvements were sustained at 2 years. More high-quality studies are needed to further validate these findings in various patient populations. Many patients with abductor tendon tears, particularly small partial tears, respond well to conservative therapy. However, if patients fail to respond, or exhibit worsening of symptoms, early surgical intervention may allow patients to regain function.

Indications for Surgery

Surgery may be recommended if patients fail conservative therapy. Larger, full-thickness tears are likely to be more amenable to repair with a greater improvement in symptoms. However, once significant functional deficits are noted on physical exam, such as Trendelenburg sign, the likelihood of regaining that strength diminishes. Surgical repair may still improve pain; however, it may not restore function to the desired level. This is important when counseling patients on management options, and for referring providers so that timely work-up and referral can be pursued if needed. Without surgical intervention, these tears do not appear to heal spontaneously, but patients may be able to compensate with surrounding musculature and strengthening of the remaining intact tissue. The tissue quality should also be evaluated as those with more severe retraction and fat atrophy are less likely to experience significant functional improvement. Patients with significant fat atrophy and functional deficits at presentation should not be considered surgical candidates.

Operative Management (Provide Brief Description of the Surgical Procedure(s))

Repairing tears of the gluteal tendon can be performed with open or endoscopic techniques. Both techniques have been shown to lead to pain reduction and improvement in function, but the endoscopic approach may be associated with less postoperative complications. With the endoscopic approach, four small portal incisions are created. The patient is placed in the lateral decubitus position, with the injured leg up in neutral rotation and slight abduction. Upon piercing the iliotibial band, the deep peritrochanteric space is entered and the tear can be visualized. An anchor is placed in the gluteal tendon insertion site on the greater trochanter, and the suture is subsequently passed through the intact portion of the tendon. It is important to note that the gluteus medius and minimus have distinct insertion sites on the greater trochanter (Fig. 10.7). Care is taken to appropriately place the anchors so that the native anatomy is restored for optimal function. Then the tendon is then securely tied back down to its anatomical insertion site. Additionally, a transosseous technique can be used to reduce tension. This is accomplished by placing the suture ends through a swivel lock that is secured just distal to the vastus ridge.

Postoperatively, patients are advised to use a progressive foot-flat weight-bearing gait with crutches. By 3 months, they may begin to return to all normal activities.

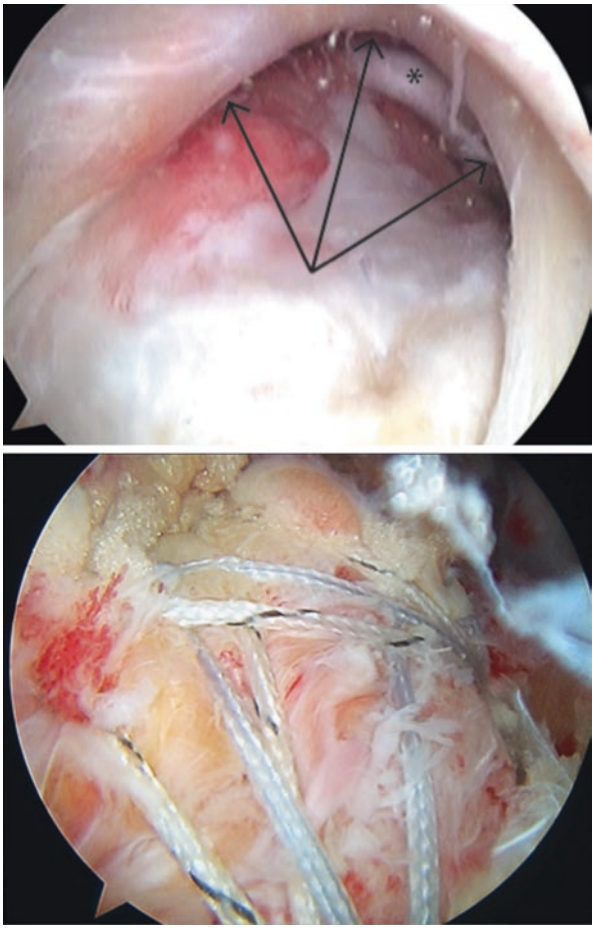

Fig. 10.7 The gluteal tendons have distinct insertion sites on the greater trochanter. When repaired, the natural anatomy should be restored, so awareness of these footprints is essential. *Top*: Arthroscopic view of tears of the gluteus medius (*arrows*) and gluteus minimus (*asterisk*). *Bottom*: Arthroscopic view of the repair

Expected Outcome (Benefits, Complications) and Predictors of Outcome

Endoscopic repair of the gluteal tendons has gained significant traction recently with studies reporting good to excellent results. Most reliably, surgical intervention provides pain relief, and patients with minimal functional deficits typically improve or maintain strength. If significant functional deficits are present before surgery, their resolution postoperatively is variable. Additionally, patients with poor tissue quality, indicated by fat atrophy and retraction, are less likely to experience functional benefits. Counseling patients on the benefits and expectations of surgical intervention is essential. Complications are rare with this procedure, but can include nerve palsy, infection, bleeding, failure of the repair, and tendon re-tear (Table 10.1).

Table 10.1 Summary of femoroacetabular impingement (FAI), labral tears, and abductor tendon tears with synopsis of presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
FAI and labral tear	Hip pain with flexion and internal rotation "C-sign" Catching, popping, locking Exacerbated with pivoting Symptoms resolve quickly, achiness suggests OA	Radiographs for bony abnormalities MRI or MRI with arthrogram Anesthetic arthrogram	Steroid arthrogram Physical therapy NSAIDS and acetaminophen Activity modification to reduce impact loading and extremes of motion	Failure of conservative management Significant FAI on imaging Younger age Minimal arthritis	Arthroscopic femoroacetabular osteoplasty with labral repair Labral debridement and/or augmentation depending on tissue quality
Abductor tendon tear	Lateral hip pain localized over greater trochanter Pain exacerbated with activity or direct pressure Weakness with abduction Trendelenburg gait or stance	Radiographs to rule out intra-articular pathology MRI Ultrasound Anesthetic injection	Steroid injection PRP injection Physical therapy NSAIDs and acetaminophen Activity modification	Failure of conservative management Full-thickness tear Minimal fatty atrophy Functional deficits not severe or chronic	Endoscopic gluteal tendon repair

MRI magnetic resonance imaging, *NSAIDs* nonsteroidal anti-inflammatory drugs, *PRP* platelet-rich plasma

Suggested Reading

Nepple JJ, Zaltz I, Larson CM, Beaulé PE, Kim YJ, Millis MB, Sierra RJ, Clohisy JC, ANCHOR Group. Surgical treatment of femoroacetabular impingement: hip arthroscopy versus surgical hip dislocation: a propensity-matched analysis. J Bone Joint Surg Am. 2020;102(Suppl 2):51–8.

Perets I, Chaharbakhshi EO, Shapira J, Ashberg L, Mu BH, Domb BG. Hip arthroscopy for femoroacetabular impingement and labral tears in patients younger than 50 years: minimum five-year outcomes, survivorship, and risk factors for reoperations. J Am Acad Orthop Surg. 2019;27(4):e173–83.

Chapter 11
Total Hip Arthroplasty and the Treatment of Hip Osteoarthritis

Michael J. Weaver

Definition and Epidemiology

Hip osteoarthritis represents one of the most common conditions within the population, with an estimated prevalence of 10% in individuals aged 45 and over. Clinically symptomatic arthritis, where hip pain due to degenerative changes impairs daily activities and quality of life, remains one of the most frequent conditions prompting presentation to an orthopedic surgeon, with costs exceeding $13 billion per year in terms of surgical treatment alone. Total hip arthroplasty (THA) remains the most common surgical intervention for primary hip osteoarthritis. THA is a re-surfacing procedure of the hip joint: diseased bone and cartilage of the hip are removed and replaced by implants made of metal, plastic, and occasionally ceramic. The new articulation allows for painless motion and weight bearing. At 10-year follow-up, over 90% of patients can expect to have a well-functioning joint without the need for revision surgery. Approximately 500,000 hip replacement procedures are performed in the United States every year. As the baby boomer generation ages, and as people continue to be more active later in life, the demand for hip replacement surgery is expected to increase over the coming decades.

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Clinical Presentation

The primary complaint of patients with hip arthritis is pain and stiffness. Typically, patients present with pain in their groin, but they may also have pain in the buttock, laterally over the greater trochanter, or in the anterior thigh. Occasionally, patients will have referred pain down the thigh into the knee. Interestingly, a small minority of patients with hip arthritis will present with knee pain only. Pain is exacerbated by activity and relieved with rest. Occasionally, patients may complain of trouble sleeping due to the pain.

Hip arthritis patients also present with stiffness in their hip. Hip flexion and particularly internal rotation can become limited and may interfere with many activities of daily living. For example, many patients complain of difficulty tying shoes and socks, discomfort sitting in a low chair, and trouble getting into and out of a car.

The pain associated with hip arthritis typically progresses over the course of several months to a year. It often begins as an intermittent ache that evolves into a constant bother. Some patients may have occasional flare-ups associated with minor injuries that can be managed with anti-inflammatory pain medications. The pain and stiffness typically progress until patients are extremely limited in their ability to walk and perform normal daily activities.

Differential Diagnosis

Hip arthritis results from degeneration of the hip joint and its articular cartilage. This process can result from a number of factors – including genetic predisposition, congenital abnormalities of the hip, or traumatic injury. The normal hip is a ball-and-socket joint lined by smooth articular cartilage. This structure allows the hip to have a wide range of motion, while being inherently stable, and be capable of withstanding bodyweight during normal activities. As the arthritic process progresses, the cartilage in the hip frays and begins to thin out. When the subchondral bone beneath the cartilage begins to wear down, inflammation, pain, and stiffness result.

Osteoarthritis

Osteoarthritis is the most common cause of musculoskeletal pain and disability. Hip osteoarthritis is thought to be due to subtle mechanical deficiencies within the hip joint. Due to slight problems in the development of the hip joint, the hip may be more susceptible to wear and damage from normal activities. As the patient ages, damage accumulates and the cartilage of the hip slowly degenerates. This is manifested by pain and stiffness in the hip and limited mobility. Osteoarthritis may affect either one or both hips, and can be synchronous or asynchronous.

Patients are commonly in their fifth, sixth, or seventh decades of life and present with progressive groin pain and stiffness. Factors associated with the development of hip arthritis include age, diabetes, family predisposition, and hypertension. Patients with developmental anomalies of their hips, including Perthes disease or hip dysplasia, are also at risk. Interestingly, unlike knee arthritis, obesity does not appear to be associated with the development of hip arthritis.

Inflammatory Arthritis

Rheumatoid arthritis (RA) is the most common inflammatory arthritis that affects the hip. The synovial lining of the hip joint becomes hypertrophic and inflamed, infiltrated by mononuclear cells, polymorphonuclear leukocytes, and macrophages. Initially, synovitis is primarily responsible for hip pain. Untreated, the inflamed synovium leads to irreversible cartilage destruction and damage to the periarticular bone. The treatment for rheumatoid arthritis has evolved significantly over the last 20 years. With the advent of disease-modifying antirheumatic drugs (DMARDs), severe joint disease and end-stage arthritis as a result of rheumatoid arthritis are now much less common. Once the disease has progressed to cartilage and bone destruction, medical management alone is often ineffective and a hip replacement may be indicated.

Seronegative spondyloarthropathies including ankylosing spondylitis, psoriatic arthritis, and Reiter syndrome can also affect the hip. These are a group of interrelated inflammatory arthritides that are often associated with the HLA-B27 gene. Ankylosing spondylitis affects the spine, sacroiliac joints, and peripheral joints. Inflammation primarily involves the entheses – the attachments of ligaments to bone. The hip is often one of the earlier joints affected. Psoriatic arthritis involves an inflammatory arthritis in association with classic psoriatic lesions. Patients also typically present with nail involvement, including hyperkeratosis or pitting of the nail bed. Reiter syndrome represents a reactive arthritis in association with eye, skin, and mucous membrane inflammation. Regardless of the underlying etiology, once advanced, the degeneration associated with these entities is best treated with total hip replacement.

Avascular Necrosis

Avascular necrosis (AVN) or osteonecrosis is a common cause of hip pain and disability that often requires treatment with a hip replacement. AVN can be associated with chronic alcohol abuse, steroid use, some viral infections such as HIV, and trauma. In many cases, an underlying cause for AVN cannot be determined. The bone of the femoral head dies due to microvascular damage and ischemia. As the bone is resorbed and repaired, it is weakened and slowly collapses, leading to

degeneration of the hip joint. Patients typically complain of groin pain. Initially, plain radiographs may be normal. MRI is useful in this situation to evaluate for the possibility of AVN. As the disease progresses, the femoral head begins to flatten and severe arthritic changes occur. There may be a role for observation or core decompression of the femoral head in the early stages of the disease, but once a subchondral fracture or deformation of the femoral head occurs, patients are best treated with a total hip replacement.

Post-traumatic Arthritis

Some patients who sustain a fracture of the pelvis around the hip joint, or the proximal femur, may develop post-traumatic arthritis as a consequence of their injury. This may be due to disruption of the blood supply to the femoral head (avascular necrosis), failure of the fracture to heal, implant failure, or simply a result of the chondral injury sustained at the time of injury. Patients with previous fractures and surgery who develop post-traumatic arthritis are at slightly higher risk for perioperative complications due to the scar tissue and altered anatomy resulting from their initial trauma as well as prior surgeries.

Suggested Diagnostic Testing

Arthritis of the hip is best diagnosed with plain radiographs. Typically, three views are obtained. An anteroposterior (AP) radiograph of the pelvis, to allow for evaluation and comparison to the contralateral hip, as well as AP and lateral views of the affected hip should be obtained.

The four cardinal signs of osteoarthritis are loss of joint space, osteophyte formation, subchondral sclerosis, and subchondral cyst formation. Figure 11.1 shows an AP pelvis radiograph demonstrating the comparison of a normal hip with preserved joint space and an arthritic hip showing signs of advanced degeneration. Mild osteoarthritis presents with thinning and asymmetry of the articular cartilage, while severe arthritis is associated with complete loss of the joint space and secondary changes in the bone around the hip. The radiographic findings of arthritis do not always correlate with the symptoms patients feel.

Inflammatory arthritis often presents with a different pattern. Initially, the joint changes are more subtle, but, as the disease progresses, the bone of the femur or acetabulum can become osteopenic and erosions of bone may appear. The femoral head often exhibits a more medial and central loss of joint space and, in extreme cases, can protrude into the pelvis.

Computed tomography (CT) and magnetic resonance imaging (MRI) are not generally useful in the work-up of typical patients with hip arthritis. MRI may be indicated in patients with hip pain where radiographs are normal, in order to assess

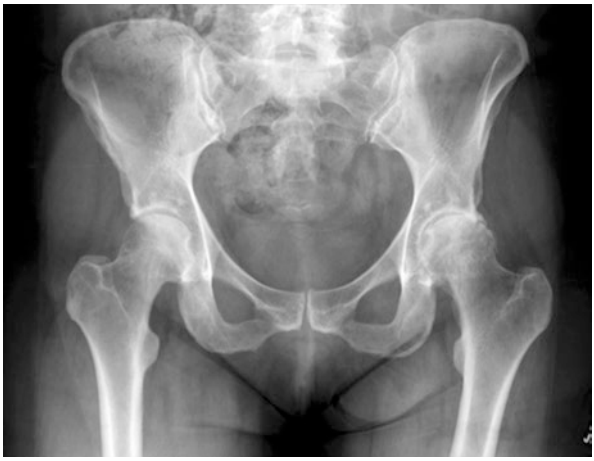

Fig. 11.1 Anteroposterior radiograph of a patient with a normal right hip and advanced arthritis of their left hip. There is complete loss of the superior joint space, osteophyte formation, and subchondral sclerosis

for lesions of the articular cartilage and labrum, or to investigate the possibility of AVN. An MRI is particularly useful in younger patients with suspected intra-articular hip pathology to better evaluate the labrum and articular cartilage and to look for signs of mechanical problems like impingement. CT is occasionally used preoperatively in some complex and revision cases to assist with preoperative planning, but is generally not useful in the diagnosis and initial management of hip arthritis.

Nonoperative Management

The primary treatment for hip arthritis is over-the-counter pain medications and activity modification. Nonsteroidal pain medications (NSAIDs) such as naproxen and ibuprofen often provide excellent relief of mild to moderate pain-related symptoms. Some patients have difficulty tolerating NSAIDs due to stomach upset or bleeding episodes. Acetaminophen is widely tolerated and also provides excellent pain relief without the gastrointestinal or hematologic side effects associated with NSAIDs. Generally speaking, it is best to avoid narcotic pain medication in the treatment of long-term arthritis pain. Studies have shown that patients who are on narcotic pain medication preoperatively tend to do worse in terms of satisfaction and function following a hip replacement.

Some patients benefit from a nonimpact exercise program focused on strengthening the muscles around the hip – particularly the gluteus medius muscle and other hip abductors. While therapy and exercise do not alter the natural course of arthritis, they may provide some symptomatic relief. Impact exercise, such as jogging, is often poorly tolerated and can exacerbate symptoms. It is also good to avoid activities that put the hip in a position of discomfort. Most commonly, patients have difficulty with deep hip flexion such as sitting in low seats. A cane can also provide

symptomatic relief and can help avoid a painful limp. It should he held in the contralateral hand to the affected hip.

While not associated with the risk of developing hip arthritis, obesity may exacerbate or potentiate symptoms. Weight loss can improve the painful symptoms associated with hip arthritis. The mechanical environment around the hip magnifies body weight, leading to forces across the hip joint of many multiples of total body weight during activities such as running and stair climbing. Preoperative weight loss also reduces the risk of complications in the perioperative period, speeds up recovery, and reduces postsurgical risks of infection and dislocation.

Weight loss in the setting of hip arthritis can be challenging as the pain associated with arthritis often limits patients' capacity to perform vigorous exercise. Nonimpact exercise programs including cycling or water aerobics may be beneficial as they tend to aggravate hip arthritis to a lesser extent. Involving a nutritionist to make dietary changes is also useful.

While steroid injections are often used in the management of knee arthritis, they are infrequently used in the setting of hip arthritis. The hip joint is much less accessible and requires the use of either fluoroscopy or ultrasound to accurately place medication into the joint itself. A steroid injection may be useful to control an exacerbation of symptoms or to try to delay surgery. There is some evidence that repeated steroid injections may lead to faster progression of cartilage degeneration. There is little role for visco-supplementation with hyaluronic acid in the management of hip arthritis.

Injections can be helpful from a diagnostic perspective when they are used to tease out how much of a patient's symptoms can be attributed to their hip arthritis. For example, in a patient with lumbar stenosis and moderate hip arthritis, an injection of local anesthetic into the hip joint – a diagnostic injection – that alleviates most of the patient's pain will confirm that the hip is the primary pain generator.

Indications for Surgery

The indications for hip replacement surgery are multifactorial and patient dependent. Patients with significant limitations to their activities of daily living, quality of life, and employment who also have radiographic signs of moderate to severe hip arthritis are generally considered to be good candidates for hip replacement surgery. However, the severity of arthritis, patient age, medical comorbidities, and tolerance for surgical risk all play a role in the decision-making process.

As progress has been made in hip replacement surgery, and the recovery and complication profile has improved, younger and more active patients are undergoing such procedures. However, the risks associated with surgery are real, and only patients with significant functional limitations should be considered for total hip replacement. Given the expected longevity of hip replacements, younger patients who undergo a hip replacement can expect to have one or more revision surgeries during their lifetime.

Perioperative complications as well as the risks of infection and dislocation are higher in overweight patients. These risks are particularly elevated in patients with morbid obesity. Some orthopedic surgeons will avoid hip replacement surgery in patients with a body mass index (BMI) of over 40 or 50 kg/m^2. Similarly, the risks of infection are higher in patients with poorly controlled diabetes mellitus (HbA1C > 7.5) and smokers. Some centers and surgeons may require improved glucose control or smoking cessation prior to proceeding with an elective surgery.

Operative Management

A total hip replacement is a re-surfacing procedure of the hip joint. The diseased and arthritic bone and cartilage of the hip joint are removed, and implants made of metal, plastic, and occasionally ceramic are used to create a new articulation between the femur and pelvis. This new joint glides easily and allows for painless motion and weight bearing. Figures 11.2 and 11.3 demonstrate an example of a contemporary hip prosthesis and the radiograph of a patient with a similar implant in place following their hip replacement surgery.

Fig. 11.2 A contemporary hip prosthesis with an acetabular shell, a polyethylene liner, a metallic head component, and a proximally porous-coated femoral stem. The porous coating allows for a cementless press-fit into bone. The components are fit snugly into the bone creating a friction fit. The bone then grows into the porous surface of the implants

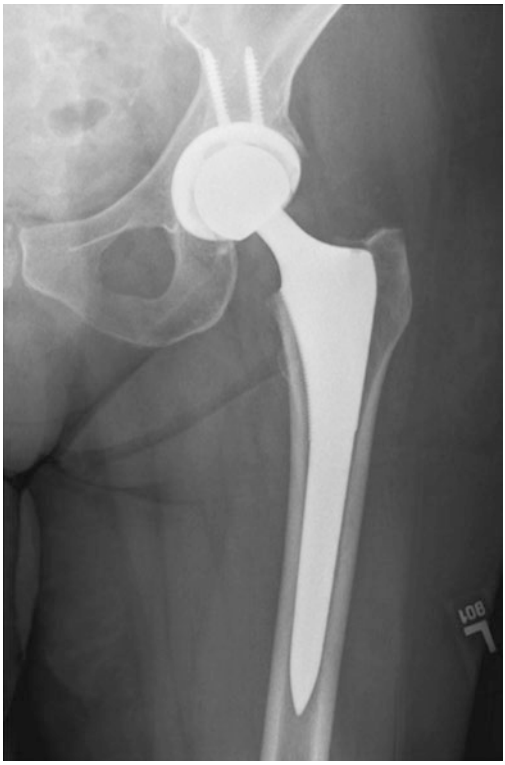

Fig. 11.3 An anteroposterior radiograph of a patient following left total hip replacement. The radiolucent liner of the acetabulum can be seen with the femoral head located securely within the acetabular component

The hip joint is surgically exposed and the diseased bone removed with specialized tools that help remove and reshape the remaining bone to accept the new prosthesis (Fig. 11.4). Hip prostheses are manufactured in a wide range of shapes and sizes to match the particular anatomy of a patient. The specific implants selected for a surgery depend upon the patient's anatomy, bone quality, and the preferences of the treating surgeon. Often computer software is used to help plan the procedure and determine the optimize implants and size ranges for a given patient.

Implant Design

Most hip replacement surgery performed in the United States uses cementless joint prosthesis. These implants are covered in a rough, gritty surface that is biologically friendly. Once implanted into the bone, they first achieve fixation through a tight friction fit. The surrounding bone then grows into, or onto, the prosthesis to crease a durable biologic bond.

Occasionally, bone cement may be used to fix either, or both, the femoral or acetabular components to bone. Bone cement is a polymer of methyl methacrylate

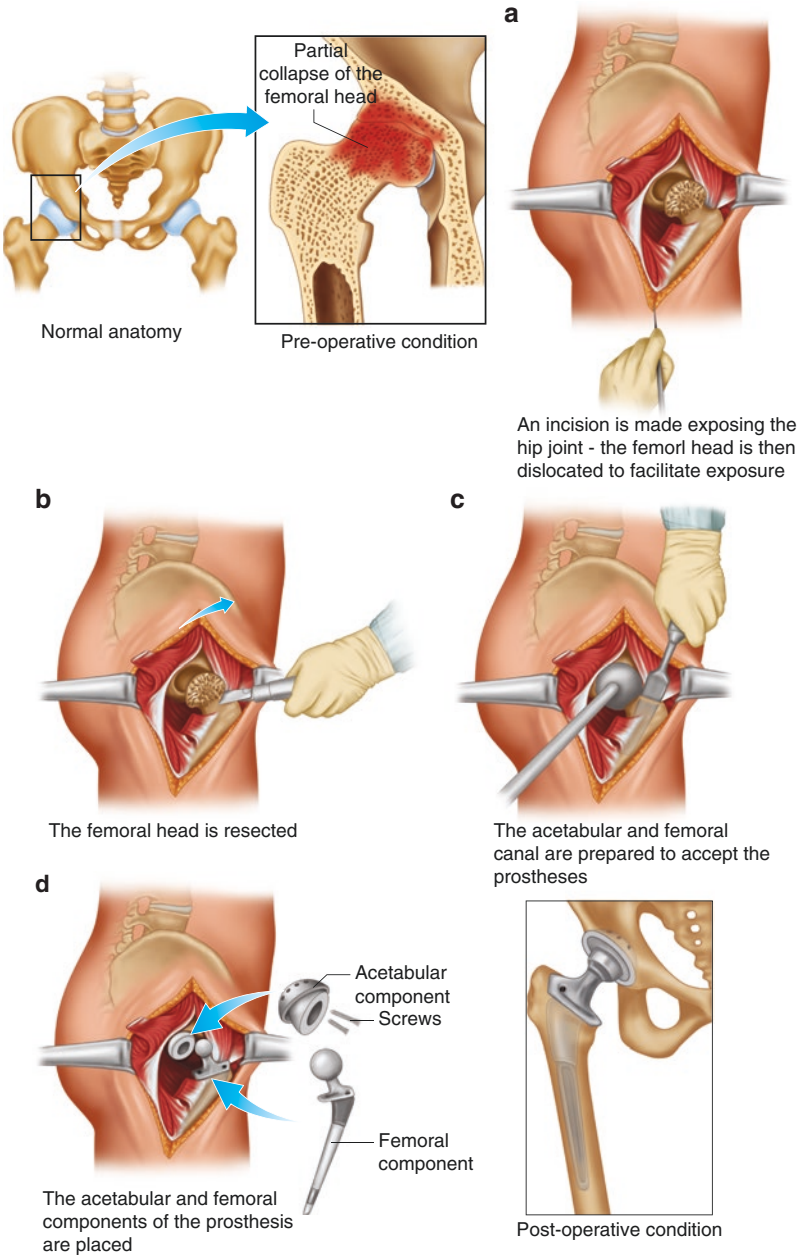

Fig. 11.4 Schematic depicting the surgical approach and procedural steps. THA surgical approach and procedural steps associated with a total hip replacement. (**a**) The hip is exposed through an incision through the skin and the surrounding muscles are reflected to expose the hip joint. The hip is dislocated to expose the femoral head and the hip socket. (**b**) The femoral head and neck are removed. (**c**) The bone is re-shaped to accept the hip implants. (**d**) The implants are impacted into the bone in the approprate position and the hip is reduced

that acts as a grout to affix the components to the host bone. The advantage of bone cement is that it achieves an immediate and strong bond. The bone cement does not require strong host bone, and if a patient has particularly poor quality, bone cement may be selected to affix the prosthesis.

One of the most important aspects of implant design is the bearing surface. This is the interface between the acetabular socket and the femoral head. Most contemporary total hip systems utilize a highly cross-linked polyethylene liner and either a ceramic or cobalt-chrome femoral head. These combinations are thought to offer the best balance between longevity and implant-related complications. Patients who have had a hip replacement that utilizes a metal on metal (cobalt-chrome head and liner) are at increased risk for developing a reaction to the wear particles generated by everyday activity that can cause pain and tissue destruction and may require revision surgery.

Approach

Total hip replacements may be performed through a number of approaches. The approach is the muscular interval used to access the hip joint. The most common are the posterior approach, anterolateral approach, and direct anterior approach. All of the approaches are similar in terms of the implants used and the expected long-term outcome. Different surgeons prefer different surgical approaches for a number of reasons including training, complication profile, specific anatomy, and patient variables. There is some emerging data that the direct anterior approach may help speed initial recovery, but it appears that the anticipated long-term functional outcome and complication profile a patient can expect is similar regardless of the surgical approach used. There are some technical issues related to patients' specific anatomy that may cause a surgeon to favor one approach over another.

Computer- and Robotic-Assisted Surgery

Over the last decade, there has been an increase in the number and types of computer- and robotic-assisted surgery used for hip replacement surgery. In general terms, these systems utilize standard hip implants and use navigation technology to improve implant position and possibly reduce the size of the surgical incision. While there may be some small benefit in the accuracy of restoring the patients' normal leg length and hip offset, there is no data to suggest improved function in the long term. The use of computer- or robotic-assisted surgery is related to surgeon preference.

Perioperative Period

Patients are typically admitted to the hospital following the surgery and stay between 1 and 3 days. Fit and active patients are often able to be discharged home the day following surgery, while more frail patients may benefit from a short stay in a skilled nursing facility to assist in their recovery.

Postoperative Rehabilitation

Physical therapy and exercise are crucial to recovery following hip replacement surgery. In the hospital, therapists work with patients to mobilize them from bed and safely ambulate with the assistance of a walker or crutches. Often patients are mobilized out on the same day as their surgery. Generally speaking, the more active and aggressive patients are with their mobilization and exercise, the better their ultimate result.

Depending on the type of implant used, and the strength of the patient's bone, most patients will be allowed to put full weight on their affected hip immediately following surgery. Occasionally, patients may have a limitation to weight bearing for the first 1–3 months following surgery.

Patients can expect some form of home physical therapy program for the first few weeks following surgery with a transition to an outpatient program once they are comfortable leaving their home. Most patients use a walker for crutches during this initial period. Many patients able to walk well without assistive devices by about 1 month following surgery and can return to light work. By 3 months, patients are often ale to return to most of their normal activities.

Hip Precautions

Following surgery and dependent upon the surgical approach and patient characteristics, surgeons may place their patients on hip precautions. These are a set of instructions regarding mobility and exercise to avoid putting the hip in a position that may lead to a dislocation. For posterior approaches, precautions typically involve avoiding flexing the hip beyond 90 degrees or sitting in low chairs. For anterior approaches, precautions typically avoid hyperextending the hip or putting the leg in extreme external rotation such as bridging exercises. Many surgeons employ hip precautions during the immediate postoperative period, but reduce or remove them once the patient has sufficiently recovered from surgery.

Expected Outcomes

The vast majority of patients who undergo total hip replacement have significant pain relief and improvement in their symptoms, with studies showing durable satisfactory outcomes achieved in greater than 90%. Once their rehabilitation is complete, most patients have little or no pain associated with their hip. Many patients have concomitant improvements in their total body range of motion, endurance, strength, and gait.

While patients may have activity limitations in the short term, within a few months, most patients can participate in normal activities without difficulty. Most surgeons limit their patients from impact exercise, such as running or jogging, but there is no limit on nonimpact exercise like cycling, swimming, or using an elliptical exercise machine. Sports such as downhill skiing, soccer, and tennis are also appropriate, but may increase the risk of periprosthetic fracture or dislocation if the patient falls or is otherwise injured.

Wear/Longevity

Hip replacements are mechanical devices – with every step, the femoral component rubs against the acetabular liner. Microscopic imperfections on the femoral head lead to abrasive damage to the liner within the hip joint. Despite significant advancements in the design of total hip replacements, from the metallurgy of the components to the composition of the liners and heads, we still expect that hip replacements will slowly wear out over time. With current implants, the wear rates are extremely small, measured in microns per year. In the past, wear was a significant problem and the most common cause of hip replacement failure and need for revision. It appears that with contemporary implants, wear will be less of an issue than it has been previously.

As technological advances have been made in materials and surgical technique, the life expectancy of hip replacements continues to improve. It is generally anticipated that approximately 90% of patients will have their hip replacement last at least 10 years without the need for revision surgery and as many as 50% of patients can extend joint longevity to 20 years or more postoperatively.

Dislocation

The hip replacement allows for improved motion following surgery. However, if the patient puts their hip in an extreme position, or falls poorly, it is possible for the hip to become dislocated. A periprosthetic hip dislocation affects about 1% of patients who have a hip replacement.

If a prosthetic hip becomes dislocated, the patient will have severe pain and a significant leg length inequality and will be unable to ambulate. Radiographic evaluation shows the femoral head to be dislocated from the acetabulum. The treatment for a dislocation is a reduction performed under sedation, most commonly in the emergency department. Occasionally, if a patient's hip cannot be reduced, or the patient has risk factors that prevent conscious sedation in the emergency room, they may need to be taken to the operating room. Most patients who have a dislocation are treated with a simple closed reduction and do not go on to have further problems. About a third of patients who do have one dislocation event go on to recurrent instability. In this case, they may be treated with a course of bracing, or potentially surgery, to try to correct any possible mechanical problems leading to the dislocations.

Periprosthetic Fracture

As hip replacements are increasingly performed within a more active population, the incidence of periprosthetic fractures is also growing. Periprosthetic fractures may result from high-energy trauma such as car accidents or falls from height or from ground level falls if the patient has osteoporosis or other compromise to their bone quality.

Periprosthetic hip fractures are challenging to treat and typically involve either surgical repair of the fractured bone or removal of the previous prosthesis and revision to a new – more extensive – prosthesis. Some minor fractures, particularly those isolated to the greater trochanter, may be managed nonoperatively.

Thromboembolic Disease

There is a small risk of developing a deep vein thrombosis (DVT) or pulmonary embolism (PE) following hip replacement surgery. The risk of a fatal PE is approximately 1 in 10,000. To reduce the risk of PE, most surgeons encourage rapid mobilization to promote circulation and use noninvasive measures such as compression stockings or sequential compression devices while the patient is in the hospital.

Chemical prophylaxis is also utilized. There is a considerable debate as to the optimal medication and duration. A balance must be found between reducing the risk of thromboembolic disease and increasing the risk of serious bleeding events requiring transfusion or a return to the operating room. Typically, chemical prophylaxis is achieved with aspirin (ASA), a direct oral anticoagulant (DOAC) such as rivaroxaban or apixaban, low molecular weight heparin (LMWH), or warfarin. Prophylaxis is typically continued for 2–4 weeks following surgery.

Infection

While rare, occurring in less than 1% of primary hip replacements, infection is a devastating complication. A deep infection is typically treated with surgical irrigation and debridement. The modular parts of the prosthesis are exchanged. If the infection becomes entrenched, then the prosthetic components have to be removed and exchanged with new components either at the same time or after 3 or more months of antibiotics and surveillance.

Due to the significant impact a periprosthetic infection has upon the patient, most surgeons utilize a number of methods to try to reduce the risk of infection. Patients are often tested preoperatively for colonization with MSSA or MRSA and treated if present. Preoperative antibacterial soap is often employed in the days before surgery. Perioperative antibiotics are used to reduce the risk of infection, and meticulous sterile technique is required.

In the long term following a hip replacement, there is a slight risk of seeding bacteria onto the hip components if patients develop bacteremia. For this reason, the American Academy of Orthopaedic Surgeons (AAOS) recommends that patients with a hip replacement receive prophylactic antibiotics prior to any dental work or other invasive procedure for the first 2 years following their surgery. Some orthopedic surgeons recommend lifetime prophylaxis. The risk of infection following dental procedures is extremely small, but the consequences are significant. For patients with no penicillin allergy, a single dose of 1 g of amoxicillin given orally 1 hour prior to a dental procedure is a common regimen. Clindamycin may be used in patients who are allergic to penicillin.

Leg Length Inequality

Hip replacement surgery alters the leg length, restoring height lost on the side of the degenerative hip joint. Occasionally, leg lengths may be asymmetric following hip replacement surgery. The difference is typically small and most patients will acclimate to it. If patients complain of a symptomatic leg length inequality, often a shoe lift is sufficient.

Summary

Patients with hip arthritis typically present with progressive groin pain and stiffness in the hip that limits normal activity. Radiographs show loss of joint space, osteophyte formation, subchondral sclerosis, and cyst formation. Nonsurgical treatment consists of over-the-counter pain medications (such as NSAIDs or acetaminophen) and low-impact exercise. Once symptoms progress to the point where they have

Table 11.1 Summary of hip osteoarthritis with a synopsis of presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Hip osteoarthritis	Groin pain Hip stiffness and limited range of motion	Plain film radiographs of the affected hip and pelvis	Nonsteroidal pain medication Physical therapy and non-weight-bearing exercise Weight loss	Failure of nonoperative treatment Total hip replacement

significant limitations in activities of daily living or employment, patients may be considered good candidates for hip replacement surgery. Most patients can expect to have a significant improvement in their symptoms with a durable result that should last for more than 10–20 years. Hip replacement surgery, when used appropriately, can have a massive impact on a patient's life, relieving pain and restoring function (Table 11.1).

Suggested Reading

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Chapter 12
Hip-Spine Syndrome

Christina Y. Liu, Jeffrey Lange and Richard Wilk

Many people develop hip and back pain which can have a significant impact on their function and quality of life. Hip-spine syndrome was first described in the 1980s [1] as a clinical scenario for patients who present with concurrent stiffness and decreased mobility in the hip and spine, most often the result of osteoarthritis and degenerative changes. Patients with hip-spine syndrome can present with complex symptoms and findings on physical exam, and with appropriate diagnostic tests, a practitioner can identify the etiology of pain and guide patient care. Employing a multidisciplinary care team to manage hip-spine syndrome is often beneficial. The care team may include providers from primary care, pain management, physical therapy, physiatry, rheumatology, neurosurgery, and orthopedic surgery. While nonoperative treatment is the mainstay of care, some patients may require collaborative surgical consultation with a joint replacement and spine surgeon to achieve relief of pain.

Summary of Epidemiology and Pathophysiology

Osteoarthritis (OA) is the most common musculoskeletal (MSK) problem that occurs with aging [2]. OA is second only to heart disease as a cause of morbidity and functional decline in the elderly [3]. Hip-spine syndrome most commonly manifests as concurrent osteoarthritis of the hip and spine, which may be present in a significant portion of the older population. Spine OA is more commonly referred to as degenerative spine disease (DSD) or spondylosis, which can progress to spinal

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stenosis or narrowing of the spinal canal. More than 90% of adults over the age of 50 have radiographic evidence of degenerative disease of the spine [4, 5], and up to 40% of patients with hip OA undergoing total hip arthroplasty (THA) have concurrent lumbar spine pathology [6].

Normal biomechanics of the hips and spine are closely related. The normal movements of the hips and spine rely on proper biomechanics of one another and are intimately connected through motion of the pelvis. Figure 12.1 demonstrates physiologic orientation of the hips, spine, and pelvis in the sitting and standing positions. In patients with hip-spine syndrome, the hips and lumbar spine lose the ability to rotate, flex, and extend normally, leading to abnormal orientations in sitting, standing, and other functional positions (Fig. 12.2).

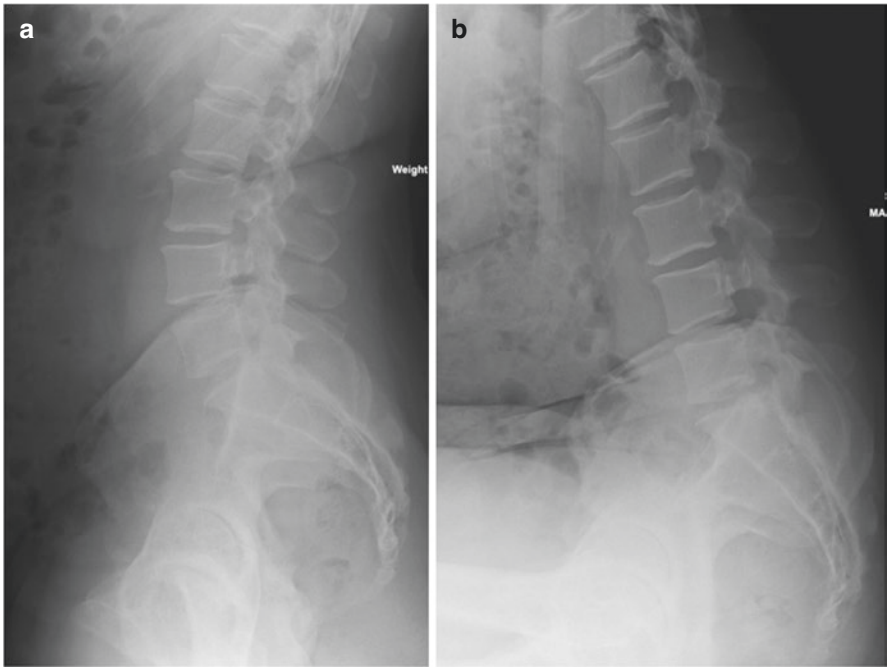

Fig. 12.1 Physiologic orientation of the hip, spine, and pelvis demonstrates physiologic orientation of the hips, spine, and pelvis in the standing (**a**) and sitting (**b**) positions. In the standing position, the lumbar spine has a lordotic curve, the pelvis tilts anteriorly, and the hip center is shifted backward to remain centered under the spine. In the sitting position, the lumbar spine straightens, the pelvis tilts posteriorly, and the hip shifts forward

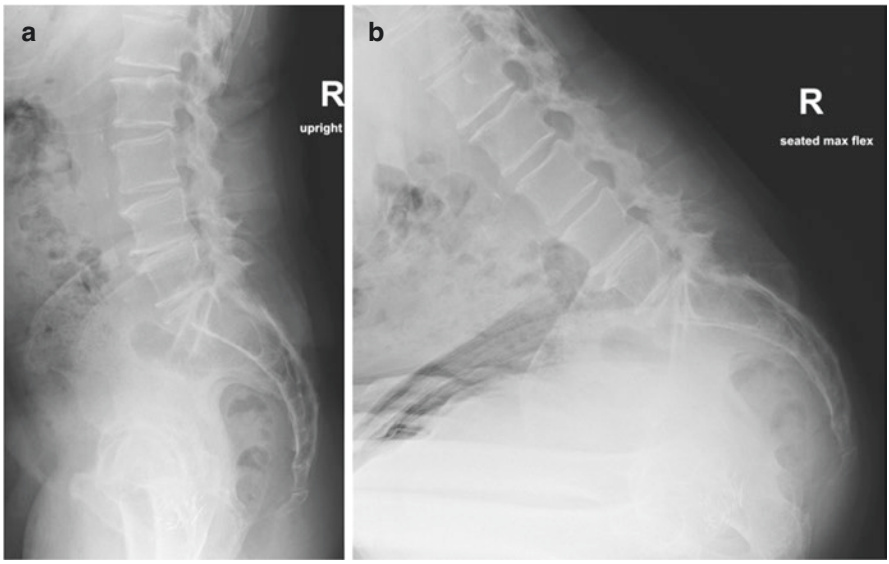

Fig. 12.2 Abnormal sagittal radiographs in hip-spine syndrome demonstrates hip and spine arthritis in a patient with hip-spine syndrome. In patients with hip-spine syndrome, the hips and lumbar spine lose the ability to rotate, flex, and extend normally, leading to abnormal orientations in standing (**a**) and sitting (**b**)

Clinical Presentation

Patients with hip-spine syndrome typically suffer with symptoms from both hip OA and DSD. It is important to differentiate which symptoms may be emanating from the hip versus those symptoms arising from the spine to best target therapy.

Like all health conditions, a thorough history and physical examination are necessary for the accurate diagnosis of patients with hip and back pain. The location and quality of pain may vary among patients with hip OA, who most often present with groin pain, which has been found to be 84% sensitive and 70% specific [7]. The presence of groin pain is more likely to suggest hip pathology or hip-spine syndrome, rather than isolated spinal degeneration [8]. Patients with hip OA may also report pain that radiates to other areas, particularly the buttocks region in 70% of patients [9] and the knee in 50% of patients [10]. Patients may also present with lower extremity weakness, either from deconditioning or as a result of pain.

A general musculoskeletal evaluation of the hip should include watching the patient walk to see if they have a limp, followed by inspection of the skin; identification of focal tenderness, including the lateral hip at the greater trochanter; and

comparison of passive and active range of motion of both the hip and knee to assess pain and stiffness. Passive flexion and rotation of the hip may often cause pain as a result of hip degeneration. Muscle weakness can be the result of pain, age-related changes, or atrophy related to nerve root impingement from spine disease.

In patients with DSD, back pain alone is neither specific nor sensitive. A significant component of DSD symptoms can be attributed to spinal stenosis, which can compress nerves that provide motor and sensory function to the lower extremities. Radiculopathy or radiating pain in the buttocks and legs is commonly reported. Patients with neurogenic claudication will experience worsening lower extremity pain or tingling with standing and ambulation, particularly when going downhill, but have immediate relief with sitting and forward flexion of their spine. The classic sign of neurogenic claudication is the "shopping cart sign," which allows a patient to walk while leaning forward on their shopping cart, thereby relieving compression of the spinal canal. Severe DSD can cause nerve impingement, leading to weakness, decreased sensation, or myelopathy.

A focused physical exam of the spine includes inspection of spinal alignment in extension and flexion from behind, as well as evaluating for kyphosis or lordosis from the side. Examination of the lumbar spine should also include range of motion with forward flexion and backward extension, along with right and left lateral bending, as well as neurovascular testing of sensation, motor strength, reflexes, and pulses in both lower extremities. The passive straight leg raise test may reproduce radiculopathy as a result of excessive tension on lumbar nerves that are compressed by bulging discs or arthritic joints in the lumbar spine. When neural compression is suspected, it is critical to assess for signs of upper motor neuron dysfunction.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis for hip pain can be vast and is beyond the scope of this chapter, but many of the more common causes are listed in Table 12.1. A thorough history and physical exam along with comprehensive radiologic studies of the hip are necessary to help reach the appropriate diagnosis.

Similarly, back pain can be caused by a wide array of pathologies. Trauma and osteoporosis can cause fractures of the spine, while degenerative changes may include spondylosis and diffuse idiopathic skeletal hyperostosis. It is important to consider a rheumatologic workup, especially in younger patients or patients with a personal or family history of autoimmune disorders. Less commonly, back pain may result from neoplastic disease or spinal infections which may manifest with significant neurologic compromise before patients seek treatment. For additional information on DSD, please refer to Chaps. 6 and 7.

The clinical presentation of hip OA and DSD can often overlap, and thus further diagnostic workup is critical to identifying which disease is causing each symptom. Depending on the primary complaint of pain, the diagnostic workup should include x-rays of the hips or lumbar spine, or both. Radiographic evidence of hip

Table 12.1 Differential diagnosis of hip pain

Intra-articular causes	Extra-articular causes
Labral tears	Trochanteric bursitis
Articular cartilage injuries	Ischial bursitis
Degenerative arthritis	Psoas bursitis
Osteonecrosis	Osteitis pubis
Loose bodies	Sports hernia
Synovial diseases Pigmented villonodular synovitis Synovial chondromatosis Gout Pseudogout	Deep gluteal syndrome
Fractures Traumatic Pathologic Stress	Piriformis syndrome
Dislocations	Hip flexor tendinitis
Infection Septic arthritis Osteomyelitis Psoas abscess	Hip adductor tendinitis
Neoplasm Primary neoplasm Metastatic disease	Hip abductor tendinitis
Adhesive capsulitis	Internal snapping hip
Inflammatory/autoimmune arthritis	External snapping hip
Transient osteoporosis of the hip	Gluteus medius tear
	Gluteus minimus tear
	Adductor tear
	Hamstring tear
	Sacroiliac joint pain
	Spine problems
	Genitourinary problems
	Endometriosis
	Ovarian cyst

osteoarthritis includes the classic findings of joint space narrowing, subchondral cysts, sclerosis, and osteophyte formation as seen in Fig. 12.3. If further clarification is necessary, advanced imaging such as MRI, MR arthrogram, or CT scan may be warranted. Similarly, XR of the spine may show osteophyte formation, collapsed disc space, sclerosis, and subchondral cyst formation as seen in Fig. 12.4. If further investigation is necessary, MRI or CT scan may be pursued. Of note, it is not recommended to obtain MRI of the spine for isolated back pain as 80% of adults over the age of 50 have degenerative disc disease on imaging and few require surgical intervention [11]. MRI is usually obtained when history or physical exam suggest neural impingement, as evidenced by significant motor weakness, loss of sensation, absent reflexes, bowel incontinence, or urinary retention.

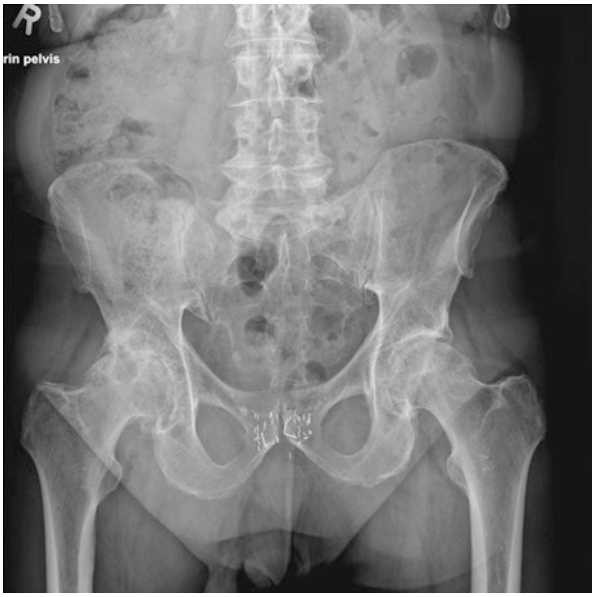

Fig. 12.3 Radiographic evidence of hip osteoarthritis demonstrates characteristic findings of hip osteoarthritis on radiographs, including loss of joint space, sclerosis, subchondral cysts, and osteophyte formation. As seen in this example, there is more advanced arthritis in the right hip compared to the left hip

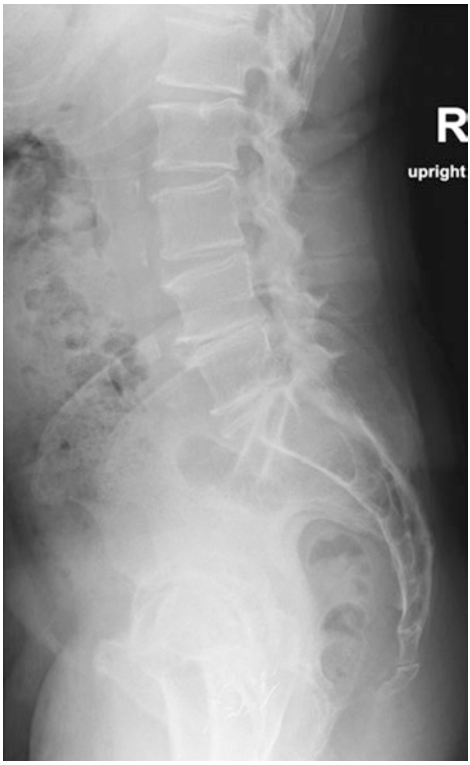

Fig. 12.4 Radiographic evidence of degenerative spine disease demonstrates classic findings including osteophyte formation, collapsed disc space, sclerosis, and subchondral cyst formation

In addition to advanced imaging, fluoroscopically guided injections of the hip or spine may be both diagnostic and therapeutic. In patients with hip osteoarthritis, studies have shown that patients who achieve more than 50% pain relief after intra-articular hip injection are more likely to achieve pain relief after total hip arthroplasty (THA) with high sensitivity and specificity [12, 13]. A lack of pain relief following an intra-articular hip injection may suggest pathology outside of the hip joint, such as trochanteric bursitis or pain radiating from spinal pathology. Similarly, in patients with back pain, epidural steroid injections (ESI) can help determine if spinal pathology alone is contributing to symptoms or if other etiologies need to be ruled out. While an improvement in pain after ESI usually indicates spinal pathology, a lack of improvement does not rule out spinal stenosis.

Nonoperative Management

Initial treatment of hip OA and DSD may include ice, rest, modified activities, and NSAIDs. Around-the-clock NSAIDs for 2 weeks can be helpful in reducing joint inflammation and pain for patients who don't have medical conditions that preclude taking NSAIDs. Physical therapy treatments including strengthening, stretching, and other modalities should also be prescribed for at least 6 weeks when possible, depending on each patient's symptoms. For some patients, bracing and assistive devices may also be helpful. In patients with high BMI, weight loss can help reduce stress on both the hip and spine. Nonoperative interventions such as intra-articular corticosteroid injections in the hip and epidural or facet joint injections in the spine may offer short-term relief of symptoms. Providers may wish to reserve cortisone injections for patients with evidence of osteoarthritis as certain steroids are toxic to chondrocytes and may accelerate cartilage degeneration. For patients who do not want surgery or may not benefit from surgical treatment, radiofrequency ablation, nerve blocks, and other nonsurgical procedures may be initiated by an interventional pain specialist [14, 15].

Indications for Surgery

Patients with hip-spine syndrome who have failed nonoperative management and whose pain and other symptoms result in functional limitations should be referred to both hip and spine specialists. Patients with DSD who have neurologic compromise, such as weakness, loss of sensation, bowel incontinence, or urinary retention, should be evaluated urgently by a spine surgeon. It is important in hip-spine syndrome that both arthroplasty and spine surgeons can coordinate early on in the decision-making process, as each case is unique and may require tailored surgical strategies.

Operative Management

THA, replacement of both the acetabular cartilage and femoral head, is the gold standard treatment for patients with end-stage osteoarthritis of the hip who have failed nonoperative management. The goal of surgery is not to cure the disease, but rather to remove the diseased cartilage and bone in hopes of offering pain relief and functional improvement.

Spinal decompression and/or fusion may benefit patients with DSD who have failed nonoperative management. The goal of spinal decompression and/or fusion is to provide pain relief and functional improvement. In cases where neurologic compromise has occurred prior to surgery, decompression and/or fusion may not always reverse that damage but will hopefully stop further progression.

While hip OA and DSD are separate pathologies, they can overlap and result in hip-spine syndrome. Indications and timing for surgical intervention may be different in each case. It behooves the patient and providers to coordinate early in the management process so that the appropriate surgical strategy can be realized.

Expected Outcome and Predictors of Outcome

Patients with hip-spine syndrome often present with complex symptoms, and physical exam findings that can make diagnostic and treatment decisions more challenging, and outcomes of treatment less successful than in isolated hip OA or DSD. Because of the overlap of symptoms and physical exam findings, patients should be counseled that separate surgeries for the hip and back may be necessary to achieve maximum relief. While most patients can be treated nonoperatively, surgery may be appropriate for patients with advanced hip arthritis, or degenerative spine disease with pain and neurologic deficits.

In general, patients report good pain relief after THA for hip OA. Most patients are ambulating the day of surgery. Depending on surgeon protocol and patient-specific factors, patients can be discharged same day (no hospitalization) or admitted for a short stay in the hospital after surgery (usually 1–2 days). Similarly, spinal decompression for select cases of DSD can offer immediate pain relief and return to normal activities of daily living. However, in the case of neurologic deficits secondary to spinal cord or nerve root compression, surgery may not reverse those deficits, but will hopefully prevent progression of those deficits in the future.

Summary

Hip and spine degenerative diseases are common among adults. In patients with hip-spine syndrome, a thorough history and exam as well as appropriate imaging studies are necessary to determine the underlying pathologies. Patients who have

Table 12.2 Summary of hip-spine syndrome with a synopsis of presentation, diagnostic testing, and suggested management strategies

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Hip osteoarthritis	Insidious onset of pain surrounding the hip region, including the groin and buttocks, and can be referred to the knee Pain worse with ambulation, extreme range of motion, and after prolonged use Stiffness	Radiograph MRI if clincal exam and radiographs are equivocal Fluoroscopically guided intra-articular cortisone injection can be both diagnostic and therapeutic	With early symptoms consider rest, ice, NSAIDs, and physical therapy May require assistive device such as cane or walker In persistent cases, cortisone injection	Surgery indicated for persistent symptoms despite nonoperative treatment Total hip arthroplasty or hemiarthroplasty
Degenerative spine disease	Insidious onset of back pain, and can refer to the hip and/or knee Radiculopathy Myelopathy Weakness Numbness Pain worse with back extension and improves with flexion Stiffness	Radiograph MRI if history or exam reveals neurologic compromise in addition to back pain Fluoroscopically guided epidural steroid injection can be both diagnostic and therapeutic	With early symptoms consider rest, ice, NSAIDs, and physical therapy May require assistive device such as cane or walker In persistent cases, epidural steroid injection	Surgery indicated for debilitating pain, pain persists despite 6 + weeks of nonoperative treatment, or presence of neurologic deficits Spinal decompression with or without fusion

exhausted nonoperative management should be referred to both hip and spine surgeons to be evaluated for possible surgical intervention (Table 12.2).

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Part IV
The Shoulder and Elbow

Chapter 13
Shoulder Soft Tissue Injuries

Courtney K. Dawson

Abbreviations

AC	Acromioclavicular
AP	Anterior-posterior
MRI	Magnetic resonance imaging
NSAIDs	Nonsteroidal anti-inflammatory drugs

Adhesive Capsulitis

Summary of Epidemiology

While many patients presenting with a painful and stiff shoulder are diagnosed with "frozen shoulder," adhesive capsulitis is a specific pathological condition wherein chronic inflammation of the shoulder capsule leads to capsule thickening, fibrosis, and adhesion to the humeral neck. As a result, there is decreased synovial fluid within the joint with diminished overall joint volume. This produces pain and mechanically restrains shoulder motion (Fig. 13.1).

Commonly encountered in the outpatient setting, the prevalence of adhesive capsulitis ranges from 2% to 5% but can be as high as 30% in patients with insulin-dependent diabetes mellitus (IDDM). Comorbid IDDM is associated with a worse prognosis and increased likelihood for surgical intervention. While the exact pathogenesis remains unclear, other factors associated with adhesive capsulitis include female sex, age over 40 years, prolonged immobilization, sedentary lifestyle, trauma, thyroid disease, stroke, myocardial infarction, and the presence of an autoimmune disease. Most cases occur in women 40–60 years of age.

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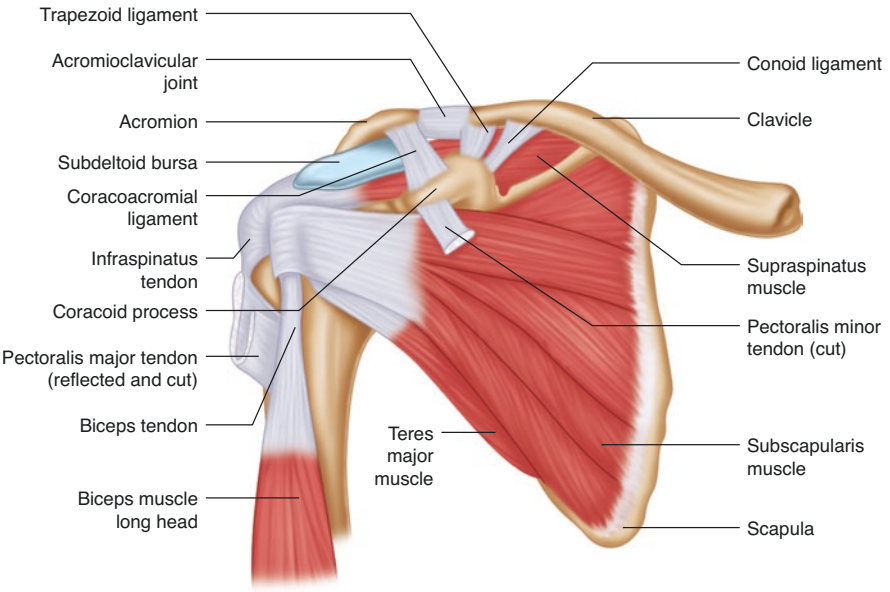

Fig. 13.1 Shoulder anatomy (anterior view)

Clinical Presentation

Adhesive capsulitis is characterized by an insidious onset of shoulder pain for several months with a global limitation of both active and *passive* range of motion. While there may be a history of recent trauma to the shoulder, this is not always the case. Pain is worsened with motion and may be referred to the deltoid region. Night pain and difficulty sleeping on the affected side are also common. The gradual loss of motion may result in difficulty dressing, combing hair, reaching backward, or fastening a brassiere.

The disease progression of adhesive capsulitis has been described as occurring in four stages. Stage 1, the pre-adhesive stage, is characterized by a fibrinous inflammatory synovitic reaction without adhesion formation. In this early stage, patients usually have full motion but with pain, often at night. Misdiagnosis is common at this stage. Stage 2 progresses to an acute synovitis with synovial proliferation and early adhesion formation. Pain becomes more prominent, but loss of motion remains mild. Stage 3 is referred to as the maturation stage where inflammation and pain have decreased; however, more fibrosis is present and range of motion becomes further limited. Stage 4, the chronic stage, is characterized by mature adhesions resulting in severely reduced motion.

Differential Diagnosis and Suggested Diagnostic Testing

Adhesive capsulitis may be challenging to diagnose in the early phases, but more readily declares itself as the symptoms progress. Perhaps the most important consideration is the loss of *passive motion* in addition to active motion. While numerous painful conditions about the shoulder can generate pain or limit active motion, relatively few substantially limit passive motion. Glenohumeral arthritis is a common cause of limited passive range of motion and should be considered in the differential diagnosis. Plain radiographs can be used to distinguish these two diagnoses as the glenohumeral joint space is usually preserved with adhesive capsulitis.

Examination of a patient with shoulder pain should proceed in a thoughtful and systematic fashion. Inspection and palpation are followed by observation of active range of motion of the shoulder joint as well as the neck. At the shoulder, active forward flexion, abduction, functional internal rotation (reaching behind and up back), and external rotation should be assessed. To evaluate passive motion, the examiner repeats the above motions while the patient is relaxed. In adhesive capsulitis, motion restriction is most pronounced with external rotation of the shoulder with the arm at the side and the elbow in 90° of flexion. Motion should be compared to the contralateral side. Typically, rotator cuff muscle strength will be preserved (although weakness may be seen due to pain inhibition), and other special tests of the shoulder will often be negative.

In patients with substantially limited passive motion of the shoulder, plain radiographs (AP, lateral, and axial views) must be obtained. In patients with a history of shoulder instability, trauma, or seizure, the axial view is of particular importance to rule out shoulder dislocation, as this can also severely limit passive motion and should not be missed. As noted above, osteoarthritis of the shoulder is common and often restricts passive shoulder motion. In patients with adhesive capsulitis, radiographs are usually normal but may show osteopenia. Magnetic resonance imaging (MRI) and other advanced imaging modalities are typically not used as initial diagnostic tools, however can be helpful to evaluate for other structural pathologies (e.g., rotator cuff tear) and to confirm the presence of findings consistent with adhesive capsulitis such as thickening of the joint capsule and fibrosis of the axillary pouch.

Nonoperative Management

Regardless of stage, physical therapy with a bridge to a home exercise program is the mainstay of treatment. The goal is gentle progressive stretching—aggressive movements are not needed and may exacerbate pain. It should be emphasized to patients that recovery can take a long period of time, up to or more than a year. While one might think the inflammatory nature of adhesive capsulitis would lend itself to successful anti-inflammatory treatments, this is frequently not the case.

Nonsteroidal anti-inflammatory drugs (NSAIDs) may be used to control pain, however have not been found to alter the disease course. Oral steroids and intra-articular corticosteroid injections may reduce pain but do not typically improve long-term outcome. Intra-articular injections can, however, be very helpful in allowing the patient to tolerate the advancing passive range of motion in physical therapy.

Indications for Surgery

Most patients do not require surgery for adhesive capsulitis. The conservative management strategies outlined above may continue for up to a year if slow progress is being made. Surgery is reserved for patients with persistent or intractable painful restriction despite an adequate trial of conservative management. Those who have more severe initial symptoms and are younger in age and those who have an ongoing reduction in motion despite at least 6 months of diligent physical therapy are more likely to be considered for surgery.

Operative Management

Before widespread availability of arthroscopy, manipulation under anesthesia was the treatment of choice for cases of adhesive capsulitis refractory to conservative measures. This involves a carefully planned manipulation technique to ensure the tightened capsule is ruptured while avoiding damage to other bony or soft tissue structures such as the subscapularis or humerus. Results are generally favorable, with most patients regaining the ability to do daily tasks within days of the procedure.

More recently, arthroscopic capsular release has overtaken manipulation as the surgical treatment of choice as this allows for intra-articular inspection and confirmation of diagnosis, followed by a more precise capsulotomy. Results are generally favorable and maintained. Postoperative range of motion is important to preserve the gains made in surgery, particularly in abduction.

Expected Outcome and Predictors of Outcome

The natural history of adhesive capsulitis is not entirely understood. Some feel this is a self-limiting process and does not need to be treated aggressively. With minimal intervention, subjective outcomes tend to be favorable; however, objective measures show that patients do not at all fully recover. Uncertainty of the natural history of adhesive capsulitis complicates studying the efficacy of various treatment options. Patients may be substantially limited for a prolonged period of time; thus, interventions typically focus on improving the speed of recovery and decreasing pain.

Biceps Tendinopathy

Summary of Epidemiology

Biceps tendon pathology is a common cause of anterior shoulder pain. This inflammatory tenosynovitis occurs as the long head of the biceps tendon courses in its relatively constrained position within the bicipital groove of the humerus. Although biceps tendonitis may exist in isolation, it is frequently associated with other shoulder pathology. This is not surprising, given the long head of the biceps has an intra-articular proximal insertion at the supraglenoid tubercle and lies in close proximity to both the supraspinatus and subscapularis tendons within the rotator interval.

The terms tendinopathy and tendonitis are often used interchangeably when referring to biceps tendon pain, although they are two different conditions. Tendinopathy is usually more chronic in nature and refers to degeneration of the collagen within the tendon substance. Tendonitis is often more acute in nature and refers to inflammation of the tendon and its surrounding tendon sheath. These two distinct conditions can exist simultaneously.

Clinical Presentation

Patients with biceps tendinopathy often describe progressive anterior shoulder pain that may be associated with repetitive overhead activities. The pain may be localized to the anteromedial shoulder in the region of the bicipital groove and may radiate downward toward the biceps muscle belly. Younger overhead throwing athletes or overhead laborers may have isolated biceps tendonitis. In the instance of proximal biceps rupture, patients may describe feeling a sudden, sharp pain in the upper arm or an audible pop or snap. This may be accompanied by subtle weakness in forearm supination or elbow flexion and a prominence of the biceps musculature (i.e., “Popeye” sign).

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis for anterior shoulder pain is broad and includes impingement syndrome, rotator cuff tendinopathy or tears, AC joint pathology, labral pathology, subacromial bursitis, glenohumeral instability, and cervical spine pathology.

In cases of true biceps tendinopathy, physical exam may reveal point tenderness elicited with direct palpation over the bicipital groove. This should be done with the arm at the patient’s side and in slight internal rotation (approximately 10°) to bring the biceps groove into a forward-facing position. Pressure approximately 5 cm below the edge of the acromion may elicit pain. The contralateral side may be tested for comparison.

Speed's test is positive if pain in the bicipital groove is elicited with resisted forward flexion of the arm with the forearm supinated, the elbow extended, and the humerus in 90° of forward flexion. Yergason's test is another special test that can be helpful in attributing a patient's pain to biceps pathology. It is important to note that the biceps muscle is a strong supinator of the arm, but a weak flexor relative to the brachialis muscle. A patient is asked to hold their elbow flexed to 90° with their arm at their side and then supinate the forearm. The examiner applies manual resistance to supination. Reproduction of a patient's shoulder pain with this test implicates the biceps as a pain generator.

As noted above, in the case of proximal biceps rupture, gross deformity of the biceps muscle may be present in the form of a "Popeye" sign. This may be made more obvious by asking the patient to contract the muscle. Bruising may be present from the upper arm down toward the elbow.

A careful and thoughtful physical examination will usually suffice in diagnosing biceps tendonitis as the source of a patient's pain and may also yield information about associated shoulder pathology. Plain radiographs (AP, lateral, and axial views) of the affected shoulder are helpful in investigating other sources of shoulder pain such as underlying bony abnormalities, particularly in the setting of recent trauma, and may show evidence of rotator cuff calcific tendonitis or suggest chronic rotator cuff insufficiency. Generally, once the diagnosis is confirmed on physical examination, conservative management may be trialed prior to obtaining advanced imaging.

In patients where the diagnosis is unclear, MRI is the imaging modality of choice given its ability to evaluate soft tissue abnormalities of the shoulder. When multiple potential pain generators or abnormal findings are present, local injection to the bicipital groove with short-acting analgesics and corticosteroids may be used (typically guided by ultrasound) to gain valuable diagnostic information regarding the primary pain generator while also providing therapeutic relief.

Nonoperative Management

The vast majority of patients with biceps tendinopathy will be successfully managed with nonoperative treatment. This includes a brief period of rest and activity modification. The use of NSAIDs can be helpful for analgesia. This should be followed by formal physical therapy to optimize scapular biomechanics and address any concomitant issues such as muscle imbalance. As biceps pathology is most often seen with concomitant shoulder dysfunction, the goal of physical therapy is to restore proper shoulder biomechanics rather than focusing solely on the biceps tendon. As opposed to other joints in the body (such as the hip joint), which have deep sockets and much inherent bony stability, the shoulder is more akin to a golf ball on a relatively shallow tee. As such, soft tissue and muscle balance are critically important. Educating patients on the purpose of the targeted physical therapy may help optimize nonoperative results and improve adherence to a program, as it may take weeks or months to see improvements.

In most cases, nonoperative treatment is similarly pursued for complete tears of the long head of the biceps tendon. In such an instance, referral to an orthopedic surgeon is warranted to discuss the risks and benefits of surgery versus nonoperative treatment. Surgery is most beneficial for higher-demand patients such as athletes or manual laborers.

Indications for Surgery

Indications for surgical management include partial-thickness tears of the long head of the biceps tendon involving more than 25–50% of the tendon thickness, full-thickness tears in high-demand patients, medial subluxation of the tendon out of the bicipital groove, and/or subluxation in the setting of a subscapularis tendon tear or biceps pulley injury. Relative indications include certain types of SLAP (superior labrum anterior-posterior) tears and persistent pain despite an aggressive trial of nonoperative treatment.

Operative Management

Optimal surgical management of proximal biceps tendon pathology remains controversial. The two most common procedures performed are biceps tenotomy and biceps tenodesis. Tenotomy is a simple procedure where the proximal biceps is cut arthroscopically and allowed to retract into the arm, without subsequent repair. This provides predictable pain relief without the need for postoperative rehabilitation. However, unsatisfactory cosmesis related to the "Popeye" deformity and discomfort from muscle fatigue, spasm, or cramping are potential challenges in some patients. Tenotomy is typically used in older, low-demand patients with satisfactory results. Biceps tenodesis involves cutting the biceps tendon with subsequent reattachment/anchoring of the tendon at a more distal point in the humerus to maintain the length-tension relationship, strength, and biceps muscle contour. Surgeons frequently prefer this method in younger, more active patients.

Expected Outcome and Predictors of Outcome

Nonoperative management of biceps tendinopathy is often successful; however, data supporting the efficacy of specific treatment modalities is lacking. In operative cases, biceps tenotomy typically results in high patient satisfaction with reliable pain relief; however, approximately 70% of patients show the classic "Popeye" sign, and approximately 38% show fatigue discomfort with resisted elbow flexion. As such, this would not be the treatment of choice for young laborers. In these

cases, biceps tenodesis has been shown to be a relatively effective and safe procedure.

Acromioclavicular Joint Pain

Summary of Epidemiology

The acromioclavicular (AC) joint is a relatively frequent source of shoulder pain, often in the setting of primary or post-traumatic osteoarthritis. The AC joint is a diarthrodial joint which supports the shoulder girdle through the clavicular "strut." The convex distal clavicle articulates with the concave acromial facet through a fibrocartilaginous meniscal disc between the articular surfaces. Degeneration of the AC joint is a natural consequence of the aging process, and the AC joint is vulnerable to the same processes affecting other joints in the body, such as degenerative arthritis, infections, and inflammatory or crystalline arthritis. Its relationship to the shoulder and superficial location makes it susceptible to traumatic injury. The complex biomechanics of the shoulder girdle lead to large loads across a small AC joint surface area, which predisposes it to degeneration with overuse. An increased emphasis on weight training and upper extremity strengthening adds stress across the AC joint.

Clinical Presentation

Patients with AC joint pathology often present with aching pain or discomfort over the anterior and superior aspect of the shoulder. The pain may radiate to the neck or deltoid region. It is often worsened by activities such as reaching across the body while driving, washing the opposite axilla, reaching behind one's back, or rolling onto the affected side while sleeping. Push-ups, bench-pressing, and repetitive overhead activities also may exacerbate the pain. It is important to inquire about history of prior acute shoulder injuries, as instability following AC joint trauma may alter treatment.

Differential Diagnosis and Suggested Diagnostic Testing

The differential for anterior and/or superior shoulder pain includes AC joint pathology (osteoarthritis, sprain or fracture, instability, dislocation), rotator cuff impingement, biceps tendinopathy, and cervical spine pathology. As with other conditions of the shoulder, a comprehensive physical exam often reveals the diagnosis, with imaging used as confirmation.

The exam begins with inspection, which may show prominence or asymmetry over the AC joint. Direct palpation may elicit tenderness. The most reliable provocative maneuver is the cross-body adduction test. The arm of the affected side is elevated to 90° of forward flexion. The examiner then grasps the elbow and passively adducts the arm across the body. Reproduction of pain is suggestive of AC joint pathology. Stability of the clavicle at the AC joint may be evaluated by holding the distal clavicle in one hand while stabilizing the acromion with the other and testing translation. Examination should also evaluate for other diagnosis such as rotator cuff pathology or biceps tendon pathology, which may coexist.

Following examination, radiologic evaluation should include bilateral AP shoulder plain films (for side-to-side comparison) and axillary views, particularly in the setting of trauma. Adding a bilateral Zanca view is frequently helpful because this offers an unobstructed view of the distal clavicle and AC joint. Patients with degenerative AC joint arthritis will have changes such as joint space narrowing, marginal osteophytes, and sclerosis. MRI may be obtained if the diagnosis remains unclear or if associated pathology, such as a concurrent rotator cuff tear, is suspected. MRI should be obtained only after a careful history and physical exam. Reactive bone edema on MRI is a more reliable predictor of symptomatic AC joint pathology than degenerative changes on x-ray or MRI. However, a patient's clinical symptoms may not correlate with changes on MRI. It has been shown that up to 82% of patients with AC joint arthritis based on MRI are asymptomatic. Similar to the evaluation of other suspected soft tissue pathologies of the shoulder, advanced imaging does not eliminate the need for careful history and physical exam.

Nonoperative Management

Initial treatment of AC joint pain is conservative, including activity modification, nonsteroidal anti-inflammatory medications, corticosteroid injections, and physical therapy. For some patients, particularly younger athletes, activity modification may involve decreasing exercises such as bench presses, dips, and push-ups. Physical therapy is useful for treating concomitant soft tissue shoulder issues such as impingement or restricted motion; however, its role is typically limited in most cases of isolated AC arthritis.

Intra-articular corticosteroid injections are an important tool for the clinician caring for a patient with AC joint pain. While history, physical exam, and imaging often point to the correct diagnosis, many patients have vague anterior or superior shoulder pain that is not easily localized to one pain generator. An intra-articular lidocaine injection provides important diagnostic information and can confirm the diagnosis if the patient experiences pain relief shortly thereafter. Corticosteroid may additionally provide longer-lasting relief.

Indications for Surgery

Surgery is considered for patients who have failed nonoperative treatment and who have history, physical exam, and radiographic evidence confirming AC joint pathology. Prior to surgery, the patient should experience pain relief after a focal AC joint injection for further confirmation of the AC joint as the source of pain.

Operative Management

Options for surgical management of AC joint pain include open versus arthroscopic distal clavicle resection. Open resection allows for direct visualization of the resected and remaining clavicle to ensure adequate bone removal. Disadvantages of this approach include interfering with the deltoid and trapezius muscles, with associated time to heal these structures. Active shoulder flexion, elevation, and abduction are avoided in the immediate postoperative period. Arthroscopic resection may be performed via either subacromial (indirect) or superior approach. One advantage of the arthroscopic approach is the opportunity to diagnose and address concomitant pathologies at the time of surgery, as well as a potentially shorter recovery time. Arthroscopic techniques avoid injury to the deltoid but are more technically demanding than an open approach and may have somewhat higher risks of inadequate resection.

Expected Outcome and Predictors of Outcome

Outcomes after distal clavicle resection are generally positive, but there is much variability in patient response. Those with post-traumatic arthritis or AC instability may have a worse prognosis. Most patients are able to return to their prior activity after distal clavicle resection. Continued pain postoperatively should raise concern for diagnostic error, which again underscores the importance of detailed history, examination, and judicious use of diagnostic and therapeutic injections.

Rotator Cuff Pathology

Summary of Epidemiology

The evaluation and management of rotator cuff tears differ according to the patient (younger vs. older, athlete vs. nonathlete) and mechanism (acute traumatic vs. chronic atraumatic). Rotator cuff pathology accounts for a significant portion of

shoulder-related complaints presenting to the primary care physician. The rotator cuff consists of four muscle-tendon units. The supraspinatus abducts, the infraspinatus, and the teres minor externally rotate, and the subscapularis internally rotates the shoulder at the glenohumeral joint. These muscles are also important for maintaining the humeral head's concentricity within the glenohumeral joint. In cases of chronic "massive" rotator cuff tears, the powerful deltoid muscle causes superior migration of the humeral head and resultant rotator cuff arthropathy (arthritic narrowing of the subacromial space) over time.

Rotator cuff pathology may occur due to trauma or may occur gradually over time. While shoulder dislocations in younger patients more commonly result in labral pathology, in older patients, dislocations are more likely to result in traumatic rotator cuff tears. Tendinopathy and tears may also occur gradually due to overuse, such as with overhead laborers or athletes.

Subacromial impingement syndrome is a common cause of shoulder pain and also represents a spectrum ranging from subacromial bursitis and rotator cuff tendinopathy to partial-thickness rotator cuff tears. While the exact pathophysiology of impingement and partial-thickness rotator cuff tears is not entirely understood, it is thought to be due in some part to external compression from the acromion.

Calcific tendinopathy is another common cause of shoulder pain. Calcium deposits can be seen within the substance of the rotator cuff tendons, but only about one-third of patients are symptomatic. The exact etiology remains unclear. Most cases of shoulder pain due to calcific tendinitis gradually improve over time as the deposit often resorbs without intervention.

Clinical Presentation

Rotator cuff pain can present acutely in the setting of trauma, chronically in the absence of trauma, or in an acute-on-chronic fashion. Trauma may occur as a result of heavy lifting, falls, or dislocations. Acute injuries to the rotator cuff are usually accompanied by pain and a significant decline in function. Conversely, in the case of overuse injuries, older patients or overhead athletes/laborers tend to present with a more gradual onset of pain. In these patients, functional decline may be more subtle, with a gradual decrease in strength and functionality affecting activities of daily living.

Rotator cuff pathology usually presents as a dull, aching pain over the anterior and lateral aspect of the shoulder. The pain is often worsened by overhead activities, such as washing one's hair, dressing, or reaching overhead. Night pain is a common complaint, and the patient may have difficulty sleeping on the affected side.

As with any patient presenting with shoulder pain, the physical exam is critical. Upon inspection, the examiner may be able to appreciate an asymmetry over the posterior scapular region, particularly in the setting of chronic rotator cuff tears wherein tendon/muscle retraction and atrophy occur. Patients with rotator cuff

tears, impingement, and calcific tendinopathy may have decreased active range of motion due to pain and, in cases of rotator cuff tears, often have adjusted their shoulder mechanics in order to compensate for the lost rotator cuff strength. The examiner should stand behind the patient to observe scapular motion with forward flexion at the shoulder, in order to assess for scapulothoracic dyskinesia or scapular winging, which may contribute to altered shoulder biomechanics and thus worsen pain.

Rotator cuff strength should be evaluated. To test the supraspinatus, the examiner asks the patient to hold their arms abducted to 90°. The patient's arms are then brought forward approximately 30°, to align their arms with the anatomic position of the scapula. A complete inability to maintain the arm elevated against gravity may produce a "drop arm" sign, which is consistent with a significant rotator cuff tear. Resisted abduction strength—referred to as Jobe's supraspinatus test—is then tested in this position (Fig. 13.2), to evaluate the integrity of the supraspinatus tendon.

The infraspinatus is tested by having the patient flex their elbows to 90°, with the elbow tucked at their sides, in neutral position. The patient is asked to externally rotate from a neutral position while the examiner resists (Fig. 13.3).

Subscapularis strength is assessed by evaluating the patient's ability to push the back of the hand off the lower back (the lift-off test) or by asking the patient to bring both their elbows forward against resistance while the hands are held pressed against the abdomen (belly-press test) (Fig. 13.4a, b).

In addition to rotator cuff strength testing, examination should also include special tests specifically looking for impingement signs. The Neer impingement test (Fig. 13.5) involves the examiner passively flexing the patient's shoulder forward

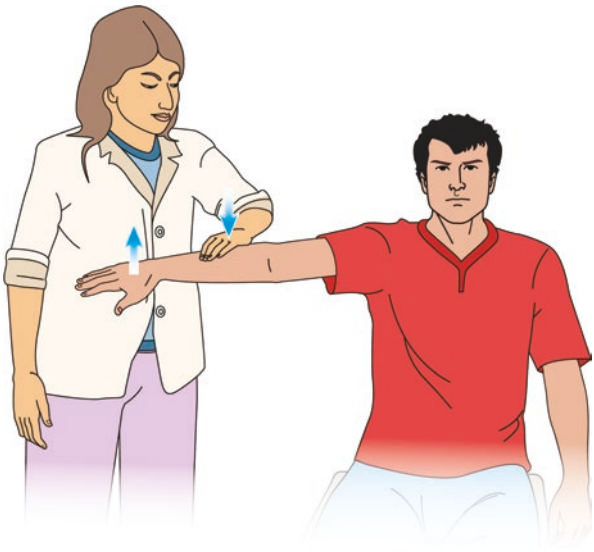

Fig. 13.2 Jobe's supraspinatus test

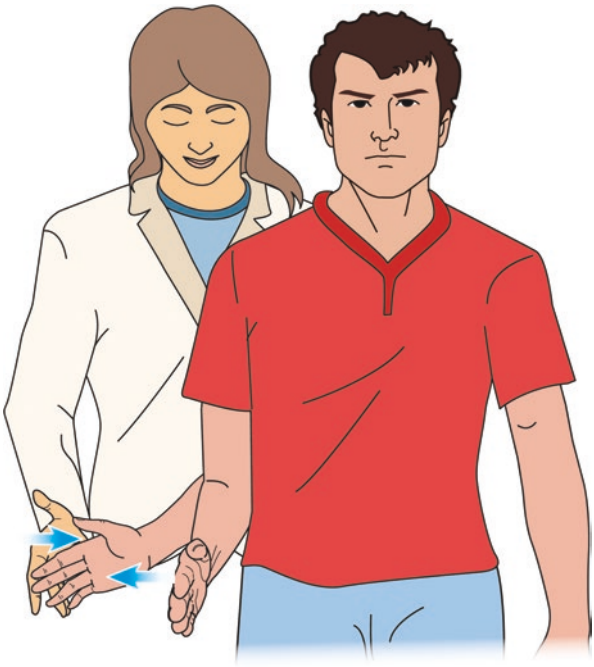

Fig. 13.3
Infraspinatus test

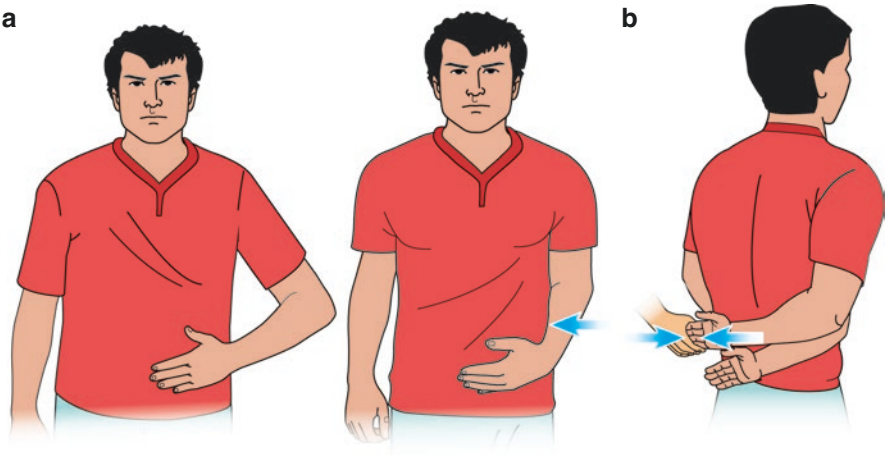

Fig. 13.4 Subscapularis tests. (**a**) Belly-press test; (**b**) Lift-off test

while using the other hand to stabilize the scapula. Pain with this maneuver, while not specific, may be indicative of shoulder impingement. The Hawkins test (Fig. 13.6) entails forward flexion of the shoulder to 90°, followed by elbow flexion and internally rotating at the shoulder. Again, pain with this test suggests impingement syndrome.

Fig. 13.5 Neer impingement test

The examiner should also consider alternative painful pathologies of the shoulder and investigate for evidence of AC joint pain (cross-body adduction test) and biceps tendinopathy (bicipital groove tenderness, Speed's test, Yergason's test). The possibility of cervical spine pathology should also be considered in appropriate patients.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis for shoulder pain is broad and depends largely on patient age and history. For young patients, the rotator cuff tendons are typically robust and pain results from acute trauma or repetitive activity. For middle-age patients, rotator cuff pathologies are a common source of pain; however, glenohumeral arthritis and adhesive capsulitis should also be considered. Biceps tendinopathy and AC joint arthritis are other common sources of pain.

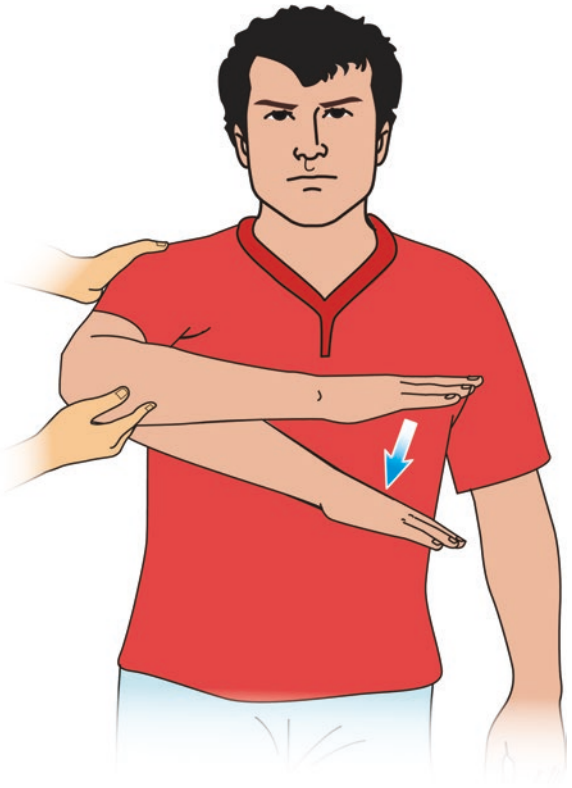

Fig. 13.6 Hawkins impingement test

Shoulder imaging does not substitute for a thoughtful history and comprehensive physical examination, as these are frequently sufficient for accurate diagnosis. For patients in whom the diagnosis is in doubt, or for whom multiple associated pathologies are suspected, imaging should begin with plain radiographs including an AP, Grashey (true AP), axillary lateral, and outlet views of the shoulder. This may reveal calcific tendonitis or other sources of pain such as glenohumeral or AC joint arthritis or rotator cuff arthropathy.

MRI is the modality of choice for imaging the soft tissue structures of the shoulder, including the rotator cuff tendons, and has been shown to reliably diagnose rotator cuff pathology. In addition to cost, an important consideration is the high false-positive rate in older patients. MRI abnormalities including rotator cuff tears are very frequently detected in older patients who are otherwise asymptomatic, and as such, MRI should be used judiciously in correlation with clinical exam to avoid overly aggressive treatment. Ultrasound is another noninvasive alternative imaging technique and allows for dynamic assessment of the rotator cuff tendons. It is frequently limited by the availability and skill of the radiologists interpreting these studies.

Nonoperative Management

The initial nonoperative treatment of rotator cuff pathology, including rotator cuff tears, calcific tendinopathy, and impingement, is generally similar and includes NSAIDs, activity modification, physical therapy, and injections. Physical therapy is the mainstay of treatment in most cases. A comprehensive exercise program should focus on rotator cuff strengthening using resistance bands, the "sleeper stretch" for posterior capsular tightness, progressive range of motion in all planes, and scapular stabilization techniques. These can be done at home, but it is often recommended that the patient learns the correct technique under the guidance of a physical therapist to avoid further injury. Once pain is better controlled, the focus of conservative treatment is on strengthening and normalizing scapulothoracic motion.

Subacromial corticosteroid injections are often used in cases of severe pain that is limiting range of motion or activities of daily living and can be used to supplement the therapy program. Most clinicians prefer to limit the total number of injections to three per year in a given shoulder due to the potential risk of tissue degeneration and tendon weakening or rupture, although the evidence for this is lacking. Subacromial injections can be performed in the office with relative ease using the posterior and lateral acromion as bony landmarks. Patients with underlying diabetes should be cautioned to monitor their blood sugars carefully after a corticosteroid injection as values can become transiently elevated in some cases. Additional treatment modalities for calcific tendinopathy include needling, image-guided aspiration/lavage of the calcium deposit, or extracorporeal shock wave therapy.

Indications for Surgery

As with other etiologies of shoulder pain, surgery is reserved for patients who have failed to improve despite a comprehensive conservative treatment program. Acute, traumatic rotator cuff tears are the exception. These tears generally occur after a specific traumatic event and should be easily identified based on the patient's history. Acute pain, weakness, and limited active range of motion after a traumatic event should raise suspicion and guide the diagnostic workup. Plain films are used to rule out a fracture, and an MRI can be used to confirm the diagnosis of a tear (Fig. 13.7). In cases of acute, traumatic rotator cuff tears in healthy individuals, surgery is often considered; therefore, early referral to an orthopedic surgeon is warranted. A delay in treatment may lead to chronic changes which cause the tear to be irreparable.

Chronic, massive, retracted rotator cuff tears with associated muscle atrophy are usually treated conservatively and may yield good results in lower-demand individuals. Some of these patients progress to rotator cuff arthropathy, at which point a reverse total shoulder arthroplasty may be considered.

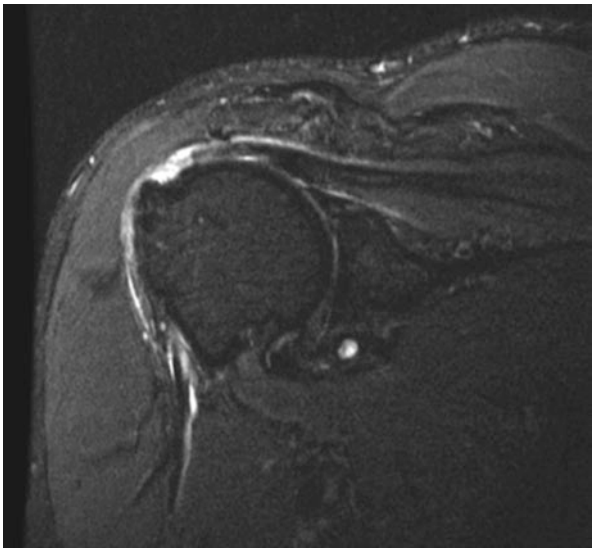

Fig. 13.7 Coronal STIR MRI: full-thickness supraspinatus tear

Operative Management

Rotator Cuff Tear

Rotator cuff repair can be performed either arthroscopically or open, depending on the size of the tear and surgeon preference. The main goal is reattachment of the torn, retracted tendons back to the humerus. Specific techniques can vary but include single- vs. double-row repairs and suture anchors vs. transosseous techniques. Biomechanical studies have shown double-row repairs to have a higher load to failure and possible improved tendon healing; however, these advantages have not been matched clinically as outcomes have been similar when compared to single-row repairs. Advances in arthroscopic instrumentation have led to better visualization and potentially faster recovery without the risk of postoperative deltoid dysfunction.

Calcific Tendinopathy

Calcific tendinopathy that is recalcitrant to conservative treatment may be treated surgically with an arthroscopic debridement of the calcium deposit under direct visualization. After debridement, the void in the rotator cuff left by the calcium deposit may require direct repair if greater than 50% of the tendon substance is affected. In these cases, the postoperative recovery is similar to a standard rotator cuff repair, requiring protection in a sling and a guided therapy program to allow for tendon healing.

Impingement Syndrome

The surgical management of impingement syndrome is somewhat controversial as most patients improve with conservative treatment. Arthroscopic subacromial decompression with or without acromioplasty is the mainstay for patients who fail to improve. There has been some debate among orthopedic surgeons as to the role of the acromioplasty as there is more recent data showing no long-term benefit when compared to a structured exercise program.

Expected Outcome and Predictors of Outcome

Outcomes after rotator cuff repair are generally favorable. The re-tear rate at 6 months is approximately 20%; however, this is usually atraumatic and often associated with massive initial tears and lower quality tissue. Despite this, the majority of patients still report clinical improvement at long-term follow-up. There is a risk of developing adhesive capsulitis postoperatively, often resolving with formal physical therapy and rarely requiring a manipulation or arthroscopic release. This is more common in diabetics or with prolonged immobilization. Arthroscopic debridement of calcific tendinopathy can yield high patient satisfaction and excellent functional results with minimal downtime. Slings are used for comfort, and physical therapy is initiated to avoid persistent stiffness. Outcomes after subacromial decompression for impingement syndrome are positive but likely no better than conservative treatment in many cases.

Summary

Shoulder pain is a common yet challenging entity presented to primary care providers. The differential is broad, and there is considerable overlap between the various etiologies of pain. Obtaining a thorough history and careful physical exam will help the provider to more clearly identify the specific diagnosis in almost all cases. Advanced imaging can be reserved for cases where the diagnosis is less clear or if the patient fails to respond to initial conservative treatment methods. Referral to an orthopedic surgeon is recommended in cases of acute traumatic rotator cuff tears, fractures, considerable weakness, and concerning physical exam findings or if there is a question as to the appropriate treatment in a given patient. In most cases, a trial of conservative treatment is warranted and is often successful.

Table 13.1 shows a standard algorithm for the evaluation and treatment of common soft tissue shoulder pathology. Note that there is considerable overlap in the initial conservative treatment of many of these common diagnoses.

Table 13.1 Summary of common soft tissue shoulder pathology, evaluation, and treatment

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Adhesive capsulitis	Insidious onset, pain, limited AROM[a] and PROM[b]	Plain films	NSAIDs, PT focusing on aggressive PROM and then AROM, intra-articular injection	Failure to respond to conservative treatment	Manipulation under anesthesia, arthroscopic capsular release
Biceps tendinopathy	TTP at the bicipital groove, positive Speed's and Yergason's tests	Clinical diagnosis often sufficient, MRI or ultrasound to confirm	NSAIDs, PT, ultrasound-guided bicipital sheath injection	Failure to respond to conservative treatment	Biceps debridement or tenotomy (older, less active), biceps tenodesis (younger, more active)
Long head biceps tendon rupture	Acute traumatic event, usually a "pop" or "snap," ecchymosis, swelling, "Popeye" sign	Clinical diagnosis often sufficient, plain films to rule out fracture if needed	NSAIDs, PT, or home exercises	None except for rare cases of young, active patients/ laborers	None except possible biceps tenodesis in young, active patient
AC joint pain	TTP at the AC joint, positive cross-body adduction maneuver	Plain films, MRI to evaluate for concomitant pathology if needed	NSAIDs, PT, image-guided AC joint or subacromial injections	Failure to respond to conservative treatment	Arthroscopic or open distal clavicle excision
Rotator cuff tear	Pain, weakness in rotator cuff testing, "drop arm" sign, positive Jobe's test	Plain films, MRI to confirm diagnosis	NSAIDs, PT, subacromial injections	Acute, traumatic tear with weakness Failure to respond to conservative treatment	Arthroscopic or open rotator cuff repair, subacromial decompression
Calcific tendinopathy	Often severe pain, AROM may be limited due to pain, positive Neer and Hawkins tests	Plain films, occasional MRI to evaluate integrity of tendon	NSAIDs, PT, injections; if persistent consider needling, aspiration/ lavage, extracorporeal shock wave therapy	Failure to respond to conservative treatment	Arthroscopic debridement of the calcium deposit, possible rotator cuff repair

(continued)

Table 13.1 (continued)

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Impingement syndrome	Pain with overhead activity, positive Neer and Hawkins tests	Plain films, clinical exam often sufficient	NSAIDs, PT, injections	Limited, failure to respond to conservative treatment	Arthroscopic subacromial decompression

[a]*AROM* active range of motion, [b]*PROM* passive range of motion, *NSAIDs* nonsteroidal anti-inflammatory drugs, *PT* physical therapy, *TTP* tenderness to palpation

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Chapter 14
Shoulder Instability

Laura Lu, Marie Walcott, and Arnold B. Alqueza

Abbreviations

CT	Computed tomography scan
MDI	Multidirectional instability
MRI	Magnetic resonance imaging
PT	Physical therapy
RTC	Rotator cuff

Introduction

The stability of the shoulder joint depends primarily on the surrounding soft tissue structures and secondarily on bony architecture. The bony glenoid does provide stability in the short arc of motion of the shoulder, whereas static stabilizers provide stability in the medium arc of motion. Finally, muscles and tendons provide stability in the extreme ranges of motion. The soft tissue structures are divided into static stabilizers (labrum and glenohumeral ligaments) and active stabilizers (rotator cuff, deltoid, biceps, and periscapular muscles) (Fig. 14.1). The bony anatomy of the shoulder provides stability in a small range of motion, since the large humeral head articulates with the small, shallow glenoid. This lack of bony constraint allows for the shoulder to move through a wide range of motion.

Shoulder instability can be due to a single traumatic injury, repetitive activities causing microtrauma, or an imbalance of the shoulder stabilizers. Shoulder instability represents a spectrum of pathology from traumatic unidirectional instability to atraumatic multidirectional instability (MDI). For the purposes of this chapter, we

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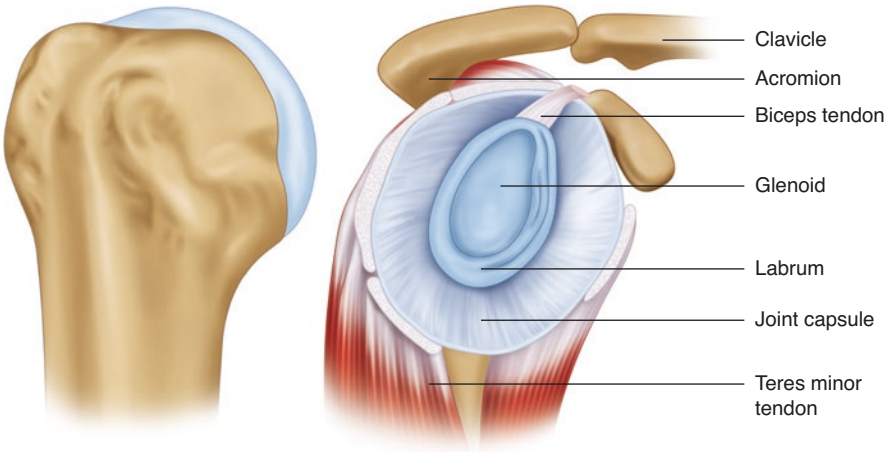

Fig. 14.1 Pertinent shoulder anatomy

group shoulder instability into three categories: unidirectional anterior, unidirectional posterior, and multidirectional. There are certainly a pathologies that do not strictly fit into these categories; however, this is beyond the scope of this chapter.

Anterior instability, commonly seen with an anterior shoulder dislocation, tends to be of traumatic origin and occurs from an anterior force on an abducted, externally rotated arm. Similarly, posterior instability can result from a posterior shoulder dislocation, caused by a seizure, electrical shock, or posterior-directed force on a forward flexed, adducted, and internally rotated arm, such as seen in blocking in football or in a motor vehicle accident. For this reason, offensive linemen have a higher incidence of posterior shoulder instability than other positions.

Multidirectional instability (MDI) is defined as symptomatic shoulder instability in two to three directions with or without hyperlaxity. Similar to the other forms of shoulder instability, MDI can result from a significant traumatic event or recurrent microtraumas, or can even be atraumatic. Patients with multidirectional instability tend to have generalized, congenital ligamentous laxity. It is important to differentiate laxity from instability when evaluating a patient's shoulder. A shoulder with hyperlaxity will typically have signs of MDI, such as a positive sulcus sign, but be completely asymptomatic. When the patient presents with both symptoms and physical findings of laxity, this is defined as instability.

Summary of Epidemiology

Anterior Shoulder Instability

In the general population, the incidence of traumatic anterior shoulder instability is approximately 1.7%. The majority of recurrences happen within the first 2 years after the initial anterior dislocation. The most important risk factor for recurrence is

age. For patients under 20 years old, research shows a >90% rate of recurrent instability. For 20- to 40-year-olds, this rate drops slightly to approximately 80%. For patients over 40, there is <20% risk of recurrent instability, but a 30% risk of concurrent traumatic rotator cuff tears. In patients over 60, the rate of concurrent traumatic rotator cuff tears increases to approximately 80%. In this group of patients, there is also an increased risk of associated greater tuberosity fracture. A particularly high-risk group is contact athletes who return to contact sports after experiencing an anterior shoulder dislocation, wherein the recurrence rate is as high as 80%.

There are several associated lesions that are seen in cases of traumatic anterior shoulder dislocation. For example, Bankart lesions (a tear of the anterior/inferior labrum) and Hill-Sachs lesions (impression fracture of the posterior humeral head) have been reported in nearly 100% of patients with an anterior shoulder dislocation (Figs. 14.2 and 14.3). Bony Bankart lesions (avulsion fracture of the anterior/

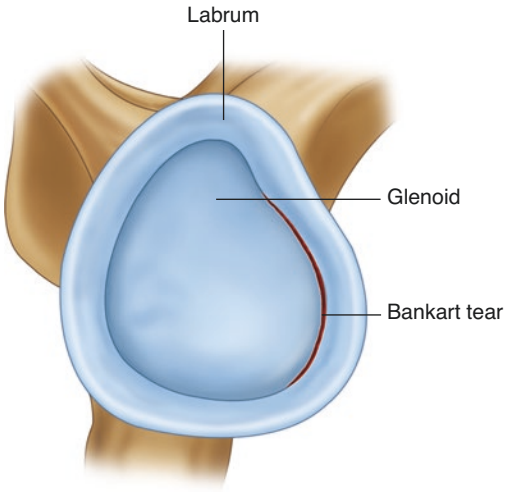

Fig. 14.2 A Bankart tear is a tear of the anterior/inferior labrum. Sometimes this is associated with a Hill-Sachs lesion

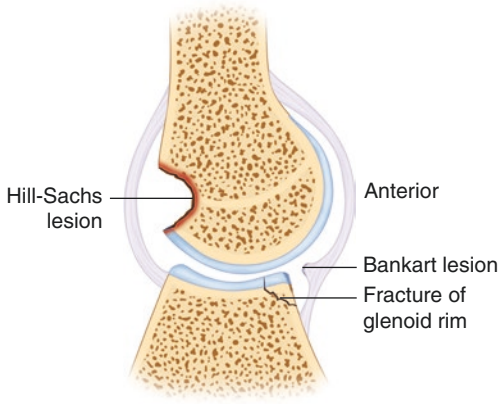

Fig. 14.3 A Hill-Sachs lesion is the result of the humeral head recoiling into the anterior glenoid when the shoulder is dislocated

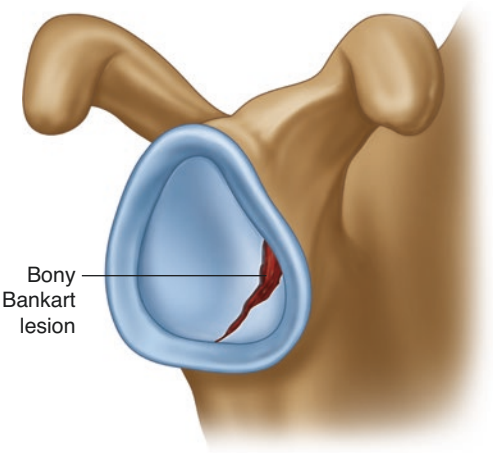

Fig. 14.4 A Bony Bankart lesion with the glenoid fracture segment attached to the labrum

inferior glenoid) are also seen frequently (Fig. 14.4). While there are other lesions associated with traumatic anterior shoulder instability, these are the most common.

Posterior Shoulder Instability

Posterior shoulder instability comprises only a minority of all cases of shoulder instability, but the incidence is increasing as it is now more recognized. In posterior instability, approximately 50% of cases are traumatic. This can be due to an isolated trauma such as frank dislocation; however, this can also be seen in cases of recurrent subtle subluxation events. Atraumatic posterior shoulder instability can be associated with a bony defect of the glenoid; thus, careful review of imaging is important.

Similar to anterior dislocations, there are associated lesions commonly seen in cases of posterior shoulder dislocation. In contrast, however, posterior shoulder dislocations result in an associated lesion only 65% of the time. These include fractures of the lesser or greater tuberosity, reverse Hill-Sachs lesions (impression fracture on the anterior aspect of the humeral head), and fractures of the posterior glenoid rim (the reverse of Fig. 14.3).

Multidirectional Instability

Multidirectional instability has an increased incidence in the second and third decades of life and an increased incidence in overhead athletes such as those who participate in volleyball, swimming, or gymnastics. Multidirectional instability can also be associated with generalized hyperlaxity.

Clinical Presentation

In cases of shoulder pain, the localization of symptoms is helpful in determining the source of the patient's pathology. For example, pain at the posterior joint line may be attributable to a posterior labrum or infraspinatus injury, while anterior shoulder pain suggests involvement of the subscapularis, biceps, or anterior labrum. Pain over the lateral aspect of the shoulder is most consistent with superior or superior-posterior rotator cuff pathology. In patients under 40 years old who present with shoulder pain, clinicians should maintain a high level of suspicion for shoulder instability.

Anterior Shoulder Instability

Anterior shoulder instability typically presents after a distinct dislocation event or in cases of recurrent subluxations, with asymptomatic periods in between. Many patients report an unstable feeling of their shoulder in the position of abduction and external rotation.

Posterior Shoulder Instability

Posterior shoulder instability can present with symptoms of subtle instability or pain, however, often without obvious symptoms of instability. For example, an athlete may complain of posterior shoulder pain at the end of a sporting event due to fatigue of the dynamic shoulder stabilizers (e.g., rotator cuff), thus unveiling pain due to injury to the static stabilizers. For patients involved in a trauma, there needs to be a high level of suspicion for posterior shoulder dislocation, as this is commonly missed at initial presentation.

Multidirectional Shoulder Instability

The diagnosis of multidirectional shoulder instability is less straightforward. Patients frequently present with the insidious onset of activity-related shoulder pain rather than a sense of instability. Symptoms are often vague and not localizable. Due to this insidious onset, patients may compensate by avoiding certain shoulder positions that provoke symptoms. A thorough history should be obtained, including assessment of other joint problems (such as patellofemoral instability) and/or a family history of collagen disorders.

Essential History

In all cases of shoulder instability, obtaining a focused history is essential. This history should include focused questions related to hand dominance, level of athletic activity, history of other joint injuries, and symptoms of generalized ligamentous laxity. Specific to the shoulder, it is important to assess factors such as direction and position of the arm at the time of initial trauma, position of the arm when symptoms recur, number and types of recurrences (dislocation versus subluxation), associated neurologic symptoms, magnitude of force to cause recurrent instability, and prior treatment.

Physical Exam

A systematic and complete shoulder exam is important to fully evaluate for instability. The exam should start with examination of the skin for muscle atrophy of the supraspinatus, infraspinatus, and deltoid. The axillary nerve can be injured in traumatic anterior dislocations, so motor function (manual muscle testing of the deltoid) and sensation (cutaneous distribution over the lateral deltoid) should be assessed and compared to the contralateral side. The axillary nerve can be stretched during an anterior dislocation event as it runs inferior to the glenohumeral joint. These are mostly transient neurapraxias and resolve spontaneously. The incidence is 5%, and X-ray may show that the humeral head is subluxated inferiorly.

Shoulder range of motion should be evaluated with the examiner first viewing the patient from a posterior direction to evaluate for scapular dyskinesis or scapular winging, which can be compensatory in posterior shoulder instability. Range of motion should then be assessed from an anterior direction. Shoulder range of motion includes forward flexion, abduction, external rotation with the elbow at the side, and functional internal rotation behind the patient's back (measured at the spinal level, the patient can reach with his or her thumb), followed by external and internal rotation with the patient's arm abducted to 90°.

Rotator cuff testing should include external rotation strength (testing infraspinatus), belly press and liftoff tests (testing subscapularis), and abduction strength (testing for supraspinatus). It is important to note both pain and weakness with these tests. For further details on how to carry out these tests, please see the chapter on shoulder soft tissue pathology.

Special tests for shoulder instability are useful to identify more subtle findings. If the patient has recently had a shoulder dislocation, these tests should be performed cautiously to prevent re-dislocating the patient's shoulder.

Anterior Instability

The apprehension test can be done with the patient sitting or lying supine, although performing the test supine leads to less guarding. The arm is abducted to 90° and externally rotated to the patient's end range. The test is positive if the patient exhibits or reports a sense of instability or apprehension about the shoulder in this position. Of note, patients with MDI may also have a positive test. The relocation test is done with the patient in the same position as the apprehension test. The examiner applies a posterior pressure on the humerus when the patient's arm is abducted and maximally externally rotated. If the patient reports relief of the prior feeling of instability, this is considered a positive test. Pain with the apprehension or relocation test is not a positive test and may be due to other shoulder pathology.

Posterior Instability

The posterior drawer test or posterior stress test is done with the patient supine. The arm is forward flexed to 90° and adducted. A posterior load is applied at the elbow in line with the humerus. A click or a clunk is indicative of a positive test. The jerk test is performed with the patient seated and the arm supported abducted to 90° in neutral position. A load is applied at the elbow in line with the humerus while stabilizing the patient's scapula. The arm is then moved into adduction. A painful click or clunk as the humeral head subluxates is indicative of a positive test. The Kim test is similar to the jerk test but is more specific for inferior labral lesions. The arm is supported and abducted to 90°, and a posterior and axial load is applied to the humerus as the arm is flexed diagonally upward 45°. A positive test is sudden onset of posterior shoulder pain.

Multidirectional Instability

The sulcus sign is done with the patient seated. With the forearm in neutral and the arm adducted, an inferior force is applied to the humerus. A positive test is noted as reproduction of the patient's pain with inferior translation of the humerus. This should be repeated in external, internal, and neutral rotation, as well as 90° of abduction. This test is graded on a 0 to +3 scale. Of note, this test can be positive in patients with asymptomatic ligamentous laxity. The load-and-shift test is done with the patient supine. The examiner applies a gentle axial load to center the humeral head in the glenoid, followed by an anterior and posterior translational force to test the degree of laxity in different degrees of abduction. If the patient is guarding, the examiner may not be able to fully evaluate the shoulder. Of note, signs of rotator cuff impingement in a young adult (<20 years old) are also suggestive of MDI.

Differential Diagnosis and Suggested Diagnostic Testing

In cases of traumatic shoulder injury, it is important to assess for other bony injuries involving the clavicle, scapula, and proximal humerus. An acromioclavicular joint injury can also present similarly. In patients over 40, it is important to also assess for a rotator cuff tear. SLAP lesions can also present in the overhead throwing athlete population; these may occur via an overuse or traumatic mechanism. The differential diagnosis of shoulder instability should also include cervical disease and thoracic outlet syndrome. Several types of imaging can be helpful in the diagnostic work-up of shoulder instability.

Radiographs (X-Ray)

Evaluation of shoulder instability starts with X-rays, to include true AP (or Grashey) and axillary lateral views (Fig. 14.5). Axillary views are critical to confirm that the shoulder is indeed reduced given that shoulder dislocations are commonly missed, particularly those in the posterior direction. Shoulder X-rays are helpful to evaluate for bony lesions of the glenoid, Hill-Sachs lesions of the humeral head, and overall glenohumeral and acromioclavicular joint alignment. Additional views specific to shoulder instability include the West Point axillary view (evaluation for a bony Bankart lesion) and Stryker notch view (evaluation for a Hill-Sachs lesion).

Further imaging is usually necessary to fully evaluate the shoulder. There are different benefits to computed tomography (CT) scan versus magnetic resonance imaging (MRI), and careful thought should be given to determine which imaging modality to choose. While general guidelines are noted below, selecting the type of advanced imaging is typically left to the discretion of the orthopedic surgeon,

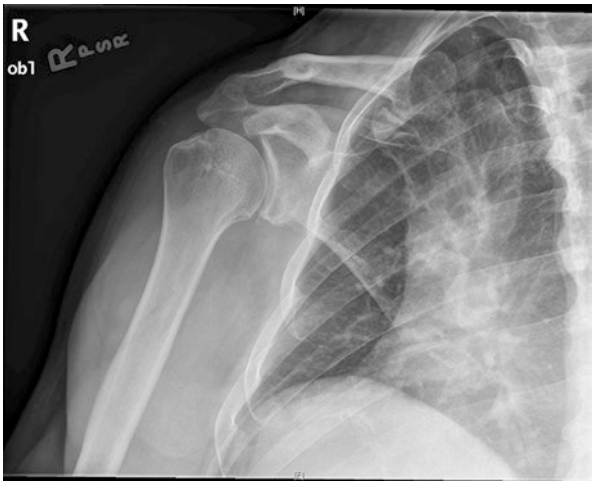

Fig. 14.5 True AP (Grashey) radiograph of a 63-year-old male status post reduction of right shoulder dislocation demonstrating an irregularity of the anterior/inferior glenoid

particularly for cases in which the patient will likely be having surgery and preoperative planning is required.

CT Arthrogram

A CT arthrogram is ideal for evaluating the bony anatomy of the glenoid and the humeral head as well as to evaluate for a bony Bankart lesion. This test tends to be ordered in cases of chronic shoulder instability, especially posterior instability, or when a patient has a history of multiple shoulder dislocations or subluxations. The use of the arthrogram is surgeon specific and should be left to the treating surgeon as to whether the advanced imaging should be done with joint contrast.

MRI Arthrogram

An MRI arthrogram is ideal for evaluating the soft tissues of the shoulder including the rotator cuff, labrum, glenohumeral ligaments, and capsular attachments (Figs. 14.5 and 14.6). This is the imaging of choice for evaluating traumatic unidirectional instability in the absence of a bony Bankart lesion or MDI. The use of the arthrogram is surgeon specific and should be left to the treating surgeon as to whether the advanced imaging should be done with joint contrast.

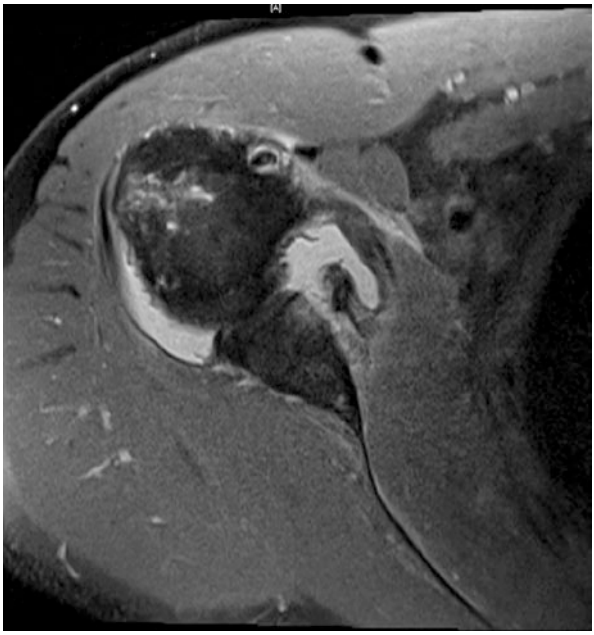

Fig. 14.6 MRI axial T2 image with a bony Bankart lesion

Nonoperative Management

Patients presenting for the first time with symptoms of shoulder instability may benefit from an initial course of nonoperative management. After a traumatic event, rest, ice, and anti-inflammatories can help control symptoms. Following these interventions, physical therapy is the mainstay of nonoperative treatment of shoulder instability.

Traumatic Anterior Shoulder Instability

There is no consensus for how long to immobilize a patient after a shoulder dislocation. Furthermore, the literature does not show any decrease in recurrence with immobilization. Thus, it is currently recommended to immobilize patients in a regular sling for 1–3 weeks for comfort only. Nonoperative management can be successful in treating many isolated causes of traumatic anterior shoulder dislocation, particularly in older adults. In these cases, short-term immobilization should be followed by a focused physical therapy program.

Posterior Shoulder Instability

The majority of patients with posterior shoulder instability can be managed with physical therapy. The program should consist of posterior rotator cuff and deltoid strengthening, along with periscapular stabilization exercises. Nonsurgical management is successful in 65–80% of cases.

Multidirectional Instability

The mainstay of treatment for MDI is physical therapy. The physical therapy protocol should focus on a periscapular stabilization program, as well as including a rotator cuff strengthening program and proprioceptive training. A prolonged course of up to 6 months of physical therapy is sometimes necessary, and research supports an even longer trial before surgical options are considered.

Indications for Surgery

First-Time Traumatic Shoulder Dislocation

Patients who are at high risk for recurrent shoulder instability should be strongly encouraged to discuss treatment options with an orthopedic surgeon. Providers can utilize the Instability Severity Index Score to identify patients who are at heightened

risk of recurrent anterior instability. The Instability Severity Index Score is a 10-point scoring system in which a score above 6 is associated with a 70% risk of recurrence (Table 14.1). Patients in this higher-risk category include age <30, glenoid bone loss, large Hill-Sachs lesion (>5/8 of humeral head), presence of ALPSA lesion (anterior labroligamentous periosteal sleeve avulsion), contact athlete, male, and positive anterior apprehension test. The data is strongest for young, athletic males, who have the highest recurrence rate with conservative management.

Posterior Instability

In cases of posterior instability, patients who fail a course of physical therapy and continue to have symptoms that interfere with daily life or sporting activities are candidates for arthroscopic labral repair. Patients who present with multidirectional instability that have gone through a complete physical therapy program and have ongoing unidirectional posterior instability may be surgical candidates.

Multidirectional Instability

In cases of multidirectional instability, only patients who have failed a prolonged course of physical therapy and continue to have symptoms that interfere with daily life and sporting activities should be considered for surgery.

Table 14.1 Instability Shoulder Index Score

Criteria	Points
Age	
≤20y	2
>20y	0
Contact sport	
Yes	1
No	0
Competitive sport	
Yes	2
No	0
Shoulder hyperlaxity	
Yes	1
No	0
Loss of glenoid contour on anteroposterior radiograph	
Yes	2
No	0
Hill-Sachs lesion visible on external rotation	
Yes	2
No	0

Adapted from Rouleau et al. "Validation of the Instability Shoulder Index Score in a Multicenter Reliability Study in 114 Consecutive Cases." *AJSM*, 2013

Operative Management

Arthroscopic Labral Repair

Arthroscopic labral repair consists of diagnostic arthroscopy to evaluate the anterior and posterior labrum, as well as the other interarticular structures. If indicated, the labrum is then most commonly repaired with suture anchors (smaller anchors than in rotator cuff repair) placed at the rim of the glenoid. The labrum is then sutured back to the glenoid. This process is similar whether the anterior or posterior labrum is torn (please see Figs. 14.5, 14.6, 14.7, and 14.8 for clinical cases).

Latarjet

The Latarjet procedure is commonly performed via an open approach, although it can be done arthroscopically. The coracoid is cut at its base and moved with the attached conjoined tendon to the anterior rim of the glenoid, in order to provide a soft tissue sling and more glenoid surface area to prevent recurrent anterior stability. It is then attached with one or two metal screws.

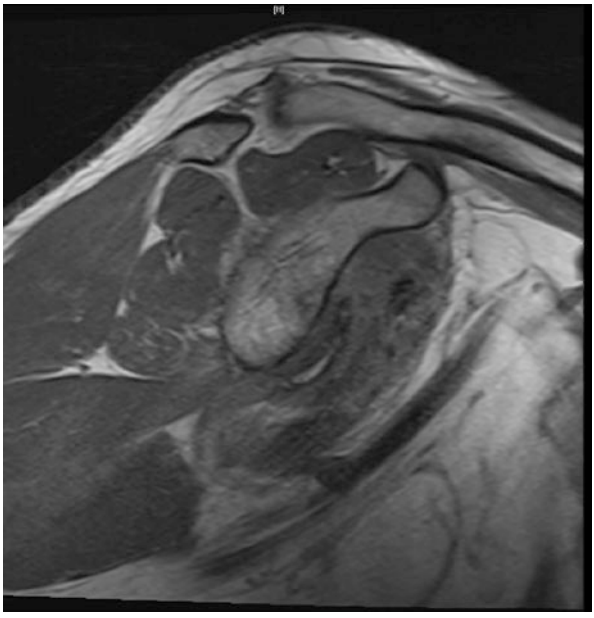

Fig. 14.7 MRI sagittal oblique T1 image showing the glenoid with a bony Bankart lesion anteriorly

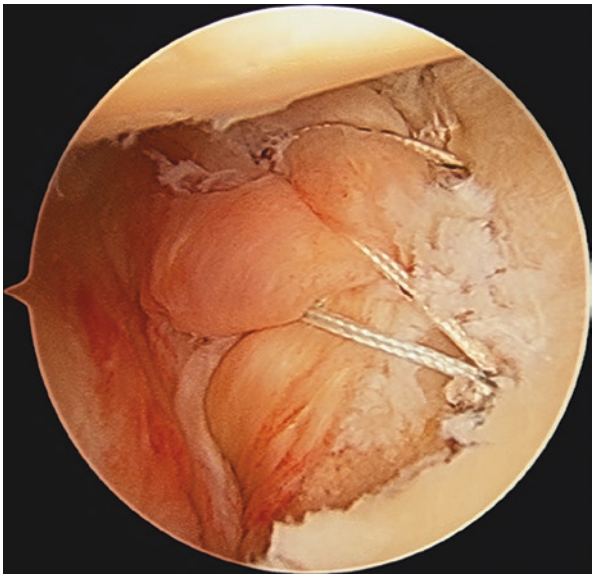

Fig. 14.8 Arthroscopic repair of a bony Bankart lesion using the double-bridge technique. The humeral head is at the *top left*, and the glenoid is to the *right*. The inferior glenohumeral ligament with labrum and fracture is centered

Arthroscopic Capsular Plication

Arthroscopic capsular plication is performed by suturing the capsule to the labrum in order to reduce the volume of the joint space. Depending on the direction of instability, the posterior, inferior, and/or anterior capsule can be plicated.

Posterior Bone Graft

This is similar to the Latarjet procedure in that a piece of bone is being attached to the glenoid to provide a robust bumper for the humeral head. The bone graft is most commonly taken from the iliac crest. A posterior bone graft can be used in the setting of posterior shoulder instability with glenoid bone loss or significant glenoid retroversion.

Expected Outcome and Predictors of Outcome

The rate of recurrent anterior shoulder instability is quite high in young, active males after their first dislocation. Older patients and those not involved in contact sports usually do well with conservative management. The Instability Severity

Index Score can be used as a predictive tool for failure after operative management, and higher scores may be useful as an indication for additional stabilizing procedures. In posterior shoulder instability, conservative treatment is reportedly successful in 65–80% of patients. For patients with a frank posterior dislocation, however, the risk of recurrence is 18%. Risk factors to having recurrence include age <40, presence of a large reverse Hill-Sachs lesion, and seizure disorder. Finally, in multidirectional instability, one study reported that 83% of patients with traumatic or atraumatic MDI had good-to-excellent results with conservative treatment. A recent study in a carefully selected population of athletes with MDI who underwent capsular plication procedure showed an 85% return to sport at their prior level.

Table 14.2 shows the three general categories of shoulder instability with presenting symptoms, clinical exam findings, and a basic treatment algorithm.

Summary

In summary, shoulder instability can be generally grouped into three categories: anterior, posterior, and multidirectional. The cause of instability may be due to trauma such as a dislocation, repetitive microtraumas, or atraumatic. A careful history and physical exam are important to correctly identify shoulder instability. In traumatic anterior dislocations, there is a very high risk of recurrence with young (<20-year-old), contact athletes, and these patients may benefit from early surgical intervention. Patients over 40 years old with a shoulder dislocation should be carefully evaluated for a concurrent rotator cuff tear. Posterior shoulder instability can be difficult to diagnose. High clinical suspicion for posterior instability is important in patients under age 40 with shoulder pain. Perhaps with the exception of young contact athletes, patients with shoulder instability benefit from starting with conservative management with a course of physical therapy. For those who fail nonoperative management, surgical management depends on the direction of the instability and if there is bone loss on the glenoid. Arthroscopic surgery can repair the static stabilizers of the shoulder, but open surgery is usually necessary if there is associated bone loss.

Table 14.2 Three general categories of shoulder instability with presenting symptoms, clinical exam findings, and a basic treatment algorithm

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Traumatic anterior instability	– Dislocation or subluxation event(s)	+ Apprehension/ relocation tests – X-ray – MRI if older than 40 y/o	– Sling for comfort for 1–3 weeks – PT: Scapular stabilization, RTC strengthening	– <20 years old, male, athletes, large glenoid bone loss, multiple dislocations	– Arthroscopic Bankart repair – Latarjet
Posterior instability	– Dislocation or subluxation event(s) – Posterior shoulder pain	+ Jerk/Kim tests – CT arthrogram or MRI arthrogram	– PT: Scapular stabilization, RTC strengthening	– Continued pain/ instability after full course of PT	– Arthroscopic labrum repair – Posterior bone block procedure
Multidirectional instability	– Insidious onset of shoulder pain and instability	+ Sulcus, load-and-shift test, symptoms of impingement in young patient – MRI arthrogram	– PT: Scapular stabilization, RTC strengthening	– Continued instability after prolonged course of PT	– Capsular plication

Suggested Reading

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Chapter 15
Glenohumeral Osteoarthritis

Daniel Plessl, Laurence Higgins, Michael Messina, and Carolyn M. Hettrich

Summary of Epidemiology

Osteoarthritis is the most common joint disease worldwide and remains the most common cause of disability in adults in the United States. With the aging population, it is projected to affect 25% of the adult population, or 67 million people in the United States alone, by 2030. The glenohumeral joint is the third most common large joint to be affected following the knee and hip. Arthritis of the shoulder glenohumeral joint can have a variety of underlying etiologies. These include primary osteoarthritis, rheumatoid arthritis, posttraumatic arthritis, infectious or crystalline arthropathy, arthritis secondary to instability, capsulorrhaphy arthritis, osteonecrosis, and rotator cuff arthropathy. While this chapter will focus primarily on primary osteoarthritis, many of the diagnostic and treatment approaches apply to other etiologies of shoulder arthritis as well.

Glenohumeral osteoarthritis can affect a wide range of patients, however is most common in patients age 60 years and older. It is estimated that between 16% and 20% of adults over the age of 65 have radiographic evidence of glenohumeral osteoarthritis. Radiographic data has found a prevalence of 94% in women and 85% in

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A. J. Schoenfeld et al. (eds.), *Principles of Orthopedic Practice for Primary Care Providers*, https://doi.org/10.1007/978-3-030-74625-4_15

men over the age of 80 years. Risk factors include female sex, Caucasian race, and obesity. Due to the shoulder being a non-weight-bearing joint, it is often less symptomatic than arthritis of the hips and knees. Patients are able to tolerate cartilage loss and degradation in the shoulder for a longer period of time, often resulting in more severe radiographic disease at the time of initial diagnosis. Despite this, given the aging of our population combined with an expectation of higher activity levels, the need for shoulder replacement surgery continues to grow at a rapid pace. While it is currently the third most common arthroplasty performed, its growth rate is higher than rates for hip and knee arthroplasty. It is projected that the demand for shoulder arthroplasty in patients 55 years and older will increase by 755% from 2011 to 2030.

Clinical Presentation

Patients with glenohumeral osteoarthritis predominantly complain of shoulder pain and stiffness that varies in location, however is most typically described as deep within the shoulder. This is in contrast to the common presentation of a patient with rotator cuff pathology or subacromial bursitis, which is typically localized to the lateral or posterosuperior aspect of the shoulder, consistent with the course of the supraspinatus and infraspinatus musculotendinous units. However, there is considerable variability in how patients perceive and describe pain around the shoulder, and this is far from diagnostic.

Pain attributable to glenohumeral osteoarthritis is often described as deep, attributed to movement, especially at the patient's end range. Patients typically have alleviation with rest and exacerbation with increased activity. Patients can have pain at night, specifically if they prefer to sleep on the side where they have arthritis. Sleep disturbance can be what leads a patient to decide to proceed with surgical treatments when nonoperative treatments have failed. Crepitus is common as well with moderate to severe arthritis and is usually described by the patients as "catching," "clicking," "popping," or "grinding." Patients with glenohumeral osteoarthritis usually will also have some degree of stiffness.

Physical examination should include inspection, palpation, range of motion, strength testing, and limited special tests. Osteoarthritis is not usually associated with abnormalities on inspection; significant deformities or muscle atrophy may suggest another diagnosis. Palpation is used primarily to rule out other pathologies such as acromioclavicular (AC) joint tenderness due to AC arthritis or bicipital groove pain from biceps tendinopathy. It is not uncommon for palpation over the bicipital groove to elicit pain, which can be either referred pain from the underlying osteoarthritis or concomitant biceps tendon disease.

Depending on the severity of the disease, the most apparent physical exam finding for patients with glenohumeral osteoarthritis is significant loss of motion. Patients frequently have a loss of active forward elevation, which is associated with pain and discomfort when further passive elevation is attempted. They also have a loss of internal and external rotation (ER). ER is best examined with the arm at the

side and the elbow flexed to 90°, with the elbow firmly adducted to the body. The degree of external rotation on the affected side will be significantly less than the contralateral side, unless the patient has stiffness (or arthritis) bilaterally (normal external rotation is from approximately 40° to 90°). Many times, patients with advanced osteoarthritis will have a complete loss of external rotation such that they are unable to actively or passively externally rotate the arm beyond neutral. Internal rotation is assessed by asking the patient to place their hand behind their back, reaching their thumb as high up the spine as is possible. With more advanced disease, the patient may be unable to position the hand much past the hip or sacrum.

As is routine for any shoulder examination, rotator cuff strength testing should be performed, although routine examination maneuvers may need to be modified secondary to a loss of motion. For the supraspinatus, this is most easily accomplished by placing the arm in forward elevation within the patient's comfortable range of motion and asking the patient to resist a downward force placed on the arm by the examiner. For the infraspinatus, the elbow should be bent to 90° and adducted against the body (similar to examining ER as described above). Beginning with the forearm in neutral rotation, the patient is asked to resist an external rotation force. The subscapularis can be tested with a belly press, or by placing the arm at the side in the same neutral rotation position as used to evaluate ER strength, and having the patient resist an internal rotation force. This maneuver, however, also engages the pectoralis muscles and does not isolate the subscapularis.

Classic osteoarthritis is associated with a full-thickness tear of the rotator cuff less than 10% of the time, and thus rotator cuff strength is typically preserved. It should be noted, however, that all of these strength tests will result in a compressive load on the shoulder joint and thus may elicit pain resulting in decreased effort, which can inaccurately be perceived as weakness. Similarly, patients will oftentimes have other associated positive special tests due to provocation of arthritis pain with certain maneuvers, such as a Hawkins test, Speed's test, or O'Brien's test, but these are not necessary (or recommended) in arthritis patients, and only lead to pain. A neurovascular assessment of the upper extremity should be performed with particular attention paid to confirming function of the axillary nerve, including an assessment of sensation over the deltoid as well as appropriate deltoid contraction force.

Differential Diagnosis and Recommended Diagnostic Testing

A patient presenting with a primary complaint of shoulder pain can have a very broad differential diagnosis; however, the most common etiologies include rotator cuff disease, glenohumeral arthritis, AC joint arthritis, biceps tendon disease, adhesive capsulitis (frozen shoulder), and various types of labral tears. It is also important to keep in mind that over 90% of patients with cervical radiculopathy have arm pain, and if the pain has a radicular component, the contribution of pain from the cervical spine needs to be determined by imaging of the cervical spine and possibly

diagnostic injections/treatments. Thus, correctly diagnosing glenohumeral osteoarthritis requires careful consideration of the history, physical exam, and imaging studies. Imaging studies play an integral role in the work-up and diagnosis of glenohumeral arthritis. It should be noted, first and foremost, that advanced imaging such as a computed tomography (CT) scan or magnetic resonance imaging (MRI) is *not* required in the initial and routine work-up of glenohumeral arthritis. Appropriate work-up should begin with radiography, which is typically all that is needed to confirm the diagnosis.

Most traditional shoulder series include approximately two to five views of the shoulder, the specifics of which can vary. At least two orthogonal views are required to adequately assess the glenohumeral joint. Thus, at the minimum, each patient should have some type of AP view (preferably a Grashey AP) as well as an axillary lateral.

There are multiple types of AP shoulder films that can be obtained. In a standard AP view of the shoulder, the X-ray beam is oriented perpendicular to the transverse axis of the patient. However, because the scapula is oriented at an angle approximately 30°–45° anterior to this transverse axis, the resulting film does not produce a view parallel to the joint line, which is ideal to assess for most shoulder pathologies. In order to obtain a truly orthogonal view to the glenohumeral joint, the beam must be oriented at an angle perpendicular to that of the scapula on the chest wall. This view is called a "Grashey" view or a "true" AP (Fig. 15.1a). The value of this in assessing glenohumeral osteoarthritis (or other shoulder conditions) is that it produces a direct view in line with the glenohumeral joint space, which is essential for evaluating many of the classic associated findings such as joint space narrowing, osteophyte formation, subchondral cyst formation, and sclerosis. The standard AP view is typically not clinically useful with exception of evaluating for AC joint arthritis, which is primarily a clinical diagnosis.

Likewise, there are different "lateral" views of the shoulder, which include, most commonly, either a "scapular Y" or an "axillary lateral." In most cases of shoulder pathology, including glenohumeral arthritis, the axillary lateral view is far superior and should be obtained as part of the initial screening series. A well-positioned axillary lateral provides a second look parallel to the glenoid face in line with the glenohumeral joint (Fig. 15.1b). From a surgical perspective, this view also yields valuable information in determining the glenoid orientation and how well the humeral head is centered on the glenoid. This is important because more severe osteoarthritis is often associated with some degree of asymmetric posterior glenoid wear and posterior subluxation of the humeral head. The scapular Y view adds very little value for the evaluation for glenohumeral osteoarthritis (or most shoulder conditions) and is unnecessary.

Similar to the clinical course, radiographs for osteoarthritis demonstrate a wide range of severity from subtle findings associated with mild, early arthritis to more severe, advanced changes. An inferior humeral head osteophyte, or "goat's beard," is the most classic X-ray finding associated with glenohumeral arthritis and is also one of the earliest. Its presence (even when small) is highly associated with at least some degree of full-thickness cartilage loss, even with generally well-persevered

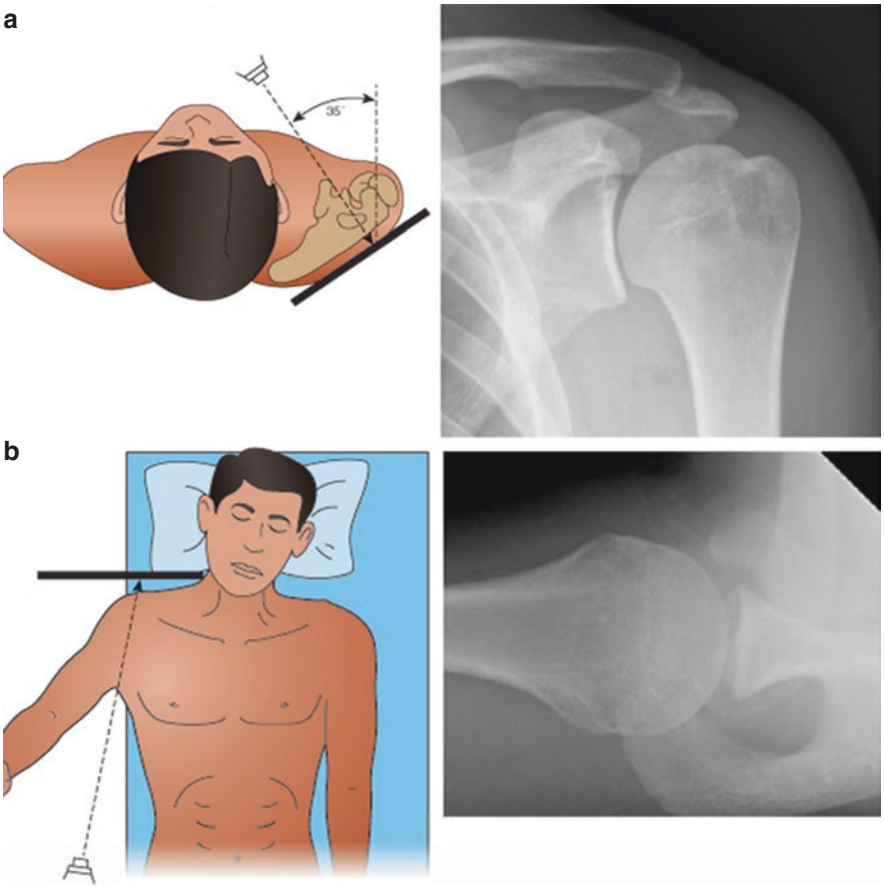

Fig. 15.1 (**a**) Proper X-ray technique and corresponding radiographic result to obtain "true AP" or "Grashey AP" view of the shoulder, which provides a view directly parallel with the glenoid face, essential for evaluating for arthritic changes. (**b**) Proper X-ray technique and corresponding radiographic result to obtain axillary lateral view of the shoulder, which provides a view directly parallel with glenoid face and orthogonal to the true AP. (Adapted from Matsen FA. http://shoulderarthritis.blogspot.com/2011/03/plain-x-ray-key-to-diagnosing-arthritis.html)

joint space (Fig. 15.2a). Over time, the size of this osteophyte can become quite large and may restrict motion (Fig. 15.2b, c).

As noted previously, there is typically no role for ordering advanced imaging beyond the X-ray images described above once the patient has been diagnosed with osteoarthritis, unless there is a concern for a concomitant, symptomatic full-thickness rotator cuff tear, which is associated with glenohumeral osteoarthritis <10% of the time. CT and MRI imaging may be required for surgical planning in the event that the patient ultimately elects to proceed with shoulder replacement surgery. These tests should be ordered by the treating surgeon, as specific protocols to allow for 3D modeling may be required for surgical planning, and imaging needs to be within 3–6 months of the surgical date.

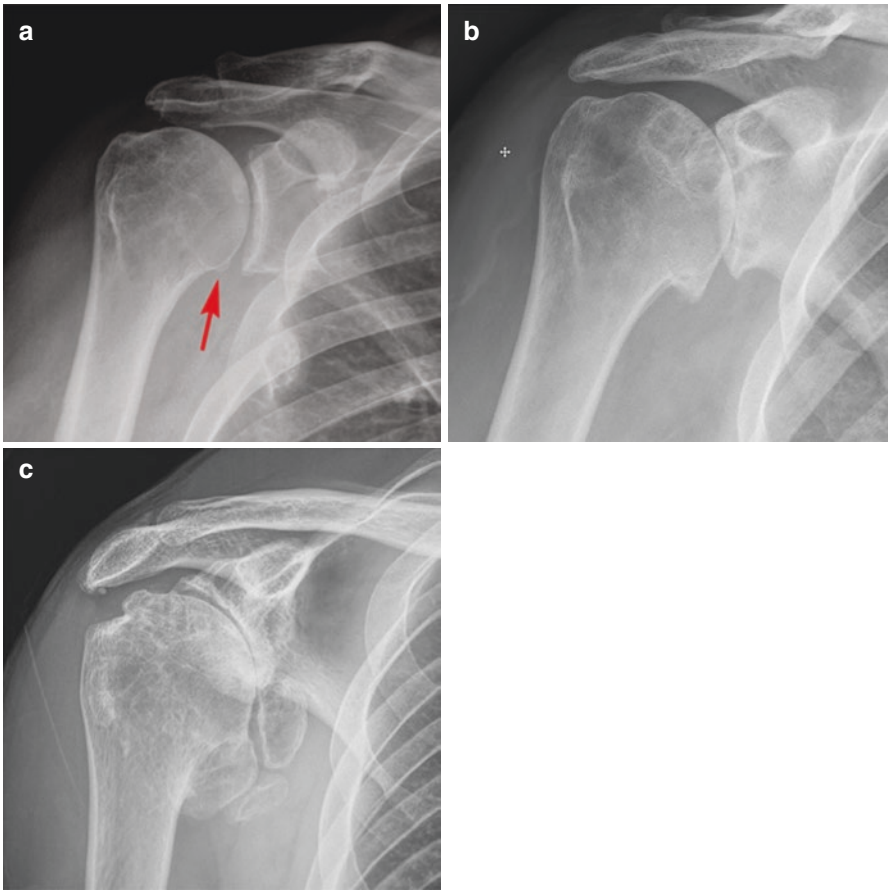

Fig. 15.2 AP shoulder X-rays demonstrating varying sizes of inferior humeral head osteophyte in shoulder osteoarthritis. (**a**) Patient with very early and subtle radiographic changes with a small osteophyte (*red arrow*). This patient underwent arthroscopy and was found to have large areas of full-thickness cartilage loss despite well-preserved joint space, and ultimately required shoulder replacement surgery soon after this image was taken. (**b**) Patient with severe osteoarthritis with moderate-sized osteophyte. (**c**) Patient with severe osteoarthritis with large inferior osteophyte

Nonoperative Management

Osteoarthritis is a dynamic and progressive process in which ongoing cartilage degradation almost always leads to worsening of pain and decreasing range of motion over time. To date, there remains no effective or proven treatment option to slow down this progression or to regenerate the lost cartilage. As a result, nonoperative treatment remains focused on symptom management. The primary goal is to minimize inflammation and pain which can be done through activity modification, weight loss, nonsteroidal anti-inflammatory drugs (NSAIDs), and corticosteroid injections.

Activity modification is a simple strategy to decrease symptoms by avoiding the particular tasks that elicit pain. This can be effective for some patients, but they must be willing to alter their lifestyle. Since the shoulder is not a weight-bearing joint, weight loss does not alleviate as much pain as it does in the lower extremities, but it does improve surgical outcomes.

NSAIDs can be a useful due to their direct analgesic effect and by decreasing the production of inflammatory cytokines. NSAIDs have a long track record of safety; however, caution should be exercised with prolonged, chronic use and in patients with a history of gastritis, gastric ulcers, kidney disease, and cardiovascular disease or in patients who require concomitant anticoagulation. For patients who cannot tolerate NSAID therapy, alternative options include acetaminophen as well as using ice. Opioids should be avoided.

Physical therapy is a common treatment in the nonoperative management of many musculoskeletal complaints but does not play a significant role in the treatment of glenohumeral osteoarthritis, particularly when symptoms and joint disease become more severe. While physical therapy is widely used in practice, much of its use is based on expert opinion, and one case-control study found no difference between formal physical therapy and a home exercise program in the management of glenohumeral osteoarthritis. Additionally, many patients with severe osteoarthritis complain that physical therapy worsens the joint pain. If a patient is insistent on trying this, a therapist can work on maintaining motion through various gentle muscular and capsular stretching techniques as well as symptom management through pain relief modalities such as electrical stimulation, iontophoresis, ultrasound, and others.

The use of injections in the treatment of many different types of shoulder pathologies is common, especially for conditions such as subacromial bursitis or rotator cuff tears, where subacromial injections can be readily performed in the office setting. It is critical to note, however, that in the presence of an intact rotator cuff, the subacromial space does not communicate with the intra-articular joint space, and thus subacromial injections are not indicated in the treatment of glenohumeral osteoarthritis. While intra-articular injections can be used successfully in the management of glenohumeral osteoarthritis, there are considerations to keep in mind. First and foremost, glenohumeral joint injections are more technically challenging to perform in the office without the use of image guidance. In fact, prior studies have shown a success rate of only 47.5% for fellowship-trained orthopedic sports/shoulder specialists. As a result, it is recommended that these injections are administered using ultrasound or fluoroscopic imaging guidance. Additionally, most evidence suggests that injections are more effective when used for mild to moderate radiographic arthritis and have less of an effect on severe arthritis.

Despite the above considerations, intra-articular glenohumeral injections do play a role in the nonoperative management of glenohumeral osteoarthritis. These injections are typically performed with corticosteroids and local anesthetic. Given a relative lack of high-level evidence supporting the use of corticosteroid for glenohumeral joint injections, much of its use is based off of its efficacy demonstrated in the knee. In the appropriately selected patient, injections can provide meaningful pain relief

and improve function. Injections can be repeated, making them a particularly attractive option for long-term pain management in patients who are poor surgical candidates, although efficacy can sometimes diminish with time. Injections should be used with caution and we recommend they be repeated no more frequently than every 3 months. Despite local delivery and limited systemic effects, intra-articular steroid injections can lead to increased glucose levels, and, as such, blood sugars should be closely monitored in diabetic patients following injections. Additionally, some recent studies have raised concern that repeated corticosteroid injections could actually hasten arthritic changes. For example, a randomized control trial involving patients with knee synovitis found that those who received corticosteroid injections every 3 months over a 2-year period had a statistically significant greater loss of cartilage thickness compared to those that received saline. Furthermore, the national Medicare database shows that patients treated with a corticosteroid injection within 3 months of shoulder arthroplasty are at an increased risk of postoperative infection. Use of ropivacaine is also recommended over lidocaine or Marcaine due to the chondrotoxicity of the latter medications.

Indications for Surgery

Simply stated, the indication for surgical treatment of glenohumeral osteoarthritis is failure of nonoperative treatment, defined as persistent pain and dysfunction despite use of modalities listed above, to the point where activities of daily living and quality of life are significantly affected, as determined by the patient. Ultimately, a shared decision-making process is essential, involving a thorough discussion about the risks and benefits of surgery. Patients must ultimately decide for themselves when their symptoms warrant more aggressive treatments.

Operative Management

Operative management of glenohumeral osteoarthritis can be divided into two general groups: arthroscopic/joint-preserving surgery and joint replacement surgery. Indications for arthroscopic treatment of the osteoarthritic shoulder are very few, however may show benefit in properly selected patients. Typically, this may be considered in patients less than 45 years of age with advanced osteoarthritis and significant symptoms as an attempt to delay joint replacement surgery due to the high risk for failure of arthroplasty performed at a young age. Generally, this procedure involves lavage and extensive debridement with some degree of osteophyte removal and axillary nerve decompression. The goals are to provide pain relief, restore motion, and improve function in order to ultimately prolong time until shoulder replacement is needed. Developed by Millet and Gaskill, this procedure has been termed comprehensive arthroscopic management (CAM) of glenohumeral

osteoarthritis. A high level of patient satisfaction with decreased pain and increased range of motion has been observed for up to 2.7 years postoperatively, however with high rates of early failure and conversion to arthroplasty in patients with less than 2 mm of joint space. In these patients, further evaluation will certainly be needed to assess for long-term success.

Shoulder replacement surgery is the gold standard for operative treatment of glenohumeral osteoarthritis. The primary indications are severe pain and dysfunction with loss of quality of life and failure of nonoperative treatment. The two most commonly used techniques are anatomic total shoulder arthroplasty (TSA) and reverse shoulder arthroplasty (RSA). As the name implies, anatomic TSA involves reconstruction of the glenohumeral joint to restore normal anatomical relationships typically with a metal humeral head component and an all-polyethylene glenoid component (Fig. 15.3). Success of anatomic TSA is highly reliant upon an intact and well-functioning rotator cuff, and on having preserved glenoid bone stock.

TSA is performed through a deltopectoral approach from the front of the shoulder which requires reflection of subscapularis for complete exposure and commonly also involves concomitant tenotomy or tenodesis of the biceps tendon at the time of surgery. Historically, humeral components have involved the use of a stem in the humeral intramedullary canal that is fixed either with or without the use of cement. Recently, stemless humeral components have been approved for use in the United States as well, with advantages of preserving bone stock and facilitating easier removal in the event revision surgery is required (Fig. 15.4). The polyethylene glenoid component is cemented, and, upon satisfactory placement of all implants, the subscapularis is repaired. Healing of the subscapularis is critical for proper function and stability following surgery, and as such, protection of the repair is a major driving force that dictates postoperative restrictions and speed of recovery. Specifically,

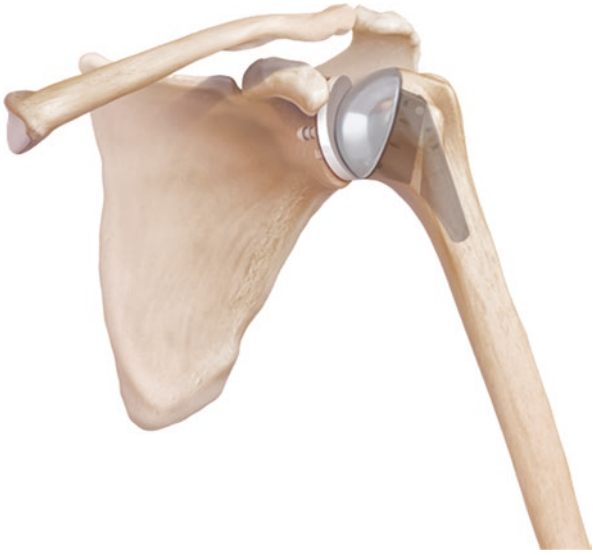

Fig. 15.3 Components of anatomic total shoulder arthroplasty. (Image courtesy of Arthrex Inc. Used with permission)

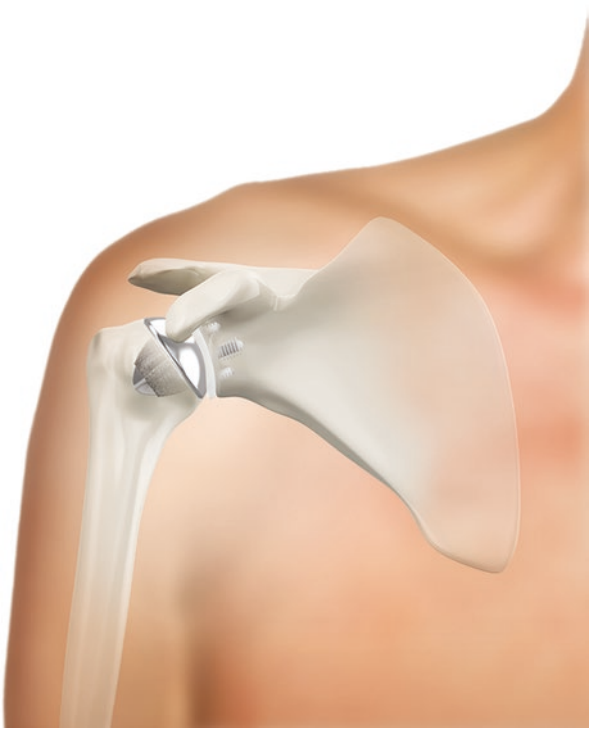

Fig. 15.4 Example of components in stemless anatomic shoulder arthroplasty. (Published with permission from Tornier, Inc., an indirect subsidiary of Wright Medical Group N.V)

it is critical to avoid any external rotation (passive and active) beyond 0°–20°, as well as any active internal rotation for up to 6 weeks after surgery, with no lifting/pushing/pulling for an additional 6 weeks.

The alternative to anatomic TSA is reverse shoulder arthroplasty (RSA) in which the "ball and socket" parts of the prosthesis are switched such that the glenoid side now consists of a spherical metal component, or glenosphere, and the humeral side consists of a stemmed, cupped, metal component (Fig. 15.5). These are most commonly performed in cases of rotator cuff deficiencies leading to rotator cuff arthropathy or significant bone loss. Postoperative recovery does not differ significantly from anatomic TSA.

Expected Outcomes and Predictors of Outcome

While specific postoperative recovery and rehab protocols vary by surgeon preference, most patients can expect to spend some length of time after surgery immobilized in a sling, typically 6 weeks. Patients usually begin therapy to work on active assist and passive range of motion. For patients who have an anatomic TSA, or a RSA where the subscapularis was repaired, careful attention is paid during the

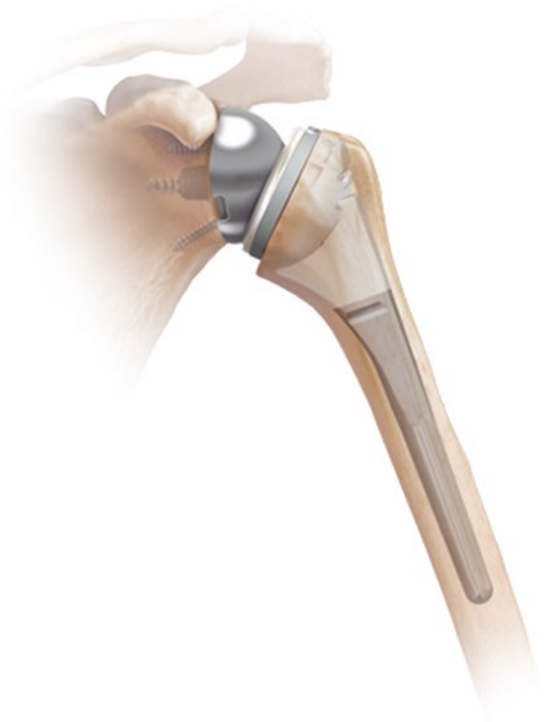

Fig. 15.5 Example of components of reverse shoulder arthroplasty. (Image courtesy of Arthrex Inc. Used with permission)

initial recovery period to protect the repair of the subscapularis tendon, and as such, they are strictly cautioned against any active internal rotation as well as passive external rotation beyond 0°–20° (depending on the surgeon and the patient). As patients begin to wean from their sling at around 6 weeks postoperatively, they are permitted to begin active ROM and use the arm for some daily activities. Strengthening begins at 3 months postoperatively.

Shoulder replacement surgery has a complication profile similar in many ways to lower extremity joint replacement, with some very pertinent differences. The common risks include bleeding, infection, nerve injury, instability, stiffness, loosening of implants, and venous thromboembolism (VTE). Blood transfusion following shoulder replacement is less common than in lower extremity joint replacement and is required in fewer than 4% of patients. One of the most dramatic differences between shoulder and lower extremity joint replacement is the higher risk for infection with *Cutibacterium acnes* (*C. acnes*) in shoulder surgery. This is due to the difference in normal skin flora of the shoulder compared to the hip or knee. Specifically, the chest and back contain a higher density of oily sebaceous glands, which harbor the growth of *C. acnes*. The clinical relevance is that *C. acnes* is a low virulence organism that can sometimes exist in the shoulder for years prior to development of symptoms, and, as such, there should always be a high index of suspicion for infection in a patient who presents with pain, stiffness, or evidence of component loosening after total shoulder replacement surgery.

Table 15.1 Clinical management of glenohumeral osteoarthritis

Clinical entity	Presentation	Diagnostic testing	Conservative management	Surgical indications and operative management
Glenohumeral osteoarthritis	– Gradual onset and worsening of deep shoulder pain; dull and aching – Loss of shoulder range of motion—particularly in internal and external rotation – Pain with shoulder motion and pain at night	– X-ray (Grashey AP and axillary lateral)—joint space narrowing, inferior humeral head osteophyte, subchondral sclerosis, and cystic changes – MRI and CT scans are not needed	– Begins with lifestyle modification – Analgesics including NSAIDs, Tylenol, and tramadol in more severe cases – Limited role for physical therapy – Corticosteroid injections can be helpful—must be given within glenohumeral joint (subacromial not effective)	– Mainstay of surgical treatment is shoulder replacement – Shoulder replacement is associated with significant pain relief and increased function as well as high patient satisfaction

NSAIDS nonsteroidal anti-inflammatory drugs, *MRI* magnetic resonance imaging, *CT* computed tomography, *AP* anteroposterior

Shoulder replacement surgery for patients who have failed conservative treatment of osteoarthritis has proven to be a highly successful operation with high patient satisfaction and long-term durability rivaling that of knee or hip arthroplasty. Shoulder replacement has consistently led to improvements in both subjective, patient-reported outcomes and objective clinical outcomes. Shoulder replacement is most reliable at relieving pain; however, most patients can additionally expect to achieve improved range of motion in forward elevation and external rotation as well as improved overall function after surgery. Given that the natural course of glenohumeral osteoarthritis is a slow progression of pain and declining function over time, shoulder replacement offers an excellent long-term solution by providing pain relief and restoring quality of life in patients who are appropriate surgical candidates. Table 15.1 shows a brief description of the clinical management of glenohumeral osteoarthritis.

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Chapter 16
Elbow Osteoarthritis and Soft Tissue Injuries

George S. M. Dyer and Stella J. Lee

Introduction

This chapter discusses various common inflammatory, traumatic, and arthritic conditions of the elbow region of the upper extremity. In each case, we address common presentation, diagnosis, options for management, and outcomes.

Elbow Septic Arthritis and Olecranon Bursitis

Septic arthritis involving the elbow is an infection within the joint resulting in inflammation of the joint synovium and painful distension of the joint capsule, followed by progressive destruction of cartilage, ultimately leading to arthritis. The pathogen is usually bacterial, most commonly *Staphylococcus aureus*. Etiologies of septic arthritis include hematogenous spread from bacteremia, direct inoculation from trauma or surgery, or local spread from adjacent cellulitis or osteomyelitis. Olecranon bursitis does not involve the joint itself but rather the olecranon bursa, a synovial-lined, fluid-filled sac overlying the triceps tendon insertion onto the olecranon. This bursa can become inflamed and filled with sterile synovial fluid, causing discomfort at the elbow. It is important to differentiate these two conditions as one may be treated with observation, whereas the other requires urgent surgical intervention.

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Summary of Epidemiology

Septic arthritis more commonly occurs in weight-bearing joints, and infections of the elbow account for only 3–9% of cases. Risk factors for septic arthritis include previous trauma, immunosuppression, rheumatoid arthritis, hemophilia, and history of intravenous drug use. Aseptic olecranon bursitis is typically associated with gout, rheumatoid arthritis, other inflammatory conditions, or intensive physical labor. Anatomic variances such as a prominent olecranon process or bone spurs are associated with higher incidence of bursitis. Although olecranon bursitis is typically aseptic, the most common cause of septic bursitis is iatrogenic infection from attempts to aspirate or drain the collection. Aseptic olecranon bursitis should never be drained.

Clinical Presentation

Septic arthritis of the elbow presents with fever, erythema, edema, and pain with range of motion of the elbow. Olecranon bursitis, on the other hand, typically presents as a non-tender fluctuant mass overlying the proximal olecranon. It may be asymptomatic until the bursa is quite distended. Alternatively, in the case of gouty or septic olecranon bursitis, the bursa can be painful and tender, and there may be overlying hyperemia or erythema.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnoses for septic elbow include crystalline arthropathies such as gout and pseudogout, trauma, hemarthrosis, and abscess not involving the joint. An elevated ESR and CRP are suggestive of infection but are not diagnostic. Imaging such as X-ray, CT, or MRI can be helpful in detecting an effusion or ruling out other diagnoses; however, it is not diagnostic of an infection. The definitive diagnosis of septic arthritis is made with joint aspiration; this should be completed prior to the initiation of antibiotics and performed using sterile technique. The needle is placed into the soft space at the center of a triangle formed by the olecranon, radial head, and lateral epicondyle. Samples should be sent to the laboratory for Gram stain, culture, cell count, and crystal analysis. A cell count of greater than 50,000/mm^3, as well as neutrophils greater than 90%, is indicative of infection.

Aseptic olecranon bursitis should be distinguished from septic bursitis, which accounts for 20% of cases. If septic bursitis is suspected, a needle aspiration should be performed. To avoid creating a chronically draining sinus, it is recommended to

use a long spinal needle, entering the bursa proximally on the radial side at a very acute angle, so that the needle traverses a long path of skin and soft tissue. With this technique, elbow flexion will squeeze the resulting aspiration tract flat and occlude it, rather than stretching it open. Care should also be taken not to enter the joint space during bursal aspiration. A white blood cell count of greater than 10,000/mm^3 with predominant polymorphonuclear cells is concerning for septic bursitis.

Non-operative Management

There is no role for non-operative treatment of septic arthritis—antibiotic therapy is an adjunct to surgical debridement. Empiric antibiotic therapy based on patient risk factors should be initiated after joint aspiration, followed by a long-term course of intravenous antibiotic therapy based on culture data. The non-operative management of aseptic olecranon bursitis consists of a combination of compressive dressings, avoidance of pressure to the area, nonsteroidal anti-inflammatories (NSAIDs), and padded splinting, usually with resolution over a period of months. Persistent bursitis is sometimes treated via serial aspirations performed under sterile technique; however, this procedure may lead to the formation of a chronically draining sinus tract. The recommended technique in this case involves placing a needle proximally through the triceps muscle and then aiming distally into the bursal sac (Fig. 16.1). Given the specificity of these procedures, it is recommended that diagnostic and therapeutic injections for olecranon bursitis be performed with the care of an orthopedic specialist.

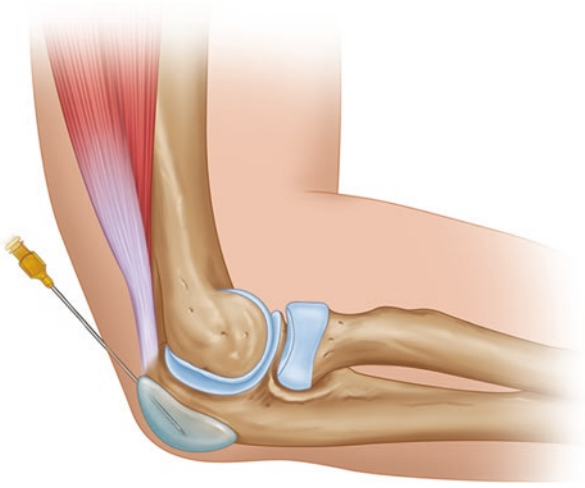

Fig. 16.1 Aspiration of the olecranon bursa. The patient is positioned with the elbow flexed at approximately 90°. The needle is placed obliquely through the triceps muscle into the olecranon bursa. The clinician should avoid inserting the needle directly into the skin overlying the olecranon bursa as this can lead to the formation of a chronically draining sinus tract

Indications for Surgery

Surgical debridement of the elbow joint should be performed if diagnostic aspiration shows frankly purulent fluid, cell count greater than 50,000/mm^3, or positive culture. Bursectomy is a treatment option for chronic symptomatic bursitis. It is also recommended for septic bursitis that is not responsive to needle aspiration and antibiotic treatment.

Operative Management

Elbow irrigation and debridement consists of either open or arthroscopic debridement of inflamed synovium, removal of any cartilage or bony debris, and irrigation with several liters of normal saline. In open bursectomy, a longitudinal incision is made over the olecranon bursa. The bursal sac is completely excised, hemostasis is achieved, and the dead space is obliterated. A drain is placed prior to wound closure.

Expected Outcomes and Predictors of Outcome

Both open and arthroscopic debridement are effective in treating septic elbow. It should be noted, however, that there is a high mortality rate associated with septic arthritis—one study showed a 50% mortality rate after the diagnosis of septic elbow. Postoperatively, the best functional outcomes are seen in patients without preexisting elbow pain who undergo debridement within 2 days of presentation. Most patients with olecranon bursitis are successfully treated with non-operative therapy. Bursectomy results in long-term pain relief but is much less effective in patients with gout.

Tendinopathies: Lateral Epicondylitis and Medial Epicondylitis

Lateral epicondylitis, or “tennis elbow,” and medial epicondylitis, or “golfer’s elbow,” are degenerative tendinopathies of the tendon origins at the lateral and medial epicondyles of the elbow. Lateral epicondylitis most commonly affects the extensor carpi radialis brevis (ECRB) and less commonly the extensor digitorum communis (EDC). Medial epicondylitis affects the pronator teres and flexor carpi radialis (FCR) tendons. Although the background cause of tendinopathy is poorly understood, in many cases, it is believed to be caused by repetitive microtrauma to the tendons.

Summary of Epidemiology

The prevalence of lateral and medial epicondylitis is 1.3% and 0.4%, respectively. Epicondylitis most often presents in patients in their 30s to 50s, favors the dominant arm, and affects men and women equally. It is associated with smoking, obesity, and forceful repetitive activities and as such may be considered a work-related condition in some cases. Medial epicondylitis in particular may affect young throwing athletes.

Clinical Presentation

In cases of lateral epicondylitis, patients complain of pain at the region of the lateral epicondyle which is aggravated by wrist extension. On exam, pain is elicited with resisted wrist extension with the forearm pronated and elbow extended, as well as with resisted extension of the long finger. In cases of medial epicondylitis, patients complain of pain at the region of the medial epicondyle. On exam, pain is elicited with resisted wrist flexion and resisted pronation. Medial epicondylitis is often associated with ulnar neuritis, and ulnar nerve dysfunction should be tested. With ulnar neuritis, patients may report pain or paresthesias with tapping the ulnar nerve at the elbow (Tinel's sign) or with cubital tunnel compression. In cases of medial epicondylitis, the elbow should also be evaluated for ulnar collateral ligament (UCL) insufficiency resulting in valgus instability. The "milking maneuver" is performed by flexing the elbow to 90° and supinating the forearm while the examiner then pulls on the thumb radially to produce valgus stress. The test is positive with pain or instability when compared to the contralateral side.

Differential Diagnosis and Suggested Diagnostic Testing

Lateral epicondylitis and medial epicondylitis are clinical diagnoses. Plain films and magnetic resonance imaging (MRI) may be obtained to evaluate for other disorders, such as arthritis, osteochondral defects, or ligamentous injuries, but are not required for diagnosis. If obtained, MRI may reveal increased signal within the common extensor or common flexor tendons on T1 and T2 sequences. In the presence of a partial tear, it may show tearing of the tendon from the bone, noted as a focal area of high T2 signal intensity. It is also important to note that lateral epicondylitis has a similar presentation to radial tunnel syndrome (i.e., entrapment of the deep radial or posterior interosseous nerve in the lateral forearm); however, the location of pain in radial tunnel syndrome is approximately 2 cm more distal when compared to lateral epicondylitis.

Non-operative Management

For both lateral and medial epicondylitis, watchful waiting and activity modification with avoidance of exacerbating activities is the first step of treatment and is effective for most. A counterforce brace may be used if it provides symptomatic relief. A counterforce brace is a simple orthosis with just an elasticized fabric band that pushes a hard plastic button into the sore spot of the muscle. This should be followed by physical therapy and gradual return to resistive activities. Local corticosteroid injections have been shown to provide a benefit in the short term but not in the long term and are thus discouraged. Up to 90% of patients experience symptom resolution with non-operative treatment, but it is important to counsel patients that this may take up to a full year. If treatment is unsuccessful after more than a year, consider an alternate diagnosis. Chronic collateral ligament tears, radial tunnel syndrome, and radiocapitellar arthritis have all been mistaken for lateral epicondylitis.

Indications for Surgery

Surgery for epicondylitis can be considered in cases that fail to improve with non-operative treatment for more than 12 months.

Operative Management

Surgical treatment for epicondylitis may be performed open or percutaneously. The affected area of the tendon is sharply excised, the surrounding area debrided, and the tendon reattached to the epicondyle as necessary. If there is progressive ulnar neuritis associated with medial epicondylitis, ulnar nerve decompression is recommended. Please refer to Chap. 19 for further information on upper-extremity compression neuropathies and ulnar nerve decompression.

Expected Outcomes and Predictors of Outcome

It is important to note that most patients experience resolution of symptoms in 8–12 months simply with activity modification. For the 10% of patients who do not improve with non-operative treatment, surgical management has a success rate of approximately 97% in lateral epicondylitis and 87–100% in medial epicondylitis.

Biceps and Triceps Tendon Ruptures

Triceps tendon injuries are generally traumatic and result from rapid eccentric muscle contraction. Distal biceps tendon ruptures also occur after an acute traumatic event, usually forced elbow flexion and supination, but can also occur in the setting of chronic tendon degeneration with chronic elbow pain. Avulsions are defined as injuries at the osseous surface, whereas ruptures are defined as injuries at the intrasubstance of the muscle or the musculotendinous junction. Tendon ruptures may be partial or complete, involving all the tendon fibers.

Summary of Epidemiology

Triceps ruptures are quite rare, accounting for less than 1% of all tendon ruptures. About 3% of biceps injuries occur at the distal biceps tendon. Both triceps and distal biceps ruptures usually occur in men, and avulsion and musculotendinous ruptures in particular are associated with anabolic steroid use. Renal failure and hyperparathyroidism are also risk factors for tendon injuries. Spontaneous tendon injuries are higher after total elbow arthroplasty.

Clinical Presentation

In cases of complete triceps rupture, patients present with pain at the posterior elbow and loss of active elbow extension. Physical exam reveals a palpable defect at the triceps tendon. Partial tendon ruptures result in some active elbow extension, however often with decreased strength and an extensor lag (inability to actively bring the elbow into full extension). In cases of distal biceps rupture, patients complain of pain at the antecubital fossa that is worsened with supination. On exam, there is often ecchymosis over the antecubital fossa and asymmetry of the biceps muscle belly when compared to the contralateral side. Pain is elicited with resisted elbow flexion and forearm supination. The examiner should perform the "hook test" to attempt to palpate and pull on the distal biceps tendon in the antecubital fossa with the elbow in 90° of flexion, using a hooked index finger. With a complete rupture, the distal biceps tendon is not palpable.

Differential Diagnosis and Suggested Diagnostic Testing

The diagnosis of triceps tendon injuries may be made clinically, but plain films should be obtained to evaluate for olecranon fracture or bony avulsion which may also result in an extensor lag. MRI may also be useful for distinguishing between

partial and complete tendon ruptures. Plain films are generally not useful in suspected biceps tendon ruptures as associated bony injuries are rare. MRI is helpful when the diagnosis of biceps injury is unclear and can distinguish between complete and partial ruptures.

Non-operative Management

Partial triceps tendon ruptures may be treated non-operatively. Both partial and complete distal biceps ruptures may be treated non-operatively. The principal flexor muscle of the elbow is the brachialis, not the biceps, thus flexion strength is typically only diminished by 20–40%, even in cases of complete biceps rupture. Forearm supination strength, however, is often greatly diminished. Non-operative treatment consists of splint immobilization in 30° of flexion for triceps ruptures and simple sling immobilization for biceps ruptures. Patients should receive regular follow-up to ensure that partial ruptures do not progress into complete ruptures.

Indications for Surgery

Surgical repair is indicated for acute complete triceps ruptures. It may also be considered for residual weakness with non-operative treatment, although surgical repair is more difficult in a chronically injured elbow, in which case, the tendon may become retracted. Surgical repair for distal biceps ruptures may be considered for patients with high functional demands.

Operative Management

In cases of triceps rupture, the triceps tendon is attached to the olecranon via nonabsorbable sutures passed through drill holes in the olecranon. Large bony fragments may be attached with additional hardware. Postoperatively, the patient is made non-weight-bearing through the arm, and the elbow is initially immobilized in partial flexion. Patient should undergo physical therapy for gradual range of motion and progression to active extension. In a distal biceps rupture, the distal biceps tendon is mobilized and then reattached to the biceps tuberosity. The repair is completed with the elbow in slight flexion in order to maximize flexion and supination strength; as the tendon attenuates, the patient can then achieve full extension. A variety of methods are used to attach the tendon to the bone, including suture anchors and bone tunnels with interference screws. For both biceps and triceps injuries, acute repair is recommended over delayed repair before significant tendon retraction occurs.

Expected Outcomes and Predictors of Outcome

Most patients regain satisfactory function following triceps tendon repair—one study reported 92% of peak strength compared to the uninjured side. Outcomes are poorer following repair of chronic tendon injuries. The most common postoperative complications include olecranon bursitis, flexion contractures up to 20°, and, rarely, re-rupture. Following non-operative treatment of distal biceps ruptures, there is an approximately 30% loss of flexion strength and 40% loss of supination strength. Most patients report satisfactory outcomes following distal biceps repair, although similar to triceps repair, there may be slight deficits in strength and endurance. Risks of a distal biceps repair include radial nerve palsy with a single-incision approach and heterotopic ossification or synostosis—fusion of the proximal ulna and radius—with a two-incision approach.

Elbow Arthritis

Elbow osteoarthritis primarily affects the ulnohumeral joint and less commonly the radiocapitellar joint. Due to the high congruence of this articulation, bony changes first occur at the margins of the joint—the tips of the olecranon and coronoid processes—and complete loss of articular cartilage does not occur until advanced stages of the disease (Fig. 16.2). Rheumatoid arthritis, by contrast, is an inflammatory disease of the soft tissue surrounding joints. The development of an inflammatory pannus ultimately results in erosion of the cartilage and subchondral bone, attenuation of ligaments, and progressive joint deformity.

Summary of Epidemiology

Primary osteoarthritis of the elbow is fairly rare compared to osteoarthritis of other major joints, affecting 2% of the population. It is seen in relatively young men involved in manual labor or throwing sports and more commonly affects the dominant arm. It can also follow elbow injury such as fracture or dislocation. Elbow involvement is common in rheumatoid arthritis, affecting about 50% of the patients affected with this systemic disorder. Patients with elbow rheumatoid arthritis are generally older than those with osteoarthritis.

Clinical Presentation

Elbow arthritis presents with pain and progressive stiffness of the elbow. In the early stages of osteoarthritis, patients complain of pain only at terminal flexion or terminal extension. As the disease progresses to involve the entire ulnohumeral joint, there is

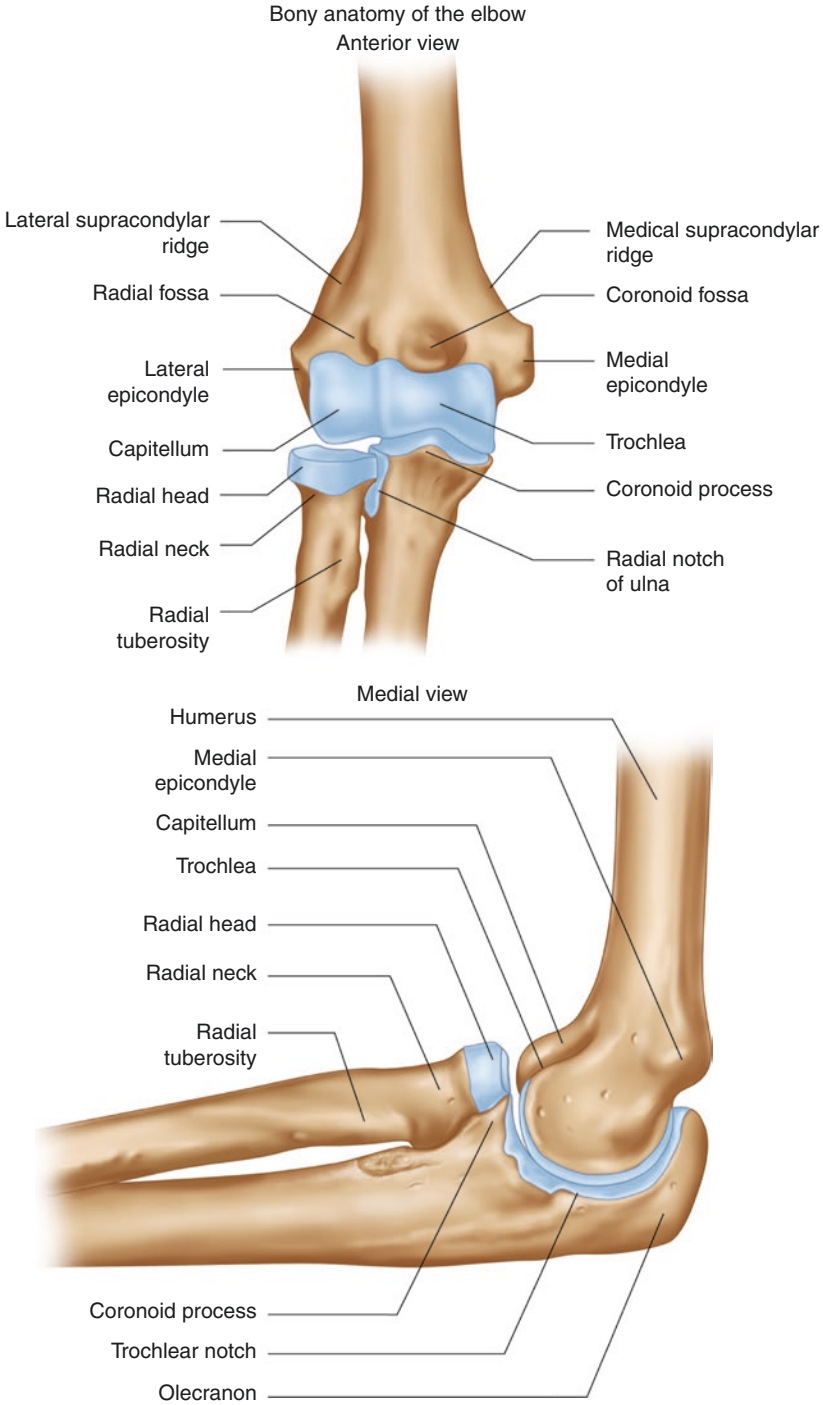

Fig. 16.2 Bony anatomy of the elbow

pain throughout the arc of range of motion. In radiocapitellar arthritis, there is lateral elbow pain with forearm rotation. Due to posterior osteophyte formation, some patients present with associated ulnar neuropathy, complaining of medial-sided elbow pain along with paresthesias in the ulnar digits. Patients should be examined for sensory deficits in the ulnar nerve distribution and intrinsic hand weakness. The physical exam should also include elbow varus and valgus stress tests for instability.

Differential Diagnosis and Suggested Diagnostic Testing

In the evaluation of arthritis, plain films of the elbow, including AP, lateral, and oblique views, should be obtained. In osteoarthritis, X-rays usually show preservation of joint spaces with anterior and posterior osteophytes at the coronoid and olecranon processes and its associated fossae. Loss of joint space does not occur until advanced stages of the disease. In rheumatoid arthritis, X-rays may be normal in early stages as pure synovitis has few radiographic correlates. As the disease progresses, X-rays may show bony erosions, joint space narrowing, and, ultimately, significant joint destruction. Regarding other imaging modalities, obtaining a computed tomography (CT) scan can be helpful in cases of osteoarthritis to evaluate for loose bodies, which is important in planning for surgical debridement. MRI is usually not helpful unless soft tissue pathology is suspected, such as involvement of the MCL in cases of instability.

Non-operative Management

In the early stages of elbow osteoarthritis, activity modification, for example, avoidance of terminal flexion and extension, can be effective. Non-operative treatments including use of NSAIDs, intra-articular corticosteroid injections, and neoprene elbow sleeves (used for comfort) may help. Hyaluronic acid injections may be helpful in the short-term but do not provide long-term benefit. The mainstay of treatment for rheumatoid arthritis of the elbow is medical management with DMARDs (disease-modifying antirheumatic drugs). The goal of medical therapy is control of synovitis and prevention of joint destruction. Corticosteroid injections may also be helpful in the early stages of RA.

Indications for Surgery

Surgery may be considered for patients with significant pain and stiffness affecting activities of daily life despite non-operative treatment. In selecting the appropriate surgical treatment for osteoarthritis, a distinction should be made between stiffness

and pain at extremes of flexion or extension, which may be treated with debridement, versus pain throughout the arc of motion indicative of total destruction of articular cartilage, treated with elbow arthroplasty. Lastly, prior to total elbow arthroplasty, the patient's current functional demands and ability to comply with postoperative activity restrictions should be assessed in order to optimize the likelihood of a successful postoperative course.

Operative Management

Surgical debridement is an appropriate choice for patients with moderate osteoarthritis complaining primarily of elbow stiffness or pain at end range of motion. Release of anterior soft tissues and removal of posterior osteophytes improve elbow extension, and similarly, release of posterior soft tissues and removal of anterior osteophytes improve elbow flexion. Debridement may be performed open or arthroscopically. An open procedure is recommended in the presence of severe contractures requiring extensive release, prior elbow operations with presumed abnormal anatomy, and associated ulnar neuropathy requiring concurrent ulnar nerve release.

For patients with severe osteoarthritis, definitive treatment consists of total elbow arthroplasty (TEA). Following TEA, patients are subjected to lifelong weight-bearing and lifting restrictions—typically 8–10 lbs. This procedure is more suitable for older, low-demand patients. For younger patients, interposition arthroplasty poses fewer postoperative restrictions. The elbow joint should never be fused. Complete resection of the joint and a "flail" elbow is less functionally limiting than fusion in any position. Isolated radiocapitellar arthritis may be treated with radial head excision or radial head replacement.

Similar to the treatment of osteoarthritis, surgical treatment of rheumatoid arthritis depends on the stage of disease. Synovectomy is effective in the earlier stages of disease marked by synovitis with some bony erosions but without significant joint destruction. Good results have been seen with both arthroscopic and open synovectomy. In some cases, synovectomy may be accompanied by radial head resection. The surgical treatment for end-stage rheumatoid arthritis is total elbow arthroplasty.

Expected Outcomes and Predictors of Outcome

Open and arthroscopic debridement for primary osteoarthritis is effective in decreasing pain and improving range of motion. However, in the long term, there is gradual loss of range of motion as arthritis progresses. Following total elbow replacement for primary osteoarthritis and rheumatoid arthritis, the most common complication is early loosening. Revision rates range from 5% to 20%. There is a higher rate of early revisions in younger patients with osteoarthritis. However, once end-stage arthritis occurs, it is favorable to proceed directly to TEA, as surgical intervention

prior to TEA results in poorer outcomes. Other complications include superficial and deep infections and transient ulnar neurapraxias.

Elbow Fractures and Fracture Dislocations

There are numerous patterns of traumatic bony and ligamentous injury to the elbow. These include simple elbow dislocations, complex fracture dislocations with radial head fractures, olecranon fractures, coronoid fractures with their associated ligamentous injuries, and Monteggia lesions—proximal ulna fractures with radial head dislocation. In the management of these injuries, the key is to identify unstable injury patterns that lead to development of arthrosis. The elbow is an intrinsically stable joint owing to its bony anatomy. There are three main articulations: ulnohumeral, radiocapitellar, and proximal radioulnar (Fig. 16.2). The ulnohumeral joint is formed by the articulation of the coronoid process and the semilunar notch of the proximal ulna onto the spool-shaped trochlea of the distal humerus. The radiocapitellar joint is formed by the radial head and the ball-shaped capitellum of the distal humerus. There are two elements to stability: sagittal, which prevents posterior subluxation of the ulna on the distal humerus, and coronal, which counters varus and valgus stresses. Sagittal stability is conferred by the prominent coronoid process anteriorly, which remains in contact with the trochlea even in full extension. Valgus stability is provided by tension on the medial collateral ligament and the bony buttress of the radial head, and varus stability is similarly provided by tension on the lateral ulnar collateral ligament and the bony buttress of the anteromedial facet of the coronoid process. Significant disruption of any of these structures may lead to elbow instability.

Summary of Epidemiology

Most fractures of the elbow occur in the elderly, predominantly women, following a low-energy mechanism of injury such as a fall from standing position. The most common elbow fractures are radial head fractures, accounting for one-third of elbow fractures and 1–4% of all fractures. High-energy mechanism elbow fractures occur more often in young men.

Clinical Presentation

The patient with an elbow fracture presents with pain and swelling around the elbow. The skin should be carefully examined for wounds that could represent an open fracture. Certain injuries, such as elbow fracture dislocations and posteriorly

and laterally displaced Monteggia fractures, may be accompanied by a neurologic deficit, usually due to ulnar nerve injury. A complete neurovascular exam should be performed for all injury types. Escalating pain and significant swelling, especially in the setting of ipsilateral distal radius and proximal ulna fractures, raise concern for compartment syndrome. There are a few exam maneuvers that are specific to various injuries. For example, in a radial head fracture, range of motion testing should include pronation and supination to assess for a mechanical block. In a displaced olecranon fracture, patients should be examined for an extensor lag or a loss of full active elbow extension.

Differential Diagnosis and Suggested Diagnostic Testing

Plain films of the elbow should be obtained at the time of injury, followed by repeated imaging obtained after any manipulation or splinting, as needed. X-rays should be carefully examined for subtle signs of instability such as coronoid fractures, which may appear as a small fleck, or subluxation of the radial head. As the radial head normally points to the center of the capitellum on both AP and lateral views, any deviation indicates instability. Obtaining a CT scan is helpful for further evaluation of a suspected coronoid fracture or radial head fracture on plain films. CT is also used for preoperative planning of complex elbow fractures. Upon diagnosis of an elbow fracture, X-rays of the humerus and forearm are recommended in order to evaluate for ipsilateral injuries. MRI is usually not necessary.

Common Injury Patterns and Management

Simple Elbow Dislocation

In these cases, X-rays show posterior, or less commonly anterior, displacement of the ulna relative to the distal humerus. Twenty-five percent of elbow dislocations are associated with fractures. Simple elbow dislocations—those without associated fractures—are treated with closed reduction under sedation or intra-articular block. Following reduction, stability should be tested with flexion, extension, and varus-valgus stresses. The elbow is temporarily immobilized in a posterior splint for one week, followed by progressive range of motion exercises.

Radial Head Fracture

Patients with radial head fractures will present with tenderness to palpation over the radial head. Range of motion testing assesses for mechanical blocks to forearm pronation and supination, which can occur with displaced fractures. The elbow

should also be tested for valgus instability. With a nondisplaced radial head fracture, X-rays may show an elbow effusion—displacement of the anterior fat pad and visualization of the posterior fat pad—without obvious fracture. X-rays should also be examined for an associated coronoid fracture, indicating instability. CT scan is helpful for identifying subtle associated injuries and for preoperative planning. Isolated nondisplaced or minimally displaced radial head fractures without block to motion can be immobilized very briefly in a sling for comfort, with early return to activity. Immobilization for more than a week may cause permanent loss of motion in the elbow—move it early! Displaced radial head fractures should be splinted, followed by surgical treatment with open reduction and internal fixation versus radial head replacement.

Olecranon Fracture

The olecranon is a subcutaneous structure, and injury often results from direct trauma. On exam, patients should be examined for skin wounds indicative of an open fracture. The integrity of the extensor mechanism should be evaluated. Patients should be examined for an extensor lag, presenting as an inability to actively extend the elbow into full extension. X-rays of the elbow are evaluated to characterize the fracture as nondisplaced versus displaced and simple versus comminuted. Imaging should be reviewed for ipsilateral injuries such as coronoid fractures, radial head fractures, ulnohumeral dislocation, and proximal radioulnar joint dislocation. Isolated olecranon fractures do not cause the elbow to dislocate, even if they are displaced.

Nondisplaced or minimally displaced olecranon fractures are treated nonoperatively with a posterior splint in extension. Displaced olecranon fractures are usually associated with disruption of the extensor mechanism on exam and require surgical fixation. They may initially be splinted with a posterior slab and then referred for surgical fixation on a non-urgent basis. Simple fractures are treated with a tension band construct, whereas comminuted fractures or those associated with other elbow injuries are treated with plates and screws. Very distal fractures may be treated with excision of the small proximal fragment and repair of triceps onto the distal fragment.

Elbow Fracture Dislocations

There are several patterns of elbow fracture dislocation: trans-olecranon fracture dislocation, Monteggia injuries, and posteromedial and posterolateral rotator fracture dislocations. Trans-olecranon fracture dislocation is a displaced olecranon fracture combined with ulnohumeral dislocation. Monteggia injury is a proximal ulna fracture with proximal radioulnar dissociation, which is apparent on X-rays as a radial head dislocation. Posteromedial and posterolateral rotatory fracture dislocations should be suspected on X-rays with small coronoid fractures or radial head

fractures. Rotatory fracture dislocations range in severity—the most severe pattern is the "terrible triad" with elbow dislocation, radial head fracture, and coronoid fracture. It is also associated with injury to the lateral collateral ligament. Dislocation is not always seen on X-ray because the elbow may "snap back" and come to rest in a congruent position, but on exam, there will be anterior-posterior ulnohumeral instability and valgus or varus instability. All of these injuries should be initially splinted and then referred to an orthopedic surgeon for further evaluation. CT is used to further characterize the injury, and management consists of surgical fixation of all bony injuries as well as repair of the lateral collateral ligament as necessary.

Expected Outcomes and Predictors of Outcome

The most common complication of elbow fractures is stiffness. Extension is typically more limited than flexion, and a flexion contracture of as much as 30°–40°may be present. Fractures of the radial head may lead to loss of forearm rotation. Other complications include (1) heterotopic ossification, which occurs more often with associated head trauma or burns; (2) ulnar neuropathy, both subacute and chronic; (3) loss of fixation, which occurs with inadequate fixation of a complex elbow injury; or (4) recurrent instability, which can occur with malreduction of proximal ulna fractures or an unrecognized LCL injury. Finally, post-traumatic arthritis may occur after high-energy mechanism injuries.

Ulnar Collateral Ligament Injuries and Valgus Instability

The medial ulnar collateral ligament (UCL) of the elbow is the primary restraint to valgus stress. There are three bundles of the UCL: anterior, posterior, and oblique (Fig. 16.3). The anterior bundle arises from the medial epicondyle of the humerus and inserts onto the sublime tubercle of the proximal ulna; this structure is the most significant contributor to valgus stability.

Summary of Epidemiology

Injuries of the UCL are most commonly seen in adult throwing athletes, such as baseball pitchers. The late cocking and early acceleration phases of throwing place the most valgus stress on the elbow. In children who participate in throwing sports, medial-sided elbow pain is more commonly a result of medial epicondyle apophysitis, or Little Leaguers' elbow, rather than UCL injury.

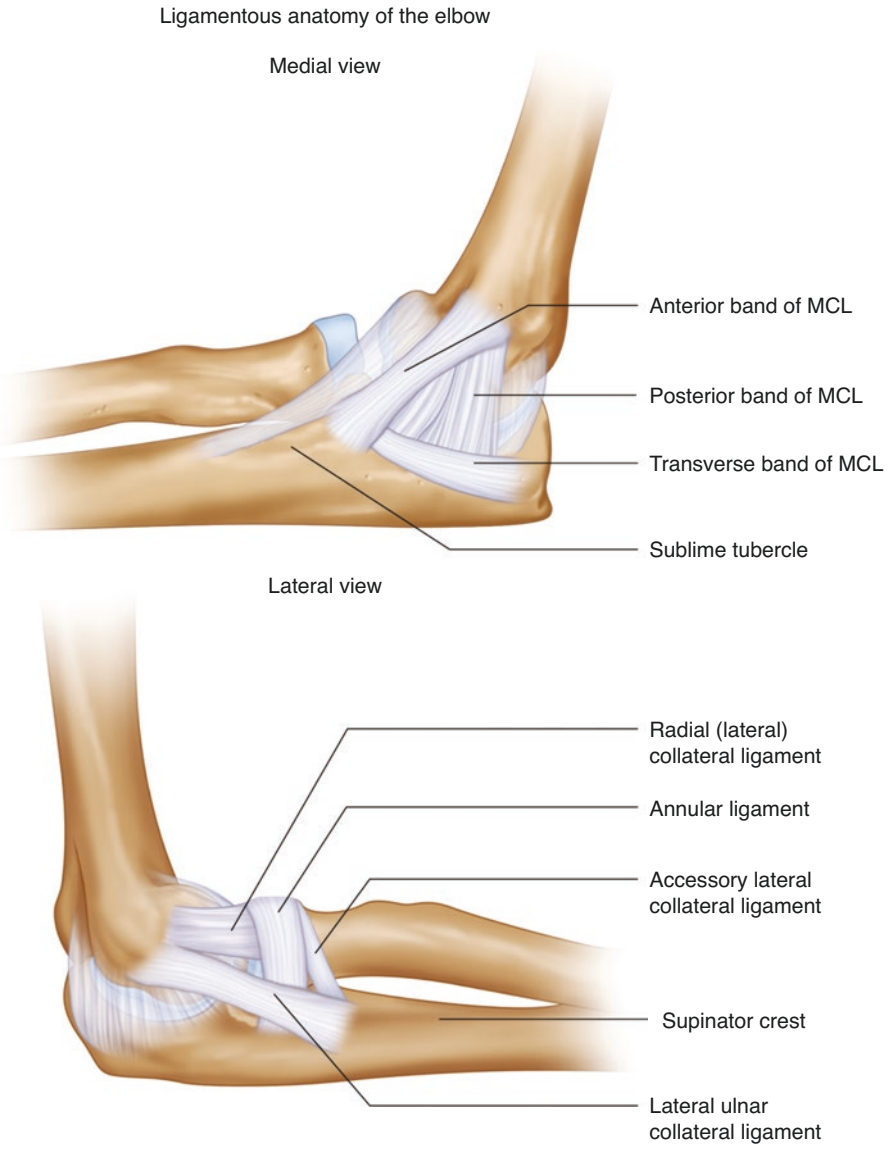

Fig. 16.3 Ligamentous anatomy of the elbow

Clinical Presentation

Patients typically present with medial-sided elbow pain. In acute injury, patients recall a "popping" sensation in the elbow during a throwing event. In chronic injury, patients complain of a dull pain associated with decreased pitching strength or

accuracy. There is tenderness along the flexor-pronator origin in acute injuries, but often no tenderness in chronic injuries. Pain with resisted wrist flexion indicates a flexor-pronator tendon injury, either in isolation or in conjunction with UCL injury. There may also be associated ulnar nerve symptoms. This should be further evaluated with Tinel's sign at the elbow and assessing for motor or sensory deficits. The physical exam should include evaluation of the entire upper extremity, including the shoulder and wrist. In the throwing athlete, there is often an associated deficit in glenohumeral internal rotation (GIRD), which may affect elbow mechanics.

Several tests are used to determine valgus stability. The valgus stress test is performed with the elbow in 30° of flexion, and laxity is compared to the contralateral side. However, this finding may also be positive in asymptomatic throwing athletes. The milking maneuver is performed by abducting the shoulder to 90°, flexing the elbow to 90°, and then supinating the forearm. The examiner pulls on the patient's ipsilateral thumb—like milking a cow—to produce a valgus stress. Pain, apprehension, or instability is indicative of UCL injury. A third test, the moving valgus stress test, is designed to recreate valgus stress during the late cocking and early acceleration phase of throwing motion. The shoulder is abducted to 90°, the elbow is placed in full flexion, and then a valgus stress is applied to the elbow. The test is positive if pain is most severe between 70° and 120° of flexion.

Differential Diagnosis and Suggested Diagnostic Testing

Medial-sided elbow pain may also be the result of flexor-pronator injuries or ulnar neuritis in the absence of a UCL injury; however, these conditions often appear in conjunction with one another. X-rays of the elbow should be obtained to evaluate for elbow osteoarthritis, bony UCL avulsions, and posterior olecranon osteophytes that may result in impingement. A valgus stress X-ray of the elbow can be used to supplement the physical exam. MRI is reliable for diagnosing full-thickness UCL tears, which are apparent as a discontinuous ligament surrounded by edema. The "T-sign," a continuation of T2-intense joint fluid along the undersurface of the UCL, may indicate a partial-thickness tear but may also represent an anatomic variant in which the UCL inserts more distally along the ulna.

Non-operative Management

Non-operative management consists of a 6-week period of rest, followed by gradual return to throwing sports accompanied by appropriate rehabilitation, including addressing shoulder motion deficits, pitching kinematics, and flexor-pronator mass strengthening. Primary prevention of injuries by promoting rest during off season and regulating the number of pitches thrown per year is also important in addressing this problem.

Indications for Surgery

Surgical intervention is favored for high-performing throwing athletes. With the popularization of "Tommy John surgery" or UCL reconstruction, many patients who are not professional athletes may request this operation, but this is not always appropriate. Surgical reconstruction may be considered for patients who do not improve with non-operative methods and are willing to commit to intensive postoperative physical therapy.

Operative Management

Operative management consists of reconstruction of the UCL with an allograft, usually the palmaris longus tendon of the forearm. In this procedure, an incision is made along the medial elbow, and the injured UCL is dissected and removed. The tendon allograft is attached to the medial epicondyle and the sublime tubercle via bone tunnels, resulting in an anatomic reconstruction. This is often accompanied by ulnar nerve release. Postoperatively, early range of motion exercises are initiated, followed by strengthening at 4–6 weeks. Return to competitive sports is deferred until 9–12 months post-op.

Expected Outcomes and Predictors of Outcome

Results are very favorable with UCL reconstruction, with about 80–90% of patients eventually returning to prior level of sports at the major league or collegiate level. The most common complication is a transient ulnar nerve neurapraxia. Other complications can include stiffness and heterotopic ossification.

Overuse Injuries: Little Leaguers' Elbow and OCD Lesions

Athletes who engage in repetitive overhead throwing activities or weight-bearing on their upper extremities (e.g., gymnasts) may be predisposed to characteristic sports-related elbow injuries. Valgus stress on the elbow results in excessive tensile forces on the medial elbow and compressive forces on the lateral elbow. At the medial elbow, this can result in injury to the ulnar collateral ligament (UCL). In children whose growth plates are open, the elbow may instead fail due to injury through the medial epicondylar apophysis. This is the bony attachment point of the flexor-pronator tendons, before it ultimately fuses with the distal humerus. Medial epicondyle apophysitis, or Little Leaguer's Elbow as it is commonly called, typically

consists of mild widening of the apophysis. Occasionally patients can develop significant displacement or medial epicondylar fracture. At the lateral elbow, repetitive microtrauma to the capitellum may result in subchondral fracture, fissure, and fragmentation of the overlying cartilage and, finally, intra-articular loose bodies. This clinical entity is called osteochondritis dissecans (OCD).

Summary of Epidemiology

As more young athletes become involved in high-performance sports, overuse elbow injuries are becomingly increasingly common. OCD is typically seen in children 10–14 years old, and medial epicondyle apophysitis more frequently occurs before then. Elbow injuries most commonly affect baseball players—almost one-third of youth baseball players develop elbow pain in-season—but can also affect gymnasts, tennis players, and wrestlers.

Clinical Presentation

Patients most commonly complain of medial or lateral elbow pain, depending on the etiology. Those with medial-sided pain may have associated symptoms of ulnar nerve irritation. Patients with OCD may present with mechanical symptoms such as clicking, catching, or giving way of the elbow. Baseball players should be questioned regarding the number, frequency, and types of pitches thrown. On exam, there may be tenderness at the medial epicondylar apophysis or at the capitellum, which is most easily palpated with the elbow in hyperflexion. Testing range of motion may show a slight flexion contracture. The elbow should be tested for valgus instability indicative of ulnar collateral ligament insufficiency. The physical exam should also include evaluation of the shoulder, which may show an internal rotation deficit in throwers.

Differential Diagnosis and Suggested Diagnostic Testing

In all cases, AP and lateral X-rays of the elbow should be obtained. In medial epicondyle apophysitis, X-rays show widening of the medial epicondylar apophysis. If there is suspicion for concomitant ulnar collateral ligament insufficiency, gravity stress X-rays should also be obtained. In the evaluation for OCD, a 45° flexion AP view of the elbow may be useful for showing the OCD lesion in profile view. MRI is more helpful for localizing and staging of OCD lesions and is becoming the

standard for diagnosis. In MRI evaluation, T2-weighted sequences that show fluid deep to OCD lesions are indicative of fragment instability. MRI is also helpful in evaluating for ulnar collateral ligament injury or other pathologies. In medial epicondyle apophysitis, MRI may also show increased T2-weighted signal at the medial epicondyle.

Non-operative Management

Non-operative treatment of medial epicondyle apophysitis and OCD consists of a 6–12-week period of rest, followed by physical therapy for flexor-pronator stretching and strengthening, as well as addressing any associated shoulder issues. Patients should gradually return to sports, paying close attention to activity-related pain. Young athletes and their parents should receive age-appropriate activity guidelines, as well as realistic expectations for timing of return to sports.

Indications for Surgery

Most cases of medial epicondyle apophysitis and small avulsion fractures can be treated non-operatively, but displaced fractures of the entire medial epicondyle should be treated surgically. The decision to treat OCD with or without surgery depends on the assessment of OCD lesions either via advanced imaging or arthroscopy. OCD lesions with stable bony fragments and intact overlying cartilage may be treated non-operatively, whereas lesions with unstable fragments and disrupted overlying cartilage, especially those with intra-articular loose bodies, should be treated surgically. Surgery may be considered for patients with OCD who continue to be symptomatic despite non-operative treatment.

Operative Management

The standard treatment for OCD is elbow arthroscopy and drilling. In this procedure, the OCD lesions are unroofed and debrided. Following this, many small drill holes are made in the subchondral bone. The resulting marrow stimulation is thought to promote healing of the subchondral bone. A relatively new procedure is the OATS procedure: osteochondral autogenous transplantation surgery. This procedure consists of debriding the OCD lesion, obtaining an autograft of cartilage and subchondral bone from a remote location (often the distal femur), and transplanting it into the capitellar lesion.

Expected Outcomes and Predictors of Outcome

Younger patients and those in earlier stages of disease are more likely to be treated with non-operative treatment than older patients with closed physes or severe disease. It should be noted that average healing time for OCD lesions exceed 12 months, and patients and families should be advised accordingly. After both OCD drilling and OATS, most patients report symptom improvement; however, rates of return to prior level of activity are variable. Complications of surgical interventions include neurovascular injury, donor-site morbidity following OATS, and recurrence of OCD lesions. Although data on long-term outcomes is limited, some papers suggest progression to elbow osteoarthritis despite surgical treatment. Table 16.1 shows a summary of the conditions discussed in this chapter.

Table 16.1 Diagnosis and management of common conditions

Clinical entity	Clinical presentation	Diagnostic testing	Non-operative management	Indications for surgery	Operative management
Olecranon bursitis	Non-tender fluctuant mass overlying olecranon	Clinical diagnosis – Needle aspiration if septic bursitis is suspected	Compressive dressings, avoidance of pressure to area	Chronic symptomatic bursitis	Open bursectomy
Tennis elbow (lateral epicondylitis)	Pain over lateral epicondyle Pain aggravated by resisted wrist extension and supination	Clinical diagnosis	Activity modification Counterforce brace Physical therapy for stretching and gradual strengthening	Failure of improvement with non-op therapy >12 months	Open debridement of affected tendon origin
Distal biceps tendon rupture	Pain at antecubital fossa Pain or weakness with supination and elbow flexion Positive hook test	MRI if diagnosis is unclear and to distinguish between complete and partial tears	Sling immobilization with close follow-up	Biceps rupture in a patient with high functional demands	Open tendon repair

(continued)

Table 16.1 (continued)

Clinical entity	Clinical presentation	Diagnostic testing	Non-operative management	Indications for surgery	Operative management
Elbow osteoarthritis	Pain and progressive stiffness of elbow Evaluate for ulnar neuropathy	Plain X-rays of elbow CT elbow if loose bodies are suspected	NSAIDs Intra-articular corticosteroid injections Activity modification	Symptomatic arthritis affecting quality of life in an older patient with low functional demands	Surgical debridement Total elbow arthroplasty
Simple elbow dislocation	Pain and deformity of the elbow	X-rays of elbow Elbow CT if subtle coronoid or radial head fractures are suspected	Closed reduction under sedation and/or intra-articular block Immobilization in a splint for 1 week, followed by progressive ROM exercises	Instability post reduction Associated fractures	Open reduction and internal fixation of associated fractures and ligament repair as needed
Radial head fracture	Tenderness to palpation over radial head	X-rays of the elbow, which may show elbow effusion without obvious fracture Elbow CT to evaluate for associated fractures or for preoperative planning	Immobilization in a sling for comfort for 1 week Early return to activity to avoid stiffness	Displaced radial head fracture with mechanical block to pronation or supination Associated fractures or dislocation	Open reduction and internal fixation of radial head Radial head replacement
UCL injury	Medial-sided elbow pain in a throwing athlete Medial elbow pain or valgus elbow instability with milking maneuver	Plain X-rays of the elbow MRI elbow shows partial- and full-thickness UCL tears Evaluate for ulnar neuropathy	Period of rest followed by gradual return to sports Pitching limits for adolescents Physical therapy for the shoulder and elbow, pitching kinematics	No improvement with non-operative management in a patient committed to intensive postoperative physical therapy	UCL reconstruction ("Tommy John" surgery)

(continued)

Table 16.1 (continued)

Clinical entity	Clinical presentation	Diagnostic testing	Non-operative management	Indications for surgery	Operative management
Little leaguers' elbow (medial epicondyle apophysitis)	Medial elbow pain in a young throwing athlete Tenderness at the medial epicondylar apophysis	X-rays of elbow MRI elbow shows increased T2 signal at apophysis, helpful for ruling out other pathologies	Period of rest followed by gradual return to activity Physical therapy for flexor-pronator stretching and strengthening Activity guidelines	Displaced fracture of medial epicondyle	Open reduction and internal fixation of displaced medial epicondyle fracture
Elbow OCD	Lateral elbow pain in a young throwing athlete or gymnast Tenderness to palpation at the capitellum	Plain X-rays of the elbow MRI elbow for localizing and staging OCD lesions	Same as above	Unstable OCD lesions based on MRI or arthroscopic appearance Failure of improvement with non-op therapy	Elbow arthroscopy and drilling of OCD lesions OATS procedure (osteochondral autogenous transplantation surgery)

CT Computed tomography, *MRI* magnetic resonance imaging, *NSAIDS* nonsteroidal anti-inflammatory drugs, *OCD* osteochondritis dissecans, *UCL* ulnar collateral ligament

Summary

In summary, a range of common conditions may affect the upper extremity. We have tried to distinguish between conditions that may appear superficially similar. Most of these evolve slowly; there are many opportunities to make the diagnosis, and conservative measures may often be successful in management. We have tried to show when it may be appropriate to refer for surgery and what you may tell your patient to expect when that happens.

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Part V
The Hand and Wrist

Chapter 17
Hand and Wrist Soft Tissue Conditions

Christina Y. Liu and Brandon E. Earp

Tendon Conditions of the Hand and Wrist

Trigger Finger (Stenosing Tenosynovitis)

Summary of Epidemiology

Trigger finger, also known as stenosing tenosynovitis, is most often an idiopathic condition that affects the flexor tendons to the digits. It occurs more frequently in patients with rheumatoid arthritis and diabetes mellitus and women more than men. The thumb is the most commonly affected digit. As the two flexor tendons to each finger (flexor digitorum profundus (FDP) and flexor digitorum superficialis (FDS)) or the single flexor tendon to the thumb (flexor pollicis longus (FPL)) enter the tighter flexor sheath region, the first pulley encountered is called the A1 pulley. It is typically here, in the palmar region by the metacarpophalangeal joints (MCP), that the pathology occurs. Either the flexor tendon becomes thickened or nodular, the sheath becomes thickened, or both. This leads to a mismatch in size, causing the tendon to get stuck on either side of the pulley, resulting in a "popping" or "triggering" sensation when the finger is flexed or extended. Tenosynovitis can also contribute to triggering in some patients. Of note, histological studies have not shown increased inflammatory cells, but rather fibrocartilaginous metaplasia, suggesting that the etiology of disease is not synovial inflammation.

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Clinical Presentation

Trigger finger typically presents without any associated trauma, although some patients will report onset after heavy gripping or other strenuous hand use. They may describe a "clicking," "popping," or "dislocating" sensation of the finger, which is often worse first thing in the morning. There may also be a tender nodule in the palm near the distal palmar crease of the affected digit. At times, the digit can get stuck in a flexed position ("locked"), and the patient may report having to use the other hand to pull the digit straight. For other patients, the finger may be stuck in extension, and the patient may be quite reluctant or unable to flex the digit into composite flexion due to pain. Patients may complain of discomfort at the PIP joint and may develop PIP contracture with long-standing trigger finger.

Differential Diagnosis and Suggested Diagnostic Testing

Multiple conditions can cause clicking or popping with finger motion. The joints themselves, especially when arthritic, may not move smoothly due to articular incongruity, leading to mechanical symptoms. Extensor tendon snapping due to sagittal band rupture or insufficiency causing central tendon instability dorsally over the metacarpophalangeal joint should also be considered.

Trigger finger is typically diagnosed clinically by physician examination and history and does not require further diagnostic workup.

Non-operative Management

Trigger finger can be treated with observation and may resolve without intervention. Oral and topical medications have not been proven effective. Extension splinting, particularly for those patients who only experience triggering first thing in the morning, may be an effective nighttime-only treatment. Full time extension splinting is not recommended due to concern about resultant joint stiffness. Injection with cortisone is typically the first intervention recommended for treatment of trigger finger. This can be effective when injected either within the tendon sheath (palmar) or around it (mid-axial). Many would recommend avoiding more than two injections for any given trigger digit due to concern about potential tendon rupture. Of note, patients with diabetes should be counselled about close glucose monitoring for the first few days after a cortisone injection as even local injections can elevate systemic glucose levels.

Indications for Surgery

Surgical treatment is recommended for patients with symptomatic triggering of their digits who have failed appropriate non-operative treatment options, or in cases of a locked trigger finger (digit is irreducibly "stuck" in a flexed position).

Operative Management

Surgical treatment involves release of the A1 pulley through a small palmar incision, either percutaneously or by open approach, followed by early motion. If there is persistent triggering or contracture despite surgical release of A1, excision of the ulnar slip of FDS can be considered. These procedures are often performed with the patient awake to allow for testing of triggering after releasing A1 and prior to closing the skin.

Expected Outcome and Predictors of Outcome

Trigger finger may resolve without invasive intervention. Single-joint immobilization of the MCP or DIP for 6–10 weeks has been shown to have up to 90% success in alleviating symptoms, but effectiveness of splinting requires a high level of compliance. Cortisone injection can be curative in approximately half of patients with a single injection. This rate is lower for patients with rheumatoid arthritis or diabetes, for younger patients, or if symptoms have persisted longer than 6 months. About 40% of patients will fail a cortisone injection, either due to initial lack of response or recurrence, and will go on to require surgical release. For patients with persistent symptoms, surgical release is a low-morbidity procedure with an excellent rate of success (97–99%) and very low rate of recurrence.

De Quervain's Disease

Summary of Epidemiology

De Quervain's disease, or first dorsal compartment tenosynovitis of the wrist, affects the radial wrist tendons of the abductor pollicis longus (APL) and the extensor pollicis brevis (EPB). De Quervain's seems to be associated with repetitive wrist motions using the thumbs and is found more commonly in women, especially in mothers with newborn infants. The tendon anatomy in this compartment is variable; the APL commonly has several slips of tendon, and there is often a sub-sheath between the two tendons. These anatomic differences may contribute to the development of symptoms in patients performing repetitive motions, as the increased bulk of the tendons with multiple slips, and the potentially more restricted space (due to a sub-sheath), may not allow as much tolerance of thickening or swelling of the tendons and/or tenosynovium.

Clinical Presentation

De Quervain's disease typically presents without any associated trauma, but most patients will report increased activity with their wrists and thumbs in the time preceding symptom onset. Patients often notice pain along the radial side of the wrist

near the base of the thumb, often associated with swelling in the region. They have difficulty with activities involving the thumb, including pinching and grasping. At times, there may be a sensation of "catching" of the tendons with certain motions of the thumb. Due to the location of the radial sensory nerve branch, which crosses just superficial to the affected tendons, at times patients will experience radial sensory nerve symptoms, including numbness or tingling in the dorsal radial skin of the hand and thumb.

On examination, patients typically have tenderness to palpation over the tendons at the radial styloid and also have pain with resisted radial thumb abduction. A Finkelstein test is commonly performed. The patient's thumb is opposed across the palm and the other digits closed around the thumb. The wrist is then ulnarly deviated, which causes discomfort along the tendons in patients with this disorder.

Differential Diagnosis and Suggested Diagnostic Testing

It is important to evaluate the other structures in the area which may also produce radial wrist and thumb base pain, including thumb carpometacarpal arthritis. Traumatic conditions such as distal radius and scaphoid fractures may also mimic symptoms.

De Quervain's disease is typically diagnosed clinically by physical examination and history, and does not require further diagnostic workup. At times, the practitioner may obtain radiographs to assess the joints in the area for trauma or arthritis and to evaluate for other local conditions.

Non-operative Management

De Quervain's disease is most often treated non-operatively with the goals of decreasing inflammation and maintaining motion. Immobilization with a splint incorporating the thumb up to the IP joint is the mainstay of management. Splints may be custom-made or prefabricated and are typically called thumb spica splints or long opponens splints. Therapy may incorporate exercises to maintain motion and enhance tendon gliding in the region. Injection with cortisone into the first dorsal compartment tendon sheath is another common intervention and is particularly helpful in patients with symptoms of shorter duration.

Indications for Surgery

Surgical treatment is recommended for patients with persistent symptoms which are functionally limiting, despite appropriate non-operative care.

Operative Management

Surgical treatment involves release of the first dorsal compartment tendon sheath through a small radial wrist incision. Care must be taken to protect the superficial radial sensory nerve in the area, to be cognizant of the anatomic variations in the first dorsal compartment, and to ensure the complete release of all tendon slips and any sub-sheath within the compartment.

Expected Outcome and Predictors of Outcome

De Quervain's disease most commonly resolves without invasive intervention via activity modification, rest, and short-term immobilization. Cortisone injection can be curative. For chronic symptoms, combined splinting and cortisone injection may be the most effective. For patients with persistent symptoms, surgical release is a low-morbidity surgery with an excellent rate of success (greater than 91%) and very low rate of recurrence.

Other Nontraumatic Tendon Disorders: Intersection Syndrome, EPL, ECU, and RA-Related Pathology

Summary of Epidemiology

Any tendon in the hand or wrist can be affected by tenosynovitis or tendinosis. Other areas of pathology which are seen with some frequency include (1) intersection syndrome, irritation at the site where the tendons of the first dorsal compartment (extensor pollicis longus (EPL) and abductor pollicis brevis (EPB)) cross over the second dorsal compartment (extensor carpi radialis longus and brevis (ECRL and ECRB)) approximately 4 cm proximal to the wrist; (2) extensor pollicis longus (EPL) irritation as it curves past Lister's tubercle at the distal radius; and (3) extensor carpi ulnaris (ECU) irritation as it passes through a sheath at the level of the distal ulna.

Some tendons are affected by inflammatory conditions such as rheumatoid arthritis (RA) and are prone to invasive tenosynovitis which can lead to attritional ruptures. The tendons most commonly affected by RA are the flexor pollicis longus (FPL) volarly and the fourth and fifth dorsal compartments (extensor digitorum communis (EDC) and extensor digitorum quinti (EDQ)) dorsally.

Clinical Presentation

The clinical presentation of the non-RA-related tendon disorders is quite similar to that of De Quervain's disease. Patients present with localized pain, focal tenderness, and discomfort with active motion. Intersection syndrome can sometimes present with audible crepitus of the tendons.

The clinical presentation of the RA-related tendon disorders typically presents with swelling along the tendons, which is often painless however may be symptomatic at the extremes of motion. The associated tenosynovitis will often be seen to move with active motion of the tendons. There is typically underlying degenerative joint disease on radiographs. With tendon rupture, the patients will be unable to actively move their digits affected by the injury (e.g., with a rupture of the finger extensor tendon to digit 5, the patient will be unable to actively extend the small finger at the MCP joint).

Differential Diagnosis and Suggested Diagnostic Testing

It is always important to assess the underlying joints for arthritis, fracture, or other pathology, which can be seen on X-ray. At times, ultrasound or MRI may be of utility.

Non-operative Management

Non-inflammatory tendon disorders are most often treated non-operatively. Typically, this involves immobilization of the affected structures with splinting and progressive return of motion and function via occupational therapy (OT). Injection with cortisone may be performed for some tendon disorders, but the practitioner should be aware that tendon rupture can occur after injection for some tendon conditions (particularly involving the EPL), and primary surgical treatment may be a safer choice. Management of RA-related tendon disorders is typically via medical management of the underlying disease process, with surgical intervention reserved for failure of that treatment.

Indications for Surgery

Failure of conservative treatment of tendon disorders, including medical management of RA-related conditions, is an indication for surgery. Tendon ruptures secondary to RA are usually treated surgically.

Operative Management

Operative management typically involves freeing the tendons of any constrictive sheaths, debriding areas of tenosynovitis, possibly transposing the tendon(s) to a new position in order to avoid tension or friction (particularly helpful for the EPL), and potentially addressing underlying joint pathology, such as in cases of RA-related distal radioulnar joint (DRUJ) involvement.

Expected Outcome and Predictors of Outcome

Most tendon disorders resolve without invasive intervention via activity modification, rest, and short-term immobilization. Medical management of underlying disease can be a definitive treatment. Cortisone injection can be a valuable tool, but the practitioner should exercise caution as some tendons may be predisposed to rupture after injection. For patients with persistent symptoms, surgical release, tenosynovectomy, and surgically addressing any underlying pathology are an excellent and appropriate choice to alleviate symptoms and minimize future risks of tendon rupture.

Traumatic Tendon Ruptures

Summary of Epidemiology

Trauma can lead to rupture of tendons, often at their distal insertions. The two most common are mallet finger (terminal extensor tendon rupture) and jersey finger (flexor digitorum profundus (FDP) rupture). A mallet injury can occur on any finger and typically is related to a sudden impact to the digit, forcing it into flexion. A jersey finger occurs most commonly in the ring finger and can occur when the flexed digit gets caught on an opponent's jersey and is suddenly pulled into extension. In both cases, the tendon can avulse at its insertion site or can avulse the attached bone, creating a bony fragment.

Clinical Presentation

With a mallet finger, the patient is unable to extend at the distal interphalangeal (DIP) joint of the finger and reports that the fingertip "droops." It is able to be extended passively, but the patient cannot maintain this passive extension unassisted. If the patient has laxity of their joints, they may notice a "swan-neck deformity" as the unopposed pull of the distally ruptured extensor leads to hyperextension at the PIP joint with volar plate laxity.

With a jersey finger (FDP rupture), the patient is unable to flex at the DIP joint of the finger, and therefore cannot tuck the digit into full flexion actively when attempting to make a tight fist. The DIP joint can be passively flexed. The patient will be able to actively flex at the PIP joint due to an intact flexor digitorum superficialis (FDS) tendon.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis for these injuries includes traumatic fracture and dislocation. In theory, nerve palsy could also present as weakness with certain attempted movements, and a thorough neurologic examination of the hand should always be

performed. It would be exceptionally rare to have an isolated digital tendon affected due to nerve injury; however, it is important to ensure that no other weakness exists that has not yet noticed (e.g., a weak FDP to the index could be related to an anterior interosseous nerve palsy, and one would expect to also see weakness of flexor pollicis longus (FPL)).

Radiographs are typically performed to assess the underlying phalanges and articulations for fractures and/or joint incongruity. Both mallet and FDP injuries can occur in conjunction with fractures; thus, imaging is an important part of the assessment.

Non-operative Management

Mallet injuries are typically treated non-operatively with full time (24–7) splinting or finger casting in extension for at least 6 weeks continuously. The PIP joint should be allowed motion to avoid later stiffness. Mallet injuries with fracture are also typically treated non-operatively unless the joint is incongruent due to subluxation. Patient compliance with full-time extension splinting can be challenging.

FDP ruptures are important to recognize early (within the first week) and are nearly always treated with surgery to re-attach the tendon. At times, late presentation or patient characteristics may lead to a choice of non-operative treatment, which involves symptom management and adjusting to the lack of DIP flexion in a specific digit.

Indications for Surgery

Indications for surgical treatment of a mallet injury include joint subluxation and can also be considered in cases wherein a patient has an inability to comply with non-operative care. In cases of FDP rupture, surgery is the treatment of choice in nearly every patient who presents acutely. With a delay in presentation, and dependent on how retracted the tendon is, the tendon may not be reparable, and the decision for or against surgical reconstruction is based on the particular patient and surgeon.

Operative Management

Operative management of a mallet finger may involve isolated extension pinning of the DIP joint or open repair of the tendon to its insertion. Both are followed by maintenance of extension and delayed motion after healing has initiated. Operative treatment of an FDP rupture involves repairing the FDP tendon to its distal insertion site, most often followed by an early motion flexor tendon protocol.

Expected Outcome and Predictors of Outcome

Mallet injuries are nearly always treatable non-operatively and yield acceptable results. Many patients will ultimately have an improved extensor lag (i.e., less "droop"); however, few will regain full active extension. Most do not find this functionally limiting. FDP ruptures are typically treated early with surgery, followed by OT and an early motion therapy protocol. There are risks of re-rupture, and tendon adhesions may limit active motion. A second surgery to address adhesions can be performed if indicated.

Benign Masses of the Hand and Wrist

Ganglions

Summary of Epidemiology

Ganglion cysts are benign masses which can arise from any joint or tendon sheath. They are attached to the joint by a pedicle or stalk and are filled with gelatinous fluid which is much thicker than typical joint fluid and primarily composed of hyaluronic acid. The etiology of ganglion cysts is controversial.

Clinical Presentation

Patients present with a mass which is firm and feels tethered to the underlying tissue. Cysts can be singular or multi-lobulated. In the wrist, the two most common locations are the dorsal scapholunate region and the volar radial wrist near the radial artery. In the digits, the two most common cysts are volar retinacular cysts (cysts of tendon sheath), which are found near the bases of the fingers by the A1 and A2 pulley regions, and mucous cysts, which are found dorsally over the DIP joints.

Ganglion cysts are typically asymptomatic or mildly symptomatic but may create symptoms if they restrict motion and can be cosmetically bothersome. Mucous cysts may lead to nail plate changes due to their location near the germinal matrix of the nail.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis for cysts includes other common masses in the hand, such as hemangiomas, giant cell tumors, glomus tumors, nerve sheath tumors, and lipomas. Typically, these can be distinguished clinically by an experienced practitioner. Superficial ganglion cysts transilluminate, which can help differentiate it from solid masses. Radiographic evaluation is rarely necessary for diagnosis but may be used to assess for other underlying joint conditions. If concerned for an occult wrist ganglion, an MRI may be indicated.

Non-operative Management

Most cysts can be treated non-operatively as they are neither dangerous nor particularly symptomatic, and it has been suggested in the literature that half will spontaneously resolve. For those that do not resolve or are symptomatic, aspiration of the cyst is highly successful at short-term alleviation of the mass and confirmation of the diagnosis due to the pathognomonic clear gelatinous fluid obtained. This said, the patient must understand that recurrence after aspiration is more likely than not. Patients may mention techniques of closed rupture of the cyst by "hitting it with a large book"; however, this is not recommended.

Indications for Surgery

Surgery is indicated for patients with ganglion cysts who are symptomatic and have not responded to or are not appropriate for an aspiration.

Operative Management

Surgical excision involves removal of the cyst, its stalk, and a small portion of the surrounding capsular (or tendon sheath) tissue. Careful dissection and avoidance of injury to nearby neurovascular and tendon structures are important. These are usually performed via an open approach, but arthroscopic data has yielded promising results.

Expected Outcome and Predictors of Outcome

Open surgical excision for focal ganglion cysts is reported to be 95% curative, but recurrence can occur.

Other Benign Soft Tissue Masses of the Hand and Wrist: Hemangiomas, Giant Cell Tumors, Glomus Tumors, Nerve Sheath Tumors, and Lipomas

Summary of Epidemiology

Benign tumors or masses of the hand and wrist are common, and appropriate recognition will often allow for diagnosis without biopsy and treatment with observation. Lesions with a rapid change in size, appearance, or symptoms warrant further evaluation and possible treatment.

The most common benign tumors of the hand and wrist, excluding ganglions, are hemangiomas (vascular masses), giant cell tumors of tendon sheath (histologically similar to pigmented villonodular synovitis), glomus tumors (arising from a glomus body, often in the peri-ungal region of the fingertip), nerve sheath tumors including neurofibromas and schwanommas, and lipomas (fatty tumors).

Clinical Presentation

Most patients with benign hand and wrist tumors present with the discovery of a mass. Hemangiomas are often reddish or purplish in color and compressible. Giant cell tumors are typically non-tender masses along the flexor tendons. Glomus tumors may present as a bluish subungal mass; however, some may not be visible, and instead, patients experience cold hypersensitivity and disproportionate focal finger pain. Nerve sheath tumors will often present with neurologic symptoms and demonstrate a positive Tinel's with percussion. Lipomas are soft, well-circumscribed lesions that typically present as mobile subcutaneous masses.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis includes other masses found in the hand as well as masses related to systemic disease, such as gouty tophus, RA nodules, and xanthomas. Malignant hand tumors are rare; the most common malignancies are attributable to skin cancers followed by metastatic tumors and quite rarely primary soft tissue malignancy such as epithelioid sarcoma. Diagnostic testing may include radiographs. MRI is not commonly indicated but should be used if uncertainty exists. Specific to glomus tumors, MRI can be helpful when a high degree of clinical suspicion exists but no definite mass is noted clinically.

Non-operative Management

Observation is appropriate for many benign tumors of the hand and wrist which are not particularly symptomatic. Routine scheduled follow-up is common, with an understanding that the patient should call to be seen if there are changes in appearance, size, degree of pain, or development of any concerning signs such as fever, weight loss, or neurologic deficits.

Indications for Surgery

Surgery is indicated for symptomatic masses or masses with features concerning for aggressive or malignant lesions.

Operative Management

Surgery typically involves an excisional biopsy. Minimizing the morbidity of the surgery is important. Nerve tumors may have unavoidable postoperative neurologic deficits, and patients must be made aware of this prior to surgery. Glomus tumor excision may lead to nail plate changes due to the often subungal location of these masses. Giant cell tumors have a high rate of recurrence, and thus adjuvant therapy may also be indicated.

Expected Outcome and Predictors of Outcome

Outcomes from treatment of benign lesions and recurrence after surgery depend on the type of lesion, location, size, and surgery performed.

Dupuytren's Disease

Summary of Epidemiology

Dupuytren's disease is a fibroproliferative condition of the palmar fascia. It is benign, typically painless, and variably progressive. There is a genetic basis for Dupuytren's, as the condition is commonly associated with patients of northern European descent and is also found more commonly in patients with diabetes mellitus. Eighty percent of patients are men, and most patients present after the age of 50. Some patients will have associated fibromatoses, including Peyronie's disease (penile fibromatosis) and Ledderhose disease (plantar fibromatosis). The myofibroblast is the affected cell, and histologically, the nodules are comprised of Type III collagen.

Clinical Presentation

The typical patient presents with nodularity noted in the palm in the line of the ring and/or small fingers. There may be "pitting" or dimpling of the skin in the area. Over time, the nodule(s) can form longitudinal cords which lead to flexion contracture of the MCP and/or PIP joints. At times, patients will also present with soft tissue thickenings over the dorsum of their PIP joints, called "knuckle pads." The nodules and cords are typically painless, although can be mildly tender.

Differential Diagnosis and Suggested Diagnostic Testing

Dupuytren's disease is a clinical diagnosis and does not require further imaging or testing. However, it is important to rule out other conditions causing joint contracture (arthritis, prior trauma, camptodactyly, and isolated palmar fibromatosis) and other conditions causing palmar nodules. Of note, isolated palmar fibromatosis affects all digits of both hands and can be associated with malignancy.

Non-operative Management

There is no data to suggest that splinting, stretching, and exercises will prevent progression of disease. Cortisone injection has not been shown to decrease Dupuytren's cords, but may help alleviate pain temporarily. While there is no cure for Dupuytren's, therapeutic interventions can help with disease symptoms. There are two procedurally based non-operative treatments, needle aponeurotomy, which mechanically breaks apart the cords, and collagenase injection, which enzymatically breaks apart the cords. Both can be performed in an office-based setting and serve to disrupt the Dupuytren's cord and allow manipulation of the digit to alleviate the contracture.

Indications for Surgery

Surgery is indicated to address the contracture associated with Dupuytren's disease. There is not an exact amount of contracture that constitutes the threshold for surgical treatment, but most agree that functional impairment with greater than 30 degrees of MCP joint contracture or greater than 20 degrees of PIP contracture is an indication for treatment. The type of surgery or percutaneous approach depends on the patient and surgeon preferences.

Operative Management

Surgery involves excision of the Dupuytren's cords, which is called a fasciectomy. The overlying skin may also be excised. Occasionally, skin grafts are indicated, or intentional open wounds are left to heal secondarily. Joint contractures are commonly addressed simply by removing the diseased tissue, but with more advanced contractures, releases of the contracted tendon sheath or joint may be necessary.

Expected Outcome and Predictors of Outcome

Treatment with either percutaneous approaches (needle aponeurotomy and collagenase injection) or surgery is quite effective at improving contractures. Patients must understand that recurrence occurs after all of these treatments and full correction of contracture may not be possible, especially with long-standing or more severe PIP joint involvement. Needle aponeurotomy is 80% effective for MCP involvement and 65% for PIP involvement, but there is up to 85% recurrence at a 5-year followup. Collagenase injection has been shown to have over 50% recurrence at 3 years. Despite the high recurrence rate with both percutaneous approaches, there remains a high patient satisfaction given the quick recovery and minimal pain. Open surgery is more effective at alleviating symptoms with low recurrence rate of about 20–25% at 5 years, but recovery can be slower given the more invasive approach.

Soft Tissue Injuries of Wrist Joint

Scapholunate Ligament Injury

See Chap. 18.

Triangular Fibrocartilage Complex (TFCC) Injury

Summary of Epidemiology

The TFCC is a localized group of soft tissue structures including cartilage and ligaments on the ulnar side of the wrist, which serves to stabilize the distal radioulnar joint (DRUJ) and the ulnar carpus. The structures comprising the TFCC include the articular disk, the meniscal homologue, the volar and dorsal radioulnar ligaments, the extensor carpi ulnaris sub-sheath, the ulnar capsule, and the ulnocarpal ligaments. Combined, these form a trampoline-like structure which extends from the distal radius to the distal ulna to the ulnar carpus, supporting the surrounding articulations. The central portion of the TFCC is avascular which leads to limited healing potential of injuries to that area. The edges of this triangular structure are perfused via their attachments, making repair possible at the periphery.

Two types of TFCC pathology exist. The first is related to a traumatic injury. These are more commonly seen in athletes and manual laborers but can also be associated with falls. The second type is degenerative in nature. The TFCC thins with age, and half of people over age 60 will have degenerative perforations of the articular disc.

Clinical Presentation

It is important to evaluate the time course of symptoms, conduct a complete history of the patient's condition, and perform a thorough examination. Patients with symptomatic TFCC pathology present with complaints of ulnar-sided wrist pain, often associated with clicking. Traumatic-type injuries are frequently related to a specific event leading to symptom onset. Degenerative-type injuries may be related to repetitive pulling or twisting motions, and those patients may recall a remote history of wrist injury. Symptoms typically improve with rest and are exacerbated by twisting, pushing, pulling, or lifting activities.

Common exam findings for patients with TFCC pathology include reduced grip strength, tenderness in the ulnar fovea (soft spot just distal and volar to the ulnar styloid), a click or pop with motion of the wrist, and pain with combined ulnar deviation and extension of the wrist.

Differential Diagnosis and Suggested Diagnostic Testing

There are many causes of ulnar wrist pain which need to be distinguished from a TFCC injury. These include fractures, joint injuries or other conditions, tendon disorders, and nerve-related pain. Bony injuries may include ulnar styloid, hamate, pisiform, and metacarpal base fractures. Joint conditions can be related to lunotriquetral ligament tear, DRUJ instability, Kienbock's disease, Madelung deformity, DRUJ arthritis, or midcarpal instability. Tendonitis can be seen in both the extensor carpi ulnaris and the flexor carpi ulnaris, both of which are localized to this region. Nerve-related pain can be caused by Guyon's canal syndrome.

After a thorough history and physical exam, X-ray of the wrist should be obtained. Patients with ulnar positive variance on the PA view (the ulna is "longer" than the radius), whether occurring by native anatomic difference or post-traumatic malunion, are at a higher risk for TFCC pathology. The films can also evaluate for other bone and joint conditions, such as fractures, congenital differences, and arthritis. MRI is also helpful to visualize TFCC pathology. The addition of an arthrogram to the MRI may increase both the sensitivity and specificity of the study by demonstrating the passage of dye through small perforations in the TFCC. The Palmer classification is used to stratify patients with TFCC pathology (Fig. 17.1).

Non-operative Management

The goals of non-operative treatment of TFCC pathology include decreasing swelling and pain, which can be accomplished by immobilization for 3–6 weeks, icing, and NSAIDs. During this time, the unaffected joints of the arm and hand should be kept moving to minimize functional loss and stiffness. After the immobilization period, rehabilitation aims to restore motion and then strength.

Class 1 -traumatic injury

a) Central perforation

b) Ulnar avulsion

- May involve the proximal or distal lamina (foveal or styloid attachment, respectively), or both

c) Distal avulsion

d) Radial avulsion

Class 2 - degenerative injury

a) TFCC wear

b) TFCC wear with lunate and/or ulnar chondromalacia

c) TFCC perforation with lunate and/or ulnar chondromalacia

d) TFCC perforation with lunate and/or ulnar chondromalacia and lunotriquetral ligament perforation

e) TFCC perforation with lunate and/or ulnar chondromalacia, lunotriquetral ligament perforation, and ulnocarpal arthritis

Fig. 17.1 Palmar classification for TFCC abnormalities. The Palmar classification was first described by Palmar in 1989 and divides TFCC pathology into traumatic and degenerative categories to help guide management

Indications for Surgery

Surgery is indicated for patients who have persistent, functionally limiting symptoms despite non-operative management, or for those with an acute unstable DRUJ.

Operative Management

Operative decision-making is guided by the history, physical exam, and imaging studies and may include arthroscopic debridement or repair, open repair, and various forms of ulnar shortening, such as wafer excision or osteotomy. The decision of whether to debride or repair is based on the location of the pathology. Due to the avascularity of the majority of the TFCC, tears in the central region cannot heal and are treated with debridement. Peripheral tears have healing potential and should be reattached to the surrounding tissue. Ulnar shortening is performed if ulnocarpal impaction (abutment of the distal ulna on the carpus) is contributing to symptoms and can be completed in several different ways.

Expected Outcome and Predictors of Outcome

Non-operative treatment is successful at achieving symptom improvement in most patients with ulnar wrist pain, including those with TFCC pathology. Arthroscopic debridement of central TFCC tears can achieve full symptom resolution with a stable DRUJ despite the altered anatomy. Treatment of peripheral TFCC tears with arthroscopic or open repair leads to symptomatic improvement in most patients, with variable rates of actual TFCC healing depending on the location of the tear. Ulnar shortening osteotomy or wafer resections for ulnar positive variance also lead to alleviation of pain in the vast majority of patients.

In conclusion, hand and wrist soft tissue conditions are multiple, varied, and extremely common. Accurate diagnosis can lead to appropriate and successful management for most of these conditions. Both the pathologic condition and various patient factors may influence decision-making and the likelihood for successful treatment (Table 17.1).

Table 17.1 Summary of hand and wrist soft tissue condition

Clinical entity	Presentation	Diagnostic Testing	Conservative management	Surgical indications & operative management
Trigger finger (stenosing tenosynovitis)	Insidious or acute onset of catching or clicking of the finger with motion Tender to palpation in the palm at the distal palmar crease of the affected digit Pain with motion (may lead to decreased motion and even flexion contracture of the PIP joint)	Primarily a clinical diagnosis Imaging is not typically performed	With early symptoms, consider rest, ice, and night extension splinting for morning-only symptoms In persistent cases, cortisone injection	Surgery indicated for persistent symptoms despite conservative treatment Release of the A1 pulley is curative

(continued)

Table 17.1 (continued)

Clinical entity	Presentation	Diagnostic Testing	Conservative management	Surgical indications & operative management
De Quervain's disease	Insidious or acute onset of pain at the radial wrist with thumb/wrist motion Tender to palpation over the first dorsal compartment tendons at the radial styloid Finkelstein test Pain with resisted radial thumb abduction	Primarily a clinical diagnosis X-rays may be used to evaluate for other pathology in the area (fractures, CMC arthritis)	Acute: rest, ice, NSAIDs, splinting (thumb spica or long opponens) OT: motion/tendon glide In persistent cases, cortisone injection	Surgery indicated for persistent symptoms despite conservative treatment Release of the first dorsal compartment tendon sheath (and any sub-sheath) is curative
Other nontraumatic tendon disorders (intersection syndrome, EPL, ECU, and RA related)	Insidious or acute onset of pain with or without swelling along the tendon sheath Tender to palpation over the involved tendon(s) Pain with active movement of the involved tendon(s) Tenosynovitis may move with active motion of tendons RA-related conditions may be painless but can result in tendon ruptures (lack active motion of involved tendon) Intersection syndrome may have audible crepitus	Primarily a clinical diagnosis X-rays may be used to assess for underlying joint pathology	Acute: rest, ice, NSAIDs, immobilization In persistent cases, cortisone injection (be aware that some tendons can rupture after injection, notably EPL) For RA-related conditions, medical management of RA	Surgery indicated for persistent symptoms despite conservative treatment May involve release of tendon sheath, tenosynovectomy, and tendon transposition For RA-related conditions, surgery may need to address the underlying joint and tendon grafting/transfers may be indicated for ruptures

(continued)

Table 17.1 (continued)

Clinical entity	Presentation	Diagnostic Testing	Conservative management	Surgical indications & operative management
Mallet finger	Usually acute onset Frequently related to a direct impact to the digit (can be mild trauma) Extensor lag (inability to extend the DIP joint)	X-rays: evaluate for associated fracture of the dorsal base of the distal phalanx and volar subluxation of the DIP joint	Acute: usually full-time extension splinting or finger casting of DIP for 6 weeks followed by night splinting for 6 weeks (total of 12 weeks) PIP must be kept mobile during this time to avoid stiffness	Surgery is indicated for failure of non-operative treatment or inability to tolerate non-operative treatment May involve extension pinning or open repair of the terminal extensor tendon to its insertion
FDP rupture (jersey finger)	Usually acute onset Most common in the ring finger Frequently related to sudden extension force against a flexed digit Inability to flex the DIP joint May be tender in the palm, depending on the degree of proximal retraction of the torn tendon stump	X-rays: evaluate for associated fracture of the volar base of the distal phalanx and dorsal subluxation of the DIP joint Fracture fragments that have retracted proximally may give clues as to the location of the tendon stump	Acute: surgery For chronic conditions, late presentation, or due to patient factors, non-operative symptomatic management may be appropriate	Acute: surgical repair of the FDP tendon to its distal insertion, followed by an early motion flexor tendon protocol

(continued)

Table 17.1 (continued)

Clinical entity	Presentation	Diagnostic Testing	Conservative management	Surgical indications & operative management
Benign masses	May present with painful or painless mass of the wrist, hand, or digit	X-rays: evaluate associated bones and joints for pathology MRI rarely necessary but may be indicated for atypical presentations or to evaluate for glomus tumors	Benign tumors (asymptomatic, stable, and classically appearing masses): observation may be appropriate Routine instructions to call re: changes in size, symptoms, or appearance are required Aspiration can be performed for cystic masses for both diagnostic and therapeutic purposes	Surgical excisional biopsy is performed for both diagnosis and treatment when appropriate Type of biopsy, recurrence rate, and follow-up are dependent on the type of tumor, location, and patient characteristics
Dupuytren's disease	Typically painless insidious onset Often begins with palmar nodules that progress to cords and lead to digital contractures Skin pitting and knuckle pads may also be seen Most commonly affects ring and small fingers	Primarily a clinical diagnosis X-rays: may be used to assess for arthritic changes in associated joints	Observation can be appropriate for isolated palmar nodules or small MCP contractures that are not functionally limiting Non-operative procedures include needle aponeurotomy (mechanical) and collagenase injection (chemical) followed by physical manipulation to alleviate the contracture	Surgical excision (fasciectomy) of the Dupuytren cord with or without joint release may be performed to address significant or recurrent contractures Decision-making is based on the patient's condition and patient and surgeon preference

(continued)

Table 17.1 (continued)

Clinical entity	Presentation	Diagnostic Testing	Conservative management	Surgical indications & operative management
TFCC injury	Insidious or acute onset of pain with ulnar wrist pain Etiology: traumatic or degenerative Tender to palpation in the ulnar fovea Pain may be elicited with combined ulnar deviation and extension of the wrist	X-rays: evaluate ulnar variance MRI +/− arthrogram: visualization of TFCC and other soft tissue pathology Be aware that 50% of patients over 60 will have TFCC degenerative perorations, and these are commonly asymptomatic	Rest, ice, NSAIDs, and immobilization are appropriate for most patients without DRUJ instability	Surgery indicated for persistent symptoms despite conservative treatment Debridement or repair of TFCC tear depends on whether tear occurred in the avascular central region or along the vascularized periphery Ulnar shortening procedures may be indicated for ulnar positive variance

Suggested Reading

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Chapter 18
Hand and Wrist Arthritis

Dafang Zhang and Barry P. Simmons

Abbreviations

CMC	Carpometacarpal joint
CPPD	Calcium pyrophosphate deposition disease
DIP	Distal interphalangeal joint
DRUJ	Distal radioulnar joint
MCP	Metacarpophalangeal joint
MRI	Magnetic resonance imaging
NSAID	Nonsteroidal anti-inflammatory
PA	Posteroanterior
PIP	Proximal interphalangeal joint
SLAC	Scapholunate advanced collapse
SNAC	Scaphoid nonunion advanced collapse
STT	Scaphotrapeziotrapezoid

Introduction

Arthritis is a degenerative disease of articular cartilage and can be considered either primary, wherein genetic predisposition may play a role, or secondary, in the setting of prior trauma, inflammatory or autoimmune disease, infection, or other identifiable events. Clinically, patients with arthritis present with pain, decreased range of motion, and progressive deformity. Radiographic hallmarks of arthritis include joint

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space narrowing, osteophyte formation, subchondral sclerosis, and cyst formation or erosion seen on plain radiographs.

The prevalence of primary osteoarthritis of the hand and wrist increases with age and is more common in women, with the exception of MCP joint and wrist osteoarthritis. In the hand, the most common joints affected by osteoarthritis include the distal interphalangeal joint (DIP), followed by the thumb carpometacarpal joint (CMC), the proximal interphalangeal joint (PIP), and finally the metacarpophalangeal joint (MCP).

There are a number of inflammatory, autoimmune, and systemic etiologies of arthritis in the hand and wrist, the most common of which is rheumatoid arthritis. The cause of rheumatoid arthritis is multifactorial, with genetic and environmental components. The wrist is most commonly affected in rheumatoid arthritis, and the MCP is affected more commonly than the interphalangeal joints.

Post-traumatic Wrist Osteoarthritis: Scapholunate Advanced Collapse (SLAC) and Scaphoid Nonunion Advanced Collapse (SNAC)

The wrist is a complex arrangement of eight carpal bones that work in concert to facilitate motion in multiple planes (Fig. 18.1). Generally speaking, these carpal bones are divided into two distinct rows. The proximal row of carpal bones is

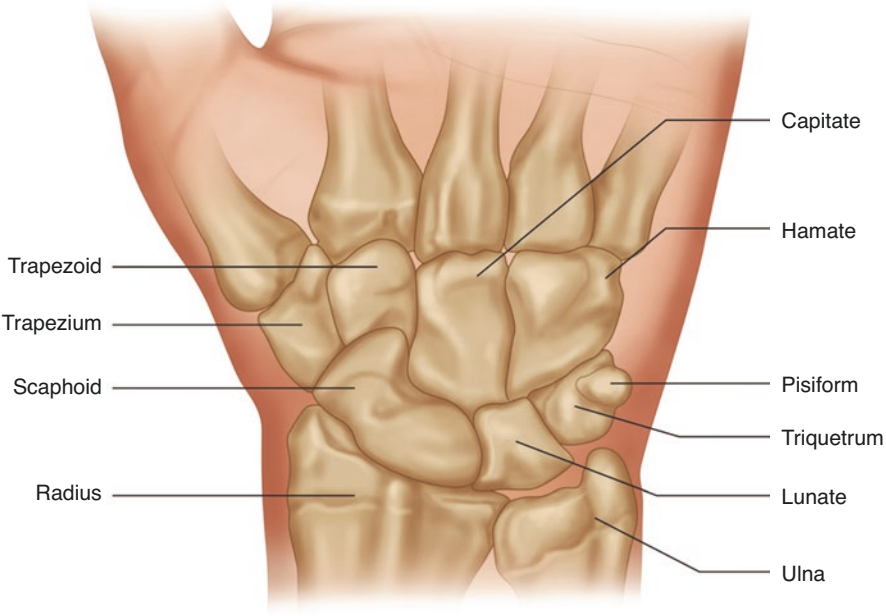

Fig. 18.1 Anatomy of the wrist joints. There are eight carpal bones, which form the radiocarpal, midcarpal, and carpometacarpal joints of the wrist

comprised of the scaphoid, lunate, triquetrum, and pisiform. This proximal row articulates with the distal aspect of the forearm bones, the radius and the ulna, to form the radiocarpal joint and the ulnocarpal joint. The distal row of carpal bones is comprised of the trapezium, trapezoid, capitate, and hamate. This distal row articulates with the metacarpal bones to form the carpometacarpal joints. The articulation between the proximal and distal carpal rows is referred to as the midcarpal joint. The scaphoid acts as an integral link in the kinematics of the proximal and distal carpal rows.

Primary osteoarthritis of the wrist is uncommon; however, secondary osteoarthritis due to trauma or vascular disease is more prevalent. The overall reported prevalence of wrist osteoarthritis is 1%, with a slight male predominance.

Fractures of the carpal bones or disruptions of the ligaments that stabilize the carpal bones and allow them to move in a coordinated fashion result in altered wrist kinematics and associated degenerative changes. Specifically, patients diagnosed with unhealed scaphoid fractures and scapholunate ligament injuries are at a risk of developing a characteristic pattern of wrist arthritis, known as scaphoid nonunion advanced collapse (SNAC) and scapholunate advanced collapse (SLAC), respectively.

Pathoanatomy

In normal wrist motion, the scaphoid bone flexes with wrist flexion and radial deviation and extends with wrist extension and ulnar deviation. The scapholunate ligament stabilizes the joint between the scaphoid and the lunate, and disruption of this ligament leads to an abnormal movement pattern in which the scaphoid flexes while the lunate extends during wrist motion. These altered mechanics lead to an abnormal distribution of forces across the radiocarpal and midcarpal joints, with resultant wrist osteoarthritis known as scapholunate advanced collapse (SLAC). Acute injuries of the scapholunate ligament are distinct from degenerative tears which can be seen in greater than 50% of patients over 80 years of age.

Scaphoid fractures are inherently at a high risk of nonunion due to the tenuous retrograde blood supply of the scaphoid bone. The more proximal the fracture location, the higher the risk for nonunion. The scaphoid bone bridges the proximal and distal carpal rows; thus, scaphoid fracture nonunion disrupts the synchronous movement of the carpal bones during wrist motion. Over time, degenerative changes of the wrist joint can occur, referred to as scaphoid nonunion advanced collapse (SNAC).

Clinical Presentation

Scapholunate ligament injuries occur following a sudden impact to the hand and wrist. The most common mechanism is a fall onto an outstretched wrist. Acute scapholunate ligament injuries typically present as dorsal, radial-sided wrist pain

with associated decreased grip and pinch strength. Patients will often report difficulty with wrist extension. More specifically, patients will report that their symptoms are exacerbated with "push-off" activities, which require loading across an extended wrist. They may also report clicking or catching across the wrist due to abnormal translation of the scaphoid.

On physical exam, there may be a joint effusion with swelling seen over the dorsal aspect of the wrist. Wrist extension will typically cause increased pain. Palpation over the scapholunate interval, which is located distal to Lister's tubercle, elicits tenderness. The scaphoid shift test is a provocative test used to detect instability of the scapholunate ligament. Dorsally directed pressure is applied over the volar aspect of the scaphoid while the wrist is brought from ulnar deviation to radial deviation. Dorsal wrist pain while performing this maneuver reflects abnormal dorsal subluxation of the scaphoid due to loss of scapholunate ligament stabilization. A clunk can sometimes be appreciated when the dorsally directed pressure is released and reflects relocation of the scaphoid into the scaphoid fossa. In chronic injuries, the scaphoid shift test may no longer be positive as advanced arthritic changes stabilize the scaphoid and wrist stiffness prevails. Patients may have tenderness at the radioscaphoid joint.

In cases of scaphoid fracture, patients typically report a remote history of a fall onto an outstretched wrist. SNAC presents with weakness with grip and pinch as well as joint stiffness, particularly with wrist extension and radial deviation. There may also be localized tenderness about the radioscaphoid articulation.

Differential Diagnosis and Suggested Diagnostic Testing

The diagnosis of acute scapholunate ligament injury is based on physical exam and imaging. Other causes of dorsal-sided wrist pain include wrist sprain, dorsal wrist ganglion cyst, extensor tenosynovitis, and intersection syndrome. Intersection syndrome is inflammation in the region where the extensor tendons of the wrist and thumb cross, commonly localized 5 cm proximal to the wrist joint, and is associated with repetitive wrist extension.

Acute scapholunate ligament injury is initially evaluated with posteroanterior (PA) and lateral radiographs of the wrist. A clenched fist view is a stress view that may reveal diastasis between the scaphoid and lunate bones, which may not be appreciated on static views. Magnetic resonance imaging (MRI) can also be used to evaluate for scapholunate injuries but must be evaluated carefully due to the high sensitivity but low specificity for this type of pathology. Wrist arthroscopy is the gold standard to diagnose and appropriately grade the severity of a scapholunate ligament injury.

Advanced arthritic changes associated with chronic scapholunate injury are best evaluated on PA and lateral radiographs (Fig. 18.2). On PA radiographs, widening of greater than 3 mm is seen between the scaphoid and lunate. The severity of

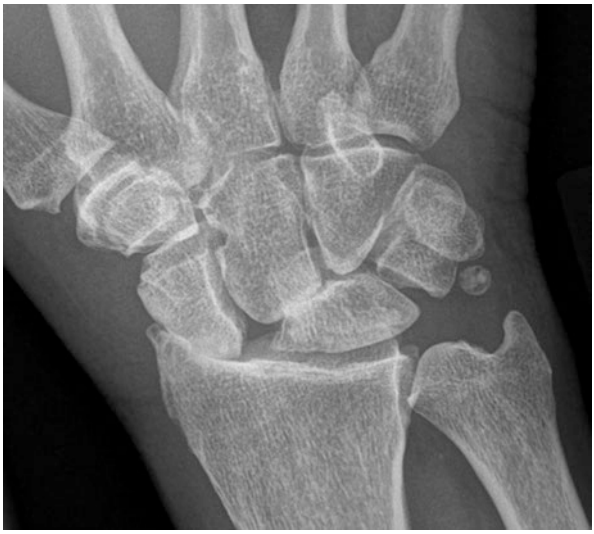

Fig. 18.2 Plain radiograph of Stage III SLAC wrist with sclerosis and joint space narrowing of the radioscaphoid joint and lunocapitate articulation

arthritic changes seen in SLAC is classified from Stage I to Stage III. In Stage I disease, there is narrowing of the joint space between the radial styloid and the scaphoid. In Stage II disease, arthritic changes have progressed to involve the entire radioscaphoid articulation, such that narrowing is seen between the scaphoid and the entire scaphoid fossa. Finally, in Stage III disease, sclerosis and joint space narrowing is also seen between the lunate and capitate and proximal migration of the capitates into the widened scapholunate interval ensues. There may also be localized tenderness about the radioscaphoid articulation.

SNAC can be diagnosed based on plain PA and lateral radiographs of the wrist. Radiographic changes seen in SNAC progress in a similar fashion as described for SLAC. In Stage I disease, there is joint space narrowing and sclerosis involving the radial styloid and scaphoid. In Stage II disease, degenerative changes are seen at the scaphocapitate articulation. Finally, in Stage III disease, periscaphoid degenerative changes are seen. Radial-sided wrist pain in SNAC wrist should be distinguished from de Quervain's tenosynovitis or adjacent base of thumb carpometacarpal osteoarthritis.

Non-operative Management

Treatment decisions for post-traumatic wrist arthritis are based mainly on symptom severity. Non-operative management is indicated as the first-line treatment in the majority of symptomatic patients. Non-operative management includes a trial of nonsteroidal anti-inflammatory (NSAID) medications, immobilization with a removable wrist brace, and activity modification. Corticosteroid injections can also provide significant symptomatic relief.

Indications for Surgery

Surgical options may be considered in patients who continue to have debilitating symptoms despite an adequate trial of non-operative management.

Operative Management

The most appropriate surgical intervention for post-traumatic wrist osteoarthritis depends on the severity of arthritic changes. In early Stage I disease, open or arthroscopic approaches can be used to perform a radial styloidectomy to prevent impingement between the scaphoid and radial styloid. In select patients who have a symptomatic SNAC wrist, with minimal arthritic change, excision of the distal non-united scaphoid fragment can be considered.

In more advanced Stage II disease, there are two different techniques that eliminate the painful radioscaphoid articulation while preserving motion through the wrist joint. A proximal row carpectomy involves excision of the scaphoid, lunate, and triquetrum in their entirety. The capitate, originally part of the distal carpal row, comes to rest in the lunate fossa of the distal radius, and wrist motion occurs through this new articulation. Alternatively, a scaphoid excision and four-corner fusion, or partial wrist fusion of the lunate, capitate, hamate, and triquetrum, can be performed. This procedure preserves motion through the wrist joint via the maintained articulation between the lunate and distal radius.

In Stage III disease, a total wrist fusion is typically recommended, often providing a stable and painless joint. Total wrist joint replacement is rarely used to treat wrist osteoarthritis due to high implant failure rates over time and the need for activity restrictions following the procedure. Partial denervation of the wrist capsule via posterior interosseous nerve excision is often performed concurrently with the above procedures. The role of partial wrist denervation of the anterior and posterior interosseous nerves in lieu of a bony procedure for wrist osteoarthritis is an evolving area of interest and may provide patients temporary relief.

Expected Outcome and Predictors of Outcome

There are no studies that examine the long-term success of non-operative management of SNAC and SLAC wrists; however, non-operative management is typically more successful in the early stages of disease. With regard to surgical outcomes, proximal row carpectomy and scaphoid excision and four-corner fusion have similar functional outcomes. Patients generally report satisfactory pain relief, strength, and function following these procedures. Patients can expect to achieve postoperative wrist motion that is about 60% compared to that of the contralateral unaffected

wrist. On average, patients achieve 80% grip strength compared to the contralateral unaffected wrist. After proximal row carpectomy, younger patients are at a higher risk of developing secondary degenerative changes of the capitate, which may necessitate secondary procedures. Only a minority of patients who undergo proximal row carpectomy and scaphoid excision with four-corner fusion require secondary procedures for persistent pain or nonunion.

Total wrist fusion results in reliable pain relief and patient satisfaction at the expense of wrist motion. Patients often must adapt the way they perform certain activities that require manipulation of the hand in tight spaces and self-hygiene. Complications are rare but include nonunion, extensor tendon adhesion, infection, poor wound healing, and painful hardware.

Wrist Rheumatoid Arthritis

Rheumatoid arthritis is an inflammatory arthritis that results from a T-cell-mediated destructive process of the joint. The inflammatory process of the synovial lining of the joint leads to synovial hypertrophy, weakening of the supporting ligaments of the joint, and articular destruction. The cause of rheumatoid arthritis is unknown, but is thought to result from a combination of genetic and environmental factors.

Epidemiology

Rheumatoid arthritis is the most common form of inflammatory arthritis and affects as much as 1% of the general population, women more so than men. More than 70% of patients with rheumatoid arthritis have hand and wrist manifestations. However, the medical management of rheumatoid arthritis has significantly improved since the development of disease-modifiable pharmacologic therapies, and severe manifestations of rheumatoid arthritis are fortunately less commonly seen.

Clinical Presentation

While the wrist is most commonly affected, rheumatoid arthritis may present with polyarticular involvement of the shoulders, elbows, knees, ankles, or cervical spine. Synovitis of the wrist and distal radioulnar joint (DRUJ) leads to a classic deformity in which the carpus supinates away from the head of the ulna, termed the caput ulna syndrome. In the hands, MCP and PIP joints may be involved, while the DIP joints are generally spared. Joint stiffness in the morning, symmetric joint involvement, joint swelling, and rheumatoid nodules are also frequent presenting symptoms and signs.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis for rheumatoid arthritis includes other inflammatory, autoimmune, or systemic causes of arthritis. These include psoriatic arthritis, systemic lupus erythematosus, systemic sclerosis, and crystalline arthropathies such as gout and calcium pyrophosphate deposition disease (CPPD).

PA and lateral radiographs are important in the workup of inflammatory arthritis and may show bony erosions and decalcifications. In advanced cases, collapse of the carpal bones and the classic caput ulna appearance of the distal ulna can be seen on radiographs. As the head of the ulna dislocates dorsally, congruity of the DRUJ is lost, and carpal impaction may be seen.

Laboratory studies may be useful to establish the diagnosis of rheumatoid arthritis. Acute phase reactants, including erythrocyte sedimentation rate and C-reactive protein, are nonspecific markers for inflammation. Antibody tests, including rheumatoid factor, anti-citrullinated peptide (anti-CCP) antibodies, and antinuclear antibodies (ANA), are helpful with various degrees of sensitivity and specificity, but must be interpreted in the context of the clinical presentation and titers.

Non-operative Management

Medical management is the mainstay of rheumatoid arthritis and consists of nonsteroidal anti-inflammatory medications (NSAIDs), corticosteroids, and disease-modifying antirheumatic drugs (DMARDs). Control of rheumatoid arthritis by medical management has been revolutionized by the advent of DMARDs, resulting in a decrease in the surgical management of rheumatoid arthritis.

Indications for Surgery

Surgical treatment for rheumatoid arthritis is indicated in patients who continue to have functionally limiting symptoms despite optimal medical management.

Operative Management

Patients with refractory rheumatoid arthritis of the DRUJ may be candidates for the Darrach procedure or the Sauvé-Kapandji procedure. The Darrach procedure is a resection of the ulnar head, which alleviates pain from the DRUJ arthritis and distal ulna impaction against the carpus. The Sauvé-Kapandji procedure fuses the distal ulna to the distal radius while maintaining rotatory motion of the forearm by creating a surgical pseudarthrosis of the distal ulna, just proximal to the level of the radioulnar fusion.

Total wrist fusion is a reliable procedure for pain relief for patients with refractory rheumatoid arthritis of the radiocarpal joint, at the expense of joint motion. Plate-and-screw constructs are commonly used. Meticulous soft tissue handling is important, as wound complications are a potential concern in patients with rheumatoid arthritis. Total wrist arthroplasty offers pain relief with the preservation of limited wrist motion; however, wrist arthroplasty carries the unique risks of implant loosening, prosthetic wear, and progressive loss of bone stock. The role of total wrist arthroplasty in the treatment of rheumatoid arthritis is evolving, and certain patients, such as those with bilateral wrist disease requiring surgical treatment, may benefit.

Expected Outcome and Predictors of Outcome

While DMARDs have greatly improved the non-operative management of rheumatoid arthritis, patients who are refractory to pharmaceutical agents benefit from surgical treatment.

Comparisons of total wrist fusion with total wrist arthrodesis have shown comparable results. Total wrist fusion predictably relieves pain, stabilizes the wrist, and corrects deformity. Loss of motion at the wrist is generally tolerated, but can limit certain activities such as self-hygiene. In light of these functional considerations, when both wrists are involved with rheumatoid arthritis, some surgeons advocate total wrist arthroplasty on the dominant side and total wrist fusion on the non-dominant side.

Kienböck Disease

Kienböck disease is idiopathic necrosis of the lunate bone, characterized by fragmentation and progressive collapse of the lunate and subsequent degenerative changes of the wrist joints.

Epidemiology

Kienböck disease is most commonly seen in men between the ages of 20 and 40 years and is usually unilateral. The etiology is Kienböck disease remains poorly understood, but it is generally accepted that it is a multifactorial disease process. While some suggest that arterial insufficiency is to blame, others believe venous congestion plays a larger role. There is also evidence to suggest that certain anatomic variations of the wrist joint lead to increased force transmission across the radiolunate joint, which may lead to Kienböck disease.

Clinical Presentation

Patients typically present with non-activity-related dorsal wrist pain and limited wrist motion, without a clear history of trauma. Dorsal wrist swelling may be appreciated on exam. Patients typically do not seek medical attention in the early stages of disease; thus, the true prevalence and natural history of the disease remain unknown.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis for dorsal wrist pain includes scapholunate ligament injury, dorsal wrist ganglion cyst, and extensor tenosynovitis. The unique feature of Kienböck disease is that pain is typically persistent both at rest and with activity.

Early Kienböck disease may be accompanied by normal radiographs. Many patients with Kienböck disease exhibit a relatively shorter ulna compared to the distal radius, known as ulnar negative variance, but this finding is nonspecific. In more advanced cases, lunate sclerosis, fragmentation, and eventual collapse can be seen. Normal plain films in a young adult with persistent non-activity-related wrist pain warrant further evaluation with magnetic resonance imaging (MRI). MRI may reveal diffuse changes of the lunate, with low signal on T1-weighted images and increased signal on T2-weighted images.

Kienböck disease is staged using the Lichtman classification (Fig. 18.3). In Stage I disease, radiographs are relatively normal, and changes are only noted on MRI. In Stage II disease, sclerosis of the lunate can be seen, but without collapse. In Stage III, lunate collapse has occurred. Stage III disease is further divided into IIIA and

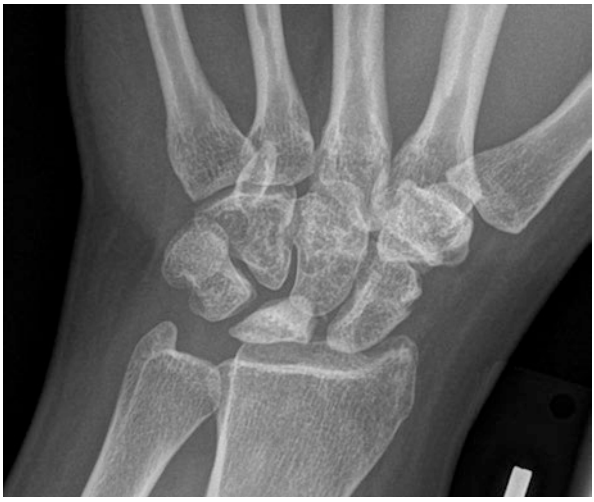

Fig. 18.3 Plain radiograph of Kienböck disease, noting sclerosis and collapse of lunate

IIIB, with IIIB disease associated with reduced carpal height due to proximal migration of the capitate and fixed flexion deformity of the scaphoid. In Stage IV disease, degenerative changes are seen in the radiocarpal and/or midcarpal joints.

Non-operative Management

Immobilization with a wrist brace or cast for 6–12 weeks is the initial treatment choice for the majority of patients and should begin at the time of initial diagnosis.

Indications for Surgery

Surgical treatment for Kienböck disease is indicated in patients who continue to have functionally limiting symptoms despite a period of immobilization.

Operative Management

A number of surgical procedure have been described for Kienböck disease. In early disease (Stage II or IIIA) and in patients with ulnar negative variance, a radial shortening osteotomy can be performed in order to level the joint and "offload" the lunate. Other procedures such as capitate shortening osteotomy and distal radius core decompression also aim to mechanically offload the lunate. A variety of vascularized bone grafting procedures have also been described in attempt to revascularize the lunate. Vascularized procedures are typically reserved for Stage II disease, when lunate avascularity is present but lunate collapse has not yet occurred. In late stages of disease (Stage IIIB and IV), namely, once carpal height has been lost and the capitate has migrated proximally, partial wrist fusions, proximal row carpectomy, and total wrist fusion are considered.

Expected Outcome and Predictors of Outcome

While some providers have reported success with non-operative management of Kienböck disease, many others have reported either no improvement in symptoms or progression of disease in most cases.

In early stages of disease, radial shortening procedures result in improved pain in over 90% of patients along with evidence of lunate revascularization in one third of patients. The majority of patients experience improved range of motion and grip strength following these joint-leveling procedures. Similar outcomes have been

described with re-vascularization procedures, and high-quality comparative evidence is still lacking. In late-stage disease, partial wrist fusion and proximal row carpectomy have been shown to have comparable results in terms of grip strength, pain relief, and wrist range of motion.

Thumb Carpometacarpal Arthritis

Epidemiology

Thumb carpometacarpal (CMC) joint osteoarthritis is the second most common arthritis of the hand. There is increasing prevalence of thumb CMC osteoarthritis with age over 40 years, especially in women. The overall prevalence of radiographic thumb CMC osteoarthritis in patients over 80 years of age has been reported to be over 90% in women and over 80% in men. Advanced destructive joint changes are more frequently seen in women compared to men. However, while radiographic arthrosis is common with advancing age, it correlates only moderately with clinical symptoms.

Clinical Presentation

Patients present with insidious onset of pain at the base of the thumb and report difficulty with grip and pinch activities that impart stress across the joint. Classically, patients report trouble opening jars and turning doorknobs. On inspection, patients often have a prominent thumb carpometacarpal joint, which is reflective of dorsoradial subluxation of the metacarpal on the trapezium. In order to maintain a wide grip, compensatory hyperextension through the thumb metacarpophalangeal (MCP) joint is often seen. Pain is elicited with a grind test or axial compression across the thumb CMC joint.

Differential Diagnosis and Suggested Diagnostic Testing

Pain at the base of the thumb or radial side of the wrist can be caused by de Quervain's tenosynovitis, scaphoid fracture or scaphoid nonunion, radioscaphoid arthritis, or scaphotrapeziotrapezoid (STT) arthritis. STT arthritis often accompanies thumb CMC osteoarthritis and involves degenerative changes between the scaphoid, trapezium, and trapezoid.

Thumb CMC osteoarthritis is evaluated using plain radiographs of the thumb, with the beam centered on the trapezium and the first metacarpal (Fig. 18.4). The

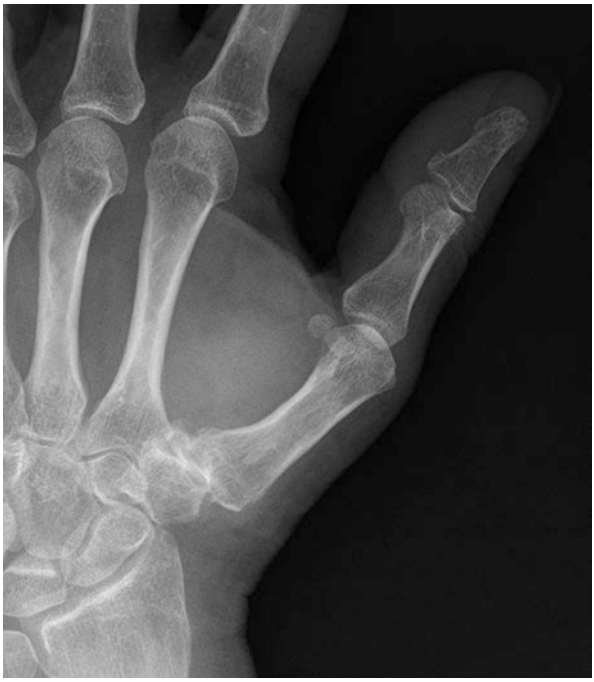

Fig. 18.4 Plain radiographs of thumb carpometacarpal osteoarthritis

radiographic stages of disease are graded based on the Eaton and Littler classification. In Stage I disease, radiographs remain unremarkable with preserved joint space. In Stage II and III disease, progressive joint space narrowing and osteophytes are seen at the thumb carpometacarpal joint, with Stage III having osteophytes greater than 2 mm in size. Stage IV disease is characterized by involvement of the adjacent STT joint, also known as pan-trapezial osteoarthritis.

Non-operative Management

The first-line treatment of thumb CMC osteoarthritis is non-operative management with anti-inflammatory medications, activity modification, and immobilization with a hand-based opponens splint, which encompasses the thumb metacarpophalangeal joint. The thumb interphalangeal joint can be left free if there is no osteoarthritis at this level, as this makes the splint better tolerated and allows the thumb to be more functional. These splints can be prefabricated or custom molded by an occupational therapist using thermoplastic material. Some patients prefer a soft neoprene sleeve, which offers less support but is less restrictive and may allow for greater ease of use with daily activities. Patients with persistent symptoms despite immobilization can consider corticosteroid injection. When corticosteroid injections are performed in patients with diabetes, patients must be made aware of the potential for temporary elevation of blood glucose levels, which may require supplementary treatment.

Indications for Surgery

Surgical treatment is elective and can be considered in patients who continue to be functionally limited despite non-operative management.

Operative Management

Multiple procedures for thumb CMC osteoarthritis have been described which include trapezium excision with or without tendon interposition and ligamentous reconstruction. All procedures have similar excellent results, and the surgeon's choice of procedure should depend on his or her comfort level. The use of non-biologic implants should be avoided, as they have been shown to be associated with higher rates of implant-related complications and reoperations. If the scaphotrapeziotrapezoid joint is also involved, which is quite common in advanced cases, removal of the proximal half of the trapezoid is additionally recommended. Thumb MCP joint hyperextension can be concurrently addressed with tightening of the volar capsule or MCP joint fusion.

Thumb metacarpal extension osteotomy is a joint-preserving option in early thumb CMC osteoarthritis and aims to shift the focus of articular cartilage loading and thereby decreased arthritic symptoms. In relatively younger patients in the early stages of disease, some have advocated for arthroscopic partial excision of the trapezium with or without soft tissue interposition. Thumb CMC fusion has been historically recommended in relatively younger manual workers with thumb CMC osteoarthritis; however, patients can be dissatisfied with the inability to lay their hand flat after this procedure.

Expected Outcome and Predictors of Outcome

Splint immobilization is an effective non-operative treatment modality, which has been show to dramatically improve symptoms within 6 months of use. Studies have reported that non-operative management can be successful in over 70% of patients, with better results seen in patients with earlier-stage disease. Corticosteroid injection accompanied by splint immobilization has also been shown to have encouraging results, particularly in patients with earlier stages of thumb CMC osteoarthritis.

Surgical treatment of thumb CMC osteoarthritis is often successful. The majority of patients report improvement in pain, pinch strength, grip strength, and function. Over 80% of patients report complete pain relief or only mild pain with certain activities at 1 year postoperatively. Pain relief is maintained in the long term in the

majority of these patients. Although a variety of surgical techniques have been described, as noted above, no technique has demonstrated superiority over another.

Metacarpophalangeal Joint Arthritis

Summary of Epidemiology

Primary metacarpophalangeal (MCP) joint osteoarthritis is rare, and thus secondary causes, including trauma and systemic diseases such as inflammatory arthritis, hemochromatosis, and calcium pyrophosphate deposition disease (CPPD), must be considered. Moreover, it is notable that men more frequently develop MCP joint osteoarthritis than women, with a reported prevalence of 12% compared to 7% in women.

Clinical Presentation

Patients may present with pain, swelling, and limited motion across the MCP joint.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnosis includes an underlying hemochromatosis, CPPD, and inflammatory arthritis. Arthritis of the index finger and middle finger MCP joints occurs in 42% of patients with hereditary hemochromatosis and may be the presenting symptom in 28% of patients. The appropriate diagnostic tests for hemochromatosis include serum ferritin and serum transferrin saturation levels.

Proliferative synovitis is most commonly seen at the MCP joint in rheumatoid arthritis, as well as characteristic deformities of the joint such as volar subluxation and ulnar deviation. If these findings are noted, a rheumatologic workup should be considered.

Non-operative Management

Activity modification and anti-inflammatory medications are the first-line treatment for MCP joint arthritis. Corticosteroid injections of the joint typically result in excellent relief and can be repeated for several years if necessary. Medical treatment of any underlying primary disease processes is of utmost importance.

Indications for Surgery

Surgery is indicated for debilitating pain and stiffness despite non-operative treatment and optimization of the medical management of any associated systemic disease.

Operative Management

The operative procedure of choice in cases of MCP osteoarthritis is joint replacement using either silicone or pyrocarbon implants. Fusion of the MCP joint is avoided due to unacceptable functional and cosmetic results. Since the arc of motion for pinch and grasp is initiated at the MCP joint, fusion of the MCP in any position can severely limit hand function.

Expected Outcomes and Predictors of Outcome

The majority of patients report pain relief, functional range of motion, and high satisfaction following joint replacement for MCP joint osteoarthritis. MCP joint range of motion is generally maintained postoperatively. MCP joint implants have demonstrated excellent durability, with over 80% survivorship after 10 years.

Proximal Interphalangeal and Distal Interphalangeal Joint Arthritis

Epidemiology

The distal interphalangeal (DIP) joint is the most common joint affected by primary osteoarthritis in the hand. The proximal interphalangeal (PIP) joint is the most common joint affected by post-traumatic osteoarthritis.

Clinical Presentation

DIP joint osteoarthritis is often asymptomatic. Symptomatic patients may report an aching pain across the DIP joint. Clinically, nodular deformities known as Heberden nodes can be observed at the DIP joint due to underlying osteophytes. Mucous cysts are also associated with DIP joint osteoarthritis, which can occasionally be painful or become secondarily infected. Mucous cysts may also lead to nail deformities due to pressure effects on the germinal matrix cells, which form the nail plate. At the PIP

joint, nodular deformities from underlying osteophytes are referred to as Bouchard nodes. The natural history of PIP joint osteoarthritis is progressive loss of motion and pain secondary to joint contracture and collateral ligament fibrosis.

Differential Diagnosis and Suggested Diagnostic Testing

The diagnosis of interphalangeal joint osteoarthritis can be made on plain radiographs of the hand (Fig. 18.5). The hallmark findings of osteoarthritis including joint space narrowing, osteophytes, subchondral sclerosis, and subchondral cysts can be seen.

The differential diagnosis of DIP joint and PIP joint arthritis is broad and includes inflammatory arthritis such as rheumatoid arthritis. It is important to remember that in rheumatoid arthritis, however, the DIP joints are often spared. PIP joints can be affected in rheumatoid arthritis; thus, it is important to examine whether there are accompanying deformities across the PIP joint that are characteristic of rheumatoid arthritis. For example, hyperextension of the PIP joint can occur due to attenuation of volar structures, while hyperflexion deformities of the PIP joint occur due to attenuation of the extensor mechanism. Seronegative spondyloarthropathies can also affect the interphalangeal joints. In psoriatic arthritis, more aggressive erosive changes are typically observed, namely, "pencil-in-cup" deformities at the DIP joint. Crystalline arthropathies, such as gout and CPPD, present with a more acute onset of symptoms with considerable pain, swelling, and erythema. In addition, gout can be accompanied by soft tissue tophi.

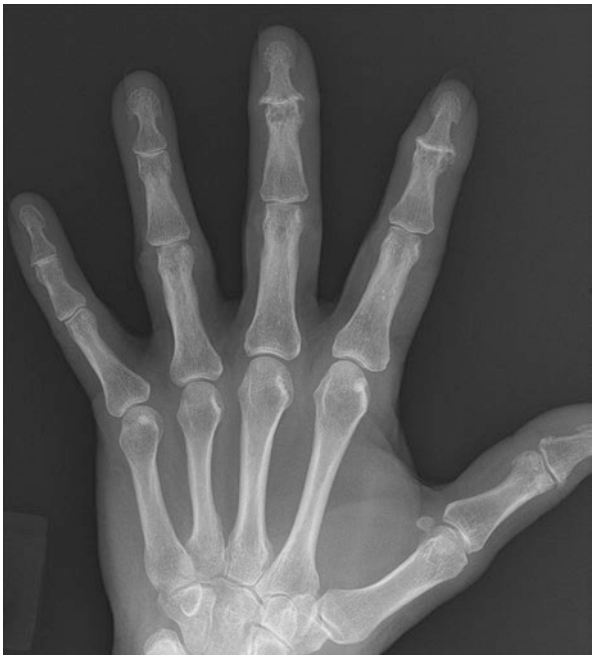

Fig. 18.5 Plain radiographs of distal interphalangeal joint osteoarthritis of the index finger and long finger. There is evidence of joint space narrowing, osteophytes, subchondral sclerosis, and subchondral cystic change

Non-operative Management

Anti-inflammatory medications and activity modification are the initial treatment for patients with DIP or PIP joint osteoarthritis. Intermittent corticosteroid injections of the interphalangeal joints often provide good-to-moderate relief.

Indications for Surgery

Surgery is indicated for patients with pain despite the above non-operative measures.

Operative Management

DIP joint osteoarthritis can be addressed with fusion across the DIP joint. Fusion of the DIP joint in slight flexion typically enables the patient to maintain excellent function. A variety of methods can be used to achieve successful fusion, including wire fixation or headless compression screws. In the setting of symptomatic mucous cysts, cyst excision with removal of underlying osteophytes is performed. Occasionally, local soft tissue must be rotated in order to achieve adequate soft tissue closure following cyst excision.

PIP joint osteoarthritis is treated either with joint fusion or joint replacement. When the index or small finger joint is involved, joint fusion will reliably result in a painless and stable joint. In these cases, a stable index finger still allows for a strong pinch. When the long or ring fingers are affected, joint replacement can be considered to preserve motion across the PIP joint. Joint denervation for PIP joint osteoarthritis focuses on division of the articular branches of the radial and ulnar digital nerves; PIP joint denervation is able to provide good pain relief at short-term follow-up.

Expected Outcomes and Predictors of Outcome

DIP joint fusion results in reliable patient satisfaction and pain relief. Successful DIP fusion can be achieved using wire fixation or headless compression screws; however, nonunion rates have been reported to be as high as 30%. Recent studies examining outcomes of DIP fusion for degenerative arthritis using headless compression screws have reported lower rates of nonunion and overall complications, but there is no clear evidence that one method of fixation is superior to another.

Although PIP joint replacements and joint fusions have similar pain relief initially, long-term results of joint replacements are quite variable, with a failure rate of over 30%. Subsequent revision surgeries are difficult due to bone loss and soft

tissue changes. Following PIP joint replacement, it is important to counsel patients that there is typically no significant improvement in range of motion. In general, postoperative PIP joint range of motion is determined by preoperative range of motion. On average, patients achieve roughly 45 degrees of motion across the PIP joint.

Table 18.1 shows a summary of the clinical presentation, recommended diagnostic testing, and management of osteoarthritic conditions of the hand and wrist.

Table 18.1 A summary of the clinical presentation, recommended diagnostic testing, and management of arthritic conditions of the hand and wrist

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
SLAC and SNAC wrist osteoarthritis	Wrist pain, weak grip strength, limited wrist motion	Plain radiographs	Anti-inflammatory medications, wrist brace, activity modification	Pain and functional limitations despite conservative management	Determined by stage of disease: radial styloidectomy, proximal row carpectomy, scaphoid excision and four-corner fusion, wrist denervation, or total wrist fusion
Wrist rheumatoid arthritis	Wrist pain, swelling and deformity, morning stiffness, and polyarthritis	Plain radiographs	Anti-inflammatory medications, corticosteroids, DMARDs	Pain and functional limitations despite optimal medical management	Various procedures, including Darrach procedure, Sauvé-Kapandji procedure, total wrist fusion, and total wrist arthroplasty
Kienböck disease	Wrist pain at rest, limited wrist motion	Plain radiographs; consider MRI if radiographs normal and high clinical suspicion	Cast immobilization	Pain and functional limitations despite immobilization	Determined by stage of disease; radial shortening osteotomy, vascularized bone grafting procedures, proximal row carpectomy, scaphoid excision and four-corner fusion, wrist denervation, or total wrist fusion

(continued)

Table 18.1 (continued)

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Thumb carpometacarpal osteoarthritis	Insidious onset of pain at base of thumb, difficulty with pinch and grip	Plain radiographs	Anti-inflammatory medications, hand-based opponens splint, corticosteroid injection	Pain and functional limitations despite conservative management	Various procedures, including trapezium excision and ligamentous reconstruction
MCP joint arthritis	Pain, swelling, and limited range of motion across MCP joint	Plain radiographs, workup for underlying systemic disease including hemochromatosis, CPPD, and rheumatoid arthritis	Anti-inflammatory medications, activity modification, corticosteroid injection, medical management of underlying systemic disease	Pain and functional limitations despite conservative management and optimal medical management	Joint replacement
Interphalangeal joint osteoarthritis (PIP and DIP joints)	Pain, swelling, and limited range of motion; Heberden nodes, Bouchard nodes, mucous cyst	Plain radiographs	Anti-inflammatory medications, activity modification, corticosteroid injection	Pain and functional limitations despite conservative management	DIP joint fusion, PIP joint fusion or joint replacement, PIP joint denervation

CPPD Calcium pyrophosphate deposition disease, *PIP* proximal interphalangeal joint, *DIP* distal interphalangeal joint, *MCP* metacarpophalangeal

Summary

Arthritic conditions of the hand and wrist are common. Various forms of arthritis in the hand can often be diagnosed with a thorough clinical history, physical exam, and plain radiographs. Surgery may be indicated for patients who have continued pain and functional limitations despite conservative management. While primary osteoarthritis in the wrist is uncommon, an understanding of the etiologies of secondary wrist arthritis as well as the functional demands of individual patients can help guide treatment.

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Chapter 19
Upper Extremity Nerve Entrapment

Philip E. Blazar and Ariana N. Mora

Abbreviations

CTS	Carpal tunnel syndrome
CuTS	Cubital tunnel syndrome
MRI	Magnetic resonance imaging
NSAIDs	Nonsteroidal anti-inflammatory drugs

Introduction

Neurologic complaints involving one or both upper extremities are relatively common. The most frequent diagnoses for these complaints are carpal tunnel syndrome (CTS) and cubital tunnel syndrome (CuTS). These conditions present with similar symptoms of numbness, paresthesia, and sometimes pain but are distinct in their presentation and management.

Carpal Tunnel Syndrome

Summary of Epidemiology

Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy. CTS is a mononeuropathy of the median nerve at the wrist, specifically in the carpal tunnel as the flexor tendons pass from the forearm to the hand. The prevalence of CTS has

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been studied in several populations and has been estimated to be approximately 4% in the United States. Classically, women account for approximately 75% of cases diagnosed, and the usual age of presentation, for both men and women, is around 50 years old.

A variety of risk factors have been identified, including female sex, pregnancy, or concomitant diagnosis of diabetes mellitus, obesity, and hypothyroidism. Specifically, the risk factors for CTS can be divided into systemic and anatomic factors. Systemic factors are more common and include diabetes mellitus and alcoholism resulting in neuropathy, fluid balance issues caused by pregnancy, myxedema, and renal failure/hemodialysis. Obesity and mucopolysaccharidosis or mucolipidosis are unclear if the increased ris systemic or anatomic. It is uncertain whether smoking affects the prevalence of CTS. Anatomic factors include paraplegia, position during sleep, deformity after fractures or other trauma, carpal bone anomalies, acromegaly, anomalous muscle bellies, hematoma resulting from anticoagulation therapy, lipoma and other neoplasms, hypertrophied synovium, infection, and increased adipose tissue volume in the carpal tunnel due to obesity.

Women who develop a first-time presentation of carpal tunnel syndrome during pregnancy are diagnosed with gestational CTS. Gestational CTS has not been well characterized in the literature. It is unclear as to what factors lead to the development of CTS during their pregnancy—though some risk factors are the same as classical CTS, including diabetes mellitus and obesity. Interestingly, the majority of women who develop CTS during their pregnancy will have symptoms abate within 1–2 weeks postpartum, yet others will develop persistent CTS.

Over the past few decades, there has been a trend for CTS to be diagnosed in younger patients, a phenomenon that some have speculated is due to an increase in occupational repetitive motion activities. The link between CTS, occupational tasks, and repetitive activity is not clearly supported by the scientific literature. Most of the epidemiologic studies that have examined the correlation between CTS and repetitive motion have not found an association. However, exposure to some occupational factors, specifically vibration, has been consistently linked to compressive neuropathy, especially CTS. Some investigators have reported that occupational CTS is epidemiologically distinct, presenting at a younger age and at a nearly 1:1 sex ratio.

Clinical Presentation

The classic presentation of CTS is a gradual onset of numbness, paresthesia, and sometimes pain in the radial three and one half digits (Fig. 19.1).

The carpal tunnel is composed of a semicircular ring of carpal bones and an overlying, unyielding fibrous band, the transverse carpal ligament. Chronic compression and increases in pressure have been shown to reduce epineural blood flow and diminish axonal transport in peripheral nerves. For both unaffected patients and those with CTS, positioning the wrist in extreme flexion or extension further

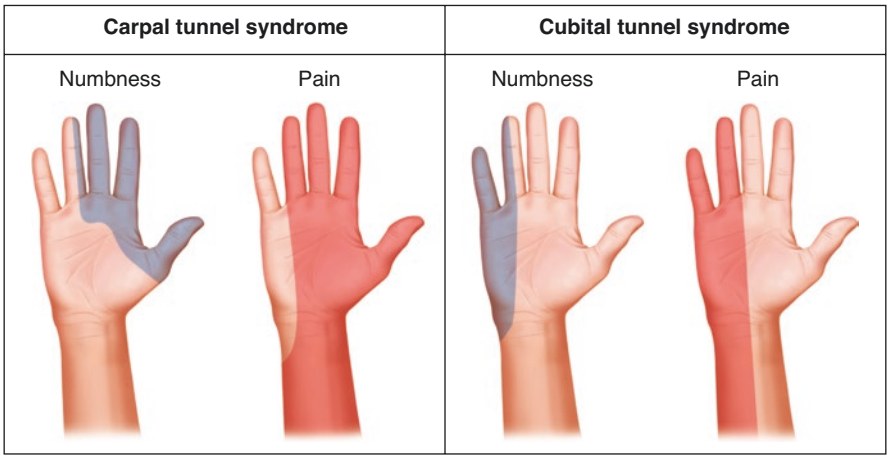

Fig. 19.1 Common symptom presentation of CTS and CuTS. *CTS* Carpal tunnel syndrome, *CuTS* cubital tunnel syndrome

increases the pressure on the median nerve. The median nerve contributes motor fibers to the thenar muscles and sensory fibers to the thumb and the index and middle fingers, as well as to half of the ring finger.

Patients typically have subacute or chronic symptoms involving the median nerve distribution. However, a substantial number of patients will report symptoms involving the entire hand. Development or worsening of symptoms at certain times or with particular activities is very characteristic and aids in diagnosis. While taking a patient's history, it is important to ask if the patient experiences numbness and pain in their hand while sleeping, driving, talking on the telephone, or reading. If so, carpal tunnel syndrome is very likely the cause.

Infrequently, patients will present with acute nerve compression secondary to swelling from trauma, spontaneous bleeding while on anticoagulation, or a rapidly progressing infection. In these scenarios, the acute process has likely caused complete intraneural ischemia and must be treated as a surgical emergency.

While most cases are idiopathic, it is important to check thoroughly for coexisting systemic disorders and local predisposing factors, which may have a substantial impact on the selection of appropriate treatment. Patients with bilateral symptoms are more likely to have metabolic or systemic risk factors. CTS may be the presenting complaint for a process with wide-reaching health implications; therefore, signs of secondary causes should always be sought, particularly in patients with bilateral symptoms.

The microvascular and anatomic changes in CTS create a spectrum of dysfunction, but patients generally can be grouped into one of three clinical stages:

(a) *Mild-stage CTS*: Characterized by intermittent paresthesia and frequent resolution of symptoms when predisposing activities are modified or ceased. In this

stage, patients may respond well to nonsurgical treatment, but a moderate percentage still progress further.

(b) *Moderate-stage CTS*: Complaint of constant or near constant paresthesia/numbness. Pain and the severity of the paresthesia may be episodic. Conservative treatment is unlikely to be of long-term benefit to this population. This group shows the largest improvement in symptoms post-surgery.

(c) *Severe-stage CTS*: Characterized by distinct sensory loss and thenar muscle atrophy. Chronic elevated pressure in the tunnel and reduced epineural blood flow are likely to lead to epineural fibrosis if untreated. A degree of persistent neurologic dysfunction despite treatment is likely after surgery, although the majority of patients will report significant symptom improvement, particularly in regard to pain relief.

The most sensitive and specific physical examination maneuver commonly employed is the carpal tunnel compression test, in which direct pressure is applied over the median nerve (Fig. 19.2). Other physical examination maneuvers that aid in the diagnosis of CTS are listed in the table below (Table 19.1). Other components of the peripheral nervous system in other limbs should be examined to exclude systemic neuropathic predisposition. Examination of the median nerve more proximally, including at the cervical spine, should always be included because patients with proximal nerve entrapment are sometimes misdiagnosed and treated for CTS.

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnoses for CTS include various conditions that present with paresthesia, pain, or weakness involving the upper extremity:

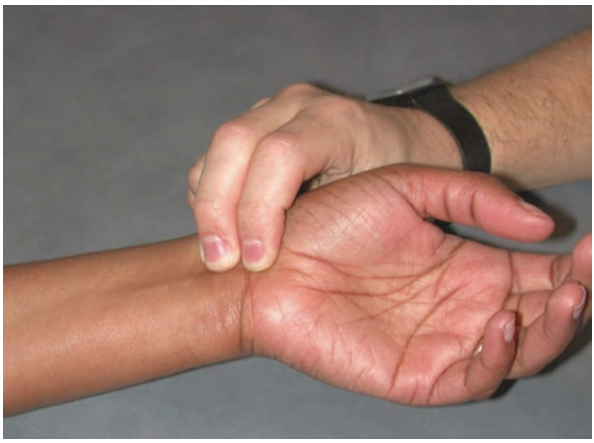

Fig. 19.2 CTS compression test. Apply direct compression of the median nerve for 30 s to elicit paresthesia. *CTS* Carpal tunnel syndrome

Table 19.1 Physical exam maneuvers that aid in the diagnosis of CTS

Test	Technique	Condition response	Positive result
Phalen's sign	Patient places arms on table, elbows extended, wrists in full flexion	Paresthesia in response to position	Numbness or tingling on radial sided digits within 60 s; probable CTS
Tinel's sign	Examiner lightly taps along median nerve at the wrist, proximal to distal	Site of nerve lesion	Tingling response in fingers at site of compression; probable CTS if response at the wrist
CTS compression test	Direct compression of median nerve by examiner	Paresthesia in response to pressure	Paresthesia within 30 s; probable CTS
Hand diagram	Patient marks sites of pain or altered sensation on an outline diagram of the hand	Patient's perception of site of nerve deficit	Diagram marked on palmar aspect; probable CTS

CTS Carpal tunnel syndrome

(a) *Cervical radiculopathy* typically although not always presents with neck pain exacerbated with neck movement, reflex changes, and weakness of proximal arm muscles including elbow extension/flexion and arm pronation in addition to CTS symptoms.
(b) *Cervical spondylotic myelopathy* usually progresses to bilateral motor/sensory dysfunction in the hands not confined to the median nerve, unlike CTS.
(c) *Brachial plexopathy* is typically unilateral and accompanied with motor/sensory dysfunction in areas beyond the median nerve distribution.
(d) *Median neuropathy in the proximal forearm* is much less common than CTS. Symptoms overlap with CTS but include thumb flexion weakness and sensory loss over the thenar eminence, as these structures receive innervation from the median nerve proximal to the wrist.
(e) *Motor neuron disease* (e.g., *amyotrophic lateral sclerosis*) presents without pain, which is a hallmark of CTS.
(f) *Fibromyalgia* is characterized by chronic widespread pain and fatigue not isolated to the region affected by CTS.

Electrodiagnostic testing is often considered the diagnostic test of choice for CTS, but it remains operator dependent. As with any test, false-positive and false-negative rates are dependent on the threshold levels used by the particular practitioner. Systemic conditions (e.g., diabetes mellitus or aging) and laboratory conditions (e.g., limb temperature) can influence test results. The data are typically compared to population norms, but often information from the contralateral extremity or the ulnar nerve in the same wrist is more useful in diagnosis. The staging system for electrodiagnostic testing is similar to the clinical staging system, but it does not necessarily correlate. Electrodiagnostic tests will detect cervical spine or more proximal upper extremity nerve compression only if the test examines the extremity proximal to the wrist.

Nerve conduction velocities and electromyography provide the only objective evidence of nerve dysfunction in CTS. Therefore, electrodiagnostic testing is especially useful for documentation in patients where there is an expected need for objective tools to monitor improvement, such as in the case of active workers' compensation claim. Because patients frequently find the electrodiagnostic tests painful, recent literature suggests that electrodiagnostic tests may be substituted with ultrasound imaging in certain situations. Radiographs and magnetic resonance imaging (MRI) are rarely indicated, except in cases of limited wrist motion, trauma, or arthritis (radiographs) or for suspected soft tissue masses (MRI).

Nonoperative Management

The severity of the clinical stage of CTS usually dictates treatment choices. Any underlying systemic processes should be investigated and treated as appropriate. Mild-stage CTS at first presentation is usually treated with splinting and/or activity modification. Nonsteroidal anti-inflammatory drugs (NSAIDs) have not been shown to be effective for CTS but are frequently effective for other hand conditions that also affect this population. However, if conservative management is unsuccessful, further activity restriction, corticosteroids, or surgery may be necessary.

(a) *Splinting*: The majority of patients are initially treated with wrist splinting in a straight position or in slight extension. Splinting only at night is typically sufficient for the vast majority of patients who have exclusively or predominantly nocturnal symptoms. Occasional patients may benefit from the additional use of splints during daytime activities that produce symptoms. In general, full-time splinting should be avoided to reduce the risk of atrophy and loss of motion. Disuse and atrophy are especially important to keep in mind for workers' compensation cases as this may delay or complicate the return to work.
(b) *Activity modification*: For patients with mild-stage CTS who wish to avoid splinting or who are not appropriate candidates for it, some benefits can be achieved through activity modification. The patient may wish to consider adjusting his or her work schedule, taking frequent breaks from repetitive activities, or making ergonomic changes to the workstation. Activity modification is frequently unsuccessful. Patients' options at this time may be limited to long-term work restrictions, surgical intervention, or continuing to manage the symptoms as above, as long as there are no signs of progressive neurologic dysfunction.
(c) *Diuretics and anti-inflammatories*: Patients with substantial peripheral edema may experience symptomatic improvement with diuretics. Frequently, NSAIDs are paired with diuretics. The use of NSAIDs has not been shown to be effective for idiopathic CTS in randomized trials; however, use of these agents for short periods is generally well tolerated if appropriately monitored. They are most beneficial for reducing inflammation in patients with multiple complaints or

comorbidities. Anti-inflammatory medications are clearly indicated for those patients who have tenosynovitis from an inflammatory process.

(d) *Corticosteroids*: Injection with corticosteroids can be an effective treatment when the previous management strategies have not been successful. The response rate is best for patients who have experienced mild symptoms for less than 12 months. Injection is likely to reduce or eliminate symptoms in the majority of these patients; however, only a minority of these will have continued relief 1 year post injection. Complications with carpal tunnel injection are uncommon, but injection into the nerve must be avoided. The goal is to introduce the steroid into the tenosynovium within the carpal tunnel but not directly into or immediately adjacent to the nerve. Any paresthesia in the median (or ulnar) distribution elicited during the procedure should prompt the physician to withdraw and redirect the needle. Complications from multiple injections at this site have not been reported; however, skin atrophy and tendon rupture from multiple corticosteroid injections at other sites have been documented. Therefore, multiple injections (more than two or three) are discouraged, except in the rare patient for whom surgery is medically contraindicated.

Indications for Surgery

Patients who present with moderate- or severe-stage CTS are typically managed surgically. Additionally, patients with mild-stage CTS who have failed nonsurgical treatment may be considered surgical candidates. Moderate-stage CTS patients typically have a limited or transient response to conservative treatment options. In these cases, surgical intervention is recommended to reduce the likelihood of permanent neurologic changes, and most patients report pain relief postoperatively. It should be noted, however, that even after surgery, many have some permanent neurologic dysfunction that can be seen through electrodiagnostic testing or subtle findings apparent during physical examination.

Patients presenting with severe-stage CTS are best managed by surgical decompression of the carpal tunnel. In patients with CTS from coexisting systemic morbidities, it is less common to see prompt diminution or resolution of symptoms with corrective measures (e.g., thyroid replacement therapy, improved control of blood glucose level). Thus, referral for consideration of surgery is appropriate even while actively addressing these comorbidities.

Clear indications for referral to a surgeon include:

(a) Patients who present with moderate- or severe-stage CTS, i.e., all patients with constant paresthesia. Those with limiting comorbidities also fall into this category. Carpal tunnel release can be performed expeditiously under local anesthesia with little physiologic stress; therefore, medical comorbidities are only considered to be an absolute contraindication for surgery in very extreme or unusual situations.

(b) Patients who have acute CTS as a result of trauma, suspected infection, or bleeding. Although rare, such patients should be referred emergently for surgical consultation and, if conclusively diagnosed, should be treated with emergent surgical decompression to avoid further neurologic injury.
(c) Patients with progressive neurologic dysfunction during nonsurgical treatment, such as progressive hand weakness noted in median nerve innervated muscles.
(d) A patient who fails nonsurgical treatment in the early stage of the disease is also an appropriate indication for referral. Patients must be counseled that decompression is an appropriate, but elective, procedure in these situations.

Operative Management

It remains controversial whether a particular surgical technique is superior to others for treatment of CTS. Surgical division of the transverse carpal ligament has been shown to rapidly return pressure in the carpal tunnel in most patients with CTS to the same level as that in controls; however, standard surgical techniques have produced moderate rates of tenderness in the area of the palmar incision (Fig. 19.3). These symptoms almost always resolve, but improvement may take several months. Although temporary, this tenderness may slow rehabilitation and resumption of occupational and recreational activities.

Endoscopic techniques were developed to reduce this complication and to hasten rehabilitation. These techniques have been shown to decrease but not eliminate incisional pain. Although patients who undergo endoscopic surgery return to work slightly sooner, they may have very low but slightly elevated rates of neurovascular injury compared with patients treated with standard techniques.

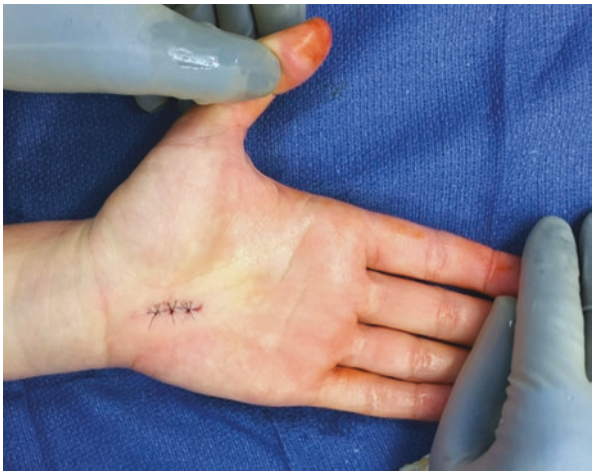

Fig. 19.3 Postoperative incision for open carpal tunnel release

Expected Outcome and Predictors of Outcome

Studies of surgical treatment for CTS have typically focused on recovery of strength, resolution of paresthesia, and time to return to work. The persistence of symptoms after surgical release varies widely between studies and is highly dependent on patient selection; in well-selected case series with long-term follow-up, about 75% of patients report complete resolution of symptoms. Patients with intrinsic neurologic dysfunction and symptoms of more chronic and severe compression have generally experienced worse outcomes. Although many studies have documented that some patients will have persistent symptoms, reported satisfaction among patients is very high (over 90%) after surgical release. Little has been written on the outcomes of CTS in populations treated nonoperatively for extended time periods.

Additionally, studies suggest that patients with earlier surgical release have better outcomes. A study found that patients who underwent surgery within 3 years of diagnosis were twice as likely to have symptom resolution compared to those who underwent surgery more than 3 years after diagnosis. Other studies have suggested that after surgical release, patients with intermittent paresthesia (mild-stage CTS) had better return of sensation than did those with constant paresthesia (moderate- or severe-stage CTS).

Cubital Tunnel Syndrome

Summary of Epidemiology

Cubital tunnel syndrome (CuTS) is the second most common upper extremity entrapment neuropathy after carpal tunnel syndrome (CTS). The prevalence of CuTS has not been studied as extensively as CTS; however, CuTS is estimated to have an annual incidence rate of 25 per 100,000, affecting men more than women, and has been associated with smoking and increased age. Besides the difference in gender prevalence in CTS, CuTS also differs from CTS in that BMI, diabetes, and hypertension are not predisposing factors for this syndrome.

Extrinsic compression of the ulnar nerve can be from acute trauma or prolonged pressure on the nerve caused by elbow flexion or leaning on the elbow, given that the ulnar nerve is situated tightly around the medial epicondyle in these positions. Trauma can lead to focal compression from osteophytes or scar tissue encroaching upon the nerve. Instability of the ulnar nerve can lead to repetitive subluxation or dislocation of the nerve during flexion, which in turn can lead to motor and sensory loss. Intrinsic factors around the elbow that can contribute to symptoms include arthritis, synovitis, mass lesions, and bone, muscle, or fibrous tissue anomalies.

Clinical Presentation

Patients with CuTS present with numbness and paresthesia in the volar aspect of the fourth and fifth digits of the hand and medial elbow pain, often with nocturnal symptoms. These symptoms worsen during sustained elbow flexion, when leaning on the elbow, or while performing activities that require repetitive lifting. While sensory complaints are more common, motor symptoms do occur, ranging from mild weakness of the intrinsic muscles of the hand to severe wasting of ulnar-innervated hand and forearm muscles, which is largely dependent on the duration of symptoms. Some patients will complain of difficulty performing tasks requiring fine dexterity and/or pinch such as using a key to open a door. Unlike CTS, this loss of dexterity in patients with CuTS is largely due to intrinsic hand muscle weakness and not sensory loss. This loss of intrinsic muscle strength can greatly affect grip strength.

The severity categories for CuTS are less defined than they are for CTS. Mild-to-moderate CuTS is characterized by intermittent or persistent sensory loss and weakness without wasting and without a causative structural lesion. When visible muscle atrophy accompanies symptoms of pain and paresthesia, CuTS should be classified as severe.

The ulnar nerve is positioned in the groove between the medial epicondyle and the olecranon process of the posterior elbow. It can be readily palpated to check for lesions, swelling, or nerve subluxation over the medial epicondyle. The examiner should inspect the elbow and forearm for deformity or atrophy. Visible muscle wasting, particularly of the intrinsic hand muscles, indicates a significant duration of ulnar nerve compression ranging from months to years. Examination maneuvers that assist in the diagnosis of CuTS are listed in the table below (Table 19.2).

Differential Diagnosis and Suggested Diagnostic Testing

The differential diagnoses of CuTS include various conditions that present with paresthesia, pain, or weakness involving the upper extremity:

(a) *Carpal tunnel syndrome* presents with numbness in the thumb, index, and middle fingers (palmar aspect) in addition to possible thenar muscle wasting.
(b) *Cervical radiculopathy* presents with neck pain exacerbated with neck movement, reflex changes, and weakness of proximal arm muscles including elbow extension/flexion and arm pronation in addition to CuTS symptoms.
(c) *Medial epicondylitis* is characterized by tenderness over the medial epicondyle; it is distinct from CuTS as it will not result in distal weakness, paresthesia, or numbness.
(d) *Thoracic outlet syndrome* includes neck and shoulder pain in addition to distal pain and numbness; patients will have normal nerve conduction studies at the elbow, and it rarely includes wasting in the hand.

Table 19.2 Physical exam maneuvers that aid in the diagnosis of CuTS

Test	Technique	Condition response	Positive result
Tinel's sign	Percussion over ulnar nerve distally from ulnar groove to cubital tunnel	Site of nerve lesion	Tingling response in ulnar distribution of hand; likely CuTS
Elbow flexion test	Place index finger over ulnar groove throughout maximum elbow flexion	Ulnar nerve subluxation and presence of paresthesia	Ulnar nerve slips out of groove and patient reports paresthesia in ulnar distribution after 60 s of flexion; likely CuTS
Froment's sign	Patient actively adducts thumb to index finger	Thumb adductor muscle weakness (innervated by ulnar nerve)	Interphalangeal thumb joint flexes; in addition to positive sensory tests, could indicate more advanced CuTS
Abduction/adduction strength test	Cross index/middle fingers; spread fingers (abduction), fingers together (adduction)	Finger abductor/adductor muscle weakness (innervated by ulnar nerve)	Inability to cross fingers, weakness to resist antagonistic movement; in addition to positive sensory tests, could indicate more advanced CuTS

CuTS Cubital tunnel syndrome

(e) *Ulnar nerve entrapment at the wrist* will result in maintenance of strong wrist flexors and ulnar deviators, and sensation will remain intact over the dorsomedial hand and the dorsum of the little and ring fingers as these structures receive innervation from the ulnar nerve proximal to the wrist.

The clinical diagnosis of CuTS can be confirmed with electrodiagnostic testing. Electrodiagnostic studies can localize the lesion on the ulnar nerve, determine the severity, and provide objective evidence of the presence of CuTS. It should be noted that there are a high percentage of cases with mild CuTS, particularly among musicians, that present with clinically significant findings of CuTS however with negative electrodiagnostic tests. Many of these individuals have progressive symptoms and frequently elect to have surgery. Unlike electrodiagnostic tests for CTS, CuTS electrodiagnostic findings do not typically distinguish between mild and moderate categories; however, severe findings are usually noted as such. If elbow trauma has occurred, radiographs of the elbow should be ordered in addition to electrodiagnostic testing.

Nonoperative Management

The severity of paresthesia and pain due to CuTS usually dictates treatment choices. Mild-to-moderate CuTS at first presentation is usually treated with splinting and/or activity modification. Corticosteroid injections are not recommended. Nonoperative management is also recommended for those who are ineligible for surgical treatment.

(a) *Splinting:* The majority of patients are initially treated with nocturnal elbow splinting limiting flexion. Alternatively, and perhaps with better rates of compliance, patients can wrap the effected elbow with a pillow or towel instead of a traditional hard splint as this may be better tolerated during sleep.
(b) *Activity modification*: Patients should be instructed to avoid excessive or repetitive elbow flexion, putting pressure directly on the medial elbow during rest or activities, e.g., crossing arms or supporting significant weight on arm rests. Workplace modifications may also be necessary to limit repetitive lifting, elbow flexion, and direct pressure on the ulnar nerve.

Indications for Surgery

Those presenting with muscle atrophy and weakness in conjunction with sensory symptoms indicative of CuTS should be treated more aggressively and are recommended for surgery. Those who have had severe CuTS symptoms existing beyond 2 years will have variable improvement in sensory-related symptoms postoperatively, but surgery is beneficial to alleviate pain associated with CuTS. Surgery is also indicated in individuals with mild or moderate CuTS whose symptoms have progressed or those who have been unsuccessfully treated after 2–3 months of conservative management.

Operative Management

Typically CuTS is surgically treated with ulnar nerve decompression, which consists of cutting the flexor carpi ulnaris aponeurosis to decompress the ulnar nerve. However, in cases where the ulnar nerve subluxates upon flexion, ulnar nerve transposition is necessary. Ulnar nerve transposition is a more involved surgery that mobilizes and frees the ulnar nerve from the ulnar groove and repositions it anteriorly in the forearm. Ulnar nerve transposition requires a larger incision and can have higher rates of complications when compared to simple ulnar nerve decompression.

Expected Outcome and Predictors of Outcome

Patients with mild symptoms of intermittent ulnar sensory loss often improve with conservative treatment alone within 3–6 months. Many of these patients will never present to clinic. Those with more persistent sensory issues, ulnar nerve subluxation, or atrophy have less predictable outcomes. Duration of symptoms plays a significant role in both conservative and surgical treatment outcomes. The rate of

Table 19.3 Summary of carpal tunnel syndrome and cubital tunnel syndrome

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Carpal tunnel syndrome	Paresthesia in thumb, index, long, and half of ring finger; nocturnal hand pain	Electrodiagnostic testing for atypical clinical presentations and when surgery is indicated	Splinting, activity modification, diuretics (those with substantial peripheral edema), corticosteroid injections	Failed conservative treatment, muscle atrophy or weakness, constant paresthesia, CTS as a result of trauma	Division of the transverse carpal ligament (carpal tunnel release)
Cubital tunnel syndrome	Paresthesia in the ring finger or little finger, medial elbow pain, can have intrinsic muscle weakness with grasping	Electrodiagnostic testing for atypical clinical presentations and when surgery is indicated	Splinting and activity modification	Failed conservative treatment, muscle atrophy or weakness, constant paresthesia	Cubital tunnel release or ulnar nerve transposition

surgical complications is very low, and these are usually minor issues such as erythema. If decompression surgery is performed, at times, a revision surgery is still necessary to transpose the ulnar nerve. Overall, patient satisfaction achieved through surgical decompression is very high due to the improvements in sensory loss and pain, coupled with the short recovery time.

The syndromes discussed in this chapter are the two most common upper extremity entrapment neuropathies; thus, the primary care physician plays an important role in the initial care of these patients. A summary of carpal tunnel syndrome and cubital tunnel syndrome, including their presentation, diagnostic testing, conservative management, indications for referral and/or surgery, and operative management, is provided below (Table 19.3).

Suggested Reading

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Part VI
Sports and Activity Related Injuries and Orthobiologics

Chapter 20
Bone Stress Injuries

Erin E. Finn and Adam S. Tenforde

Epidemiology

Bone stress injury (BSI) is a common form of overuse injury in athletes and active individuals who present to sports medicine clinics. The injury is the result of cumulative microtrauma to bone that exceeds remodeling, resulting in development of bone injury. A BSI is on a continuum of injury, with imaging showing bone marrow edema in early injury stages and possibly a fracture line in advanced injury stages. Early injuries are commonly referred to as stress reactions, while advanced injuries with a fracture line are commonly referred to as stress fractures. Land-based sports, especially those with cyclical, unidirectional loading such as running, can cause repetitive overload to the skeleton, placing athletes at elevated risk for lower extremity BSI. Biological factors including states of low energy availability with resulting systemic sequelae described by the Female Athlete Triad (Triad) [1] and Relative Energy Deficiency in Sport (RED-S) [2], biomechanical influences, and bone anatomy may each contribute to increased risk for developing a BSI.

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Clinical Presentation

A. Chief Complaint and History

An athlete with a BSI will most commonly present with a complaint of focal pain directly over bone or with weight bearing. In early injury stages, an athlete with a BSI will typically provide a history of pain precipitated by sport activity. As the injury advances, the BSI may cause pain outside of sport and during weight bearing, sometimes even progressing to pain at rest and at nighttime. A detailed history may reveal causes of injury. These include changes in frequency, volume, or intensity of training or preparing for a sports competition/event. Additionally, all female athletes should be screened for the Female Athlete Triad (Triad) and have their associated risk assessment score calculated. Another helpful concept for BSI risk is Relative Energy Deficiency in Sport (RED-S), reflecting consequences of low energy availability in athletes of both sexes and athletes with disability. Other relevant history includes medical conditions that predispose to BSI or impaired bone health (including thyroid disease, rheumatological conditions, and osteopenia/osteoporosis), family history of fracture or osteopenia/osteoporosis, and review for culprit medications (including steroids, proton pump inhibitors, and anti-epileptic drugs). Assessing dietary patterns including restrictive eating, disordered eating behaviors, symptoms of malabsorption, food allergies, and amount of calcium and vitamin D intake from food and supplements should also be included as part of the initial history, as impaired nutrition is often a key component of BSI development (often through the Triad or RED-S).

B. Sex-Specific Considerations for the History

Female athletes should be screened for the Triad. The Triad is defined as the interrelationship of energy availability, menstrual function, and bone mineral density in female athletes. Each component of the Triad may fall on a spectrum of health to disease. An increased number of Triad risk factors characterized as diseased are associated with elevated risk for BSI [1]. The Triad is part of the expanded concept of RED-S [2].

1. *Energy Availability*

Energy availability is the difference between energy intake and estimated energy expenditure standardized to metabolically active tissue of fat-free mass per day. When body weight is constant, reduced energy availability is caused by decreased caloric intake, increased exercise energy expenditure, or a combination of both. If energy availability is $\leq$30 kcal per kg of fat-free mass per day, it is considered low [1, 2]. Low energy availability can alter both metabolic and reproductive hormones [3], and resulting reductions in sex hormones such as estradiol may manifest as changes in menstrual periods (increased interval of time between menstrual periods, lighter periods, or cessation of menses) and are therefore an important early marker for inadequate energy availability. Low energy availability may occur inadvertently due to mismatch of energy intake to energy expenditure or as a result of disordered

eating. It should be noted that active female athletes may have low energy availability without disordered eating or an eating disorder.

2. *Menstrual Function*

   The first step in determining menstrual function is obtaining a menstrual history. Key components include age of first menstrual period (menarche) and number of periods over the past 12 months. In the absence of pregnancy, females should have ten or more periods per year. Although patients using hormonal therapy such as oral contraceptive pills may experience withdrawal bleeding, it is important to recognize that withdrawal bleeding is not the same as having normal menstrual periods and should not necessarily be equated to sufficient menstrual health. To assess for menstrual regularity in patients on hormonal therapy, patients should be asked why the medication was prescribed. Normal menstrual function is a good initial measure of adequate hormonal function and therefore sufficient energy intake, whereas abnormal menstrual function might indicate insufficient energy availability. In addition to the insight provided by the menstrual history into the average energy availability of the patient (and the resulting increased risk for poor recovery and injury risk), it is also important to assess the menstrual history because the cyclic patterns of estradiol and progesterone associated with menstrual periods influence bone turnover and bone mass accrual.

3. *Bone Mineral Density*

   Both energy availability and menstrual function influence skeletal health, including bone mineral density. In the setting of adequate energy availability, menstrual function is preserved, hormones including estradiol promote bone health, and risk for BSI is decreased. In the setting of inadequate energy availability, reduced estradiol and nutritional deficiencies compound to result in reduced bone mass and an increased risk for BSI [1, 2].

4. *Screening for the Triad*

   The Female Athlete Triad Coalition has developed a series of screening questions that should be asked at the time of annual pre-participation evaluation (PPE) in athletes [1]. However, these questions can also be incorporated into clinical evaluation for a female athlete with a suspected BSI. The questions address the three main components of the Triad. Questions covering menstrual health include age of menarche and number of periods over the past 12 months. Late menarche is defined as first menstrual period at age 15 or older. Oligomenorrhea is six to nine periods over the past 12 months, and amenorrhea is fewer than six periods in 12 months or the absence of menses for 3 months. If the cause of this amenorrhea is chronic anovulation due to improper hypothalamic signaling, then it is referred to as functional hypothalamic amenorrhea. To assess energy availability, diagnosis of an eating disorder or disordered eating is important to elicit. Additionally, other markers of inadequate energy availability may include reduced body mass index (defined as less than 18.5 kg/m^2 in female athletes), concerns about weight, or abnormal dietary patterns or attitudes. The third component of the Triad, bone health, is more challenging to assess without direct measure of bone density

using dual-energy x-ray absorptiometry (DXA); screening questions include history of prior stress fractures or stress reactions or diagnosis of low bone mineral density.

5. *RED-S*

Female and male athletes with low energy availability may experience effects on health and performance as described by the concept of RED-S. Health features impaired in the setting of RED-S include metabolic rate, bone health, immunity, protein synthesis, and cardiovascular health [2]. These markers should be evaluated, both because they signal a potential associated risk of BSI and because they have their own serious health consequences. As mentioned previously, RED-S is an umbrella under which the Triad exists. The Triad screening tool, therefore, has been modified to screen for BSI risk in men by removing the sex-related components (age at menarche and oligomenorrhea/amenorrhea). Recent studies have shown that increases in this male-adapted cumulative risk score are associated with increased risk of prospective BSI [4]. Including this screening tool in the PPE has the potential to identify male athletes at increased risk for BSI who should be targeted for therapy or early intervention in the case of a suspected BSI.

C. Physical Examination

On physical examination, body mass index should be measured. The initial assessment includes the following key features:

1. *Pain with weight bearing* or single leg hop
2. *Tenderness with direct palpation* over the bone
3. *Pain with direct or indirect percussion* over the site of injury

Clinical judgment should guide the examination. For example, a patient with a suspected advanced BSI should not perform a single leg hop if there is concern for a fracture that could worsen or progress to a more severe injury.

Special tests may be added to assess by anatomy:

4. *Hip Internal Range of Motion*

A femoral neck BSI may have pain with movement through range of motion of the affected hip, particularly at the end range of internal rotation. Lesser trochanteric BSI may also have pain elicited with resisted hip flexion due to associated hip flexor tendinopathy that may be associated with lesser trochanteric BSI [5].

5. *Fulcrum Test*

A femoral shaft BSI can be difficult to evaluate on physical examination because tissue limits ability to directly palpate or percuss the femur. The fulcrum test is useful because it can elicit pain without palpation. This test is performed with the patient seated. The examiner places one forearm beneath the thigh. With the other hand, the examiner presses in an anterior to posterior force through the thigh. The examiner uses the forearm as a fulcrum that can be moved along the length of the femur to stress the bone. Pain localizing to the site of the fulcrum may represent an underlying BSI at that location. Alternatively, this test may be performed with the patient sitting on the edge of the table with their feet hanging above the ground and the edge of the table as the fulcrum.

6. *Sacroiliac Joint Maneuvers*
   A BSI to the sacrum or pelvis may be evaluated using sacroiliac joint provocative maneuvers (such as thigh thrust, flexion abduction with exertional rotation, FABER), although these examination findings are typically nonspecific for pelvic BSIs.
7. *Lumbar Extension*
   Pars interarticularis fractures are overuse injuries localized primarily to the lumbar spine. They may occur at the most distal vertebral segments, including the fifth lumbar vertebrae, due to repetitive extension-based activities of sport. The injury is typically evoked with extension-based stress to the spine, including instructions for the patient to stand on a single leg and go into lumbar extension (commonly known as the stork test).
8. *Calcaneal Squeeze Test*
   The calcaneal squeeze test can be helpful in identifying a fracture in the calcaneus. The examiner places both hands on the calcaneus and applies a compressive force to the calcaneus to attempt to reproduce bone pain.

Differential Diagnosis

The differential diagnosis for a BSI is based on injury location. Magnetic resonance imaging (MRI) is the most sensitive test to evaluate for a BSI, may exclude other regional soft tissue etiologies, and uses nonionizing radiation to acquire diagnostic imaging. In addition, MRI can be used to grade the severity of a BSI and guide return to play for sports [6]. Unfortunately, the costs of MRI may limit its use. Consideration for MRI should be based on exam and clinical decision-making, including evaluation for high-risk fracture locations. As alternatives, x-ray or ultrasound may be used. Plain film x-ray is less expensive and reasonable for initial evaluation of a suspected BSI in non-spine locations (MRI is often necessary for BSI identification in the spine). However, x-ray may have a high rate of false negative findings, especially in early stages of injury, and utilizes ionizing radiation. Ultrasound may demonstrate presence of a cortical step off or hyperemia and can evaluate for soft tissue pathology. However, ultrasound typically has low yield for detecting most forms of BSI. A bone scan is an alternative imaging technique for identifying a BSI, but involves radiation exposure and is not specific for a BSI, oftentimes requiring follow-up imaging for diagnosis confirmation.

A. Pars Interarticularis

   1. *Differential diagnosis* includes common causes of axial low back pain (such as zygapophyseal joint-mediated arthropathy and discogenic low back pain) or sacroiliac joint-mediated pain. Visceral-referred pain should also be considered. In women, obstetric and gynecological sources of pain and symptoms must be considered, including pregnancy.

2. *Diagnostic testing* for spinal BSI has traditionally included plain film radiographs with anterior/posterior, lateral, and oblique views. Computed tomography of the lumbar spine with thin slices oriented through the pars can evaluate for presence of fracture. However, x-ray is commonly negative and CT involves greater exposure of ionizing radiation, a concern that is particularly important in patients of reproductive age. MRI of the lumbar spine oriented with thin slices (2–3 mm) using STIR sequence oriented through the pars interarticularis is preferred to evaluate for presence of BSI. MRI of the lumbar spine is also useful to evaluate for the presence of non-BSI spine-related conditions.

B. Sacral/Pelvic BSI
   1. *Differential diagnosis* includes tendinopathy, referred pain from primary hip pathology, spine-referred pain (including discogenic low back pain or S1 radiculopathy), piriformis strain/syndrome, tendinopathy in the pelvis (adductor, gluteal tendons, or hamstring), and sacroiliac joint-mediated pain.
   2. *Diagnostic testing* for a suspected BSI requires use of MRI. Most sacral BSIs are high-grade injuries and are thus important to identify to prevent injury progression and address underlying causes to prevent future injury. Sacral and pelvic bone stress injuries occur in bone that is more cancellous in composition, making it more hormonally sensitive to low sex hormones. In athletes of both sexes, presence of a pelvic BSI should prompt a more extensive endocrine workup and consideration for DXA to measure bone density. Triad and RED-S screening should be performed.

C. Femoral Neck
   1. *Differential diagnosis* includes hip-related pathology including femoroacetabular impingement, intra-articular pathology (such as labral tear), or iliopsoas tendinopathy.
   2. *Diagnostic workup* may initially include plain film x-ray to evaluate for presence of a displaced fracture and to provide information on bony anatomy, including presence of femoroacetabular impingement. MRI is more sensitive and should be considered if femoral neck BSI is suspected, as femoral neck or lesser trochanteric BSIs may progress to full fractures requiring surgery if undetected [5].

D. Femoral Shaft
   1. *Differential diagnosis* includes spine-referred pain; hip-referred pain; quadriceps, hamstring, or adductor tendinopathy; or myofascial pain.
   2. *Diagnostic testing* includes x-ray of the femur that may reveal fracture; however, MRI should be ordered if fracture is not visualized.

E. Leg
   1. *Differential diagnosis* for exertional leg pain is broad. More common causes of leg pain include medial tibial stress syndrome (MTSS)/shin splints, chronic exertional compartment syndrome (CECS), peripheral nerve entrapment, and vascular etiologies (including popliteal artery entrapment syndrome or vascular claudication).

2. *Diagnostic testing* for fractures of the tibia or fibula may be visualized using x-ray or MRI. The typical location for a tibial BSI is the posterior distal third of the tibia. However, a BSI localized to the anterior tibia is considered high risk due to its tension-sided location, which can contribute to higher risk for nonunion. Presence of "the dreaded black line" on x-ray, defined as radiolucency extended horizontally through the anterior tibial cortex, requires close evaluation to ensure fracture healing. MRI may also reveal presence of MTSS. The workup for other causes of exertional leg pain, including vascular, neurological, and CECS, are beyond the scope of this chapter and should be guided by experienced medical providers.

F. Foot/Ankle
1. *Differential diagnosis* is anatomically based and broad. For hindfoot fractures, primary ankle joint pathology (including ankle impingement or intra-articular pathology including osteochondral lesion or osteoarthritis), insertional Achilles tendinopathy, plantar fasciopathy, ankle sprain, chronic ankle instability, peripheral nerve entrapment, and presence of tarsal coalition may all cause pain. Mid-foot pain may be caused by metatarsalgia, ligament injury including Lisfranc ligament sprain, or neuroma. Forefoot pain may result in tendinopathy, arthritis, neuroma, or sesamoiditis.
2. *Diagnostic testing* should start with a weight-bearing x-ray of the foot or ankle for initial evaluation; this will provide information on bone alignment, presence of fracture, and joint disease. MRI can identify and grade a BSI and exclude soft tissue etiologies of pain, including presence of ligament sprain, osteochondral defect, or neuroma.

Nonoperative Management

A. Physical Activity

Most BSIs respond favorably to nonoperative management. Non-weight bearing with crutches (and a controlled ankle motion boot in certain circumstances) should be prescribed if the patient experiences pain with ambulation. Typically, 6 weeks of non-weight bearing is recommended for a BSI in a high-risk location (see the following section). Once ambulation is pain-free, the patient may progress to aerobic weight-bearing activities such as the elliptical trainer or a partial weight-bearing treadmill (e.g., AlterG, LightSpeed Lift, HydroWorx). For running sport athletes, deepwater running may help maintain aerobic fitness without impact loading. Conservatively managed high-risk and more severe BSIs require extended restrictions on weight bearing. Time for return to full sport participation is determined by the severity of the BSI, anatomical location, and addressing the underlying etiology of the injury to reduce risk for recurrence.

B. Diet

Due to the extensive role low energy availability can play in the development of BSI, it is essential to assess a patient's energy intake and expenditure to make sure they are consuming enough calories to meet their energetic demands. Athletes with Triad risk factors or RED-S should be referred to a nutritionist with experience in managing athletes with low energy availability. In the presence of disordered eating or an eating disorder, referral to mental health provider is critical in order to address the underlying disease and reduce the risk for injury recurrence and even death from the underlying eating disorder. For medical providers not familiar with managing the Triad/RED-S, referral to a sports medicine professional with experience managing these conditions is prudent. A workup for menstrual dysfunction is also important in female athletes. The Female Athlete Triad Coalition has helpful information on physician referral network and educational materials for patients and medical professionals (www.femaleathletetriad.org).

Micronutrients and vitamins also play a role in maintaining bone health and preventing BSIs. Calcium and vitamin D, for example, are both important for overall skeletal health. In the absence of food allergy, milk and dairy products provide an excellent source of dietary calcium. Additionally, calcium-rich foods often contain macro- and micronutrients that promote bone health, such as protein and phosphorus, and may be fortified with vitamin D. A benefit from athletes obtaining calcium from food includes increased energy intake and energy availability. However, vitamin D supplementation is also reasonable to ensure target 600 IU daily based on the Institute of Medicine 2010 guidelines [7].

C. Medications

Typically, use of analgesics is discouraged for management of BSI because pain is a useful symptom for the patient to monitor for fracture healing. Furthermore, anti-inflammatory medications including NSAIDs should be avoided due to concerns for impaired bone healing with this medication. Alternatively, screening for vitamin D deficiency is reasonable by obtaining serum 25-hydroxy vitamin D level; supplementation should be considered to ensure vitamin D levels are sufficient (25-OH >30 ng/dL), although a target of 45–50 ng/dL is being considered in athletes with a history of recurrent BSIs or athletes deemed higher risk for this form of injury (e.g., distance runners).

D. Physical Therapy

Physical therapy should be prescribed to address the biomechanical contributors to injury after the initial rest period following BSI diagnosis. Because muscle should serve as a primary shock attenuator for the bone, ensuring adequate muscle mass is important; a physical therapist can help identify and address these imbalances. The guiding principle for management of a BSI is for physical therapy to be focused on helping the patient develop a softer and more well-aligned landing during their sport activity.

E. Specialist Referral

Referral to a bone endocrinologist should be considered for athletes of both sexes who present with a fracture in a cancellous site, including the pelvis, or

with recurrent BSI. The workup and management is sex-specific. Female athletes with menstrual dysfunction require a full workup, as functional hypothalamic amenorrhea is a diagnosis of exclusion. Male athletes may require screening for low sex steroids and other endocrine conditions, such as thyroid disease, hyperparathyroidism, malabsorption diseases, or renal disease. Referral to an orthopedic specialist should be considered for athletes with atypical presentations of injury or who sustain a BSI at a high-risk location.

Operative Management

Rarely does a BSI require operative management. Exceptions include displaced fractures, some fracture localized to high-risk locations, or some BSIs that recur (depending on clinical context).

High-risk fracture locations should be evaluated by an orthopedic surgeon and/or someone with advanced clinical knowledge and expertise in bone stress injury management in the following locations: the femoral neck (particularly tension side), lesser trochanter, anterior tibial cortex, medial malleolus, talus, tarsal navicular, fifth metatarsal diaphyseal fracture (also known as Jones fracture), base of the second metatarsal, or sesamoids of the forefoot.

Surgical management may require use of rods, pins, or screws to approximate the bone and ensure bony union. Additionally, high-risk locations with excess biomechanical forces may fail to respond favorably to nonoperative management and recur if the athlete returns to the same activity levels prior to injury. For this reason, orthopedic surgeons may sometimes offer early surgical management to select patients who sustain injuries such as Jones fracture, given the more predictable healing response and return to sport with surgery as compared to conservative treatment.

Expected Outcomes

Most patients will have healing of a BSI with nonsurgical management. However, education regarding the underlying etiology of the injury is important to reduce risk for recurrence. This includes management of biological risk factors described by the Triad and RED-S. Biomechanical contributors including appropriate strength and conditioning exercises and gait retraining should be encouraged prospectively to reduce risk for future injury. Finally, prevention strategies should be employed to reduce future risk of injury.

Prevention

Prevention of a BSI includes optimizing biological and biomechanical risk factors, nutrition, and sleep.

A. Biology

Coaches, physicians, and athletic trainers should be familiar with the Triad/RED-S to ensure it is addressed. In addition to developing healthy attitudes about diet, exercise, and body image, childhood and adolescence are times of growth and peak bone mass accrual; therefore prevention or limitation of progression of consequences of low energy availability is critical for long-term bone health. Medical concerns of low energy availability should be screened at times of medical encounters of Triad and RED-S and managed appropriately during annual PPE prior to sports participation. The PPE can serve as the first line of defense against developing a BSI.

B. Biomechanics

Bone loading during childhood and adolescence in sports and activities that encourage multidirectional jumping may result in stronger and more fracture-resistant bones. Review of the literature suggests early participation in ball sports or jumping activities may improve bone quality and reduce the risk for future fractures, even on discontinuation of the activity [8].

C. Sleep

Sleep quality is important for overall health. Although the role of sleep in BSI development has not been determined in athletes, compelling evidence for the role of sleep contributing to bone health has emerged from military investigations. In one study, a subset of subjects randomly assigned to sleep upright or in sleep-deprived conditions had elevated bone turnover markers and a 5% bone mass loss over a 7-day period [9].

D. Nutrition

Ensuring adequate energy availability is critical to promote bone health for both sexes. Adequate energy availability is typically 45 kcal per kg of fat-free mass. Additionally, all athletes and active individuals should be encouraged to meet the calcium and vitamin D intakes as recommended by the Institute of Medicine [7]. For both sexes ages 9–18, calcium intake target is 1300 mg daily. Pre-menopausal female adults and males until age 70 should consume 1000 mg of calcium daily, whereas females ages 51 or males ages 70 or older should consume 1200 mg daily. Recommended vitamin D dietary intake is 600 IU daily for ages 9 and older.

Summary

In conclusion, a BSI is a common form of overuse injury in athletes and active individuals. The history and physical examination are the most important components of evaluating BSI risk. MRI is the most useful diagnostic test given its high sensitivity and ability to visualize soft tissue, as well as its ability to allow providers to grade the severity of injury. However, MRI is higher cost than x-ray, potentially limiting its use. Injuries in low-risk locations may be managed without advanced imaging, with an emphasis on repeat clinical examination to ensure bone healing.

Injuries in high-risk locations, however, should be imaged with MRI and potentially referred to a specialist. Management of injury includes activity modification, addressing underlying biological risk factors including the Triad and RED-S, physical therapy to address biomechanical contributors to injury, and optimizing nutrition. Prevention includes identifying and correcting low energy availability, addressing faulty sports movement mechanics, promoting osteogenic bone loading activities at a young age, and nutrition that includes a diet with adequate energy availability and foods rich in calcium and vitamin D.

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Chapter 21
Hamstring and Calf Injuries

David M. Robinson and Kelly C. McInnis

Abbreviations

CECS	Chronic exertional compartment syndrome
MRI	Magnetic resonance imaging
MTSS	Medial tibial stress syndrome
NSAID	Nonsteroidal anti-inflammatory drugs

Introduction

Hamstring and calf injuries are common causes of lower extremity pain in athletes. Depending on the severity of injury, they may be associated with significant functional impairments and morbidity. Etiologies range from acute strains to chronic overuse conditions. In this chapter, we discuss the presentation, diagnosis, conservative management, and operative indications for hamstring and calf pathologies commonly encountered in clinical practice.

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Hamstring Injuries

Hamstring Strain

Definition and Epidemiology

Hamstring muscle injuries are common as it is a biarticular musculotendinous unit with a high composition of type II fast-twitch fibers. From a functional standpoint, the hamstring decelerates the lower limb during walking, running, and sports-specific activities. High-speed run-type hamstring strains are most common, typically occurring at the myotendinous junction. The injury occurs during the terminal swing phase when the hamstring is at greatest tension and eccentrically contracting, or during the explosive take-off phase of running. The long head of the biceps femoris is frequently affected, thought in part due to its dual innervation, larger musculotendon stretch, and shorter muscle fascicles. Athletes participating in soccer, sprinting, and American rules football are at highest risk.

Proximal hamstring strains typically develop from progression of proximal hamstring tendinopathy or in the setting of excessive lengthening, in which case they are labeled stretch-type strains. Stretch-type hamstring strains are common in activities involving hip flexion and knee extension such as dancing, high kicking, and gymnastics. The semimembranosus is typically affected, often with partial-thickness tearing and minimal retraction. Conjoint tendon strains are less common and may occur from a forceful eccentric contraction during the late swing phase. Complete proximal avulsion injuries are seen following an eccentric contraction with the hip in a flexed position and concurrent knee extension, such as with ballistic activities like weightlifting. Avulsion fractures of the ischium involving the hamstring muscle origin rarely occur in adults but may occur in skeletally immature athletes.

Grade I injuries are mild with no significant strength or functional deficits, minimal muscle fiber damage, and pain that typically develops the day after injury. Grade II injuries include partial-thickness tears and are associated with loss of strength and acute pain. Grade III injuries are full-thickness tears or ruptures and cause significant disability.

Clinical Presentation

Patients with hamstring strain injuries typically present with acute posterior thigh pain. Often, this develops immediately following a high-risk running activity. Late swing and early stance phase injuries involving eccentric contraction are typical. An audible “pop” may be described and usually occurs with stretch-type injuries and complete avulsions. Inspection revealing broad ecchymosis and swelling may indicate a high-grade myotendinous injury or proximal avulsion. The ischial tuberosity

may be tender with proximal injuries and a palpable defect may be noted with higher-grade injuries.

Hamstring flexibility can be assessed with the passive straight-leg raise and bent-knee stretch tests. Pain may be provoked with active knee flexion or hip extension against resistance. Resisted prone knee flexion should be performed at 90, 45, and 15 degrees. If pain and weakness are evident at 90 degrees, when the hamstring has optimal length and leverage, prognosis may be worse. Passive knee extension is often more painful with partial-thickness tears.

A stiff-legged gait is frequently seen for both partial and full-thickness tears as patients attempt to avoid hip and knee flexion. Symptoms of sciatic neuralgia may occur resulting in radiating pain beyond the knee, paresthesias, and foot and ankle weakness (rarely); therefore, a thorough lower extremity neurologic examination should be performed.

Differential Diagnosis

In most cases, the mechanism of injury and localization of symptoms make the diagnosis of acute hamstring strain straightforward. However, in patients with long-standing gluteal and posterior thigh pain, recurrent symptoms, or an unclear mechanism, a definitive diagnosis can be more difficult. The differential diagnosis for gluteal and posterior thigh pain includes lumbosacral radiculopathy, sacroiliac joint dysfunction, piriformis syndrome, proximal hamstring tendinopathy, ischial bursitis, and ischiofemoral impingement.

Diagnostic Testing

Plain radiographs are helpful in identifying ischial avulsion injury; however, they are negative in most hamstring strains. Ultrasound and MRI are most frequently utilized to confirm clinical suspicion and provide detail regarding location and severity of the injury (Fig. 21.1). Ultrasound allows for dynamic correlation with the physical examination, providing useful information regarding tendon and muscle integrity. Sensitivity is greatest when done acutely when edema and/or hematoma is present in the soft tissue.

MRI may be more sensitive for detecting subtle partial-thickness tears, deeper myotendinous injuries, and proximal avulsion injuries. The “sickle sign” is suggestive of a partial-thickness tear and is seen as a crescent-shaped linear T2 signal intensity at the bone-tendon interface on axial and coronal images. In the case of severe hamstring injury and for surgical planning, MRI is preferred. This is because MRI can precisely define the injury site and quantify the degree of damage and number of involved tendons, extent of retraction, and chronicity given that sensitivity does not decrease with time.

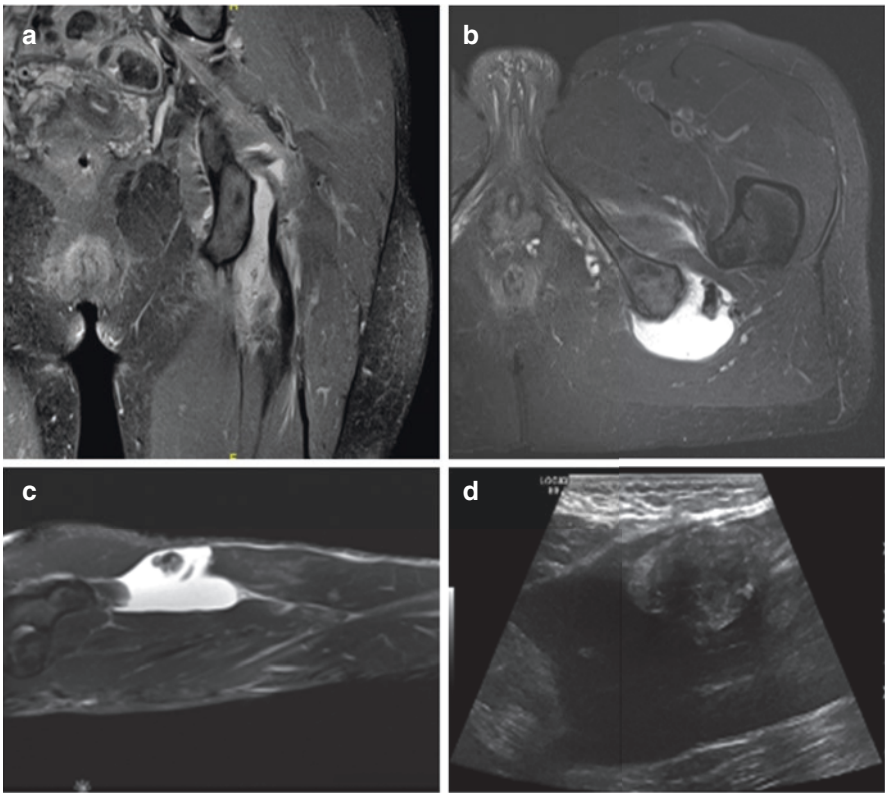

Fig. 21.1 MRI and ultrasound demonstrating full-thickness proximal hamstring tear with tendon retraction and associated hematoma. (**a**) Coronal T2 MRI, (**b**) axial T2 MRI, (**c**) sagittal T2 MRI, (**d**) sagittal ultrasound view

Nonoperative Management

Nonoperative management is indicated for single-tendon and two-tendon partial-thickness tears with less than 2 cm of retraction. Despite the differences in mechanism of injury, tissues involved, and recovery rates, current rehabilitation approaches do not differ greatly when treating high-speed running vs. overstretch injuries. A three-phase rehabilitation approach is recommended, with initial management focused on limiting local swelling and controlling pain. Though weight-bearing restriction with crutches may be necessary in the short term for pain control, early mobilization is recommended. NSAID use is controversial due to theoretical impairment of healing, but currently, there is no clinical evidence that short-term use in the treatment of hamstring injuries has deleterious effects. However, the benefit of NSAID use is questionable as well.

As acute symptoms improve, gentle stretching followed by hamstring isometric strengthening can be implemented. Core strengthening and stabilization is an important facet of rehabilitation. Deficits of strength and flexibility are targeted throughout the

lower extremity kinetic chain. This often includes gluteus maximus and hip abductor strengthening. Hamstring isotonic strengthening is introduced gradually as tolerated. Jogging and eccentric exercises can be prescribed when isometric knee flexion at 90 degrees and walking are pain-free. Once jogging at 50% of maximum speed is pain-free, more extensive neuromuscular control and sports-specific training can be implemented. Return to play is recommended when strength is at least 90% of the normal contralateral side. Platelet-rich plasma injection may be considered for refractory cases. Steroid injections are typically avoided due to fear of extending tissue damage.

Prevention of hamstring injury recurrence is essential. Eccentric exercises are emphasized and have been shown to produce greater strength gains than similar concentric hamstring movements. Prevention strategies are likely most beneficial as part of a preseason training program.

Nonoperative management of complete tears may be indicated in sedentary patients with significant medical comorbidity, or in those who cannot comply with postoperative restrictions. The rehabilitation approach is similar to partial-thickness tears and persistent strength deficits are often noted.

Indications for Operative Management

Surgical referral is indicated for two-tendon injuries with more than 2 cm of retraction, for complete proximal avulsion, and in selected partial-thickness tears that fail nonoperative management. For complete proximal avulsion of the tendinous origin from the ischium, early surgical repair is advised as this approach leads to more favorable outcomes in active individuals.

Expected Outcomes

Prognosis for return to sport is generally favorable for most hamstring strains. Mild strains may result in weeks of lost playing time, whereas more severe injuries may take many months to fully recover. Recovery from stretch-type hamstring strains has been shown to be prolonged compared to high-speed run-type hamstring strains. Recurrence rates are very high, estimated at 30%, and most occur within the first 2 weeks of returning to play.

Proximal Hamstring Tendinopathy

Definition and Epidemiology

Proximal hamstring tendinopathy, relative to hamstring strains, has a more insidious and progressive symptom onset. Athletes who perform distance running, hurdling and sprinting, and sports with frequent changes of direction are at risk. Contributing factors may include compression of the hamstring tendons at their attachment to the

ischial tuberosity during repetitive hip flexion and adduction and high-energy storage in the late swing to early stance phases. Resultant tendon degeneration and thickening develop secondary to repetitive cumulative microtrauma, often with concomitant enthesopathy or degenerative tears. Notably, the proximal hamstring is subjected to higher energy storage with forward trunk leaning, uphill running, and overstriding. Most commonly, the semimembranosus is affected.

Clinical Presentation

Patients typically describe deep buttock pain at the ischial tuberosity that is worse at initiation and upon completion of activity. Provocative activities include running (uphill in particular), acceleration, deep hip flexion (lunging), and direct pressure to the ischial tuberosity, as with prolonged sitting. Symptoms tend to worsen with sustained static stretching in end-range hip flexion postures. Physical examination consists of a series of palpatory, stretching, and loading maneuvers. There may be tenderness over the ischial tuberosity. Passive stretch tests include the modified bent-knee stretch test and the Puranen-Orava test. Loading maneuvers, working from lower to higher loads, include the single-leg bent-knee bridge, the supine plank with straight-leg raise, and the single-leg deadlift (Fig. 21.2).

Differential Diagnosis

The differential diagnosis for proximal hamstring tendinopathy includes hamstring strain or avulsion, lumbosacral radiculopathy, ischiofemoral impingement, piriformis syndrome, intra-articular hip pathology with radiation to the buttock, inferior-pubic rami bone stress injury, gluteal tendinopathy, and sacroiliac dysfunction.

Fig. 21.2 Supine plank test with straight-leg raise for identifying proximal hamstring tendinopathy. Pain is felt on the side where the leg remains in contact with the exam table

Diagnostic Testing

In many cases, the history and examination alone are sufficient to make the diagnosis. Plain radiographs are largely inconclusive but may show enthesopathy or intra-tendinous calcifications. Ultrasound or MRI is the preferred imaging modality when the diagnosis remains in question and may help identify tendinosis and partial tears and characterize the severity. Ultrasound often demonstrates thickening and degenerative changes of the proximal hamstring tendinous insertion. MRI may show tendon thickening, peritendinous edema with a feathery distal pattern, and ischial tuberosity bone marrow edema.

Nonoperative Management

Nonoperative management is successful for most cases. Therapy should initially focus on core and lumbopelvic stabilization, including gluteal muscle activation. Load modification emphasizing less repetitive hip flexion activities is recommended. Hamstring stretching is generally not recommended, as in other lower extremity insertional tendinopathies. Exercises should be progressed, guided by pain, from isometric to isotonic with increasing degrees of hip flexion, to eccentric and energy storage. With these activities, low levels of pain are acceptable provided the pain does not last longer than 24 hours. Activities that stress the proximal hamstring tendons such as running hills and hurdles should be avoided in the early stages of rehabilitation. Additional recommendations to reduce pain include postural modification, cycling in a standing position, and using seat cushions.

In refractory cases, procedures that attempt to incite a proinflammatory response and stimulate tendon healing may be trialed. These include ultrasound-guided needle tenotomy with or without platelet-rich plasma and extracorporeal shockwave therapy. Additionally, a one-time corticosteroid injection under ultrasound guidance, targeting the hamstring tendon sheath (provided there is no partial tear), may be helpful to reduce pain and facilitate full participation in a progressive loading program.

Indications for Operative Management

Surgical referral is indicated for those with chronic disabling symptoms unresponsive to the aforementioned treatments, or when the tendinopathy has progressed to high-grade partial- or full-thickness tear. Operative management may consist of open tendon debridement or primary repair.

Expected Outcomes

This condition can be difficult to treat. Nonoperative management is successful in the majority of cases, often resulting in return to sport within 3–6 months. Low levels of pain may persist for a few weeks upon resumption of normal activity.

Surgical candidates typically have more severe pathology and thus require a longer rehabilitation phase.

Distal Hamstring Injuries

Definition and Epidemiology

Distal hamstring tendon injuries occur less frequently than myotendinous and proximal pathologies. Injuries vary from low-grade strains to avulsions. The biceps femoris appears to be more commonly affected in distal avulsion injuries, again likely secondary to its dual innervation and resultant dissimilar force vectors. High tension loads are placed on the distal biceps femoris with forceful hip flexion and knee hyperextension, as can be seen when kicking a soccer ball.

Clinical Presentation

Similar to other hamstring strains, patients often present acutely with posterior thigh or knee pain from a noncontact injury. Often, these injures are accompanied by a feeling of being kicked in the posterior knee. Ecchymosis and stiffness may be observed. Examination demonstrates tenderness to palpation of the fibular head and posterolateral corner of the knee. Knee flexion weakness and weight-bearing difficulty are common.

Differential Diagnosis

The differential diagnosis for distal hamstring tendon injuries include deep vein thrombosis, ruptured Baker's cysts, intra-articular knee pathology, popliteus injury, lateral collateral ligament sprains, and proximal gastrocnemius strains.

Diagnostic Testing

Radiographs should be obtained to rule out fibular avulsion fractures. Ultrasound and MRI again are the preferred imaging modalities with similar findings to those seen in more proximal strains. Notably, distal semitendinosus strains are often larger than those seen proximally.

Nonoperative Management

Treatment is similar to those of myotendinous and proximal strains. Acute focus is on pain and limiting inflammation, with progression to a rehabilitation program ultimately working toward pain-free eccentric and sports-specific exercises. Grade I distal strains tend to do very well when managed conservatively.

Indications for Operative Management

Literature on operative management for distal hamstring injuries is limited. Generally, higher-grade distal hamstring injuries and full-thickness tendon tears require surgical repair for a better outcome. Surgical options include anatomic repair to the fibular head for distal biceps femoris tears and occasionally tenotomy for distal semitendinosus tears.

Calf Injuries

Gastrocnemius and Soleus Strains

Definition and Epidemiology

Gastrocnemius and soleus muscle injuries typically occur in sports involving acceleration and deceleration or high-volume running loads such as tennis, soccer, and running. These muscles constitute the triceps surae and blend distally with the plantaris to form the Achilles tendon. The gastrocnemius is at highest risk of strain given its biarticular nature and primary composition of type II fast-twitch fibers. These features predispose the gastrocnemius to excessive stretches and greater internal forces from rapid muscle contraction. Most commonly, the medial head of the gastrocnemius is injured at the musculotendinous junction. The classic mechanism of injury is a rapid eccentric contraction with knee extension and ankle dorsiflexion. Colloquially, this has been termed "tennis leg." Soleus injuries are less common as the muscle crosses only one joint and is primarily composed of type I slow-twitch fibers. Typically, soleus injuries are more subacute and occur when the knee is flexed.

Clinical Presentation

The acuity of symptom onset, injury mechanism, and focused physical examination are often sufficient for accurate diagnosis and differentiation between gastrocnemius and soleus strains. Most patients with gastrocnemius strains recall a sudden onset of pain occasionally accompanied by an audible pop. Often, patients report a sensation of being "kicked" in the posterior leg. Swelling typically occurs acutely within hours to days of the injury. Soleus injuries classically present with calf tightness and pain in the distal third of the poster leg that worsens over days to weeks and is associated with a period of overuse. Impact exercise including walking or jogging tends to provoke symptoms. Swelling and disability are generally milder when compared with gastrocnemius strains.

Examination begins with inspection for visible swelling and ecchymosis. In more severe injuries, there may be visible calf asymmetry and a palpable muscle defect. Pain with plantar flexion when the knee is extended is more common with

gastrocnemius injuries, whereas the soleus is preferentially activated when the knee is flexed. Functional movements such as calf raises and circumferential calf measurements should also be done. Neurological examination, including positions of neural tension, can be performed to rule out S1 radicular pain.

Differential Diagnosis

The differential diagnosis for calf strain includes Baker's cysts with or without rupture, popliteus injury, deep vein thrombosis, MTSS, popliteal artery entrapment syndrome, Achilles tendinopathy, acute or chronic exertional compartment syndrome, lumbar radiculopathy, and claudication.

Diagnostic Testing

In many cases, the history and examination are sufficient to diagnose calf strain. When the diagnosis is unclear or there is concern for a more severe injury, imaging is helpful to better characterize injury extent and guide return to play protocols. Ultrasound or MRI is the first-line imaging modality. Ultrasound findings may include muscle fiber disruption, hypoechogenicity within the muscle, and fluid between the medial gastrocnemius and soleus that is most prominent at the myotendinous junction (Fig. 21.3). Fluid collection size and presence of hemorrhage are informative regarding the extent of injury. Axial views allow visualization of the entire muscle belly and may help distinguishing partial- from full-thickness tears. MRI will demonstrate T2 high-intensity signal, indicating fluid. Muscle fiber discontinuity or rupture and fiber retraction may also be found.

Nonoperative Management

Nonoperative management is the mainstay for calf muscle strain. Acutely, treatment aims to provide symptom relief and limit hemorrhage. For the first few day's post-injury, patients should rest the calf through limiting stretch and contraction. Ice, compression, and elevation may be utilized. NSAID use is controversial given potential increased bleeding risk. Crutches, walking boots, and heel lifts may be used in the short term to decrease pain with ambulation. Thereafter, a rehabilitation regimen should be implemented to maintain range of motion and prevent contracture. The regimen begins with passive stretching to elongate the intramuscular scar and prepare the injured muscle for strengthening. When ankle dorsiflexion is essentially pain-free, isometric and dynamic calf strengthening exercises can be added progressively as tolerated.

Although rare, myositis ossificans and compartment syndrome may complicate acute strains. If symptoms do not improve as expected, reexamination and consideration for imaging studies should be done to evaluate for these complications.

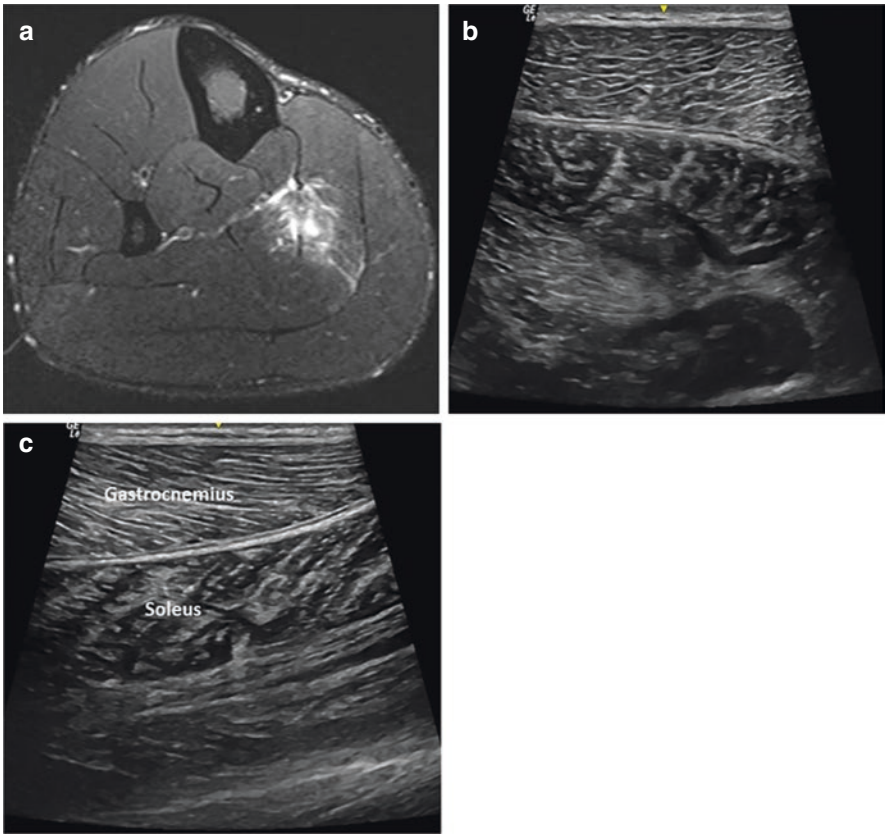

Fig. 21.3 MRI and ultrasound demonstrating a soleus strain. (**a**) Axial T2 MRI with hyperintense fluid signal within the soleus musculature. (**b**) Axial ultrasound with corresponding anechoic fluid and (**c**) sagittal ultrasound view with muscle fiber discontinuity and hypoechogenicity. Note the difference in fiber organization compared to the superior gastrocnemius muscle

Indications for Operative Management

The only reported absolute surgical indication is when a patient with a calf strain subsequently develops compartment syndrome, at which point fasciotomy is indicated.

Expected Outcomes

Prognosis is typically good, although those with more severe injuries may experience prolonged symptoms. Those with grade I or II strains can often return to sport within 2 months. Grade III strains often require at least 6 months to return to previous activity level. Recurrence rates are estimated at 15% and typically occur within 6 months of the initial injury.

Medial Tibial Stress Syndrome

Definition and Epidemiology

Medial tibial stress syndrome (MTSS), commonly referred to as "shin splints," is a clinical syndrome of pain along the posteromedial border of the tibia that occurs during exercise. It is commonly seen in runners, military recruits, and jumping athletes. While the underlying pathophysiology is debated, both traction from repetitive musculature contraction causing periostitis and local bone stress reactions from repeated tibial loading have been postulated as the primary causes. Muscular attachments of the soleus, flexor digitorum longus, and tibialis posterior have all been implicated in the traction theory. Risk factors for MTSS include elevated body mass index, increased navicular drop, increased ankle plantar flexion or hip external rotation range of motion, and previous history of MTSS. Ischemic causes of pain and stress fractures should be excluded prior to formal diagnosis.

Clinical Presentation

MTSS presents as exercise-induced posteromedial leg pain, usually along the distal two-thirds of the tibia. Early on, symptoms develop upon exercise initiation but dissipate with continued activity. As pathology progresses, pain may persist throughout exercise and even after its completion. When this is the case, bone stress injury should be excluded.

Physical examination aims to localize symptoms and evaluate for contributing risk factors while excluding alternate pathologies. Tenderness at the posteromedial tibial border over a length of ≥ 5 consecutive centimeters is characteristic. In contrast, pain is more focal and horizontal with tibial bone stress injuries. The anterior tibia is usually non-tender. Biomechanical risk factor assessment and general screening for proximal lower extremity and core musculature strength deficits are recommended. Footwear should be inspected for tread wear, which may implicate certain gait abnormalities.

Differential Diagnosis

Other causes of posteromedial leg pain include tibial bone stress injury, chronic exertional compartment syndrome, gastrocnemius or soleus strain, popliteal artery entrapment syndrome, deep vein thrombosis, Achilles tendinopathy, and lumbar radiculopathy.

Diagnostic Testing

MTSS is a clinical diagnosis. Plain radiographs are normal in patients with MTSS but may be helpful to evaluate for tibial stress fracture. Radiograph finding of the "dreaded black line" is indicative of anterior tibial stress fracture, although notably radiographs are often negative with early bone stress injuries. MRI can be utilized if needed to rule out bone stress injury and confirm MTSS with characteristic finding of periosteal edema.

Nonoperative Management

Treatment of MTSS is nonoperative. Temporary reduction of provocative activities is initially recommended. Patients are advised to decrease running volume and intensity and incorporate nonimpact cardiovascular fitness modalities such as cycling and swimming. Running on uneven surfaces or hills should be avoided while symptomatic. Oral analgesics and post-activity cryotherapy can be utilized as needed. As symptoms improve, a targeted therapy program with graded tibial loading, calf strengthening (including soleus- and posterior tibialis-specific exercises), and proprioceptive balance training can be implemented. Modifiable biomechanical deficits in the lower extremity kinetic chain should be addressed. Once pain-free, athletes may slowly increase training intensity and duration, add hill running, and begin sports-specific activities. Expectations for recovery should be discussed upon diagnosis as this condition may take months to resolve. For those with recalcitrant pain, gait retraining or shockwave therapy can be considered.

Indications for Operative Management

Surgical treatment is rarely indicated for MTSS. Consultation can be considered when pain persists despite exhaustive conservative management and in those with multiple prolonged recurrences. Surgical options include posteromedial fasciotomy with or without periosteal stripping.

Expected Outcomes

Full recovery can typically be expected with activity modification and rehabilitation. Patient education about the nature of MTSS and its relation to inadequate load management is necessary. Symptoms will often persist or recur when pre-injury activity levels are resumed too quickly.

Chronic Exertional Compartment Syndrome

Definition and Epidemiology

Chronic exertional compartment syndrome (CECS) is a condition characterized by exercise-induced leg pain caused by supraphysiologic compartmental tissue pressures. It commonly presents in young athletes participating in distance running, with similar incidence between men and women. Symptoms are typically bilateral. The primary risk factor is repetitive and strenuous exercise, as this can lead to greater muscle volume from increased blood flow and edema, thereby raising intra-compartmental pressures. Pain arises from tissue ischemia or disproportionate oxygen supply versus demand of the compartment muscles. Of note, increased muscle volume, as can be seen with anabolic steroid or creatine use, is an additional risk factor. The anterior compartment is most often affected, followed by the lateral. Deep and superficial posterior compartments are less frequently involved.

Clinical Presentation

CECS should be suspected in athletes presenting with anterior leg pain that worsens with prolonged use and resolves upon activity cessation. Patients often describe the pain as occurring predictably at the same time, distance, or intensity of exercise, and daily activities are typically non-provocative. Burning, aching, or a pressure-like pain quality is common. As the condition worsens, weakness of compartment-specific muscles (such as transient foot drop) and paresthesia in an affected peripheral nerve distribution may develop. Physical examination aims to exclude alternate pathologies and identify which compartments are affected through a thorough neurologic exam. Ideally, the exam should be performed after the athlete completes the provocative exercise. At rest, the examination may be completely benign without findings. The venous system is typically affected, and thus arterial pulses are normally intact.

Differential Diagnosis

Conditions that may present similar to CECS include MTSS, tibial stress fracture, peripheral nerve entrapment, claudication, fascial defects, popliteal artery entrapment syndrome, and deep vein thrombosis.

Diagnostic Testing

The definitive diagnosis of CECS is made through intra-compartmental pressure testing. Notably, this is invasive and thus it is imperative to first rule out other causes of lower leg pain. Compartment pressures are taken prior to exercise and at both 1 and 5 minutes after completion. Patients should perform the offending activity until

symptoms develop. A resting pressure >15 mmHg, 1-minute post-exercise pressure >30 mmHg, and a 5-minute post-exercise pressures greater >20 mmHg are diagnostic of compartment syndrome. MRI may show fascial thickening, edema, and increased T2 signal intensity in the involved muscle. Electromyography can be considered to rule out peripheral nerve entrapment.

Nonoperative Management

Notably, there is often significant delay in the diagnosis of CECS from time of symptom onset. The only nonoperative treatment certain to alleviate the pain from CECS is cessation of causative activities. Other treatments occasionally trialed include avoidance of running on hard surfaces, changing footwear, and gait retraining. Cycling may be substituted for running to maintain cardiorespiratory fitness in patients who wish to pursue nonoperative management.

Indications for Operative Management

The definitive treatment of CECS is fasciotomy, with only the involved compartments being released. Open and endoscopic fasciotomies are the most common techniques. Some evidence exists for ultrasound-guided fasciotomies, although it has not been studied well enough to be considered a first-line option.

Expected Outcomes

Success rates after anterior or lateral compartment fasciotomies have been reported to be up to 80%, while posterior compartment fasciotomies tend to fare worse. Postoperatively, a graded rehabilitation regimen should be initiated with the goal of return to full activity by 6–12 weeks.

Plantaris Tendon Ruptures

Definition and Epidemiology

Plantaris tendon injuries and ruptures are relatively rare and occur via similar mechanisms to those of gastrocnemius strains. However, these injuries can occur in isolation as the muscles' biarticular nature predisposes it to injury. The plantaris is a small muscle of the superficial posterior compartment that originates on the posterolateral femoral condyle and descends along the medial Achilles tendon with variable insertion. Its primary function is thought to be more proprioceptive than locomotive. Ruptures of the plantaris typically occur at the proximal myotendinous junction and may mimic a deep vein thrombosis. Distal ruptures occur less commonly.

Clinical Presentation

Plantaris rupture presents similar to gastrocnemius strain, with acute posterior leg pain and possible ecchymosis. Often, athletes describe feeling as though they were struck in the calf, despite the injury usually being noncontact. A palpable mass may be felt between the lateral gastrocnemius and popliteus. Thompson's test will typically be negative, and plantar flexion will cause pain.

Differential Diagnosis

Other conditions that present similar to plantaris rupture include gastrocnemius or soleus strain, deep vein thrombosis, and ruptured Baker's cyst.

Diagnostic Testing

Imaging, such as venous Doppler ultrasound, may be helpful to exclude alternate pathologies. Musculoskeletal ultrasound will show plantaris discontinuity. Fluid accumulation is typically present between the medial gastrocnemius and soleus muscles. In the setting of rupture, MRI and ultrasound may demonstrate proximal muscle retraction with a mass between the popliteus tendon anteriorly and the lateral gastrocnemius posteriorly.

Nonoperative Management

Plantaris injury-specific management literature is sparse. However, rehabilitation protocols similar to those for gastrocnemius and soleus strains are often recommended. Initial management aims to limit pain and inflammation while promoting early mobilization. A progressive strengthening and proprioceptive rehabilitation program should be prescribed. Notably, the plantaris may be preferentially targeted when strengthening exercises are performed with the ankle in external rotation.

Indications for Operative Management

Nonoperative management is successful for most cases of plantaris tendon injury. Fasciotomy may be necessary if a superficial posterior compartment syndrome develops secondary to a hematoma or swelling.

Table 21.1 demonstrates a summary of hamstring and calf injuries with synopsis of presentation, diagnostic testing, and suggested management.

Table 21.1 Summary of hamstring and calf injuries with presentation, diagnostic testing, and suggested management

Clinical entity	Presentation	Diagnostic testing	Nonoperative management	Surgical indications and operative management
Hamstring strain	Acute posterior thigh pain Pain +/− weakness with resisted knee flexion and hip extension	Primarily clinical U/S—myotendinous defect or hematoma MRI—evaluate for full avulsion at ischial tuberosity	Protected weight bearing, rest, ice PT	Complete proximal tendon avulsions, three-tendon tears, and two-tendon tears with >2 cm of retraction
Proximal hamstring tendinopathy	Insidious deep buttock pain over ischial tuberosity Pain provocation with bent-knee stretch test, single-leg bridge, resisted hip extension and knee flexion	U/S or MRI—tendinosis or partial tear of proximal hamstring tendinous insertion, intra-tendinous calcifications	PT U/S-guided tenotomy with injection	If failure of nonoperative care Open tendon debridement or primary repair
Distal hamstring strain	Acute posterior knee pain Pain +/− weakness with knee flexion	Radiographs to rule out avulsion US or MRI—tendinous defect, hematoma, avulsion	Protected weight bearing, rest, ice PT	Tendon avulsion at the fibular head
Gastrocnemius and soleus strains	Acute posterior calf leg pain for gastrocnemius, often subacute for soleus Pain with ankle plantar flexion, push-off, passive dorsiflexion	Primarily clinical US or MRI—hematoma or swelling Defect in muscle fibers with possible retraction	Protected weight bearing, rest, ice PT	
Medial tibial stress syndrome	Posteromedial tibial pain worse with activity Tenderness ≥5 cm of the posteromedial tibia	Primarily clinical MRI—periosteal or bony edema	Activity modification PT Gait retraining or shockwave therapy	
Chronic exertional compartment syndrome	Exercise-induced leg pain May be neurologic findings in involved compartments	Compartment testing	Impact activity cessation Gait retraining	Open or endoscopic fasciotomy

(continued)

Table 21.1 (continued)

Clinical entity	Presentation	Diagnostic testing	Nonoperative management	Surgical indications and operative management
Plantaris tendon rupture	Acute calf pain Pain with plantar flexion	US or MRI—plantaris discontinuity Consider Doppler to rule out deep vein thrombosis	Protected weight bearing, rest, ice PT	

Suggested Reading

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Chapter 22
Running Injuries

Matthew Zinner and Rebecca G. Breslow

Introduction

In 2011, the American College of Sports Medicine updated its position on exercise prescription and recommended that most healthy adults engage in moderate-intensity physical activity for at least 150 minutes per week, vigorous-intensity physical activity for at least 75 minutes per week, or a combination of the two to achieve 500–1000 metabolic equivalents per week. Many Americans meet these requirements through recreational long-distance running. In the 2018 Running USA National Runner Survey, a survey of more than 4000 runners, 81% of respondents categorized themselves as either fitness or recreational runners. Additionally, three in four respondents reported a running-related injury in the previous 12 months, and 22% experienced an interruption in their training of 4 days or more due to a running-related injury at least once during that time period. Only 25% of these reported they would seek advice from a doctor – just slightly more than those who reported they would either seek advice from other runners or online – and nearly one-third admitted they would continue to run through their injury. Medical providers who treat recreational runners must be well-versed in the typical injury patterns that befall them, and in the management and prevention of these injuries, in order to engender trust and encourage runners to seek care when needed. The prevalence and characteristics of specific running-related injuries and management strategies are described below and summarized in Table 22.1. A return to running protocol, as well as a discussion of running injury prevention, also follows.

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Table 22.1 Summary of running-related injuries, evaluation, and management

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Hip flexor tendonitis	Anterior hip pain	Stinchfield's test Ludloff's test Snapping hip sign Ultrasound MRI	NSAIDs Activity modification Physical therapy	Failure of conservative treatment	Open repair of affected tendon
Femoral bone stress injuries	Anterior hip/groin pain Female athlete triad/ RED-S	X-ray MRI	Restricted weight-bearing Gradual, progressive return to activity after 6–12 weeks	Femoral neck tension side fracture Nonunion femoral shaft fracture	Percutaneous cannulated screw placement
Iliotibial band syndrome	Lateral knee pain	Noble test Ober test Ultrasound MRI	NSAIDs Activity modification Physical therapy	Failure of conservative treatment	Excision or release of distal ITB
Popliteal artery entrapment syndrome	Calf/lower leg pain	Ankle-brachial index Dynamic duplex ultrasound CT with angiography MRI with angiography	N/A	Surgical referral is appropriate for anatomic and functional variants	Myotomy and rerouting of artery or abnormal gastrocnemius insertion
Chronic exertional compartment syndrome	Lower leg pain with exertion	Needle manometry X-ray Bone scintigraphy MRI	NSAIDs Activity modification Physical therapy Gait retraining	Failed conservative management Intractable symptoms	Open fasciotomy Endoscopy-assisted compartment release Single minimal incision fasciotomy Percutaneous fasciotomy under local anesthesia Ultrasound-guided fasciotomy

(continued)

Table 22.1 (continued)

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Tibial bone stress injury	Lower leg pain Female athlete triad/ RED-S	X-ray MRI	Immobilization Cross-training Physical therapy Gradual progressive return to activity after 6–12 weeks	Delayed union fractures Nonunion fractures	Compression plating, drilling, intramedullary nailing
Peroneal tendinopathy	Lateral ankle pain	Ultrasound MRI	NSAIDs Activity modification Orthoses Physical therapy	Failure to respond to conservative treatment Peroneal subluxation or dislocation	Direct repair of superior peroneal retinaculum
Tibialis posterior tendinopathy	Medial ankle pain	"Too many toes" sign Excessive pronation Ultrasound	NSAIDs Physical therapy Orthoses ESWT	Failure of conservative treatment for significant deformity	Tendon transfer and bone realignment
Metatarsalgia/ metatarsal bone stress injury	Forefoot pain	Silfverskiold test Drawer test X-ray MRI	NSAIDs Activity modification Physical therapy Boot immobilization Toe sleeves Contrast bathing Orthoses	Failure of conservative treatment	Gastrocnemius release Distal metatarsal osteotomies Metatarsal shaft osteotomies Dorsal plate fixation and autogenous bone graft for recalcitrant stress fractures

Running-Related Injuries of the Hip

Hip Flexor Tendonitis

Summary of Epidemiology and Definition

Hip flexor tendonitis is inflammation of the hip flexor tendons caused by overuse, repetitive actions, or acute injury. In a cross-sectional study of 110 male athletes, 1/3 of all acute groin injuries affected the hip flexor; the iliopsoas was most commonly implicated, followed by rectus femoris, pectineus, and sartorius. In a retrospective case-control study of 2002 running injuries, the incidence of iliopsoas injuries was 0.8%.

Clinical Presentation

Runners with hip flexor tendinitis present with a history of insidious anterior hip pain that has developed over the course of months. They may report a recent increase in running distance, intensity, or frequency. The pain will usually be present during the initial phase of running, improve after warming up, and then reemerge as the run becomes more strenuous. The pain can also be provoked when changing positions from sitting to standing. The runner may also complain of accompanying pain in the contralateral gluteal region as well as the lower back.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis for hip flexor tendonitis includes hip osteoarthritis, hip labral tears, or referred pain from lumbar degenerative disc disease.

Hip flexor tendonitis is often diagnosed based on the history and physical exam. Key findings include pain with hip flexion and tenderness at the insertion of the iliopsoas. The clinician should observe the runner walking and may notice a shuffling gait if symptoms are more severe. The runner may also have a shortened stride length on the unaffected side due to limited hip extension, and hip flexors may be tight during range of motion exercises. Pain with resisted hip flexion in a supine position (Stinchfield's test), or seated (Ludloff's test), and pain and/or weakness of the iliopsoas during resisted flexion of the hip with the hip externally rotated are other positive findings. Many runners with hip flexor tendinitis will also demonstrate obligate external rotation of the hip on passive hip flexion, known as a positive Drehmann sign.

Both ultrasound and MRI may be helpful in confirming a diagnosis of hip flexor tendonitis. Positive findings include edema of the involved structures, architectural disruption, tears, and avulsions.

Non-operative Treatment

Initially runners with this condition should be treated conservatively. If no contraindications, NSAIDs may be used to manage pain in the acute phase. Runners should modify their activities and avoid those that cause pain until symptoms are improved. A physical therapy program focusing on stretching and strengthening the core and peri-pelvic stabilizers, as well as iliopsoas-specific stretching and strengthening, is recommended. The runner should also complete hip range of motion, pelvic mobilization, and anti-lordotic exercises. Most patients regain normal hip function with 6 to 12 months of activity modification and physical therapy.

Indications for Surgery

If the runner does not respond after extensive conservative treatment or is unable to run or perform other activities of daily life, surgery may be indicated. Surgical candidates should be prepared to undergo postoperative rehabilitation. Of note, it is extremely rare for runners with hip flexor tendinitis to progress to the point of having to consider surgical intervention.

Operative Treatment

Operative treatment approaches may be open or arthroscopic. The open technique involves lengthening the iliopsoas muscle or releasing the tendon. The arthroscopic procedure also involves lengthening the iliopsoas tendon and has fewer complications.

Expected Outcomes

Open surgical procedures for hip flexor tendinitis have been associated with a high rate of complications, including persistent hip pain, recurrent snapping hip syndrome, persistent hip flexor weakness, and wound healing issues. Arthroscopic approaches are better tolerated, with fewer complications. A case series of 15 athletes with refractory painful snapping hips and who underwent arthroscopic iliopsoas tendon release reported full return to sport at an average of 9 months post-operation for all participants.

Femoral Bone Stress Injuries

Summary of Epidemiology and Definition

In a descriptive study of stress fractures in NCAA athletes, Rizzone et al. found the incidence of femoral stress fractures in runners (cross-country, indoor track and outdoor track) to be 19.4% for women and 7.0% for men. In another study, athletes who ran more than 32 km per week were twice as likely to suffer a stress fracture of any kind, and athletes with a body mass index (BMI) of less than 19 were nearly three times more likely. Low energy availability, which frequently includes an eating disorder, menstrual dysfunction, and low bone density, places an athlete at further risk. In a cohort study of 259 physically active women, the risk of suffering a bone stress injury increased 15–20% for participants with 1 risk factor for the female athlete's triad and 30–50% for those with 2 or more risk factors. For more information regarding these pathophysiologic risk factors, please see Chap. 20.

Clinical Presentation

The runner's history may include an increase in running distance, pace, or hill running. Pain worsens with activity and improves with rest. The pain is usually described as vague, localizes to the anterior groin, and may be exacerbated by weight-bearing, in particular jumping, hopping, or running.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis includes femoroacetabular impingement, labral tear, iliopsoas tendinopathy, hip osteoarthritis, and inguinal hernia.

On physical exam, hip range of motion is painful, especially hip internal rotation. Though not validated for femoral bone stress injuries, a positive hop test is a common finding (between 70 and 100%) in patients with presumed lower extremity stress fractures, including femoral bone stress injuries.

Stress fractures may be diagnosed by X-ray, though, in most cases, they will not be visible for 3–4 weeks post-injury. Therefore, MRI is the modality of choice for suspected bone stress injuries when X-ray is inconclusive. A systematic review determined that MRI has a sensitivity of 68–99% and a specificity from 4 to 97% for lower extremity stress fractures.

Non-operative Treatment

Treatment approaches vary depending on the location of the femoral bone stress injury. Strategies for specific fracture locations are outlined below:

Femoral Shaft Fracture

The runner should be non-weight-bearing with crutches for an initial rest period of 3–4 weeks. Symptom control may be achieved with oral analgesics, such as acetaminophen. After this period, runners may begin weight-bearing and non-weight-bearing exercises. Many runners can resume running by 12 weeks post-diagnosis, but this should be cautiously introduced through a graduated, walk-run interval program.

Femoral Neck Compression Side Stress Fracture

Stress fractures on the compression side of the femoral neck are rarely displaced, so these may be managed non-operatively. The runner should be non-weight-bearing on crutches for the first 2 weeks. At 2 weeks they may begin partial weight-bearing

if pain-free. At 4 to 6 weeks, most can resume full weight-bearing if pain-free, as well as non-weight-bearing exercises. Running can resume at 12 weeks as above.

Femoral Neck Non-displaced Tension Side Fractures

Non-displaced tension side fractures are generally referred for surgical intervention. However, in a cohort of 70 military recruits with non-displaced femoral neck fractures, followed longitudinally, none developed fracture displacement or adverse complications with conservative treatment. Conservative management includes 2 weeks of bed rest plus an additional 4–8 weeks of strict non-weight-bearing with crutches. The patient may then progress to partial and then full weight-bearing. Finally, at 12 weeks post-injury, the patient may begin a gradual 6-week return to running program.

Indications for Surgery

Femoral neck stress fractures may require surgery, even when not displaced, if the runner will not be compliant with a strict recovery program or desires a quicker return to running. Displaced femoral neck fractures require urgent referral to an orthopedic surgeon for operative intervention.

Operative Treatment

Operative approaches are outlined below:

Femoral Neck Tension Side Fractures (Non-displaced)

The patient is treated with surgical percutaneous cannulated screw placement.

Femoral Neck Tension Side Fractures (Displaced)

These fractures are treated with open reduction and internal fixation. Emergent surgery may reduce the chance of avascular necrosis of the femur; therefore, operative intervention should occur as soon as possible.

Outcomes

Patients with non-displaced fractures generally make a full recovery within the appropriate time frame for the specific injury. Displaced fractures have less optimistic outcomes. In a study of 23 athletes with displaced fractures, 60% were unable to return to pre-injury levels of activity, and in another study of 6 cases in the British military, 50% of cases required more than 12 months to achieve union.

Greater Trochanteric Pain Syndrome

For additional detail on greater trochanteric pain syndrome, please see Chap. 9.

Proximal Hamstring Tendinopathy

For additional detail on proximal hamstring tendinopathy, please see Chap. 21.

Running-Related Injuries of the Knee

Distal Iliotibial Band Syndrome

Summary of Epidemiology and Definition

Distal iliotibial band syndrome (ITBS) is caused by the repetitive friction of the iliotibial band (ITB) rubbing against the lateral femoral condyle during knee flexion. The ITB is a continuation of the tendinous portion of the tensor fascia late muscle that crosses the hip joint to insert on the patella, tibia, and biceps femoris tendon. In a systematic review including 3500 runners, the incidence of iliotibial band syndrome was 1.8–9.1%.

Clinical Presentation

Runners with ITBS present with an insidious onset of pain localized to the lateral knee where the ITB crosses the lateral femoral condyle. The pain can vary from a sharp, transient burning sensation to a persistent deep pain that lasts for the duration of the activity. The lateral knee is tender approximately 2 cm above the joint line, which worsens with standing or knee flexion to 30 degrees. Runners will often report a history of recent increases in running distance, pace, or hill running.

Differential Diagnosis and Diagnostic Testing

Other causes of lateral knee pain include patellofemoral pain, lateral meniscal injury, popliteus tendinopathy, lateral hamstring tendinopathy, and lateral collateral ligament injury. ITBS can be distinguished by historical details and physical exam findings.

Special tests for ITBS include the Noble and Ober tests, though neither has been validated for ITBS in a clinical trial.

Ober test This is performed by placing the runner on their side with the unaffected hip on the table and affected side up in the air. The examiner then places one hand on the patient's affected hip and the other on the lower leg for support. The hip and knee are held at zero degrees extension and allowed to passively adduct with gravity. If the runner is unable to passively adduct past midline, then the test is positive.

Noble test This is performed by having the runner lie on their unaffected side with the affected knee up and flexed 90 degrees. The physician will place pressure on the ITB at the lateral femoral condyle and then extend the knee. The test is positive if pain occurs as the knee approaches 30° of flexion.

X-rays are generally negative in ITBS. Musculoskeletal ultrasound and MRI may show a thickened ITB over the lateral femoral condyle and a fluid collection deep to the ITB.

Non-operative Treatment

Non-operative treatment of ITBS involves activity modification, massage, stretching, and strengthening in order to help reduce friction as the ITB passes over the femoral condyle. In low-mileage runners, a stretching regimen and NSAIDs may be sufficient to relieve pain, but more competitive or higher-mileage runners will likely require a longer treatment plan. Initially, the runner should avoid any activity that involves repetitive flexion and extension of the knee. They should also use ice and NSAIDs if not contraindicated. In a randomized control trial of 43 runners, the combination of NSAIDS and physiotherapy reduced running pain and increased the distance and time the runners were able to run.

After acute inflammation subsides, a stretching routine focused on the ITB, hip flexors, and plantar flexors should be initiated. Once the runner is able to complete the stretching routine without pain, a strengthening program with specific attention paid to peri-pelvic stabilizers, such as the gluteus medius muscle, can be started. After the runner is able to complete the strengthening exercises pain-free, they may begin a gradual return to running at a slow pace on flat ground. Most are able to progress through the program and return to running within 3–6 weeks.

Anti-inflammatory measures such as oral or topical anti-inflammatories, or corticosteroid injection of the ITB bursa, may also be helpful for short-term control of symptoms so progress with rehab can be achieved. A randomized control trial of 18

runners found a significant reduction in pain while running on the treadmill after corticosteroid injection.

Indications for Surgery

The runner should only be referred for surgical consultation after at least 6 months of failed conservative treatment. They should be advised on the risks of surgery and prepared to complete the rehabilitation protocol.

Operative Treatment

Surgery for ITBS may be performed arthroscopically or via an open approach. Treatments include excision or release of the distal ITB to help loosen or lengthen it as well as to prevent the ITB rubbing against the lateral femoral condyle.

Outcomes

The outcomes for operative management of ITBS are largely positive. In a systematic review of 200 athletes undergoing surgery, between 81 and 100% of patients were able to return to their sport. The mean time to return to sport ranged from 6 weeks to 4 months. In one case series of 36 runners, 74.2% were able to return to running after 2 months, and 100% were able to run 3 months postoperatively.

Popliteal Artery Entrapment Syndrome

Summary of Epidemiology and Definition

Popliteal artery entrapment syndrome (PAES) is a condition in which the popliteal artery is compressed, leading to lower leg pain or paresthesia. Compression is either anatomic, caused by abnormal muscular or tendinous structures compressing the artery, or functional. Functional PAES may be caused by hypertrophy of the gastrocnemius or soleus compressing the artery during leg movement. The overall incidence of PAES in runners is unknown, but the reported incidence in the general population is between 0.6% and 3.5%. In a study of 327 symptomatic athletes presenting to a clinic, 35 were diagnosed with PAES, of which 83% were male. In a study of 68 popliteal entrapment releases, 70% of patients with anatomic entrapment were sedentary, middle-aged males, whereas 70% of patients with functional entrapment were younger, active females.

Clinical Presentation

The runner presents with an achy, cramp-like pain that occurs after intense exercise. This presentation is often mistaken for exertional compartment syndrome but can be distinguished by the pain location primarily in the calf, and not the anterolateral aspect of the lower leg. PAES also may present with paresthesia in the lower leg and feet, foot swelling, pallor, cramping, blanching, and cold feet.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis includes chronic exertional compartment syndrome, popliteal artery aneurysm, and neurogenic claudication.

When PAES is suspected, the clinician should first analyze the ankle-brachial index (ratio of the blood pressure in the ankle to that of the upper arm) at rest and then after exercise. A normal ankle-branchial index is between 1 and 1.4; when the ratio decreases between 30 and 50% with the recreation of symptoms after exercise, the test is considered positive. Positive ABI testing should prompt a dynamic duplex ultrasound of the popliteal artery with the patient in active plantar flexion and the knee flexed to 15 degrees. A decrease in the peak flow with this movement is considered a positive result. X-ray should also be performed to rule out any potential osseous causes of arterial compression. Additionally, CT and CT angiography as well as MRI and MR angiography are important to confirm the diagnosis and help in planning operative treatment. Catheter-injected contrast angiography with provocative maneuvers of the affected leg is the preferred modality for preoperative planning.

Non-operative Treatment

There is currently no proven non-operative treatment for PAES. However, in a case series of 27 patients with PAES, a novel treatment using Botox A resulted in a 60% favorable response rate with no complications. This management technique is currently being evaluated in a clinical trial.

Indications for Surgery

Anatomical PAES should be surgically corrected, regardless of symptoms. If left untreated, the disease will progress to full occlusion, with resultant ischemic damage to the lower leg that may ultimately require amputation. In functional PAES, surgery is only indicated when the patient desires symptom relief and is prepared to undergo the rehabilitation process.

Operative Treatment

The specific operative treatment differs for the anatomical and functional versions of PAES. In both cases the treatment involves a surgical myotomy and rerouting of the popliteal artery or offending gastrocnemius insertion. In cases where the popliteal artery entrapment syndrome has gone untreated and vascular injury has occurred, a saphenous vein graft is used for repair or revascularization.

Outcomes

In a retrospective study, 25 limbs on 18 patients were treated with musculotendinous division without arterial reconstruction, interpositions of the damaged popliteal artery, or artery bypass. The 5-year patency for patients overall was 84%. The 5-year patency was 100% for patients that underwent myotomy alone or surgery for lesions restricted to the popliteal artery. In another retrospective study of 29 limbs, 25 required revascularization and musculotendinous division, and the authors reported 1- and 5-year patency rates of 96.3% and 91.9%. Return to play outcomes in athletes have not yet been determined.

Patellofemoral Pain Syndrome

For additional detail on patellofemoral pain syndrome, please see Chap. 28.

Running-Related Injuries of the Lower Leg

Achilles Tendinopathy

Summary of Epidemiology and Definition

Achilles tendinopathy involves chronic pain and degenerative change in the Achilles tendon. In a systematic review including 3500 runners, the incidence of Achilles tendinopathy was 9.1–10.9%, and rates are even higher in elite runners. In an epidemiological study of 125 track athletes, 43% of elite track and field runners (83% middle-distance runners) developed this condition.

Clinical Presentation

Runners present with a history of insidious onset pain, stiffness, and reduced function, especially in the morning or after long periods of sitting. Many report pain to palpation or with running and jumping, primarily during the beginning of the activity. Performance metrics frequently suffer, even prior to the onset of pain. Achilles tendinopathy may be associated with Haglund's deformity, or enlargement of the bony section of the heel at the Achilles insertion, or an os trigonum, an accessory bone behind the talus. Both can cause increased irritation of the Achilles tendon during or after running.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis includes posterior ankle impingement, acute Achilles tendon rupture, soleus muscle injury, and calcaneal bursitis.

Pain upon palpation and subjective reporting of pain are consistent with midportion Achilles tendinopathy. Special tests include the arc sign, the Royal London Hospital test, and the Thompson test.

Arc sign The tendon is palpated to identify the presence of thickened nodules. If thickening is present, the examiner pinches the area while the runner actively dorsiflexes and plantarflexes the ankle. The test is considered positive if the evaluator feels a mobile, thickened nodule.

Royal London Hospital test The examiner pinches the tendon to identify the most symptomatic location while the foot is relaxed. The runner then actively dorsiflexes the foot and the examiner pinches the previously identified location. Reduced pain with palpation while the ankle is dorsiflexed indicates a positive test.

Thompson test This assesses for Achilles tendon rupture. The runner lies prone on the examination table with the knee on the affected side flexed. The examiner gently squeezes the calf and watches for plantar flexion of the foot. If present, the tendon is presumed to be at least partially intact. No movement suggests a full rupture of the Achilles tendon.

Ultrasound is the imaging modality of choice to confirm the diagnosis. The examiner should assess tendon length, thickness, and cross-sectional area for signs of tendinopathy and inflammation.

Non-operative Treatment

First-line therapy for Achilles tendinopathy is rehabilitation designed to strengthen the tendon, decrease pain, and stimulate remodeling. The initial phase should involve symptom management and load reduction, followed by recovery, then

rebuilding, and finally a return to sport. Initially, exercises should feature high loads and slow contractions. Complete rest is not recommended, as it may be detrimental to the runner's quality of life and sporting performance and is not associated with improved outcomes. In a randomized clinical trial of 38 patients, those in the Achilles tendon loading group did not experience any adverse effects from continuing activity during rehab compared to controls.

In addition to exercise programs, treatment with extracorporeal shock wave therapy (ESWT) and platelet-rich plasma (PRP) injections has been proposed. A systematic review of randomized and non-randomized studies assessing ESWT in patients with Achilles tendinopathy found that ESWT is comparable to eccentric training at 4 months, and superior to expectant management, especially for midportion and insertional tendinopathy. In contrast, a double-blind, randomized controlled trial of 54 patients with Achilles tendinopathy, aged 18 to 70 and undergoing an eccentric training program, showed no clinical and ultrasonographic superiority of PRP injection over placebo 1-year post-intervention.

Indications for Surgery

For Achilles tendinopathy, runners who do not respond to 3 to 6 months of conservative treatment may consider alternative operative and non-operative treatment options and should consult with a physiatrist or orthopedic surgeon.

For acute Achilles tendon ruptures, surgery may be indicated, though definitive evidence is lacking. A systemic review including 577 patients with Achilles tendon rupture treated surgically did find that patient could return to work and sport more quickly, which was associated with positive outcomes. However, there was no difference in re-ruptures observed between the two groups, and surgical patients suffered minor, resolvable complications at a higher rate. Surgery may be a better option for younger runners or those seeking to return to activity more quickly. Non-operative treatment may be better suited for older runners content with less robust early functional outcomes and fewer complications.

Operative Treatment

The procedure involves end to end repair of the tendon via a posteromedial approach.

Outcomes

In a randomized, controlled trial of 100 patients undergoing surgery for acute Achilles tear, there was no difference in functional results, physical activity, or quality of life in the operative versus non-operative groups.

Chronic Exertional Compartment Syndrome

Summary of Epidemiology and Definition

Compartment syndrome is a painful muscle injury caused by elevated intramuscular pressure that compromises tissue perfusion. The most common form to afflict runners is chronic exertional compartment syndrome (CECS), and one in seven patients presenting with exercise-induced leg pain is diagnosed with CECS. Of 149 patients who underwent fasciotomy for a diagnosis of CECS in an 18-year period at 1 clinic, 38% participated in track, cross-country, or road racing as their primary sport. The mean age of presentation in athletes is between 26 and 28 years old.

Clinical Presentation

Runners with CECS present with severe pain that localizes to one or more specific compartments of the lower leg during exercise. The pain worsens with increased exercise intensity and duration before quickly improving with rest. The runner will often report associated symptoms including paresthesia, numbness, or even transient foot drop in the affected leg.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis includes medial tibial stress syndrome, stress fracture of the tibia or fibula, deep vein thrombosis, or popliteal artery entrapment.

The physical exam is often nonspecific, but patients may have point tenderness or muscle fascial herniation of the affected compartment. Measurements of intracompartmental pressures with needle manometry are the first-line diagnostic test. An intracompartmental pressure of ≥15 mmHg at rest, ≥30 mmHg 1-minute post-exercise, or ≥20 mmHg 5 minutes post-exercise is a positive test and confirms the diagnosis. Because CECS occurs bilaterally in 70% to 80% of the population, the clinician should consider testing the contralateral limb, even if the runner is asymptomatic. Other tests may be useful to rule out other potential causes of pain including radiography, bone scintigraphy, MRI, and electromyography.

Non-operative Treatment

Non-operative management involves rest, physical therapy, gait retraining, and experimental therapies such as botulinum toxin. Though some modalities seem promising, the literature is still emerging. In one historical cohort study of 75 military members with compartment syndrome, 65% of patients who underwent a conservative training program emphasizing gait retraining were able to return to duty

without surgery, though 28% were eventually referred for surgery. In a case series of ten patients with CECS who underwent a gait retraining intervention over 6 weeks, 70% reported asymptomatic running after 1 year.

Indications for Surgery

Generally, most cases of CECS that are confirmed by needle manometry are referred for surgical intervention.

Operative Treatment

The standard surgical technique is fasciotomy of the affected compartment(s), either via traditional open fasciotomy, endoscopy-assisted compartment release, single minimal incision fasciotomy, percutaneous fasciotomy under local anesthesia, or ultrasound-guided fasciotomy.

Outcomes

Fasciotomy for CECS leads to significant improvement in pain and overall satisfaction in 75–90% of patients. A case series of 43 athletes had a return to sport rate of 84.4% after fasciotomy, though a return to running rate of only 56.3%. Other studies have shown that fasciotomy of all four compartments versus fewer than four compartments leads to significantly greater percentage of subjects (91% versus 66.7%) able to return to desired exercise levels.

Tibial Bone Stress Injuries

Summary of Epidemiology and Definition

In a systematic review including 3500 runners, the incidence of tibial stress fractures was 9.1%. In a cohort study of 211 NCAA track and field and cross-country runners, 34 athletes suffered 61 bone stress injuries of which 31 affected the tibia.

Clinical Presentation

Runners with tibial bone stress injuries often report a recent, significant change in duration, intensity, or frequency of runs without proper recovery. Historical details may include signs of Female Athlete Triad–Relative Energy Deficiency in Sport (RED-S), such as a history of amenorrhea, disordered eating, and low bone density.

Clinicians should note body mass index and body composition and should obtain a history of dietary habits. When evaluating female runners with a tibial bone stress injury, obtaining a menstrual history is appropriate, given the association between abnormal menstrual cycles and increased stress fracture risk.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis includes medial tibial stress syndrome, muscle strain of the posterior tibialis or soleus, posterior tibialis tendinopathy, and acute compartment syndrome.

The most common symptoms are pain on ambulation, focal tenderness along the tibia, and edema at the site of injury. The tuning fork vibratory stress test may be useful for confirming the diagnosis and is positive when a tuning fork produces vibratory pain at the suspected site of fracture. The test has sensitivity of 75% and specificity of 67%.

Imaging may confirm the diagnosis; however, plain radiographs may be negative in the acute phase of the injury. Therefore, MRI is the modality of choice and has a sensitivity of 88% and a specificity of 99%. Positive findings include increased signal in the endosteum, reactive soft tissue, and marrow edema with periosteal reaction.

MRI findings for tibial bone stress injuries may be categorized by severity according to standardized grading scales, such as the Fredericson scale, which also has a predictive value for return to running trajectory. In a cohort study of 211 NCAA track and field and cross-country runners, each one unit increase in grade correlated with an additional 48 days elapsed before return to running was possible. Mean return to sport for grade 4 was 8 months, significantly longer than for lower severity grades.

Non-operative Treatment

If the tibial bone stress injury has not progressed to a full fracture, treatment includes 2 to 4 weeks of weight-bearing restriction and anti-inflammatory measures. More severe injuries require longer healing times of up to 6 to 8 weeks. Most tibial fractures are located in posterior medial cortex and heal with 4 to 8 weeks of rest and low-impact training. NSAIDs and acetaminophen may be used to help manage pain. In more severe stress fractures, bracing or casting for 3 to 12 weeks is recommended. Runners should be encouraged to maintain cardiovascular fitness through biking, swimming, or other non-weight-bearing exercises and can continue to strength train with core and upper body exercises.

Anterior tibial fractures require longer rest periods and are less likely to respond to conservative treatment due to decreased vascular supply. In a study of 11 athlete patients (6 runners) with anterior tibial stress fractures treated conservatively, 4 (36%) required surgical intervention for refractory symptoms lasting 12 to

34 months after injury. At follow-up 60 months after the initial injury, only one patient (9%) was symptom-free.

Indications for Surgery

Surgery should be considered for fractures that fail conservative management, those with delayed union or nonunion, and in runners who desire a rapid return to sport. In particular, anterior fractures have a high rate of failure to return to sport and nonunion and often require surgical treatment.

Operative Treatment

Operative treatments for anterior tibial fracture include compression plating, drilling, and intramedullary nailing. Compression plating includes tension band plating, laminofixation, and open reduction with internal fixation.

Outcomes

Surgical outcomes are generally good. In a systematic review of 115 athletes (including 38 runners and 23 track and field athletes), symptom resolution was achieved in 87.8% of athletes, and 94.7% were able to return to sports. However, there was an overall complication rate of 27.8% and 14.8% underwent a subsequent surgery.

Medial Tibial Stress Syndrome

For additional detail on medial tibial stress syndrome, please see Chap. 21.

Gastrocnemius Strain

For additional detail on gastrocnemius strain, please see Chap. 21.

Running-Related Injuries of the Foot and Ankle

Peroneal Tendinopathy

Summary of Epidemiology and Definition

Peroneal tendinopathy in runners is most commonly due to overuse, poor running mechanics, or improper shoes. However, it can also be a sequelae of ankle trauma: in a study of 58 runners that had experienced an acute ankle sprain, 55 (95%) had subsequent peroneal tendinosis.

Clinical Presentation

Runners with peroneal tendinosis present with pain and swelling in the lateral ankle or hind foot. The swelling is often posterior to the fibula or along the lateral calcaneus.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis includes ankle sprain, ankle fracture, stress fracture of the proximal fifth metatarsal, and os perineum or os trigonum.

Palpation of the tendons may produce local tenderness. Hind foot cavovarus, an abnormally high arch with the foot turned inward at the heel, is a common concomitant finding: in a retrospective review of 22 patients with peroneus longus tendinopathy, 82% had a cavovarus alignment. Provocative physical exam maneuvers that suggest this diagnosis include eversion against resistance, passive inversion of the ankle, and plantar flexion of the first metatarsal against resistance.

Weight-bearing anteroposterior and lateral radiographs of the ankle and foot are useful to determine if injuries such as pes cavus, stress fractures, or osteophytes are contributing to the symptoms. However, the diagnostic modality of choice is musculoskeletal ultrasound. In study of 60 patients with suspected peroneal tendinopathy, ultrasound had a sensitivity of 100%, a specificity of 85%, and 90% accuracy in diagnosing peroneal tendon pathology. Peroneal tendinosis can also be diagnosed on T2-weighted MRI images, which demonstrate increased signal intensity within the tendon and fluid in the surrounding tendon sheath.

Non-operative Treatment

In mild cases, the treatment plan involves NSAIDs, rest, activity modification, and orthoses with lateral forefoot posting. A rehabilitation program focusing on eccentric strength exercises is also key for recovery of function. In cases that do not

improve after rest and therapeutic exercise, immobilization in a walking boot or short leg cast for 6 weeks may be needed. While use of corticosteroids theoretically poses an increased risk of tendon rupture, a retrospective cohort study of 96 patients (109 injections) found that the complication rate was only 1.8% and 36.8% of patients experienced more than 12 weeks of pain relief. Alternative therapies such as ESWT and PRP injections may also be considered.

Indications for Surgery

Surgical treatment may be indicated in runners that fail to respond to conservative treatment for 6 months or in runners with peroneal subluxation or dislocation.

Operative Treatment

For patients with peroneal tendinosis, the standard procedure is an open synovectomy. For patients with subluxations or dislocations, the mainstay of operative management is direct repair of the superior peroneal retinaculum.

Outcomes

The success rates for surgical treatment of peroneal tendinosis are high, and 70 to 80% of patients return to activity at an average of 12 weeks post-surgery.

Tibialis Posterior Tendinopathy

Summary of Epidemiology and Definition

Tibialis posterior tendinopathy is a cause of medial ankle pain commonly found in runners. In a systematic review including 3500 runners, the incidence of tibialis posterior tendinopathy was 3.6%.

Clinical Presentation

The injury presents as the insidious onset of pain at the affected tendon that worsens with sustained activity. Early on, the pain decreases with an adequate warmup, but as the injury progresses, the pain persists at rest and worsens with further activity. The progression of pain may last months to years before eventually leading to partial or complete rupture.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis includes medial ankle sprain or fracture, plantar fasciitis, flexor hallucis longus tendinopathy, and medial malleolus stress fracture.

A common physical exam finding is the "too many toes sign": the examiner can see more toes on the lateral side of the affected foot when looking from behind compared to the non-affected foot. A flattened arch is also often present, as well as excessive pronation and relative weakness of the tendon. Ultrasound is the imaging modality of choice and should be performed with a high-frequency, linear-array transducer. The exam should include transverse and longitudinal analysis of the retromalleolar area as well as dynamic studies.

Non-operative Treatment

The Alvarez Protocol is commonly used in patients with this condition. Patients are treated with foot orthoses, followed by strengthening exercises for the superficial and deep muscle-tendon complexes of the lower leg, including isokinetic exercises, exercise band therapy, heel raises, and toe walking. The median rehabilitation length with this protocol is 120 days, and it is highly successful, with an 89% satisfaction rate. ESWT is another emerging non-operative treatment. In a study of 94 runners, 6 of 7 runners with tibialis posterior tendinopathy (86%) achieved symptom relief with this treatment.

Indications for Surgery

If conservative treatment fails for 3 to 6 months or significant deformities are present, the runner should be referred for a surgical consultation.

Operative Treatment

Surgical treatment consists of debridement of the tear, repair of the posterior tibial tendon and deltoid ligament, and transfer of the flexor digitorum longus tendon.

Outcomes

In limited studies of athletes, surgical outcomes are positive. In a study of eight professional, collegiate, and high school athletes, all returned to sport after surgery, and seven reported the absence of symptoms at 22-month follow-up.

Metatarsalgia/Metatarsal Bone Stress Injuries

Summary of Epidemiology and Definition

Metatarsalgia is the presence of pain in the forefoot under a metatarsal head and is secondary to other causes of foot pain including trauma, structural causes, or systemic diseases. The overall incidence of metatarsalgia in runners is unknown, though metatarsal bone stress injuries are common in this group. In a study of stress fractures in athletes, 27% of the 73 fractures suffered by long-distance runners and 37.5% of the 8 fractures that occurred in sprinters were metatarsal stress fractures.

Clinical Presentation

Runners with metatarsalgia present with pain that localizes to the forefoot, often described as akin to "walking on pebbles." Other structural deformities, such as stress fractures, metatarsophalangeal instability, and hallux rigidus or valgus, are frequently present. Symptoms may be difficult to differentiate from those of metatarsal bone stress injury; however, the latter is often characterized by chronic, dull, achy pain that limits running. Tenderness to palpation at the location of the suspected bone stress injury is also commonly described.

Differential Diagnosis and Diagnostic Testing

When evaluating metatarsalgia the clinician should rule out traumatic fracture or bone stress injury to the metatarsal as the source of pain.

On a physical exam, signs of first ray deformities, hallux valgus, and medical column (first ray) instability or rigidus may be present, as well as gastrocsoleus complex tightness that may be increasing pressure on the forefoot. Pain with hop testing suggests bone stress injury and should prompt further evaluation with imaging.

Standard radiographs include AP, lateral, and oblique views, but may not identify bone stress injuries in early stages. In the setting of a negative plain film and a high index of suspicion for metatarsal bone stress injury in a weight-bearing athlete, MRI is the modality of choice for confirming the diagnosis. Positive findings include hypointense fracture lines on T1 and bone marrow edema on fluid-sensitive images.

Non-operative Treatment

Metatarsalgia is treated conservatively and resolves with identification and management of the underlying primary cause. Physical therapy focuses on lengthening of the calf muscles to reduce forefront loading. Commercially available toe sleeves or

toe stretchers such as Yoga toes may be used to relieve pain. Contrast baths may also provide symptom relief and have been shown to reduce acute edema and inflammation in the metatarsals.

If a metatarsal bone stress injury is identified, the initial treatment includes activity modification with nonimpact cross-training, as well as immobilization in a short leg walking boot for 4 weeks. Once pain resolves, patients can wean out of the boot and gradually return to activity. The runner may consider orthotic inserts to address biomechanical abnormalities and should maintain stretching programs focused on the calf and Achilles to reduce stress on the forefoot. Metatarsal stress fractures generally heal after 6–8 weeks of conservative treatment, but a gradual approach to return to running is recommended.

Indications for Surgery

Patients with metatarsalgia are rarely referred for surgery, and the mainstay of treatment involves determining the underlying cause while considering anatomical and biomechanical factors. For displaced metatarsal stress fractures, or nonunion fractures that have failed conservative management, surgical referral is indicated.

Operative Treatment

Surgeries for underlying causes of metatarsalgia include gastrocnemius release, distal metatarsal osteotomies, and metatarsal shaft osteotomies. Surgical treatment for metatarsal stress fractures involves dorsal plate fixation and autogenous bone graft from the calcaneus, tibial metaphysis, or iliac crest.

Outcomes

The surgical treatment and outcomes vary significantly and depend on the location of the fracture and the cause of injury. In a study of 42 elite athletes with stress fractures of the fifth metatarsal, a high-risk fracture, all were able to return to their pre-injury level of sport after surgery with modified tension band wiring. Four patients suffered refracture but were able to return to play after conservative treatment for an average of 12 weeks. Definitive outcomes for metatarsalgia are less clear and depend on the primary source of pain and elected surgical procedure.

Plantar Fasciitis

For additional detail on plantar fasciitis, please see Chap. 33.

Return to Running

Return to running after an injury can be difficult for runners, especially when the deadline of a goal event is imposed on the recovery timeline. Runners will wish to return to running as soon as possible and will have anxiety about missed training time and the effect on performance or their ability to complete their goal race. Brief clinical assessments to help guide return to run decision-making are in development. However, the predictive value of successful return to running based on these assessment scores is currently unknown. In the absence of validated tools to assess readiness to run, it is reasonable to trial a "test run," of 10 to 20 minutes of easy running once the runner is pain-free with activities of daily life and with regular walking. Successful completion of a test run without exacerbation of pain is often a good indicator that the runner can return to training. If the rest period from running has not been prolonged (i.e., less than 2 weeks) and the runner has maintained fitness with cross-training, they will likely be able to resume their training plan as if uninterrupted.

Longer duration rest periods require a more graduated return to running protocol in order to avoid reinjury. A 10- to 20-minute test run may be too great an initial trial in this case; an alternative is to try 30–60 seconds of hopping on the affected lower extremity. If pain-free, the runner can be cleared to begin a return to running protocol for several weeks prior to resuming full training. Published protocols are available in the literature and emphasize a gradual increase in volume and intensity and inclusion of nonimpact activity days. We favor the use of walk-run intervals; Table 22.2 illustrates our approach. Prior to initiating intervals of 15 minutes or longer, we challenge the runner with a greater cumulative running time of 36 minutes to ensure they can tolerate increasing amounts of continuous running.

Runners who are used to a high training volume may be resistant to embarking on a walk-run program, even for several weeks. Thus, it is important to emphasize the need to recondition the injured site gradually and to point out that there may be a disconnect between the runner's perception of training load and the actual stimulus to the musculoskeletal system. Conventional recommendations to increase weekly running mileage no more than 10% per week have not been shown to reduce running-related injuries in novice runners; however, they may still provide a safe guideline to prevent runners from progressing too quickly and suffering reinjury. It is also critical to manage expectations about the recovery trajectory in order to avoid disappointment and discouragement. Setbacks are common, and should be expected, as the runner regains fitness.

Running Injury Prevention

Medical providers must understand the process by which runners gain fitness in order to guide them on injury prevention strategies. Run training can be manipulated by changing how much distance a runner trains (volume), how hard a runner

Table 22.2 Return to running progression

Run #	Run interval (min)	Walk interval (min)	Repetitions
1–3	1	1	10
4	2	1	5
5	3	1	4
6	3	1	5
7	4	1	5
8	4	1	6
9	6	1	4
10	8	1	3
11	8	1	4
12	10	1	3
13	12	1	3
14	15	1	2
15	10, then 20	1	1
16	30	0	1

Instructions:
5 minutes of walking pre- and post-run are recommended
Runs should be done at an easy, conversational pace
Do not advance to the next run until you can tolerate a run without symptoms
If a run causes symptoms, rest or cross-train until pain-free and then repeat that run

trains (intensity, e.g., speed or hill workouts), or how often a runner trains (frequency). Whenever these training variables are increased, the runner's body is stressed beyond what it is accustomed to and requires recovery. During recovery, a process called supercompensation occurs; the healing process from the training stress strengthens the runner, allowing the runner to tolerate more stress the next time he or she trains. Over time, this leads to increased fitness if the runner recovers adequately between training bouts.

Failure to allow an adequate recovery period can lead to tissue breakdown and overuse injuries. A runner can adjust the volume, intensity, and frequency of their training to offset this process; thus, medical providers should ask about these components of the training program. For the runner with overuse injuries, providers can suggest that pain signals should be interpreted as potential signs of tissue mechanical fatigue, should prompt a re-evaluation of the training plan, and should warrant consideration of increasing recovery intervals. In the early stages of the injury, including these preventive measures can prevent the injury from progressing to a point where it will require a prolonged rest period from the training plan.

Medical providers can also suggest complimentary training modalities, such as strength training, to augment tissue capacity, enhance runner preparedness, and achieve injury risk reduction. A recent systematic review and meta-analysis of randomized trials of strength training-based sports injury prevention found that strength training programs reduced sports injuries in military recruits and soccer athletes by an average of 66%. This included a reduction in overuse anterior knee pain, which also commonly afflicts recreational marathoners, by 75%. This reduction was

attributed not only to the direct effect of strengthening the tissues but also to improved coordination and enhanced technique.

Finally, encouraging runners to cultivate a flexible mindset is critical to injury avoidance. Many recreational runners become wedded to the prescriptions of their training plan. The idea of missing a run can provoke anxiety, and many will "run through" pain signals in order to remain on their schedule. Reassurance that minor deviations from the training plan to prioritize healthy, pain-free running will not materially affect their fitness can help runners feel comfortable making necessary training modifications to avoid injury. Providers can help those runners who sustain injuries that prevent them from participating in their goal event focus on lessons learned from the injury. Addressing training errors and biomechanical factors that led to an injury not only reduces future injury risk but also usually improves future performance and thus may be viewed as an opportunity to improve as an athlete. Runners can be reminded of this and encouraged to adopt a long-term view of their training. Providers can use the example of elite-level athletes, whose resilience has been shown to contribute to their athletic success and well-being, to further demonstrate how adaptability can enhance goal achievement in sport.

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Chapter 23
Orthobiologics

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Introduction

Osteoarthritis (OA) and chronic tendinopathy are among the most common musculoskeletal diagnoses. OA is a leading cause of disability, especially in the ageing and obese populations, affecting an estimated 27 million adults in the United States. Chronic tendinopathy has been reported to affect anywhere between 1 and 3% of adults annually, and the prevalence of OA may be even higher. The socioeconomic costs of degenerative and tendinopathic conditions include both the impact on an individual's activities of daily living and the direct costs of treatments and the indirect costs of work productivity loss.

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Traditional Treatment Paradigm for Chronic Musculoskeletal Injuries

Traditional paradigms for treatment of common musculoskeletal complaints include nonoperative management such as rest, activity modification, weight loss, and physical or occupational therapy. Additional nonoperative options include analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), and injections including corticosteroids and hyaluronic acids. Side effects, such as gastrointestinal or renal effects of NSAIDs and the chondrotoxicity and tenotoxicity of corticosteroids, may inhibit the long-term use of these agents. As our understanding of tendon and joint degeneration evolves, so should our treatment paradigm. Historically, if a person did not improve with conservative management, a surgical intervention was often the next and only additional option. For numerous reasons however, patients may find themselves without treatment options, in a gap within this traditional paradigm, as they have failed typical nonoperative treatments but are not ready for surgical intervention. These patients may include those who are not yet surgical candidates, such as young patient with mild or moderate OA, those who are not medically optimized for surgery, or those who delay or refuse surgical interventions. This has led to a shift in the traditional treatment paradigm and the need for additional, effective nonoperative management options. Orthobiologics, as a category of substances that can be used to promote healing, are an additional option in the nonoperative treatment paradigm for refractory musculoskeletal injuries.

Pathogenesis of Pain in Musculoskeletal Injuries

Osteoarthritis is often described as a painful condition consisting of joint injury with an inflammatory component, which leads to articular cartilage degradation and pain. Pain generators include both the central factors such as depression, sleep deprivation, and catastrophizing, as well as the peripheral nervous system. In an osteoarthritic joint, a rich innervation of free nerve endings and inflammatory cytokines are found at increased levels within the joint capsule, synovium, tendons, ligaments, retinacula, fat pads, periosteum, and subchondral bone. Chronic tendinopathy is often described as a predominantly degenerative condition often secondary to an overuse injury, which results in a disorganization of tendon architecture. Tendinopathy may occur within the main tendon substance or at the insertional enthesis. Pain generators for tendinopathy include similar central and peripheral factors.

Common Orthobiologic Agents

The following sections will review four frequently used orthobiologic techniques: prolotherapy, platelet-rich plasma (PRP), bone marrow aspirate concentrate (BMAC), and adipose tissue derivatives (ATDs).While the precise mechanisms of action are not known for most orthobiologics, proposed mechanisms of action are suggested below based on basic science and animal study research. Table 23.1 summarizes generalized orthobiologic uses, contraindications, and pros and cons of each.

Prolotherapy

Mechanism of Action

Prolotherapy, also known as proliferative therapy, is a low-volume irritant solution, most commonly composed of a hypertonic dextrose solution. Prolotherapy has been proposed to repair injured tissue by an indirect stimulation of growth factor release. Some proposed growth factors involved in tissue repair include platelet-derived growth factor, epidermal growth factor, basic fibroblast growth factor, insulin-like growth factor, and connect tissue growth factor. Prolotherapy has also been proposed to create a temporary state of inflammation via an osmotic effect on local cells at higher concentration. These proposed mechanisms are theorized to trigger a healing response. Additionally, limited research suggests prolotherapy may also have a direct neuromodulatory analgesic effect that alters nerve excitability and downregulates pain receptors.

Authors' Preferred Technique

The authors prefer the use of dextrose solution for a prolotherapy injection. Typically, 50% dextrose is mixed with equal parts of normal saline and a local anesthetic, and dextrose concentrations may vary between 10 and 25%. Lower concentrations are more commonly used to perform perineural injections given its proposed neuromodulatory effect, or for an initial injection dose in a prolotherapy-naive patient. Slightly higher concentrations are often utilized for soft tissue application. The highest concentrations are often used for intra-articular application. Concentration adjustments may be made at subsequent visits, based on a patient's response to previous concentrations.

Table 23.1 Overview of orthobiologics including main uses, contraindications, and pros and cons

Orthobiologic	Main uses	Contraindications	Pros	Cons
Prolotherapy	OA: knee, CMC Tendinopathy: Osgood-Schlatter, lateral epicondylopathy, rotator cuff, Achilles, patellar tendon Others: temporomandibular joint, sacroiliac joint, axial back pain, myofascial pain	*Absolute* Acute cellulitis Local abscess Septic arthritis *Relative* Brittle diabetes Acute gouty arthritis Acute fracture	Use "in-season" Continue home exercise Alter concentration Treat large areas Few side effects/adverse events Continue anticoagulation use High-level evidence	Multiple visits Gradual effects
Platelet-rich plasma (PRP)	OA: knee, hip, ankle Tendinopathy: lateral epicondylopathy, patellar tendon, proximal hamstring, rotator cuff Others: plantar fasciitis, ankle OCD	*Absolute* Blood dyscrasias Septicemia Local infection *Relative* Antibiotic use Anticoagulant use Immunosuppressant use Severe anemia Active malignancy	Easy to harvest sample Few side effects/adverse events May continue anticoagulation use High-level evidence Cheaper than cell-based therapies	No standardized dosing Variability in quantity and quality
Cell-based therapies				
Bone marrow aspirate concentrate (BMAC)	OA: knee, glenohumeral Tendinopathy: rotator cuff, patellar tendon, medial and lateral epicondylopathy Others: osteonecrosis of femoral head, osteochondral lesions, bone nonunion	*Absolute* Active malignancy Local infection (harvest or procedure site) *Relative* Anticoagulant use Severe osteoporosis	Few side effects/adverse events May be used on tendon tears or advanced injuries	Most expensive Potential pain at harvest site Variability in quantity and quality
Adipose tissue derivatives (ATDs)	OA: knee Tendinopathy: Achilles, rotator cuff Others: talus osteochondral lesions, patella chondromalacia, meniscus tears			

Clinical Evidence for Use

There is abundant high-level evidence supporting the use of prolotherapy in knee osteoarthritis. Additionally, there are a few high-level studies that support the use of prolotherapy in carpometacarpal OA. In chronic tendinopathy and enthesopathy, high-level evidence exists in support of the use of prolotherapy in Osgood-Schlatter disease, lateral epicondylopathy, and rotator cuff tendinopathy. Emerging evidence exists for the use of prolotherapy in temporomandibular joint laxity and pain. Mixed or lower-level evidence exists for the use of prolotherapy for sacroiliac joint pain and ligamentous laxity, axial back and myofascial pain, as well as Achilles and patellar tendinopathy.

Contraindications, Side Effects, and Adverse Events

There are few absolute contraindications for prolotherapy use. These include acute cellulitis, local abscess, and acute septic arthritis. Relative contraindications to prolotherapy include patients with brittle diabetes mellitus, acute gouty arthritis, and acute fractures. The most common side effects reported in the literature include pain, often described as a sense of fullness or numbness. A postinjection pain flare may occur and is typically self-limited. Injection reactions, such as allergic reaction, infection, and bleeding, are rare but possible adverse events.

When to Refer

Formal training in prolotherapy techniques may be taught during residency or, more commonly, during a sports medicine fellowship. Prolotherapy should be considered in patients with recalcitrant musculoskeletal complaints who have failed conservative management or are nonsurgical candidates or in those who require a large area of treatment as both intra-articular and surrounding soft tissue structures can be targeted within the same visit. Prolotherapy may be considered especially advantageous for “in-season” athletes or those who do not wish to pause their daily exercise routine. It is often well tolerated, especially at low concentrations; therefore it may be desirable for patients with significant hyperalgesia and centralized pain. Management of expectations is an important part of a prolotherapy referral consultation as symptomatic improvement generally occurs gradually over months and after numerous sessions.

Platelet-Rich Plasma

Mechanism of Action

Platelet-rich plasma (PRP) refers to autologous human plasma with a supraphysiologic platelet concentration. Platelets' role in wound healing extends far beyond simple hemostasis, as platelets house several anabolic growth factors and immunologic mediators influencing inflammation, cellular proliferation, tissue remodeling, and angiogenesis within alpha-granules. Though the mechanism of PRP has not been determined, animal and human studies implicate platelet-derived growth factor, epidermal growth factor, basic fibroblast growth factor, insulin-like growth factor, transforming growth factor beta, and vascular endothelial growth factor as key anabolic mediators. Additionally, PRP has been suggested to dampen pathologic inflammation associated with catabolic matrix metalloproteinases. Overall, PRP is an immunomodulatory and anabolic therapy which may augment healing, particularly in tissues with low endogenous healing potential.

Though the first documented use of PRP dates to the 1980s, preparation techniques for musculoskeletal applications have not been standardized, and variability within PRP composition is noted within the literature. A PLRA (platelet count, leukocyte content, red blood cell content, and activation) classification system has been proposed to standardize documentation and compare composition of PRP in clinical studies. Leukocytes, in particular, have been shown to be proinflammatory. Research suggests leukocytes may have deleterious effects on chondrocytes but may stimulate the repair process in other tissue which is a beneficial response. Leukocyte-poor PRP (LP-PRP) has been suggested to be beneficial for intra-articular injections, while leukocyte-rich PRP (LR-PRP) may be more advantageous for tendinopathy use.

Author's Preferred Technique

Variation exists in the preparation and collection protocols for PRP based on the commercial system used and manufacturer's parameters and reagents. Systems may differ in their efficacy of platelet capture, the centrifuge speed and technique (one- or two-step centrifugation), and the collection system.

In the authors' preferred technique using a commercially available system, PRP preparation begins with the collection of 60–120 ml of venous whole blood, drawn with an anticoagulant such as sodium citrate. This autologous, anticoagulated whole blood is then centrifuged for 10–20 minutes. From this centrifugation, three distinct layers form: a platelet-poor plasma (PPP) fraction on top, a middle "buffy coat" fraction (consisting of platelets and leukocytes), and a red blood cell fraction on the bottom which is discarded. PPP and buffy coats are then combined and the resultant PRP is collected from the system.

A final PRP volume may be between 3 and 10 ml depending on multiple factors, with a reported three- to fivefold increase in platelet concentration compared to whole blood. During PRP preparation, an acid buffer such as sodium bicarbonate and/or a platelet activator such as bovine thrombin or calcium chloride may be added. Acid buffers have been suggested to improve platelet viability and reduce injection pain, while platelet activating agents are proposed to maximize degranulation of the aforementioned mediators.

Following the preparation phase, the patient's skin is sterilized, and a local anesthetic is applied as appropriate. Ultrasound under sterile technique is often useful for needle guidance and placement during PRP injection. The injection site is subsequently covered with a bandage.

Clinical Evidence for Use

A meta-analysis of aggregated high-level evidence demonstrates PRP may lead to superior pain relief lasting from 3 months to 1 year. Intra-articularly, numerous studies have shown the benefit of PRP in knee osteoarthritis, with improved quality of life and pain reduction. Lower-level evidence also exists for the use of PRP for ankle osteochondral defects as an adjunct to surgery. Discordant results were found for the treatment of hip OA with PRP when compared with hyaluronic acid injections, as well as ankle osteoarthritis, with only short-term benefits seen. Abundant high-level evidence exists for the use of PRP for lateral epicondylopathy with superior pain control from 6 months to up to 2 years when compared to the use of whole blood, prolotherapy, or corticosteroids. There is weaker, but promising, evidence to support the use of PRP in patellar tendinopathy, proximal hamstring tendinopathy, rotator cuff tendinopathy, plantar fasciitis, and donor site pain in patellar tendon graft bone-patellar tendon-bone autograft anterior cruciate ligament reconstructions. In general, there is limited and conflicting data reviewing the efficacy of single versus multiple PRP injections. Furthermore, while there are recommendations for PRP preparation and classification, these parameters have not been thoroughly investigated for clinical optimization.

Contraindications, Side Effects, and Adverse Events

Absolute contraindications include blood dyscrasias with platelet dysfunction, fever, overlying cutaneous or joint infection, and septicemia. Relative contraindications include antibiotic use, use of anticoagulants or systemic immunosuppressants, severe anemia, and malignancy. PRP complications are rare and typically relate to those associated with standard injections, namely, postinjection pain, infection, allergic reactions, skin discoloration, and blood clots. PRP, especially when injected directly into tendons, can be associated with increased postinjection pain. LR-PRP

may be associated with more pain and swelling due to its relative proinflammatory nature. Less commonly, adhesive capsulitis or tendon rupture, in the case of an intra-tendinous injection, has been reported.

When to Refer

Patients may be referred for PRP at any time; however most commonly they are referred after failing several conservative modalities due to its out-of-pocket expense. PRP may be considered especially advantageous for "out-of-season" athletes or those who are willing to modify their daily exercise routine for several weeks. A formal consultation with the treating provider is recommended prior to the planned procedure. This allows for adequate discussion regarding clinical diagnosis, treatment costs, procedure-day specifics, and the clinical evidence regarding PRP use with their specific aliment. The provider will also discuss pre-procedural protocol which may include the discontinuation of corticosteroids, NSAIDs, and anticoagulation, pre-procedure physical therapy in order to obtain a post-procedure rehabilitation protocol, and expectation management including that results from PRP, similar to prolotherapy, may be seen gradually over months. A thoughtful discussion should occur regarding the provider's recommended return to play protocols, particularly in athletes, given post-PRP activity restriction and physical therapy commitments may last for 6–12 weeks.

Cell-Based Therapies

Bone marrow aspirate concentrate (BMAC) and adipose tissue derivatives (ATDs) are two commonly used cell-based therapies for musculoskeletal complaints. In the United States, bone marrow-derived signaling cells are non-cultured cells, used directly after concentrating the cells from initial aspirate by centrifugation. Cultured cells, cells that undergo a multistep in vitro cell-line expansion, are currently prohibited for use to treatment musculoskeletal complaints under the US Food and Drug Administration (FDA). These cell-line expansion techniques are offered outside of the United States; however with fewer regulatory controls also come the increased safety concerns including the maintenance of sterility of in vitro expansion and the clearance of cytokines used for culture expansion, as well as the uncertainty of genetic stability of the culture-expanded cells.

Bone Marrow Aspirate Concentrate (BMAC)

Mechanism of Action

The precise mechanism of action of BMAC responsible for its clinical effects has not been clearly established within the literature. It has proposed that through a paracrine effect, bone marrow-derived mesenchymal signaling cells promote growth factors and cytokines to locally recruit neighboring cells to stimulate tissue repair. Research suggests BMAC also has anti-inflammatory, immunomodulatory, pro-angiogenic, anti-apoptotic, anti-fibrotic, and wound healing properties. Numerous animal models have shown improvement in histologic tendon properties with the use of BMAC.

Authors' Preferred Technique

Typically, a bone marrow aspirate is harvested in a clinic or same-day procedure suite setting, using a commercially available kit. Bone marrow aspirate may be obtained via the posterior superior iliac spine (PSIS) or anterior superior iliac spine. The patient is typically awake with local anesthesia or under conscious sedation or general anesthesia depending on the setting. The patient is positioned in the supine or prone position, depending on the site of harvest.

The author's preferred method for bone marrow harvest is with the patient in the prone position for access to the PSIS. The bony landmarks in this region are palpated and ultrasound or fluoroscopic guidance is often used to confirm location. The procedural site is then sterilely prepared including the use of sterile drapes to ensure an adequate sterile field. With the help of an assistant, the BMAC aspiration kit is then opened and pre-heparinized to avoid coagulation, and the battery-powered drill is sterilely covered. Under sterile technique, ample local anesthetic is injected down to the PSIS periosteum. Once relative anesthesia is obtained, a larger gauge needle is used to create a percutaneous insertion site. A blunt trocar is then manually inserted perpendicular to the PSIS, and once it has reached the periosteum, the drill is used to assist the trocar into the medullary cavity of the PSIS (Fig. 23.1). A heparinized sterile syringe is then attached, and bone marrow is manually aspirated (Fig. 23.2). Stasis is achieved with manual pressure and a sterile dressing at the harvest site. Once the bone marrow aspirate is obtained, it is processed via a density centrifuge machine, into a concentrated product described to contain mesenchymal and hematopoietic signaling cells, platelets, and cytokines. BMAC is then injected into the specific target site, often with ultrasound guidance.

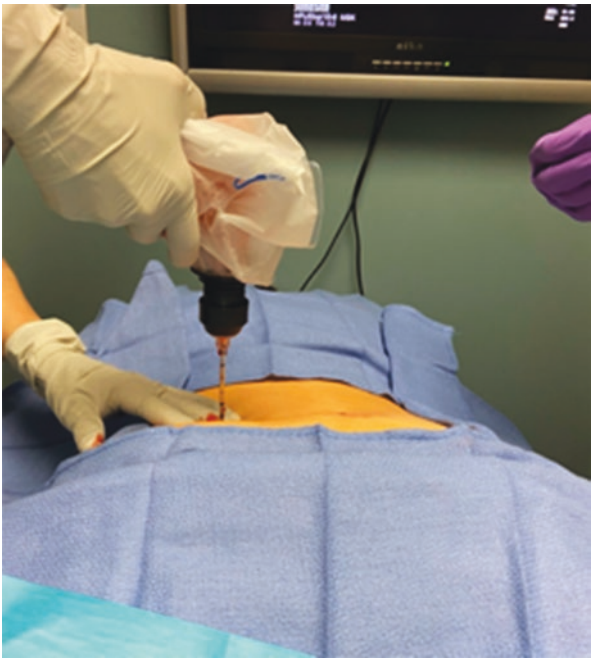

Fig. 23.1 Battery-powered drill assisting the trocar into the medullary cavity at the left posterior superior iliac spine

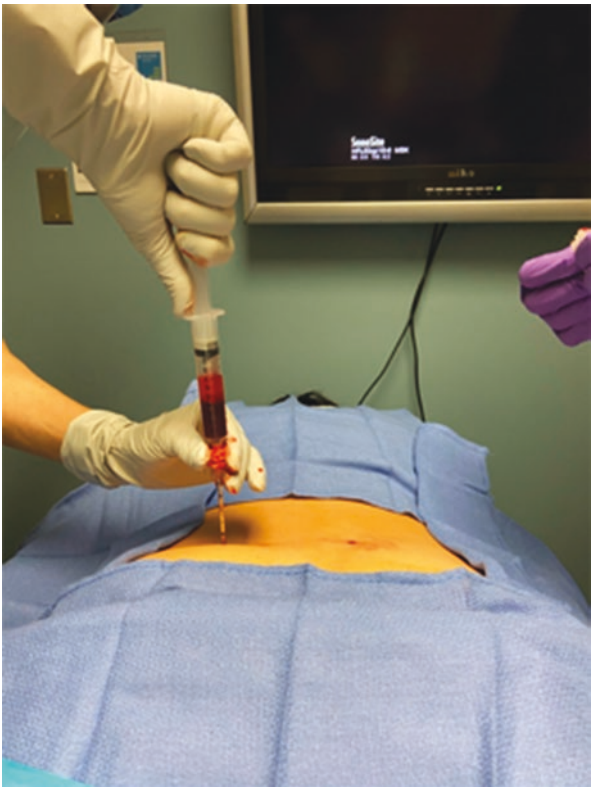

Fig. 23.2 Bone marrow aspiration of the left posterior superior iliac spine medullary cavity via a patient in a prone position

Clinical Evidence for Use

The majority of BMAC research thus far has been seen in the surgical literature, often as an adjuvant to surgery, with or without the additional use of PRP. Research of BMAC use in osteonecrosis of the femoral head has been mixed; however its use may decrease the need for hip arthroplasty. The bulk of BMAC research currently is through case series and non-randomized trials. Evidence is promising for the use of BMAC in intra-articular and osseous conditions including glenohumeral and knee OA, osteochondral lesions, and bone nonunions. Research, although limited, suggests BMAC may be beneficial in tendinopathy as well, including rotator cuff tendinopathy, patellar tendinopathy, and medial and lateral epicondylopathy.

Adipose Tissue Derivatives

Mechanism of Action

Adipose tissue derivatives (ATDs) refer to stromal vascular fractions (SVF) and micro-fragmented adipose tissue (MFAT). SVF consists of washing the adipose tissue and enzymatically digesting the extracellular matrix of the fat cell population. This process of altering adipose-derived tissue to create SVF is considered a manipulation of tissue, which is not currently approved for musculoskeletal use in the United States by the FDA. Micro-fragmented adipose tissue (MFAT), on the other hand, is a mechanical process of minimal manipulation. ATDs, as cell products, are clinical preparations proposed to contain adipose-derived mesenchymal stem cells (ASCs) and other regeneration-supportive cells including perivascular smooth muscle cells, endothelial cells, pericytes, fibroblasts, and immune cells. Research suggests that ASCs may differentiate into the damaged tissue in which they infiltrate, although this has not yet been confirmed. It has been hypothesized that ASCs also release growth factors to induce cellular proliferation, vascularization, and restructuring of the extracellular matrix and limit pathologic inflammation associated with excess fibrosis and cell death. Finally, the resultant cytokine cascade may recruit additional signaling cells from the bone marrow, although additional research is needed to confirm these findings.

Authors' Preferred Technique

A commercially available kit is used to harvest and process lipoaspirate to obtain an micro-fragmented adipose tissue (MFAT) product for same-day injection. Adipose tissue can be obtained from the abdomen, flank, peri-gluteal region, or thighs using a minimally invasive technique. Anesthetic is typically applied locally, and patient positioning depends on the harvest site. Lipoaspiration is performed using standard sterile technique involving a blunt, small-diameter cannula to minimize pain and blood vessel damage.

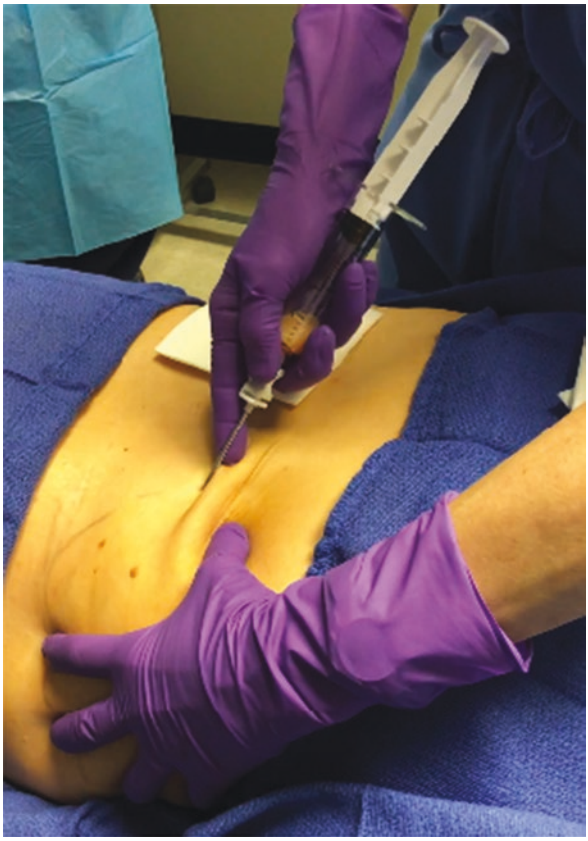

Fig. 23.3 Lipoaspiration from posterior flank with patient in prone position. Note harvested fat aspirate within the syringe

The authors' preferred harvest site in males is the posterior flank in the prone position. For females, we prefer to harvest adipose from the lateral thigh and perigluteal region in the side lying or prone position. Once the harvest site is determined, a large site is sterilely prepared including the use of sterile drapes to ensure an adequate sterile field. A lipoaspiration is then performed in the designated region under local anesthesia to obtain an adequate sample (Fig. 23.3). Stasis is achieved at the harvest site and a compressive bandage is placed over the area.

The adipose tissue sample is then processed through a closed-system device containing saline, where it undergoes a series of washing, filtering, and mechanical agitation, allowing for the release of oils and blood products from the desired cells. After lipoaspirate processing, the final product is obtained and injected into the site of interest, usually with ultrasound guidance.

Clinical Evidence for Use

Only one high-level study on adipose tissue derivatives has been published, which compared adipose to PRP in recalcitrant Achilles tendinopathy and suggested early improvement in pain, function, and activity. Beyond this study, human ATD literature is limited to case series and case reports for various osteoarthritic joints (most commonly knee), talus osteochondral lesions, patella chondromalacia, rotator cuff tears, and meniscus tears. The interpretation of data is confounded by combination with other injectable modalities such as PRP or hyaluronic acids, varying preparation kits, and poorly described postinjection rehabilitation protocols. Nonetheless, the existing literature has suggested ATD may improve pain and function without significant adverse events.

Cell-Based Therapies Overall

Contraindications, Side Effects, and Adverse Events

Contraindications for the use of BMAC and ATD include active cancer, or an acute, local infection at the harvest and/or injection site. Relative contraindications may include the use of anticoagulants and severe osteoporosis due to the increased risk of fracture specifically with a BMAC procedure. The most common side effects reported in the literature include self-limited pain, both at the harvest and injection site, as well as self-limited joint swelling.

When to Refer

Formal training in BMAC and fat-aspiration techniques are most commonly taught through fellowships or training courses. A consultation with a sports medicine physician who performs BMAC and/or ATD should occur prior to the procedure. A thorough discussion regarding rehabilitation protocols should also be discussed as the patients will require a pause in their physical activity with a slow return to activity, similar to PRP. Understanding the varying out-of-pocket expenses, the use of adjuvant orthobiologics including PRP, and management of expectations are an important part of a cell-based therapy consultation.

A good candidate may include patients with recalcitrant musculoskeletal complaints who have failed conservative management or those who want to prolong or forgo a surgical consultation. These patients should understand the relative

limitations regarding efficacy within clinical research. Younger patients may have an improved response with BMAC compared to an elderly population which may have decreased bone marrow quality. Adipose tissue has been shown to maintain stromal cell quantity and quality over time and may be more beneficial to our elderly patient population. Patients are often happy to donate their fat.

Educational Pearls

Optimizing a Patient for an Orthobiologic Referral

As a primary care provider, your role in an orthobiologic referral is vital. A patient should be medically stable to participate in exercise training. Reinforcing a lifestyle that optimizes medical and metabolic function, including smoking cessation, adequate sleep, optimizing nutrition and BMI, and dedication to exercise, is key. A conversation regarding the importance of active participation in rehabilitation should be initiated by the PCP to ensure the patient is willing to participate in therapy. It is important to have a brief conversation with your patient regarding the concept of orthobiologics and how additional orthobiologics may be offered offshore but often lack an equivalent regulatory body such as the FDA.[36] Patients should be warned of scams or false claims by those using terms such as "stem cells" or those who make claims of tissue regeneration, as this has yet to be verified in research. Expectations for improvement in patients with severe, end-stage disease, such as osteoarthritis with restricted range of motion, must be tempered as their response to any orthobiologic may be limited at best.

Proximity to a provider and a patient's willingness to travel, especially for prolotherapy which requires a series of multiple monthly visits, should be taken into consideration prior to referral. Each prolotherapy procedure, however, is often relatively quick. PRP, BMAC, and ATDs, on the other hand, are often performed via a single injection. PRP appointments typically take around 30 minutes, while BMAC and ATD procedures may take several hours depending on the form of tissue acquisition.

Setting Expectations: A Specialist's Role

One of the main goals of an initial consultation on orthobiologics is to set realistic expectations for any orthobiologic procedure. During this consultation, it is imperative that the provider acknowledge that true "regeneration" has not yet been established in high-level evidence-based research and that the exact mechanism of action of these therapies is still yet to be determined. The conversation should also include the risks and benefits of any procedure and highlight that evidence supports a good safety profile on all of the aforementioned procedures.

Expectations need to be established regarding the amount of time expected for symptom improvement to manifest. It is not uncommon for results to be seen for several weeks to months after a procedure, which is a delayed paradigm when compared to the short-onset injections readily available such as corticosteroids and hyaluronic acids. The discussion should also include an understanding of the importance of active participation in rehabilitation and exercise in order to optimize the functional gains. Lastly, a transparent discussion should occur between the orthobiologic specialist and the patient regarding out-of-pocket expenses, which can range from a few hundred dollars to several thousands of dollars per treatment.

Conclusion

Multiple orthobiologic injectables exist for a plethora of musculoskeletal complaints and offer additional nonsurgical options for osteoarthritis and chronic tendinopathy. The main take-home points have been synthesized in Table 23.2. In general, these procedures are well tolerated with a high safety profile; however they are an out-of-pocket expense. Orthobiologics may take weeks to demonstrate their full effect, yet research suggests they may be more durable and less chondrotoxic and tenotoxic than corticosteroids. Patients with recalcitrant musculoskeletal complaints or who are not surgical candidates should be referred to a sports medicine physician with a special expertise and training in orthobiologics. The consultation should include procedure options, risks and benefits of each, and the most up-to-date literature recommendations. Caution must be made when referring to practitioners that make false claims regarding "tissue regeneration" or "stem cell" use. Referral to a reliable physician, ideally one who also uses image guidance for enhanced accuracy during the procedure, is best. Limited high-level research exists on the efficacy of orthobiologics; however promising early studies and interest throughout multiple fields, including sports medicine, PM&R, orthopedics, rheumatology, and pain management, will hopefully lead to additional research in this ever-evolving field.

Table 23.2 Take-home points for PCP regarding orthobiologics

Orthobiologics: take-home points	
Ideal musculoskeletal complaints	Mild and moderate osteoarthritis who have failed conservative management Chronic joint pain or ligamentous laxity in nonsurgical candidates Chronic tendinopathies without rupture Those who wish to delay or are not candidates for surgical intervention
Ideal candidates for referral	Medically optimized by PCP Willing to actively participate in the rehabilitation process Agreeable to out-of-pocket expenses
Ideal orthobiologic specialists	Training in orthobiologic use and image guidance Up-to-date on current literature Do not make false claims regarding orthobiologics' ability to "regenerate tissue" or the use of "stem cells"

Suggested Reading

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Part VII
The Knee

Chapter 24
Knee Osteoarthritis

Jeffrey N. Katz, Kaetlyn R. Arant, and Thomas S. Thornhill

Abbreviations

APM	Arthroscopic partial meniscectomy
COX	Cyclooxygenase
MRI	Magnetic resonance imaging
NSAID	Nonsteroidal anti-inflammatory drug
OA	Osteoarthritis

Introduction

Osteoarthritis of the knee is characterized by pain, functional loss, and damage to cartilage, bone, meniscus, and other structures (Fig. 24.1).

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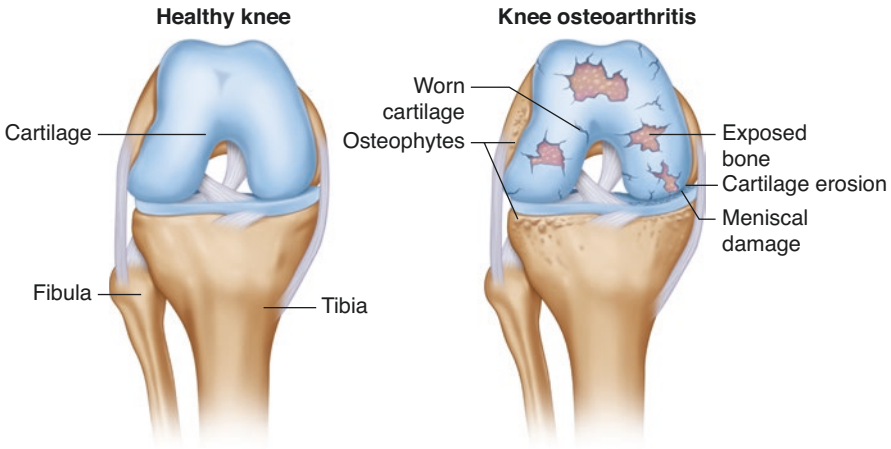

Fig. 24.1 A healthy knee and a knee with osteoarthritis, showing cartilage damage, osteophytes, and meniscal damage

Epidemiology

Osteoarthritis affects over 30 million Americans. The knee is among the most commonly involved joints, with symptomatic OA of the knee affecting over 14 million Americans and tens of millions more worldwide. OA is a costly condition. Over 600,000 persons in the USA undergo total knee replacement, at a cost exceeding $12 billion. As with many chronic conditions, the indirect costs of lost productivity are even greater than the direct medical costs of osteoarthritis.

There are several important risk factors for knee osteoarthritis. The most powerful is age. While knee OA is uncommon in persons less than 40 years old, symptomatic radiographic knee OA occurs in over 15% of persons aged 65 or greater. With age, chondrocytes lose their capacity to produce the rich matrix of highly negatively charged macromolecules that enable cartilage to imbibe and retain fluid and bear load. Genetic factors also influence the loss of chondrocyte function. Obesity confers risk of OA both because of the excess biomechanical load borne by the knees of obese persons and due to metabolic factors associated with obesity. Race and gender have also been cited as potential risk factors. Though Blacks and Whites have a similar prevalence of hip OA, Blacks have a greater prevalence of knee OA compared with Whites, as do females compared with males.

Prior injury is another powerful risk factor, increasing an individual's risk of OA by more than tenfold and often resulting in a much earlier onset (approximately 10 years sooner). Individuals who have sustained anterior cruciate ligament tears with concomitant meniscal tear by age 25, for example, face a lifetime risk of developing symptomatic, radiographic knee OA of around 30%. This is because repair of the cartilage and subchondral bone is often incomplete after injury; thus, the altered cartilage matrix is less able to buffer mechanical loads, furthering joint degradation.

Long-standing occupational exposure to repetitive squatting confers risk, as does abnormal knee alignment (varus or excess valgus). Several medical conditions also may predispose to OA including hemochromatosis.

Pathogenesis

The development and progression of OA involves articular cartilage, subchondral bone, and the synovium. As the matrix of the articular cartilage degrades, chondrocytes and cells in the synovium produce pro-inflammatory cytokines including IL-6, IL-8, and tumor necrosis factor alpha, which may result in an abnormal expression of inflammatory mediators and morphological changes. This, in turn, can cause osteophyte formation, changes to the vascularity of the subchondral bone, and destruction of the joint.

In addition to genetic predisposition, there are several other factors that may increase the risk of OA, such as the biomechanics of the joint. In the case of knee OA, a varus or valgus alignment can place additional stress on one compartment of the joint (medial for a varus alignment, lateral for a valgus alignment), significantly accelerating the progression of OA. Knee laxity and muscle weakness are also contributing factors. Hip dysplasia and femoracetabular impingement (which may arise from genetic differences or participation in high-intensity sports during adolescence) increase the risk of hip OA.

Clinical Presentation

History

The patient with osteoarthritis of the knee generally presents with gradual onset of knee pain with activity. Those with predominantly medial compartment disease typically perceive pain medially and those with lateral compartment disease laterally. It is possible, however, for patients with unicompartmental disease to feel pain on the contralateral side of the knee. Many patients will also have a global distribution of pain about the knee, reflecting concomitant involvement of the patellofemoral and one or both tibiofemoral compartments. Pain rarely occurs at rest and is usually relieved by sitting or lying down. A complaint of stiffness is common, typically lasting less than 30 minutes. Range of motion is often limited, especially late in the disease course, and a small cool effusion is common. The quality of the pain varies; some patients describe it as sharp and others dull. Pain is typically predictable and use-related, but unexpected, acute painful episodes become more common later in the course. Patients may notice intermittent swelling. Patients may also notice clicking, catching, popping, or a feeling that the knee is giving way. While

these symptoms should alert the physician to the possible presence of a symptomatic meniscal tear, they often arise from osteoarthritis per se (perhaps due to irregularities in the chondral surface of the osteoarthritic knee).

Stair climbing is frequently difficult for persons with knee OA, particularly those with involvement of the patellofemoral joint. Patients tend to seek care when they lose the capacity to perform valued activities, such as taking a walk with friends or climbing a flight of stairs in their house. Asking patients about their walking distance, the number of flights they can climb, and other functional activities that are relevant to their weekly routines is a useful way of assessing whether patients are improving or worsening. A number of patient-reported outcome measures are available and are collected in clinical practice in some settings.

Physical Examination

Patients with knee OA often have an antalgic gait, in which they limp in attempt to place as little load across the knee as possible, for the shortest period of time. It is useful to observe knee alignment in the coronal plane. The normal alignment of the lower tibia compared to the thigh is about 4° of valgus. Greater extent of valgus (tibia oriented excessively toward the lateral side) overloads the lateral tibiofemoral compartment, while varus malalignment overloads the medial compartment. Patients with varus knees and more advanced OA may manifest a varus thrust with walking, in which the varus deformity is accentuated briefly as the patient pushes off in gait. Symptomatic patients tend to reduce weight-bearing on the affected knee. As a result the examiner can often appreciate atrophy of the quadriceps (measured best 3 cm above the patella, as the vastus medialis obliquus is the most vulnerable muscle).

Tenderness is common over the medial or lateral joint line, depending on which compartment(s) are involved. Those with patellofemoral involvement often have pain with crepitus on manual compression of the patella against the femoral trochlea. Patients occasionally have palpable effusions; these are generally small and cool. Patients with effusions will sometimes have popliteal fullness on exam as well as pain, reflecting a popliteal cyst (Baker's cyst), which is a posterior outpouching of the synovium into the popliteal space. While range of motion tends to be preserved in early osteoarthritis, further in the course, patients may develop limitations in flexion and extension.

Differential Diagnosis and Suggested Diagnostic Testing

Differential Diagnosis

The differential diagnosis of knee OA is broad. The chief challenge is not so much to determine whether the patient has knee OA but rather to discern whether knee OA is the principal source of the patient's symptoms, or whether symptoms arise from

one of several associated conditions. *Anserine bursitis* is a common source of pain in patients with knee OA. The anserine bursa is located at the insertion of the medial hamstring muscles into the tibia, just inferomedial to the tibial tubercle. Patients with *inflammatory arthritis* generally have warm effusions and often have involvement of other joints and prominent morning stiffness. *Infection* can generally be excluded on the basis of the more indolent presentation of osteoarthritis and the lack of warmth, substantial swelling, systemic symptoms, or monotonic worsening. A *strain of the medial collateral ligament* may mimic medial compartment osteoarthritis and can be identified by stressing the medial collateral ligament. *Patellofemoral dysfunction* due to malaligned patellar tracking tends to cause more diffuse anterior knee pain and can usually be provoked by patellofemoral compression. It is difficult to distinguish patellar dysfunction due to maltracking from patellofemoral OA; in fact, the two problems often overlap. (For more, see the chapter on patellofemoral syndromes in this text).

Meniscal tear is a frequent concomitant of knee osteoarthritis. Over 80% of patients with established osteoarthritis of the knee have meniscal tear on MRI. It is difficult to determine whether these tears are *symptomatic*. Popping, clicking, and catching sensations alert the physician to the possibility of meniscal tear, but these symptoms are nonspecific and may arise from osteoarthritis per se. The McMurray maneuver has modest diagnostic value. The examiner flexes and extends the knee using torque on the joint to stress the medial and then the lateral compartment. The test is designed to elicit a painful clicking sensation due to the direct irritation of the torn meniscus by loading each tibiofemoral compartment through an arc of motion.

Diagnostic Testing

The diagnosis of knee OA can generally be made on the basis of characteristic history and physical examination findings, with no need for radiographs, advanced imaging, or blood tests. Radiographs obtained with the patient standing demonstrate the extent of joint space loss and osteophyte formation and are useful for assessing the severity of knee OA (Fig. 24.2). Weight-bearing views with flexion of the knee are useful for assessing the extent of lateral compartment loss. Knee MRI is not necessary to diagnose knee OA but is sometimes used to evaluate for other problems that may mimic or accompany knee OA, such as meniscal tear. MRI should be ordered with caution in this setting, since over one third of all adults have meniscal tears (most of whom are asymptomatic). Thus, the MRI may trigger a series of therapeutic maneuvers, including meniscal surgery, that may be unnecessary. MRI also provides detailed evaluation of OA features besides joint space loss and osteophytes, such as bone marrow lesions, synovitis, and effusion. Bone marrow lesions are subchondral areas of fluid signal on MRI. They are thought to arise from overload of subchondral bone (due, e.g., to destruction of the articular cartilage and/or meniscus, both of which bear load that is directly transmitted to subchondral bone when these tissues fail).

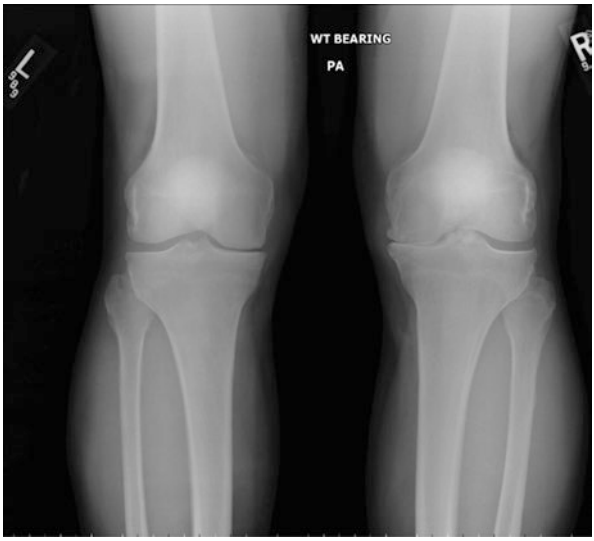

Fig. 24.2 Bilateral osteoarthritis with complete joint space loss and osteophyte formation medially on right; moderate to severe joint space loss on left

Nonoperative Management

Exercise and Core Lifestyle Changes

Substantial evidence documents that regular walking, knee strengthening, weight loss (for obese patients), and stretching to preserve a normal range of knee motion are all helpful in reducing pain and functional limitations in persons with knee OA. Consequently, the management of knee osteoarthritis should begin with patient education and engagement in self-care to initiate and sustain these lifestyle modifications. Physical activity and weight loss are notoriously difficult lifestyle changes for many patients; however weight loss has been shown to have protective effects against cartilage damage. In a secondary analysis of the Intensive Diet and Exercise for Arthritis (IDEA) RCT, Messier et al. suggest that the standard of care for weight loss in overweight and obese persons should be 10% reduction in an individual's body weight, although there is additional benefit with even greater levels of weight loss (≥20%). Weight loss significantly reduces the load on the knee joint, alters the cartilage composition in the medial tibia, reduces inflammatory biomarkers, and has clinically relevant reductions in pain and improvements in function and quality of life. Programs and strategies are available to patients in the community to help persons with OA make these lifestyle commitments; a combination of diet modification and exercise is optimal to achieve maximum weight loss.

In recent years, several professional organizations, including the American College of Rheumatology (ACR) and Osteoarthritis Research Society International (OARSI), have recommended mind-body practices including Tai Chi and Yoga as a treatment for osteoarthritis, as they promote strength, balance, and psychosocial development.

Referral to a physical therapist is often useful so that patients can learn appropriate exercise techniques for strengthening, stretching, and improving neuromotor control of the lower extremity. Physical therapy has been shown to have greater improvements in pain, function, and quality of life as compared to usual OA treatment, even in participants with moderate to severe radiographic OA.

Medications

There is no validated and commercially available disease-modifying drug capable of arresting the process of joint destruction in persons with OA. Studies of disease-modifying effects of glucosamine and chondroitin sulfate are conflicting. Glucosamine, a constituent of the extracellular matrix, promotes osteoblast proliferation and reduces inflammation, while chondroitin, a component of articular cartilage, serves as an anti-inflammatory, anabolic agent. Though several studies have reported significant reductions in joint space narrowing and improvement in pain and function in persons taking glucosamine and chondroitin as compared to placebo, others have shown no superiority of glucosamine, chondroitin, or their combination over placebo treatments. In their most recent guidelines (2019), the American College of Rheumatology strongly recommends against glucosamine and chondroitin (taken as individual agents) for knee, hip, and hand OA. They conditionally recommend the combination of these supplements for hand OA but strongly advise against the combination for treatment of hip and knee OA. The American Academy of Orthopaedic Surgeons (AAOS) also strongly recommends against chondroitin and glucosamine (individually or in combination) as treatment for knee OA and moderately recommends against glucosamine for the treatment of hip OA.

Several agents, including a Wnt inhibitor, a cathepsin K inhibitor, and an anabolic growth factor (FGF-18), have shown promise as disease-modifying drugs in randomized trials. However, for these drugs to be approved for clinical use, the US Food and Drug Administration will require evidence of both structural modification and improvement in pain or function.

In the absence of an approved disease-modifying medication, treatment of OA focuses on symptom relief and preservation of functional status. Acetaminophen is quite safe unless patients have liver dysfunction, but its analgesic effects are weak. Nonsteroidal inflammatory drugs (NSAIDs) are more potent but carry more toxicity, particularly in older patients and those with cardiac, renal, and gastrointestinal comorbidities. Thus, these drugs need to be used carefully, if at all, in patients with these comorbid conditions. Gastroprotective agents (e.g., proton pump inhibitors and H-2 blockers) reduce the frequency of gastrointestinal events in NSAID users. Several NSAIDs are also available in topical form (e.g., diclofenac). The topical formulations have similar efficacy to oral NSAIDs with less toxicity. Traditional NSAIDs such as ibuprofen and naproxen are predominantly cyclooxygenase 1 (COX-1) inhibitors. Predominant COX-2 inhibitors (such as celecoxib) do not inhibit platelets and thus are good options for patients with bleeding disorders or

who are taking anticoagulants. The COX-2 inhibitors do increase risk of hypertension and cardiovascular events and thus should be used with care in patients with cardiovascular comorbidity.

Patients with pain that does not respond to any of these measures are often prescribed opiates. This prescribing practice is controversial. On the one hand, patients have limited options for addressing their pain. On the other, opiates carry risks of somnolence, respiratory suppression, falls, cardiac events, tolerance, addiction, and diversion of pills into the community. Physicians and their patients should discuss the risks and benefits of opiates carefully in this setting. Duloxetine, a selective serotonin and norepinephrine reuptake inhibitor antidepressant, has been shown in randomized controlled trials to reduce chronic pain due to OA. Gabapentin, an antiepileptic that has been useful for neuropathic pain, has also been used in OA though there is limited evidence of its efficacy. Many physicians will suggest duloxetine or gabapentin for patients with features of centralized pain, such as amplification of pain severity and broadening of pain location.

Intra-articular Injections

Intra-articular corticosteroid injections have been shown to be safe and effective, though transient in their effect. Some patients benefit from a strategy of two or three injections annually. This is particularly useful for patients who wish to delay or avoid TKA. Injections of hyaluronate and related products—viscosupplementation—involve greater costs than steroid injections, but the effect appears to persist longer. The guidelines of various authoritative societies are mixed with respect to viscosupplementation, though it is often recommended against. Injections of platelet-rich plasma (PRP) and mesenchymal stem cells (MSCs) have also been considered as potential treatments for patients with knee OA. While several meta-analyses of RCTs have shown PRP to be more effective than HA and placebo injections at reducing pain and improving function for patients with knee OA, a recent double-blind RCT with 5-year follow-up suggests that there are no significant differences between HA and PRP in long-term evaluation. Though there may be a future for PRP in the treatment of OA, it is not currently recommended by professional societies due to a lack of conclusive evidence. Similarly, while injection of MSCs has shown some indication of pain improvement, the limited evidence does not support the use of MSCs for OA.

Indications for Surgery

Patients with knee OA who have not responded to nonoperative therapy may wish to consider surgical options. High-quality randomized controlled trial data document that arthroscopic surgery is no better than a sham control or than a physical

therapy program in reducing pain due to significant knee OA. Thus, there is no role for arthroscopic surgery in the management of knee osteoarthritis, per se.

On the other hand, if patients have suspected meniscal tear in association with their osteoarthritis, arthroscopy can be considered. This issue is covered in greater detail in the chapter on meniscal tear. Several large trials have been completed on the efficacy of arthroscopic partial meniscectomy in patients with knee OA. One trial documented a clear advantage for surgery; another showed that surgery is no more efficacious than a sham arthroscopic partial meniscectomy. In several other trials, surgery showed no advantage over a PT-based regimen in the intention to treat analyses, but better outcomes in the as-treated analyses. Experts have generally interpreted this evidence as supporting a strategy of initial rigorous physical therapy—with an emphasis on strengthening—with consideration of surgery for patients who have not responded and who recognize that the efficacy of surgery in this setting is uncertain.

For the patient with symptomatic unicompartmental osteoarthritis despite trials of nonoperative therapy, several surgical options can be considered including osteotomy, unicompartmental knee arthroplasty, and total knee arthroplasty. For the patient with unilateral medial compartment OA, the osteotomy is designed to shift load bearing to the lateral compartments. Similarly, for the patient with lateral compartment OA, the osteotomy is designed to shift load medially. This procedure tends to be particularly well suited to younger patients (e.g., those in their 40s), for whom a total knee arthroplasty would carry a high risk of failing in the patient's lifetime.

Unicompartmental knee arthroplasty (UKA) and total knee arthroplasty (TKA) are alternative treatments for end-stage osteoarthritis. For more information regarding the indications for and outcomes of osteotomy, UKA, and TKA, please refer to the chapter on management of advanced knee OA (Table 24.1).

Table 24.1 Summary of presentation and management of knee osteoarthritis

Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Use-related pain and loss of valued activities Joint line tenderness Occasional effusions	Diagnosis made by history and physical exam Radiographs to assess severity MRI occasionally useful to exclude other entities	Walking, quad strengthening, stretching Weight loss (if obese) Analgesia (acetaminophen, NSAIDs, topical NSAIDs) Intra-articular injection	Persistent use-related pain and loss of valued activities despite conservative Rx Patient understands and accepts short- and long-term risks Acceptable surgical risk	No role for arthroscopy in treating knee OA Role of arthroscopic surgery for OA with symptomatic meniscal tear evolving; requires careful discussion with physician Osteotomy or unicondylar arthroplasty if unicompartmental OA Total knee arthroplasty

OA osteoarthritis, *MRI* magnetic resonance imagining, *NSAIDs* nonsteroidal anti-inflammatory drugs

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Chapter 25
Surgical Approaches to Advanced Knee OA (TKA, UKA, Osteotomy)

Adam S. Olsen and Vivek M. Shah

Indications for Operative Intervention

As mentioned in the previous chapter, the general indication for operative intervention in knee osteoarthritis is confirmed symptomatic disease that has failed conservative treatment in patients otherwise healthy enough to tolerate surgery. While knee osteoarthritis is often apparent on physical examination, other sources of pain about the knee must always be excluded; potential sources include but are not limited to bursitis, ligamentous injuries, patellofemoral syndromes, and isolated meniscal pathology. Referred pain from hip osteoarthritis, which frequently occurs in the same population as knee OA, is also relatively common, and examination of the ipsilateral hip should always be performed in patients presenting with knee pain. Radiographic evaluation of knee OA should always include weight-bearing films of the knee, ideally standing AP, flexion weight-bearing, lateral, and patellar views. Supine radiographs are not very useful in the evaluation of OA, as the extent of cartilage loss is not appreciated well. In the past, a common belief was that only patients with radiographic evidence of severe disease ("bone-on-bone") should be offered surgery; however more recent research has demonstrated that symptomatic patients with less dramatic radiographs often experience significant relief after arthroplasty. It is still important to differentiate symptomatic OA patients from those suffering from isolated meniscal pathology in the setting of preserved cartilage space, as these patients may be candidates for alternative treatments such as arthroscopy (see meniscal tear chapter).

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General Contraindications: Absolute and Relative

Absolute Contraindications

There are few absolute contraindications to knee arthroplasty or osteotomy; the first and most obvious is current infection. This can refer to infection in the knee joint itself (native knee septic arthritis), or much more commonly remote infection. Some of the more common etiologies of remote infection are dental/gastrointestinal and chronic wounds (such as those stemming from diabetic ulcers or venous stasis). Regardless of the source, these must all be resolved prior to considering arthroplasty as they expose patients to risk of periprosthetic joint infection. Chronic extensor mechanism insufficiency is also considered an absolute contraindication, as well as severe neuromuscular dysfunction and Charcot neuropathy. Conditions that preclude a patient's ability to heal a lower extremity wound, such as severe untreated or untreatable peripheral vascular disease, are also contraindications. Finally, certain patients are simply too chronically ill secondary to medical comorbidities to tolerate these operative interventions; this decision must always be weighed against the morbidity of continued immobility and pain due to advanced knee OA.

Relative Contraindications

In contrast to the absolute contraindications discussed above, the relative indications to knee arthroplasty or osteotomy are much more numerous and also much more debatable. These often vary by surgeon and to some extent by location. Obesity is frequently encountered in the population with knee arthritis, and body mass index (BMI) thresholds are often used to determine which patients are candidates for surgery. Relative thresholds in the 35–40 kg/m^2 range are frequently used, as complications such as infection and implant loosening are significantly elevated as BMI values enter this range. Absolute BMI thresholds are somewhat controversial, as patients with morbid obesity generally experience pain relief following TKA and these patients also find it difficult to lose weight. A strategy of staging bariatric surgery, and then TKA, addresses this conundrum and is recommended occasionally.

Similar to morbid obesity, uncontrolled diabetes is associated with an elevated risk of complications following knee surgery, with infection being the most commonly cited. The optimal method of evaluating glycemic control in the preoperative period is still not entirely clear, nor is the threshold for mitigating complications; hemoglobin A1C is likely the most frequently used marker with thresholds in the 7–8% range. Other systemic contraindications include malnutrition and any other physical or mental factors that would prevent patients from participating in the necessary postoperative rehabilitation following surgery.

In addition to systemic conditions such as obesity and diabetes, local factors affecting the lower extremity may also represent relative contraindications to

surgery. Skin conditions around the knee such as venous stasis, lymphedema, psoriasis, or scarring from previous surgery or trauma must all be thoroughly evaluated and often treated. Patients with histories of native knee septic arthritis, local osteomyelitis, or recurrent cellulitis (regardless of timing) are always at increased risk of infection after knee surgery. Severe arthritis of the ipsilateral hip is a commonly cited contraindication to knee arthroplasty, as outcomes of TKA with ipsilateral hip OA are poorer (it is often preferable to address the hip first). Finally, heavy tobacco or opiate use is considered a relative contraindication for many surgeons, due to increased risk of postoperative complications in these patients.

Surgical Options

After the decision has been made to proceed with operative intervention for advanced knee osteoarthritis, several surgical options are available. The decision of what surgery to perform is guided by many factors, including patient demographics (age/activity level/expectations), anatomy (disease location/bone loss/ligamentous stability), and those related to the surgeon (preference/experience). In general, advanced knee OA is surgically treated with either total knee arthroplasty, partial (unicompartmental) knee arthroplasty, or less commonly tibial osteotomy. The next sections will discuss each of these procedures separately and will discuss some of the specific indications, procedural details, outcomes, and possible complications of each.

Total Knee Arthroplasty

In 2019, over 850,000 total knee arthroplasties were performed in the USA alone, and this number continues to rise annually due to an aging population and expanding indications for surgery. The total versus partial knee arthroplasty decision can be complex; however the typical total knee patient is one with generalized arthritic pain and evidence of degenerative changes throughout the knee on weight-bearing radiographs. Exceptions to this include patients with localized symptoms (affecting either the medial, lateral, or patellofemoral compartments of the knee) but with significant bone loss or clinical instability that would preclude consideration of partial knee arthroplasty in these patients.

General Procedure

Total knee arthroplasty (TKA) can be considered a resurfacing-type procedure, where the formerly cartilage-covered articular surfaces of the distal femur and proximal tibia are trimmed away. The articular surface of the patella is sometimes, but not always,

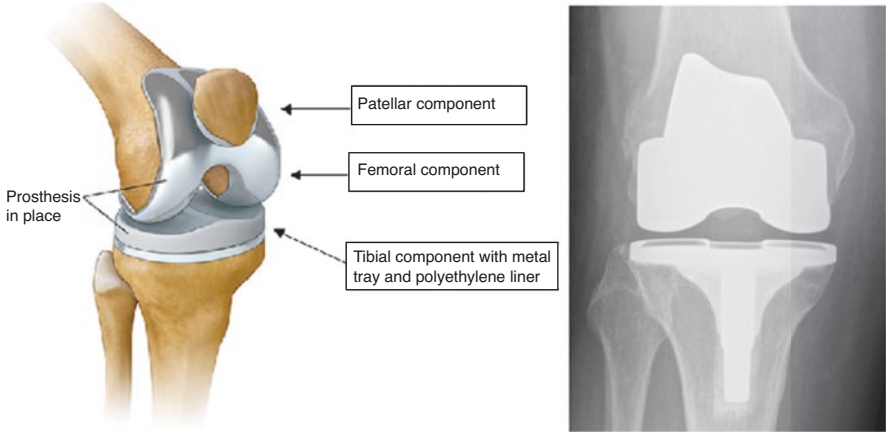

Fig. 25.1 Total knee arthroplasty illustration with representative AP x-ray

resurfaced as well, and this decision is based largely on surgeon preference and intraoperative findings (there is likely no difference in patient outcome). In the most common configuration, the distal femur is capped with a cobalt-chrome component (alternative materials exist for patients with metal allergies), and a titanium plate is secured to the proximal tibia. A polyethylene (radiolucent plastic) insert is then joined to the tibial tray, completing the construct (Fig. 25.1). The most common alternative to this configuration involves the use of an all-polyethylene tibial component, taking the place of both the titanium tibial tray and the plastic articular insert.

The operative procedure always involves the removal of the anterior cruciate ligament and remnants of the medial and lateral menisci, which are typically severely damaged from the arthritic process. A series of cutting jigs is used to remove as little bone as possible to recreate patient anatomy and restore ligamentous balance. Treatment of the posterior cruciate ligament (PCL) is variable and is based on ligament competence and surgeon preference. In general, individual surgeons may prefer to either preserve ("cruciate-retaining", CR TKA) or remove ("posterior-stabilized", PS TKA) the PCL. This distinction has become more blurred over the past several years, as advances in the wear properties of polyethylene have given surgeons many more options in the operating room. The fixation of implants to bone has also evolved over time, and while most TKA in the USA are still secured with bone cement, press-fit implants designed to grow into host bone are becoming more popular. Irrespective of technique or fixation strategy, most patients are permitted to weight-bear as tolerated without restrictions immediately after surgery.

Outcomes

Approximately 80–90% of patients report satisfaction after total knee arthroplasty, and these numbers have not changed significantly over the years despite advances in technique, implant design, and perioperative medical management. The

remaining 10–20% of postoperative patients either report incomplete paint relief (some likely multifactorial), worsening function, or unfulfilled expectations. Certain patient factors such as depression, anxiety, or a lack of social support have also been shown to put patients at a higher risk of dissatisfaction after surgery. It does appear that patients treated at high-volume centers have improved satisfaction after surgery. The revision-free survival of total knee arthroplasty is approximately 95% at 15 years.

Complications

Complications after total knee arthroplasty are largely influenced by the overall health of the patient, and approximately 3% of patients experience a serious complication in the first 3 months after surgery. In the early postoperative period, the risk of venous thromboembolism is particularly relevant, with most manifesting within 2 weeks of surgery. Without prophylaxis of any kind, the prevalence of deep vein thrombosis (detected on imaging) after TKA is around 50%. With modern strategies including pharmacological and mechanical prophylaxis as well as early patient mobilization, current rates of symptomatic deep vein thrombosis and pulmonary embolism are less than 1%.

Another dreaded complication following total knee arthroplasty is infection. Infections can occur at any time after total knee arthroplasty. In the early postoperative period, erythema around or drainage from the surgical incision may be suggestive of deep infection in the replaced joint. In these cases, it is paramount that oral antibiotics are never started before consulting with the operating surgeon, as a joint aspiration/culture is often required and antibiotic administration will interfere with these results. Later infections can manifest as gradual increases in pain over a prolonged period (chronic infections) or abrupt increases in pain and swelling (acute hematogenous infections). The surgical intervention required depends on factors such as time from surgery, duration of symptoms, and infecting organisms and can range from simple debridement to implant removal.

Other, less common complications after total knee arthroplasty include aseptic component loosening, polyethylene wear (becoming less relevant with advances in polyethylene manufacture), arthrofibrosis (postoperative stiffness/loss of motion), and periprosthetic fracture.

Partial (Unicompartmental) Arthroplasty

Partial or unicompartmental knee arthroplasty (UKA) is typically performed in patients where pain and radiographic evidence of OA is confined to either the medial tibiofemoral, lateral tibiofemoral, or patellofemoral compartments of the knee. Similar to total knee arthroplasty, and for similar reasons, the number of UKA performed in the USA is steadily increasing, and around 70,000 were performed in

2019. By far the most commonly performed UKA is the medial tibiofemoral UKA (Fig. 25.2), largely due to the prevalence of isolated OA in this compartment. Lateral UKA is performed less frequently, for isolated valgus OA affecting the lateral compartment, and the least commonly performed UKA is the patellofemoral arthroplasty (Fig. 25.3), where the articular surfaces of the patella and anterior femur (trochlea) are resurfaced. These procedures are appealing to both surgeons and patients because they may lead to faster recovery, more normal knee kinematics, and improved functional outcomes when compared to TKA.

The steady increase in the number of UKA performed in the USA every year is multifactorial. With regard to medial UKA, there have been fairly drastic changes in indications over the past several decades. Years ago, the appropriate candidate for a medial UKA was a thin (BMI under 30), older patient with absolutely no evidence of OA in the patellofemoral compartment (even in the absence of patellofemoral symptoms) and no suspicion of anterior cruciate ligament insufficiency. More recent literature has demonstrated satisfactory outcomes in older and heavier patients, as well as those with radiographic evidence of OA in the patellofemoral compartment. ACL insufficiency is also no longer considered an absolute contraindication. UKA in general are also becoming less technically challenging due to improvements in instrumentation and technique (i.e., robotic surgery and intraoperative navigation), although these technologies are not required for successful outcomes. The expanding indications and increasing popularity of these procedures will likely lead to

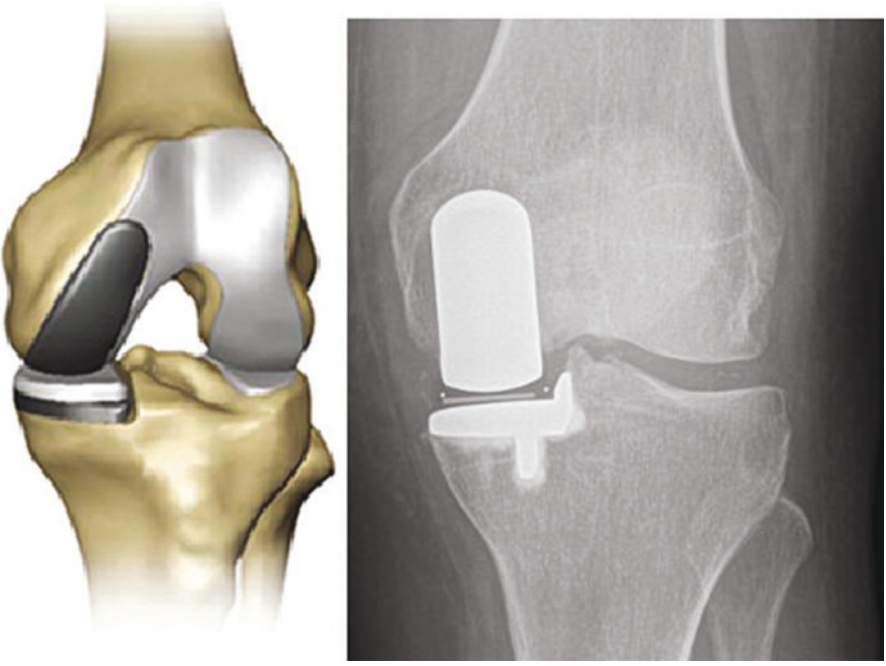

Fig. 25.2 Medial unicompartmental arthroplasty illustration with representative AP x-ray

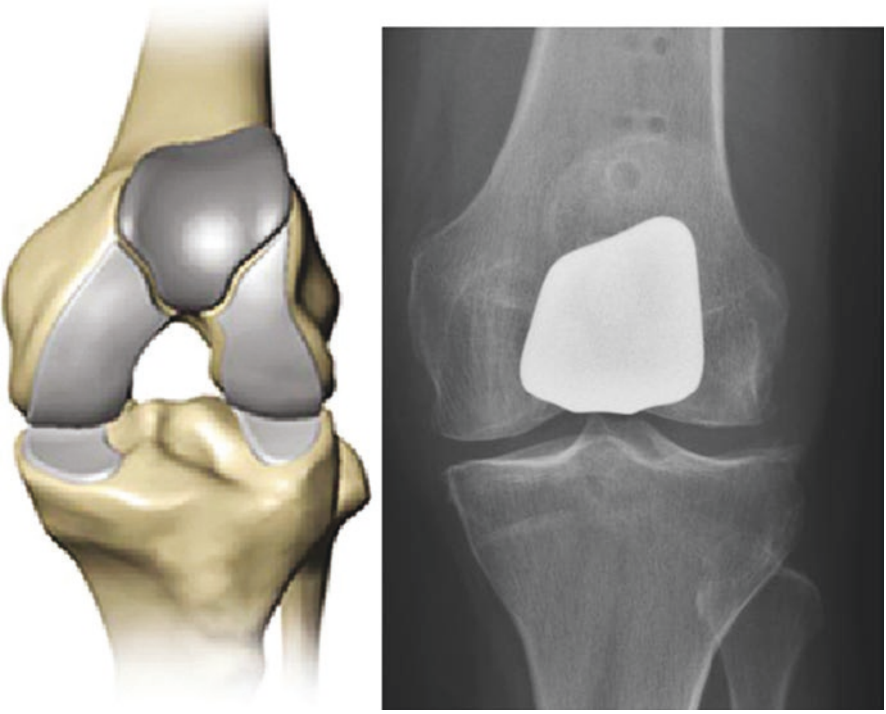

Fig. 25.3 Patellofemoral arthroplasty drawing with AP x-ray

increased trainee exposure and thus a steady increase in the number of surgeons performing these procedures in the future.

General Procedures

Similar to total knee arthroplasty, UKA procedures can be considered resurfacings, although there is a relative preservation of host bone when compared to TKA, making these procedures particularly appealing to younger patients. Also similar to TKA, the femur is typically capped with a cobalt-chrome component after the arthritic articular surface is removed, and a titanium tray is fixed to the tibia (except in the patellofemoral arthroplasty, where the tibia is not altered). In the medial and lateral tibiofemoral UKA, a polyethylene insert is placed between the tibia and femur, and the patella is not resurfaced.

All three procedures begin with an incision over the anterior knee followed by an arthrotomy. After entering the knee joint, the articular surfaces throughout the knee are always visually inspected as a final check to confirm that the patient is a candidate for a UKA. Medial and lateral UKA procedures then continue with exposure of

the affected compartment, with preservation of the ACL and PCL, as well as the meniscus in the unaffected compartment. A series of cutting jigs is used to resect the arthritic joint surfaces, and particular attention is paid to not overcorrecting the preoperative deformity, which may transfer excess force to the unaffected compartment. As with TKA, the fixation of medial and lateral UKA components can be either cemented or press-fit. After arthrotomy/inspection in patellofemoral arthroplasty cases, cutting jigs are used to resurface the anterior femoral trochlea only, and the patella is capped with a polyethylene component in a fashion similar to that of TKA.

Outcomes

A relatively common misconception about UKA is that these procedures are precursors to eventual total knee arthroplasty. While arthritic progression to other areas of the knee is one of the most common reasons for revision, the survival characteristics of UKA are quite favorable, especially in the setting of bone preservation and other potential advantages over TKA as previously discussed. Cohort studies have reported approximately 95% 10-year and 90% 15-year revision-free survival for medial UKA. Similarly, in a systematic review, lateral UKA was reported to have 91% and 89% survival at 10 and 15 years, respectively. The long-term survival of patellofemoral arthroplasty is less clear, likely in part due to technique and design changes throughout the history of the procedure. Studies have reported survival of patellofemoral arthroplasty to be approximately 95% at 5 years and 84% at 10 years; however the relevance of these values to contemporary technique/design is not entirely clear.

Complications

In addition to those associated with total knee arthroplasty, there are several possible complications that are unique to unicompartmental knee arthroplasty. Among the most common reasons for revision from partial to total knee arthroplasty is arthritic progression in other compartments of the knee. This is likely sometimes related to surgical technique, as overcorrecting (or "overstuffing") of a compartment during a medial or lateral tibiofemoral UKA can place excess force through the previously unaffected compartments, accelerating the degenerative process. There are also potential issues specific to medial and lateral UKA, one of which is aseptic loosening. While this can occur in total knee arthroplasties, it is relatively more common in medial/lateral UKA, likely due to a much smaller surface area for cement interdigitation or bony ingrowth. There is also a potentially higher risk for

periprosthetic fracture of the tibial plateau after medial/lateral UKA, likely related to both patient anatomy and surgical technique. Finally, a potential complication unique to medial UKA is bearing dislocation. Some, but not all, medial UKA feature a mobile polyethylene bearing, which is free-floating in the knee but maintained in appropriate position via conformity to the femoral component. Acute increases in pain or instability in mobile-bearing medial UKA patients (mobile bearings are not used in lateral UKA) may be secondary to bearing dislocation, with incidence in the literature around 2%.

High Tibial Osteotomy

Osteotomy of the proximal tibia can be used to treat isolated medial or lateral tibiofemoral arthritis, by redistributing force to the unaffected compartment (Fig. 25.4). These procedures have declined in frequency in the USA, partially due to the success of UKA and the longevity of modern arthroplasty materials. The ideal high tibial osteotomy patient is a younger, high-demand patient that would likely require at least one revision in their lifetime if treated with an arthroplasty procedure. Contraindications to the procedure include generalized/inflammatory arthritis (as with UKA), and also any significant bone loss, subluxation, or limitation in range of motion. Patients with larger deformities are also better suited for arthroplasty procedures, except very young patients that may require several osteotomies (femur and tibia) for deformity correction.

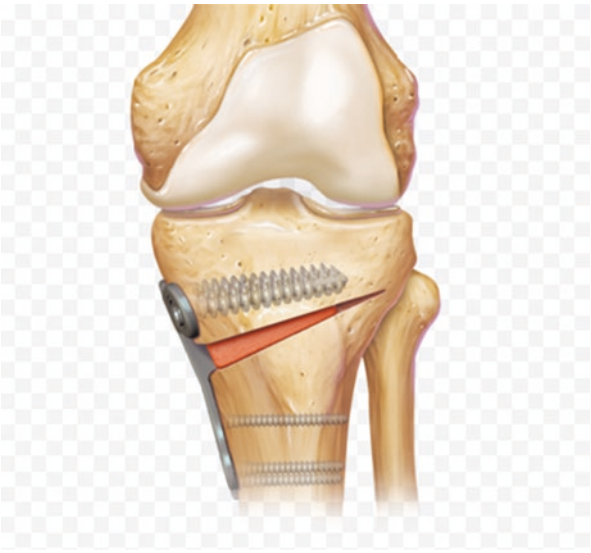

Fig. 25.4 Medial opening wedge high tibial osteotomy illustration

General Procedure

There are several technical options when performing high tibial osteotomy, and the utilization of these has changed over time. Currently, the most commonly performed osteotomies are opening wedges of either the medial or lateral tibial plateau. The procedure involves making a precise cut in the proximal tibia up to, but not through, the contralateral bony cortex. The osteotomy site is then hinged open and secured with either a plate, graft material, or both. Through precise planning and technique, deformities in several planes (coronal and sagittal) can be corrected with this procedure.

Outcomes

Unlike the other procedures listed, and especially considering the patient population, high tibial osteotomy performed for OA can be thought of as a temporary solution with the goal of delaying arthroplasty. While the 5-year survival of these procedures is in the 85–95% range, by 15 years approximately 50% of high tibial osteotomies performed for OA have been converted to arthroplasty. Recent studies have also demonstrated improved outcomes with UKA when compared to high tibial osteotomy, as well as less-satisfactory TKA outcomes in the setting of prior high tibial osteotomy.

Complications

As with the other procedures discussed in this chapter, there are several potential complications unique to high tibial osteotomy. Infection and thromboembolism are always possibilities, and as with UKA, the progression of arthritis elsewhere in the knee. Fracture of the tibial plateau may occur in the early postoperative period after high tibial osteotomy, and there is also a risk of osteotomy site nonunion. Alteration of the joint line during high tibial osteotomy impacts patellofemoral biomechanics, often resulting in a relative lowering of the patella (patella baja). This can both be a source of anterior knee pain in these patients and can make for a more difficult future conversion to total knee arthroplasty.

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Chapter 26
Cartilage Defects in the Knee: Clinical, Imaging, and Treatment Aspects

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Introduction

Cartilage lesions are common sequelae of knee injury and pose a significant health burden to patients limiting sports practicing and routine activities. Besides affecting patients' quality of life, cartilage injuries are highly associated with the development of osteoarthritis (OA), a potentially irreversible outcome. Considering that the majority of cartilage lesions are incidental and detected in non-symptomatic patients during magnetic resonance imaging (MRI) or arthroscopy procedures, its true prevalence is unknown. Recent studies, however, have reported numbers as high as 900,000 cases of cartilage lesions per year in the United States, with approximately 200,000–300,000 requiring surgery, leading to a significant socioeconomic impact.

Chondral or cartilage injury includes a wide range of entities varying from small, isolated, and contained defects to advanced end-stage OA. In addition, chondral lesions might be graded based on the depth of the lesion, such as superficial, full thickness, or osteochondral injuries. In this regard, the cartilage lesion characteristics (size, location, and depth) determine disease prognosis and guide different treatment strategies. Cartilage defects can appear in

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different areas in the knee, being most prevalent on the weight-bearing femoral condyle (up to 58%), with the majority found in the medial condyle. Patellar and trochlear lesions are less frequent, accounting for up to 36% and 16% of cases, respectively.

Articular cartilage has a poor spontaneous healing capacity as a consequence of its relatively hypocellular and avascular structure. Consequently, after a cartilage injury, spontaneous healing is usually not expected. In fact, a regenerative process can only be observed when the subchondral bone is affected, leading to a subsequent release of bone marrow-derived mesenchymal cells. However, the formed scar is usually composed of fibrocartilaginous tissue with lower biomechanical properties when compared to the native cartilage tissue. Such tissue has far inferior properties, leading to early wear, incomplete subchondral bone coverage, possible pain, and inflammation slowly propagating the clinical progress toward OA.

Another important feature of articular cartilage is that despite its uniform macroscopic appearance, it is not a uniform tissue, displaying distinct biochemical and morphological differences between the superficial, middle, and deep layers. Although chondrocytes and the extracellular matrix composition, constituted mainly by high-molecular-weight proteoglycans and type II collagen fibers, are present in all cartilage layers, its number and concentration differ in each zone. While the superficial layer contains a high cellular density formed by small-flattened and discoid chondrocytes embedded in a network of thin parallel collagen fibrils and low concentration of proteoglycans, the deep layers are formed by more spherical chondrocytes and a higher concentration of proteoglycans. Given this important tissue complexity, treatment of cartilage injury is challenging and must take these differences into account.

The nature of the cartilage injury is still unclear. Multiple factors have been associated with its development and progression. Among these, increased age and high body mass index (BMI) seemed to be the most profound risk factors for knee cartilage degeneration in non-traumatic conditions. Additionally, genetic factors might contribute to increased susceptibility to cartilage breakdown and degradation, which favors OA development. Moreover, cartilage defects can be due to other etiologies, including acute or recurrent trauma, osteochondritis dissecans, avascular necrosis, and degenerative abnormalities.

Post-traumatic OA

Articular cartilage requires a fine and regulated environment to properly maintain its homeostasis and overall health. Any insult that disrupts the intra-articular environment might impair this critical equilibrium, potentially leading to cartilage damage.

Effects of Anterior Cruciate Ligament Injury on Early Cartilage Degeneration

Joint instability after a ligamentous injury has been extensively associated with the development of chondral injuries. Indeed, biomechanical alterations that occur following an anterior cruciate ligament (ACL) tear and reconstruction commonly result in cartilage degeneration and the development of cartilage lesions. However, besides biomechanical changes, disruption of the cytochemical joint environment also plays an essential role. Proteins and inflammatory cytokines are leaked into the synovial environment after ACL injury. Matrix metalloproteinases and pro-inflammatory cytokines, which result in cartilage breakdown, impair tissue homeostasis and lead to chondrocyte death and cartilage degeneration. Therefore, even after surgical joint stabilization, the development of post-traumatic OA is not brought to a halt and may progress. While ACL reconstruction does not prevent OA development following the ligament injury, it may, in fact, accelerate the progression to post-traumatic OA in some patients. Prolonged increase of markers of chronic inflammation in the synovial fluid of patients after ACL injury showed a slightly higher increase in IL-6 in patients that underwent ACL surgery after ACL injury over patients that were managed nonoperatively. While this observation leads us to understand post-traumatic OA after ACL injury as a likely outcome, we still do not understand exactly why this progression happens and who is at greatest risk for the rapid development of post-traumatic OA.

Effects of Meniscus Injuries and Malalignment on Early Cartilage Degeneration

Meniscus tear is another well-established pathology inducing early cartilage degeneration. The proper load distribution over the joint depends on the intact meniscus, considering its function of shock absorption, joint stability, and knee proprioception. In addition, the menisci are responsible for cartilage nutrition and lubrication, which directly influence cartilage homeostasis. Lastly, alignment abnormalities, both in the tibiofemoral and patellofemoral joint, may also be associated with cartilage damage. Lower limb malalignment overloads the joint, impairing the biomechanical properties and disrupting the cartilage homeostasis.

Clinical Presentation and Evaluation

A crucial factor to adequately manage cartilage injuries is the early recognition and identification of the features of the disease. Delayed diagnosis favors the exacerbation of symptoms and progression to more severe cartilage damage. However,

symptoms are not always readily detected or reported, and when they are, a wide variety of clinical presentations might occur, making early diagnosis challenging. Because cartilage does not contain nerves, damage does not directly cause pain, and even large chondral defects can be totally painless for a long period of time. In addition, there is likely a significant delay between the original chondral injury and the actual loss of chondral tissue. Therefore, pain related to cartilage damage, if any, is usually associated with insults to the surrounding structures. In this regard, the adjacent soft tissue, the underlying subchondral bone, and the synovium may be the source of pain. Besides pain, patients with a damaged cartilage may experience mechanical symptoms, such as catching or locking. Moreover, acute effusion derived from an acute injury (e.g., hemarthrosis) or recurrent effusion may also be present and is widely considered one of the most important signs of clinical progression.

Aside from specifics in the medical history that may lead to the detection of specific causes of chondral or subchondral pathology (i.e., immunosuppression, history of steroid use leading to avascular necrosis, etc.), duration of symptoms is an important factor as longer symptoms have been associated with worse clinical results after cartilage repair. In addition, previous knee traumas and surgeries are important to take into account as well. A past trauma, even if it occurred a long time before the visit, might be the root of the current problem. Previous knee surgery, specifically when involving treatment of the subchondral bone (i.e., microfracture treatment), may not only affect the diagnosis but also the nature of the subsequent cartilage repair procedure, as it has been shown to have a marked effect on cell-based therapies. The family history deserves to be investigated, since some disorders, such as osteochondritis dissecans, might have a familial correlation. Also, a family history of OA is frequently associated with worse outcomes following cartilage procedures, although scientific confirmation is still needed. Anecdotally, large swings in weight (significant weight gain or loss) are often associated with progressive chondral breakdown and symptomatic presentation. Likewise, investigation of social habits are particularly important during the medical anamnesis. For instance, it is known that smoking impairs postoperative healing and chondral repair.

Physical examination must be part of the routine evaluation. Patient's gait pattern and body type must be assessed. An abnormal gait may occur due to several factors, such as muscle weakness, knee pain, lower limb malalignment (varus/valgus), or knee thrust due to ligament deficiency. In addition, focusing on the knee, any muscular atrophy, swelling, or prior surgical incisions should be recorded. The exact location of the patient's symptoms must be actively sought by static and dynamic evaluations. Knee range of motion – and associated flexion contractures or hyperlaxity – clicking, catching, or locking need to be recorded. A thorough ligamentous exam is important to rule out associated mechanical instability. The patellofemoral exam, including the presence of pain and crepitus, patellar tracking, tilt and glide test, as well as malalignment assessment using bony and ligamentous parameters, is important as they inform the choice of associated procedure necessary to improve the success of a chondral repair strategy. Importantly, other patient's characteristics such as age, BMI, and associated comorbidities should be investigated as well, considering the potential negative impact on cartilage repair processes.

All physical examinations must be performed bilaterally, comparing both knees. An adequate clinical evaluation will identify the characteristics of a given cartilage damage guiding the best therapeutic approach for satisfactory outcomes.

Differential Diagnosis

As mentioned before, clinical diagnosis of chondral defects is challenging. Although some symptoms are suggestive of chondral injury, there is no pathognomonic finding, and an overlap with other knee pathologies may occur. Femoral condyle defects that cause joint line pain, mechanical symptoms, and knee effusion might mimic meniscal injuries. Anterior knee pain during kneeling, squatting, descending, or ascending stairs might simulate anterior knee pain due to soft tissue inflammation. A meticulous investigation might be the key for proper diagnosis.

Imaging Examination

Radiography

Noninvasive imaging techniques are important tools to evaluate, diagnose, and monitor cartilage defects. Radiographic examinations are commonly the first method utilized and are an indispensable instrument to start the patient's evaluation. Conventional bilateral x-ray series of weight-bearing anteroposterior, lateral, 45° supine tangential patellar skyline view, and 45° bent standing posteroanterior view (Rosenberg) are the standard series. These images do not directly explore the cartilage status, unless there is bone loss, joint space narrowing, or osteophytes present. Instead, radiographs are crucial to evaluate the presence and status of degenerative diseases, given that advanced knee OA is a contraindication for cartilage repair procedures. An anteroposterior long leg alignment x-ray (MTP-2 single leg standing alignment view) (Fig. 26.1) is also crucial to determine the lower limb alignment, since malalignment must be corrected before or concomitantly to any cartilage repair. Moreover, patellofemoral abnormalities, such as patella alta or baja, patellar tilt, and trochlear dysplasia, need to be assessed during the radiographic investigation.

Computed Tomography

Computed tomography (CT) alone is not the ideal tool to evaluate the cartilage tissue. However, subchondral bone status, the presence of osteophytes, and patellofemoral joint irregularities are adequately evaluated using CT scan and might assist in further management of the patient.

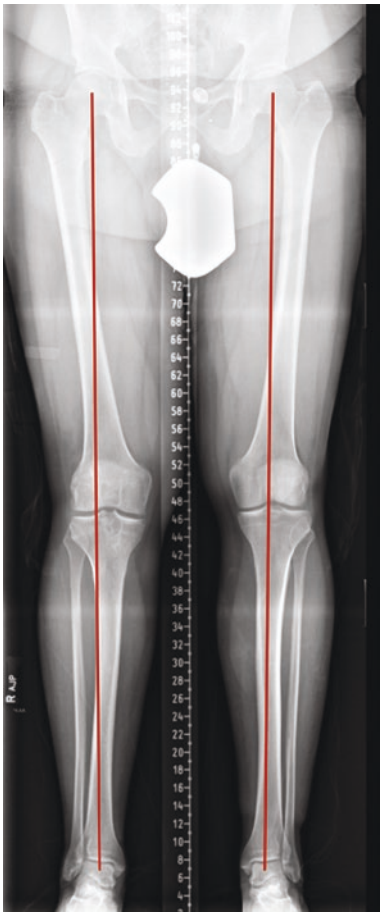

Fig. 26.1 Anteroposterior long leg alignment x-ray showing valgus alignment (right knee) and neutral alignment (left knee)

Magnetic Resonance Imaging

Patients experiencing joint line tenderness, knee swelling, or mechanical symptoms are candidates for magnetic resonance imaging (MRI) exam. MRI is widely accepted as the best imaging modality to assess the knee cartilage and subchondral bone. In fact, even in case of a small defect, MRI presents a detailed and precise information about the articular cartilage. In addition, the development of cartilage specific sequences, including T2 mapping and proton density fat suppression, has significantly enhanced the accuracy of cartilage status evaluation. Moreover, besides analyzing the cartilage in terms of the morphological conditions, presence of fissure or delamination, or size and depth of the defect (if any), the subchondral bone can be clearly examined in regard to bone marrow edema, cysts, and intralesional osteophytes (Fig. 26.2). MRI is also excellent for an overall investigation of the hard and soft tissues within the knee, which brings important considerations about associated

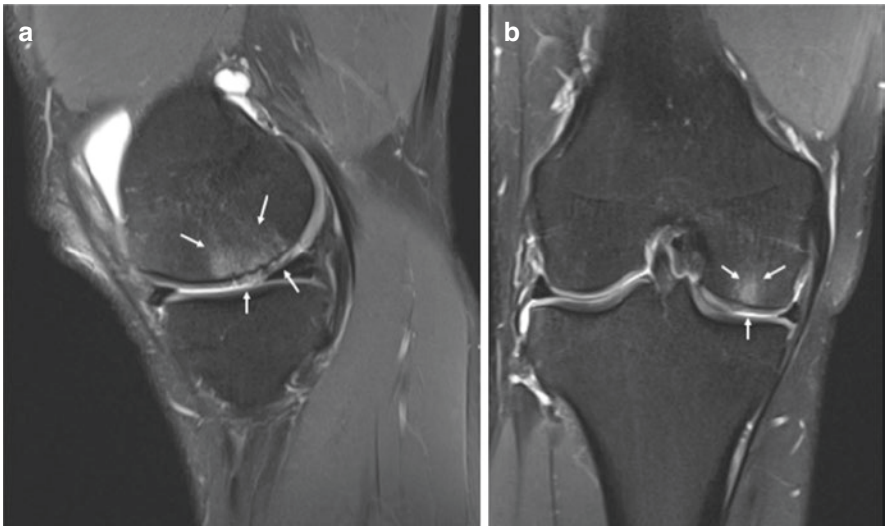

Fig. 26.2 Magnetic resonance imaging 3 T. Sagittal (**a**) and coronal (**b**) fat-suppressed intermediate-weighted images demonstrating a deep cartilage lesion with underlying bone marrow reaction

injuries (e.g., meniscus or ligament tears), synovitis, and loose bodies. Finally, MRI allows a reliable assessment of the patellofemoral joint with regard to structural damage and alignment.

Arthroscopic Evaluation

In case of any uncertainty regarding the patient's condition after physical examination and adequate imaging modalities, arthroscopic evaluation is necessary. In fact, this is the only approach that provides a direct view of the joint environment and therefore considered to be the gold standard in cartilage evaluation. Subtle, early changes, cartilage softening, and partial delamination that occasionally are not visualized on the MRI can be detected during arthroscopy. Moreover, it is the most reliable way to measure the size and depth of the defect, which are crucial characteristics to set the proper cartilage treatment (Fig. 26.3). Joint arthroscopy also allows for a possible immediate point-of-care treatment and chondral biopsy in preparation for a cell-based cartilage repair procedure. Furthermore, the lesion depth can be classified based on the International Cartilage Regeneration and Joint Preservation Society (ICRS) grading system.

Recently, the in-office arthroscopy has been proposed as an alternative strategy to investigate the joint environment. Performing the procedure in office avoids anesthesia risks, as well as reduces the costs to the patient, insurance company, and hospital. In-office arthroscopy can theoretically improve the diagnostic accuracy in

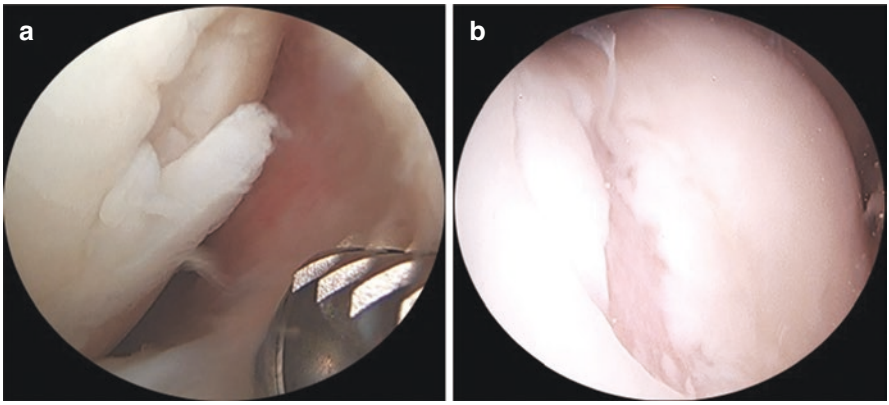

Fig. 26.3 Arthroscopy view showing a cartilage defect in the femoral condyle (**a**) and in the trochlea (**b**)

relation to MRI for intra-articular injuries; however, more quality evidence is needed. In this regard, clearly defined protocols, indications, and contraindications must be established before its widespread use.

Treatment Algorithm

The treatment of cartilage injuries has markedly improved over the years. Despite the historical register of osteochondral allograft transplantation in the beginning of the 1900s, and bone marrow stimulation techniques in the 1950s, the knowledge of the cartilage structure and physiology and its interaction with the underlying bone has only been explored recently.

Recognizing which patient needs surgical cartilage interventions and what procedure would be most suited is still challenging, and the treatment usually is personalized based upon the patients' specific situation. Imperative for success of any cartilage repair procedure is the active participation of the patient, as the rehabilitation requires prolonged periods of time, sometimes in excess of 12 months.

Osteochondral Unit

A pivotal concept that dictates the surgical treatment refers to the interaction between the cartilage and the underlying bone. Articular cartilage and subchondral bone are intimately connected through the calcified cartilage, forming the so-called osteochondral unit. This unit is essential to maintain the health and integrity of the joint, and it absorbs up to 30% of the total impact load of the joint. Moreover, because of its highly innervated nature and vascularization, the subchondral bone

actively remodels and regenerates in response to loading which affects the diffusion of nutrients and signaling molecules influencing articular cartilage.

Therefore, the status of the subchondral bone is an important part in decision-making on which cartilage repair procedure to use. Signs of impaired subchondral bone such as severe subchondral edema, intralesional osteophytes, or large subchondral cyst are taken into account to decide if the entire osteochondral unit requires treatment or a cell surface treatment may be sufficient.

Nonoperative Treatment

Nonoperative treatment approaches aim to control the symptomatology and disability of the patients, potentially slowing the progression of degenerative changes that are related to cartilage breakdown.

Improving muscular stabilization and preventing knee stiffness are the fundamental principles of the nonoperative treatment. Low-impact exercises and weight loss must be advocated. Indeed, besides improving strength and range of motion, these activities increase the feeling of well-being without adding any side effects. Physical therapy is another excellent strategy, which must focus not only on stretching and strengthening the knee muscles but also include the hip and core musculature. In addition, non-pharmacological measures to reduce the inflammatory complaints, such as ice bag application and/or moderate warmth, should be encouraged. Braces can contribute to reduce the symptomatology as they tend to alleviate the pressure of the affected compartment by transferring the mechanical axis to an unaffected weight-bearing area in the joint. Medications, such as steroidal and non-steroidal anti-inflammatory drugs (NSAIDs), are frequently used, showing good results in terms of pain and edema control, with relatively few side effects. Intra-articular injections of viscosupplement therapy or, more recently, platelet-rich plasma, bone marrow aspirate concentrate, and stem cells have been increasingly applied in clinical setting, although their efficacy remains to be clarified.

Surgical Treatment

The surgical management of cartilage defects is based on the lesion characteristics, in particular the etiology and chronicity of the defect, the number, size, depth, location, and underlying bone involvement, as well as the patient's background, including age, BMI, smoking habits, duration of symptoms, and previous traumas or surgeries (Table 26.1). The status of the surrounding articular cartilage is also crucial to dictate therapy. Moreover, associated abnormalities must be addressed in order to achieve success in the surgical treatment. In this regard, correcting knee instability due to ligament injuries, meniscus insufficiencies, and tibiofemoral or patellofemoral malalignment is as important as the cartilage treatment by itself.

Table 26.1 Summary of the management of cartilage injuries according to the lesions depth and size

Diagnosis	Presentation	Management	
		Small defects	Large defects
Articular cartilage defect	Partial-thickness cartilage defect	Nonoperative treatment Consider surgery if symptoms persist	
	Full-thickness cartilage defect (not extending through the subchondral bone)	Marrow stimulation techniques ACI	ACI Particulated juvenile allograft cartilage
	Full-thickness cartilage defect (extending through the subchondral bone plate)	OAT	OCA Aragonite-based osteochondral scaffold
Associated abnormalities must be properly treated (ligament instability, meniscus insufficiencies, and tibiofemoral or patellofemoral malalignment)			

Joint Debridement

Articular debridement is a wide term that includes chondroplasty, removal of loose bodies, articular abrasions, and synovectomy. The magnitude of this treatment depends on the size, number, and location of the cartilage lesion. Generally, articular debridement is performed to remove unstable flaps and loose bodies in patients with small lesions and with lower demands or, eventually, in athletes through the season as an interim procedure. Attention must be taken to maintain the integrity of the adjacent healthy cartilage and the subchondral bone. There is no evidence showing that joint debridement is truly effective to treat osteoarthritic patients. However, this kind of treatment is reasonable to treat symptomatic patients with unstable tissue and may offer particular patients, such as professional athletes, short-term bridging options.

Marrow Stimulation Techniques

The aim of MST is to induce the migration of potential repair cells into the chondral/osteochondral lesion. By stimulating bleeding using a sharp material into the vascularized bone marrow, stem cells are allowed to move to the cartilage defect. These cells induce a fibrocartilage tissue formation, which tends to fill the defect. However, this fibrocartilage tissue, mainly formed by collagen type I, has inferior biomechanical properties than the native hyaline cartilage (type II collagen), with reduced stiffness and higher predilection for deterioration over time.

Drilling, microfractures, and, more recently, nanofractures, which use smaller-diameter and deeper subchondral bone perforations, are described as MSTs. Microfracture is the most frequently performed surgical procedure to treat focal chondral injuries, as it is a relatively easy and accessible technique with low cost. In this method, all unstable pieces of cartilage are removed, creating a well-contained defect ideally surrounded by a stable and perpendicular-edge cartilage. In addition, a complete exposure of the subchondral bone is required. Multiple fracture holes of approximately 3–4 mm apart and 4 mm in depth are created using a specific device that penetrates the subchondral plates, allowing bone marrow cell migration and clot formation. A systematic review has shown satisfactory outcomes after microfracture in a short-term period follow-up, particularly in young subjects with small defects. However, the positive results tend to be short lived and decline over time. Additionally, bone overgrowth was observed in more than 60% of cases, which may increase the risk of failure.

The nanofracture technique is a new concept based on the same principles of microfractures but uses thinner devices (awls 1 mm-thick) that produce controlled and deeper perforations of 9 mm depth, which seems to preserve the trabecular bone. In fact, benefits of the nanofracture over the usual microfracture were demonstrated in an animal model study, but clinical evidence is still lacking.

Marrow stimulation should not be opportunistically performed in all small defects. Indeed, recent guidelines suggest that even smaller lesions should be treated using cell-based therapies rather than microfracture. In our practice, marrow stimulation technique is only indicated in young, athletic patients, with isolated and acute defects less than 2 cm^2 in the femoral condyle.

Autologous Matrix-Assisted Chondrogenesis

The use of biodegradable hydrogels or three-dimensional scaffolds to improve the clot stability has also been studied. Autologous matrix-assisted chondrogenesis (AMIC) combines microfracture with the use of a collagen scaffold, aiming to enhance the mechanical stability of the clot along with stimulating the chondrogenic differentiation. A randomized, controlled clinical trial showed superior quality and quantity of the repaired tissue using the AMIC technique in comparison to the microfracture alone; however, no differences in clinical outcomes were observed. To date, there is no sufficient evidence to indicate these techniques over the established procedures.

Osteochondral Autograft Transplantation

Osteochondral autograft transplantation (OAT) is a therapeutic modality best indicated for osteochondral defects that are less than 2 cm^2. OAT consists in harvesting cylindrical osteochondral plugs from a low-weight-bearing area of the

knee – usually the intercondylar notch or the trochlea's periphery – and transfering these plugs to fill a chondral defect. Multiple plugs can be used as a mosaic (i.e., mosaicplasty) in case of larger defects. The major advantage of OAT is that the lesion is promptly filled by mature, hyaline cartilage using an autologous tissue. In addition, for being an osteochondral plug, the unhealthy underlying subchondral bone can also be addressed. In case of numerous plugs, the gaps between the plugs and the native cartilage are filled by a fibrocartilage tissue. The difficulty in creating a congruent surface between the donor explant and the defect is a limitation of the OAT. Moreover, because of the increased donor site morbidity, the procedure is not recommended to cover large defects.

Generally considered a technically difficult procedure, osteochondral autograft transplantation yields good-to-excellent results which can be achieved in terms of durability and functional outcomes as long as defects are not too large and only single plug grafts are utilized. Indeed, a systematic review including more than 600 patients who underwent OAT to treat knee osteochondral injuries showed improved clinical outcomes, with an overall rate of implant survivor of 72% in a mean follow-up higher than 10 years. Increased rates of failure were observed in cases of previous surgery, older age (>40 years), women, and defects greater than 3 cm^2. Randomized clinical studies have shown that, when compared to microfractures, OAT presented better results in young active patients.

Failures related to the OAT technique are usually associated with errors in the plug harvesting or implantation. Inadequate restoration of the joint surface congruity leads to biomechanical wear of the implants, leading to worse graft incorporation, bony resorption, and cyst formation. In addition, chondrocytes in the plug periphery might not be viable after harvesting, which can impair the lateral integration of the plug. A randomized, controlled clinical trial has reported inferior results of OAT in comparison to ACI in defects over 2 cm^2.

Osteochondral Allograft Transplantation

Osteochondral allograft (OCA) transplantation is a versatile procedure that uses allograft tissue permitting the treatment of large osteochondral defects with a best-matched osteochondral explant. OCA transplantation has been used consistently over the last 40 years in the United States, and its use is still expanding as a better comprehension of the graft incorporation physiology and improved conditions to enhance graft survivorship are developed. These grafts are strictly regulated and tested by FDA-certified tissue banks. OCA's are usually implanted after a minimum period of 14 days post harvesting, allowing time for numerous tests including culture results and donor history assessments. Since viability of the graft is known to decrease over time after harvest, there is urgency to perform the procedure soon after the finalization of the safety test and typically within 28 days of harvest.

Current indications for OCA transplantation include large focal chondral/osteochondral lesions, failure of previous cartilage repairs, osteochondritis dissecans, osteonecrosis, and post-traumatic osteochondral lesions.

The success of OCA depends on adequate subchondral bone integration and remodeling of the allograft and the surrounding host bone. Several studies have shown good clinical outcomes using this technique to treat high-demand patients with both focal and diffuse osteochondral injuries.

In addition, the excellent rate of survivorship reported (82%) at 10 years follow-up encourages the use of OCA transplantation. More complex injuries requiring multiple grafts may present higher failure rate and need for revision in comparison with single graft. Even so, the overall improvement in patient outcomes justifies use of this technique. High cost, waiting time, and limited availability might be limiting factors for the OCA transplantation in many centers worldwide. However, substantial effort has been given to improve OCA preservation and increase its accessibility worldwide.

Autologous Chondrocytes Implantation

ACI technique comprises a two-stage procedure, in which an initial arthroscopy is performed to diagnose and evaluate the defect (size and location) and collect a small piece of healthy cartilage for biopsy that will serve as a chondrocyte culture. The biopsy is performed in a low-weight-bearing area of the knee, commonly the intercondylar notch. Next, chondrocytes are isolated and expanded in vitro and stored until their implantation. In the second-stage procedure, the lesion's bed is prepared by removing any fibrous tissue, and a contained defect surrounded by vertical edges of normal cartilage is made. Prior to 2016, it was necessary to create a sealed chamber using a collagen 1/3 porcine-based membrane or periosteum. A chondrocyte suspension was injected underneath the scaffold to deliver the cells into close proximity of the subchondral bone.

Since 2016, a third-generation ACI, the so-called matrix-assisted chondrocyte implantation (MACI), is available (Fig. 26.4). This technique constitutes a substantial improvement of the technique. It consists of a hydrated collagen 1/3 porcine scaffold carrying suspended expanded chondrocytes that is directly implanted in the defect and secured with fibrin glue. By acting as a cell transporter, MACI allows a more equal distribution of chondrocytes in the defect. Besides that, MACI is an easier and quicker procedure in comparison to the other ACI generations. Currently, MACI is the only cell-based cartilage therapy approved by the FDA.

A usual indication for ACI is patients with cartilage defects greater than 2 cm^2, when the subchondral plate is intact. Since it is a membrane technique, it can easily be used for defects of different contours and size and, therefore, can be used for both femoral and patellar lesions.

MACI represents one of the most common cell-based therapies currently employed to treat large full-thickness defects of the femur or patellofemoral

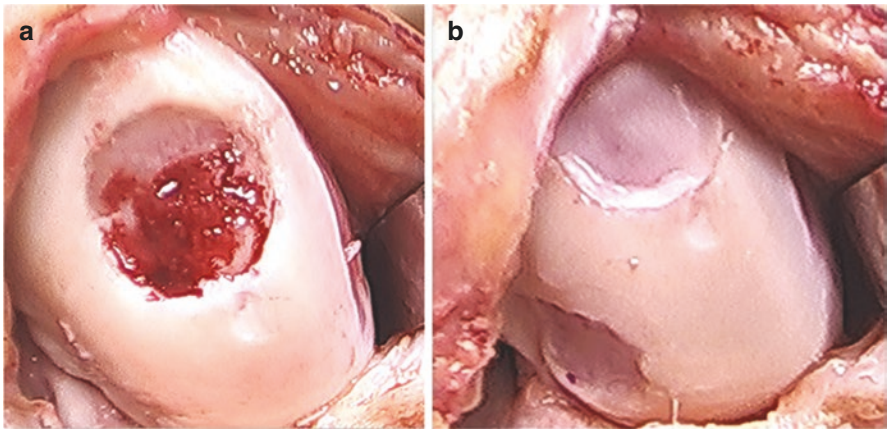

Fig. 26.4 Intraoperative imaging demonstrating a focal chondral defect in the femoral condyle (**a**) repaired with MACI technique (**b**)

compartment. Indeed, because of the unique anatomy of the patellofemoral joint, MACI appears to be an excellent option considering the difficulty patellofemoral anatomy.

MACI leads to good postoperative outcomes with a survivorship as good as 78% at 5 years. Regarding clinical outcomes, long-term studies have shown better functional results of ACI/MACI in comparison to OAT. When evaluating larger defects, significant functional improvement and satisfactory survival rate of 71% in 10 years follow-up have been reported in lesions sizing 8.4 cm^2 in average. The major disadvantages are that ACI/MACI requires two-stage procedures, long rehabilitation periods, and elevated financial costs.

Particulated Juvenile Allograft Cartilage

Particulated juvenile allograft cartilage (PJAC) consists in the implantation of allograft juvenile chondrocytes suspended in their native extracellular matrix. Considering that immature cartilage potentially has increased chondrogenic activity in comparison to the adult cartilage, PJAC fits as a promising option for cartilage restoration. In fact, when there is no immunological reaction, juvenile chondrocytes have demonstrated a faster growth and 100-fold increase in proteoglycan synthesis when compared to adult chondrocytes. This procedure can be indicated to treat focal, contained chondral defects or in combination with OAT or OCA transplantation, aiming to cover the gaps left behind. Frequently, PJAC is indicated for patellar defects after a failed nonoperative treatment, given its ability to fill different lesion shapes. Similarly to the ACI technique, PJAC requires a prior preparation of the lesion, with debridement of any fibrous tissue and stable vertical walls surrounding the defect. The subchondral bone should be intact.

A positive aspect of PJAC when compared to ACI is that the PJAC is a one-stage procedure. Short-term follow-up studies have shown a significant improvement in the symptomatology of patellar defects treated with PJAC. In addition, better functional outcomes, hyaline-like cartilage formation on histological evaluation, and near-to-normal cartilage repair findings on MRI have been demonstrated. However, randomized controlled studies comparing this technique with other cartilage restoration methods are still lacking.

Aragonite-Based Osteochondral Scaffold

Recently, innovative technologies have been developed to improve the chondral or osteochondral regeneration by using materials with interesting biological and mechanical properties. Aragonite-based osteochondral scaffold (Agili-C™, CartiHeal Ltd) is a cell-free, biodegradable, and biphasic scaffold that, by stimulating the growth of cartilage and subchondral bone, restores the osteochondral unit to the original structures of the tissue. The mechanism of action for this technique is based on the promotion of bone marrow stem cell adhesion and differentiation and chondrocyte migration and proliferation from the healthy surrounding native cartilage.

CartiHeal is indicated to treat osteochondral defects in both degenerative and non-degenerative joints, including large condylar defects, given that different sizes and shapes of this implant can be designed. Preclinical studies have shown that CartiHeal is able to induce cartilage repair and regeneration, resulting in hyaline cartilage formation and subchondral bone regeneration. These findings were further verified in preliminary clinical trials.

Postoperative Rehabilitation

Protocols for patient rehabilitation post-surgery are commonly designed depending on the performed cartilage repair technique. Regardless of the technique, the first week usually focuses on improving the range of motion to prevent knee stiffness. Also, patellar mobilization, proximal core, and quadriceps strengthening are encouraged. The weight-bearing may vary according to each surgical procedure. Overall, right after the joint debridement/chondroplasty, weight-bearing as tolerated is allowed. When the surgery involves cartilage restoration, complete weight-bearing in full extension is permitted 2 weeks post-procedure. Given the need of time for tissue maturation post-MSTs, ACI, and particulated juvenile allograft, cartilage requires at least 6 weeks of protected weight-bearing, which can be progressively increased to full weight-bearing. On the other hand, by providing immediate hyaline cartilage, osteochondral autograft and allograft techniques theoretically permit a faster recovery to full weight-bearing. Indeed, some surgeons allow full bearing within the first 6 weeks post-surgery, although this is not a consensus.

Conclusion

Articular cartilage defects can be treated successfully in most patients. Nevertheless, cartilage repair remains a challenging and ever expanding field of clinical care and research. Depending on the size, depth, and location, and the patient's history and characteristics, individualized therapy is necessary for optimal results. While several techniques have been developed to treat chondral and osteochondral defects in the knee, there is no single best therapy. Moreover, cartilage injuries do not exist in isolation, and a full organ view of the lesion (i.e., limb malalignment, ligament or meniscus insufficiency, synovial inflammation, etc.) must be taken into account in order to successfully treat the chondral defect, the joint, and the patient. Importantly, the surgeon and patient's expectations must be aligned to ensure a successful biological management of the joint disease.

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Chapter 27
Meniscal and Ligamentous Injuries of the Knee

Simon Goertz, Emily M. Brook, and Elizabeth G. Matzkin

The Meniscus

The meniscus is a semilunar fibrocartilaginous structure that articulates between the femoral condyle and tibial plateau on the medial and lateral aspects of the knee (Fig. 27.1). The meniscus is an important structure for both load sharing and stability of the knee joint.

Summary of Epidemiology

Meniscal injuries are a common source of knee pain and disability that can occur in traumatic or nontraumatic settings. In general, the medial meniscus is more commonly affected, particularly in chronic conditions. Traumatic meniscal tears most frequently occur in the young and active individuals aged 15–45. Over a third of traumatic meniscal tears are related to cutting or pivoting maneuvers. Nontraumatic or degenerative meniscal tears most frequently present in individuals over 45 years of age and are often associated with degenerative joint disease (DJD). Individuals

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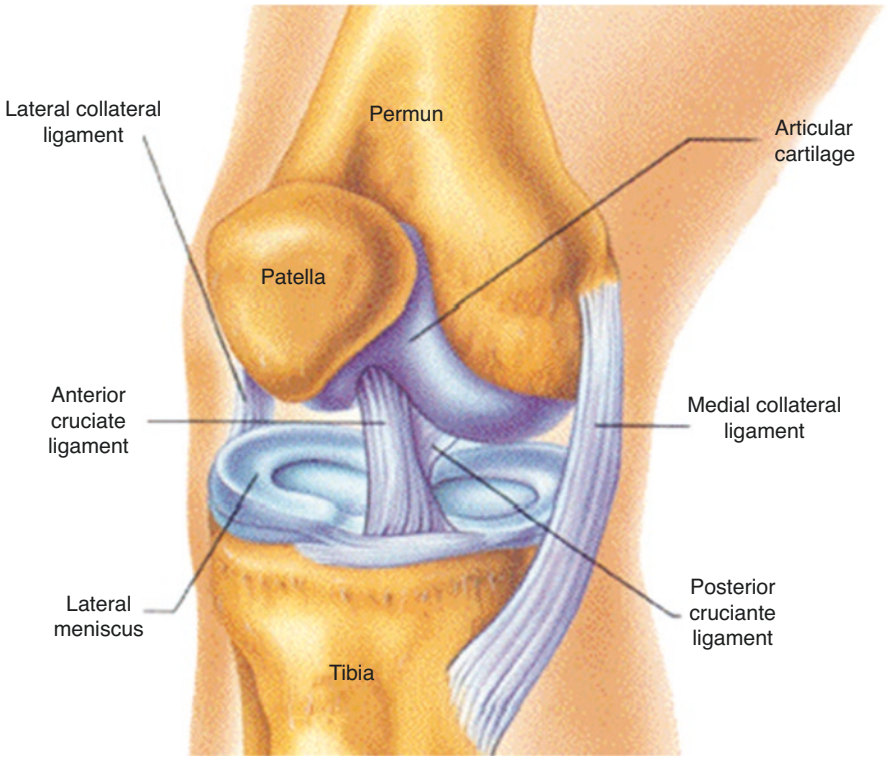

Fig. 27.1 The anatomy of the knee joint. (*Note*: Image below serves as a guide for Springer illustration. We would like the medial and lateral menisci, ACL, PCL, MCL, and LCL shown in the illustration)

with a higher body mass index (BMI) have a greater occurrence of degenerative meniscal injury due to increased weight-bearing forces on the knee.

Clinical Presentation

1. *Traumatic Meniscal Tears*

Traumatic meniscal injuries are common among the young and active population, causing pain, loss of motion, and limitations in function. Common mechanisms of injury are noncontact deceleration and cutting or pivoting movements. These mechanisms of injury can be quite traumatic to the knee joint, and often there is concomitant ligamentous injury. Traumatic meniscal tears commonly present with an effusion, pain, mechanical symptoms such as a locking or catching, or a feeling of instability, especially if a ligamentous injury is also present. After an injury mechanism described above, patients often have immediate onset of pain and

as a result are unable to continue activity or sport. Pain is localized to the overlying joint line although often the pain can be diffuse, particularly if a concurrent ligamentous injury or effusion is present. Individuals with a meniscal tear may report inability to fully flex or extend the knee because it is "stuck." Mechanical symptoms are usually caused by an interposed meniscal fragment between the femoral condyle and tibial plateau, causing a mechanical block and associated functional limitations.

2. *Degenerative Meniscal Tears*

Degenerative meniscal tears can be difficult to diagnose because of vague or variable symptoms in addition to the presence of concomitant DJD in the knee. Not all degenerative meniscal tears are symptomatic, and patients typically do not report any history of specific trauma or injury to the knee but often describe a gradual onset of symptoms associated with an active lifestyle. An effusion may or may not be present at the time of clinical presentation, although many patients report a history of swelling after an increase in activity level or duration of exercise. Degenerative meniscal tears typically present with pain and point tenderness localized to the posterior aspect of the medial or lateral joint line, correlating to the location of the tear. Mechanical symptoms such as catching and locking are not always present. Instead, the chief complaint may be activity limitation secondary to pain and/or swelling.

Physical Examination

Evaluation of knee pain starts with a thorough patient history and physical examination. It is important to gather information on whether the knee pain occurred in a traumatic versus nontraumatic setting and the specific mechanism of injury. A proper physical examination includes inspection, palpation, range of motion, and specific provocative tests for the suspected meniscal injury. The clinician should inspect both the unaffected and affected knee for effusion, soft tissue swelling, and ecchymosis. The ballottement test is performed by placing one hand above and the other below the patella and pushing inward with both hands to check for fluid in the knee and comparing it to the contralateral side.

Range of motion should be assessed with the patient supine on an examination table. A normal range of motion is 0° of extension and 135° of flexion. Tenderness to palpation along the medial and lateral joint lines should raise index of suspicion for a meniscal tear.

The McMurray test should be performed as part of a standard physical examination of the knee, particularly when meniscal injury is suspected. This is performed with the patient supine and the knee flexed to approximately 90° (Fig. 27.2). The examiner should have one hand over the patella with the fingers placed on the medial joint line and the thumb placed on the lateral joint line. The other hand is cradling the heel of the foot (Fig. 27.2a). To test for a medial meniscal injury, the examiner should rotate the tibia externally, applying valgus stress to the knee, and

Fig. 27.2 McMurray test for meniscal tears. The examiner should place one hand on the joint line and the other hand around the ankle or heel (**a**). To test the medial meniscus, the examiner should rotate the lower leg externally (**b**). For the lateral meniscus, the examiner should rotate the lower leg internally (**c**, **d**)

rotate the heel in flexion in an attempt to impinge and stress any injured meniscus tissue (Fig. 27.2b). Presence of a lateral meniscal injury should be tested by rotating the tibia internally, applying varus stress to the knee, and rotating the tibia in a similar fashion (Fig. 27.2c, d). A positive McMurray test is a "click" felt by the examiner along the joint line. However, a pseudo-positive test may not elicit a click, but rather pain with the motion. The sensitivity of the McMurray test is varied in the literature,

one study reported a sensitivity of 57% on the medial side and 77% on the lateral side in 121 patients with meniscal injuries. Another study noted that a positive McMurray sign is indicative of good postoperative outcomes in a cohort of 149 patients with a meniscal lesion and concomitant osteoarthritis. Another test for meniscal injury is the deep squat test. The examiner should have the patient perform a deep squat where the knee is loaded and flexed past 90°, which may cause an impingement of the posterior horn of the meniscus against the femoral condyle. However, if the patient has an acute onset of injury and a large effusion, he or she may not be able to perform the deep squat test.

Suggested Imaging

All patients presenting with traumatic or persistent knee pain should have plain radiographs obtained. Standing anterior-posterior, lateral, Merchant, and bilateral posterior-anterior weight-bearing flexion (Rosenberg) views should be obtained to assess for acute osseous abnormality and presence of DJD, which is associated with degenerative meniscal tears.

Magnetic resonance imaging (MRI) is particularly useful in patients with minimal or no knee osteoarthritis. A MRI should be considered in the presence of an acute onset of knee pain or persistent pain that has failed conservative treatment measures. In this study, the sensitivity of MRI in the detection of meniscal tears was 96% and the specificity was 97% with another showing that 97% of medial and 96% of lateral meniscal tears could be identified on sagittal MRI images alone. MRI is highly effective in determining the specific type, size, and location of a meniscal tear especially when planning surgical intervention.

Non-operative Management

Not all meniscal injuries require surgical intervention, and many degenerative meniscal tears that do not cause mechanical significant symptoms can be managed non-operatively. If a meniscal injury is diagnosed without it causing significant mechanical symptoms or limitations, non-operative management should be considered initially. It is important for the patient to understand that degenerative meniscal tears generally lack vascular access and thus have poor healing potential. Non-operative modalities such as physical therapy, regular use of ice and NSAIDS, and corticosteroid injections can help alleviate the symptoms of a chronic degenerative meniscal tear. Physical therapy to optimize proximal musculature control reduces the weight-bearing forces on the knee and can improve symptoms. An ice and NSAID regimen can help alleviate swelling and provide pain relief. Symptomatic episodes with effusion and pain can be treated with an intra-articular corticosteroid injection, often allowing the patient to continue physical therapy or home exercise program.

Outcomes of Conservative Treatment Measures

1. *Traumatic Meniscal Tears*

Traumatic meniscal tears tend to occur in patients under the age of 40 and often cannot be managed effectively with conservative treatment. These tears often cause joint effusion and limitation of not only athletic activities but activities of daily living. Meniscal root tears often happen in acute-on-chronic scenarios and cause complete disruption of essential hoop stresses that render the meniscus deeply dysfunctional. Bucket-handle meniscal tears indicate that there is a flipped piece of meniscal tissue, causing a mechanical block of the knee and resulting in the inability of the patient to fully extend or flex their knee. These tears generally do not respond to conservative measures and require prompt evaluation, reduction, and surgical repair.

2. *Degenerative Meniscal Tears*

Physical therapy or structured exercise programs can be successful in reducing symptoms and improving function from chronic degenerative meniscal tears. A study comparing surgical intervention versus non-operative strengthening exercises in patients with degenerative medial meniscal tears showed that both the operative and non-operative groups had significant pain relief and improved function at 2 years follow-up. Another study of patients with degenerative medial meniscal tears showed that a 6-week course of analgesics combined with a formal exercise program provided pain relief and improved function up to 6 months after initial diagnosis but began to decline long term. The study found that osteoarthritis continued to progress and was associated with worse outcomes long term. Current literature suggests an initial non-operative treatment protocol consisting of analgesics and a formal or home exercise program before considering surgical intervention in the treatment of chronic degenerative meniscal tears, at least in the absence of significant mechanical symptom component.

Indications for Surgical Intervention

Patients presenting with a traumatic meniscal injury with pain, mechanical symptoms, and a MRI confirming a torn meniscus may require surgical intervention. Bucket-handle-type meniscal injuries necessitate surgical intervention to reduce the displaced meniscus and restore range of motion. Bucket-handle tears are often amenable to suture repair unlike many other meniscal tears. These tears are commonly associated with a ligamentous injury, such as an ACL tear. Bucket-handle tears are best repaired surgically in a timely fashion to prevent plastic deformation and to preserve as much meniscal tissue as possible. Radial meniscal root tears commonly affect the posterior horn of the medial meniscus and lead to a complete disruption of the circumferential hoop stresses that convey the load sharing properties of the meniscus. Failure to restore

the meniscal root usually leads to extrusion of the torn meniscus and a biomechanical environment that functionally behaves akin to a subtotal meniscectomy, often followed by rapid arthritic degeneration of the affected compartment.

For degenerative meniscal tears, surgery may be indicated if non-operative modalities such as physical therapy or corticosteroid injections have failed. Patients with degenerative meniscal tears who have persistent mechanical symptoms with no or minimal osteoarthritis may elect arthroscopic surgery in order to debride unstable meniscal fragments. Degenerative meniscal tears are often associated with some degree of DJD. Therefore, it is important that patients understand that the symptoms of DJD may not be reliably alleviated with an arthroscopic surgery; however, symptoms stemming from a meniscal tear can be improved.

Operative Management

If surgical intervention is indicated, patients will undergo an arthroscopic meniscal repair or meniscectomy generally performed through a small anteromedial and anterolateral incision. A diagnostic arthroscopy assesses the size and location of meniscal tears (Fig. 27.3a, b) in addition to any degenerative changes to the cartilage or any ligamentous injury. The surgeon will evaluate the shape and size of the meniscal injury and determine if the meniscus can be successfully repaired or should be debrided. Arthroscopic meniscal repair involves the passing of sutures and/or fixation devices through the meniscus at the location of the tear to reapproximate the torn edges and restore the form and function of this important tissue. Complex degenerative meniscal tears are usually irreparable, and a partial meniscectomy is most often appropriate. In partial meniscectomy, mechanical biters and/or an arthroscopic shaver is used to debride the torn and unstable edges to a stable rim of meniscal tissue.

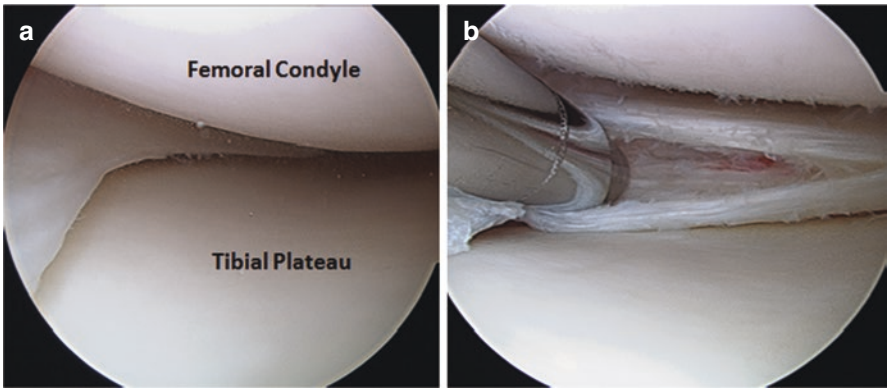

Fig. 27.3 Arthroscopic image of healthy meniscal tissue (**a**). Arthroscopic image of a meniscal tear occurring in a traumatic setting (**b**)

Expected Outcome

1. *Surgical Intervention for Traumatic Meniscal Tears*

Well-indicated and performed meniscal repair has generally high success rates reported in the orthopedic literature, regardless of technique. A systematic review of 27 studies found no significant differences in clinical failure rates between inside-out and all-inside repairs, 11% vs 10%, respectively. It is thought that preserving meniscal tissue by using repair techniques versus debridement provides better long-term outcomes in young patients as the meniscus can continue to maximize its anatomic function in weight-bearing with a repair; however, long-term data remains limited.

2. *Expected Outcome for Degenerative Meniscal Tears*

Data suggests that conservative treatment measures provide adequate pain relief and improvement in function for degenerative meniscal tears. However, patients who fail non-operative modalities may elect arthroscopic intervention to treat a symptomatic degenerative meniscal tear. A recent study evaluated 150 patients aged 45–64 with degenerative medial meniscal tears and no evidence of osteoarthritis on radiographs. Patients who underwent arthroscopic partial medial meniscectomy had substantially more improvement in pain than those having non-operative therapy. A large randomized controlled trial evaluating 351 symptomatic patients with a meniscal tear and concomitant mild-to-moderate osteoarthritis randomized patients to a standardized physical therapy regimen or arthroscopic surgery with postoperative physical therapy. The investigators found no significant differences in the study groups in patient-reported outcomes at both 6 and 12 months after randomization. Of note, 30% of the patients who were randomized into the conservative treatment with physical therapy group crossed over and elected to undergo surgery within 6 months of randomization. There are inconsistencies in the literature on the efficacy of surgical intervention for a degenerative meniscal tear, precluding definitive recommendations. The body of evidence does suggest that physical therapy should be the first line of treatment and that a partial meniscectomy remains a potential treatment for those with persistent pain and functional limitation despite adequate conservative treatment. It is important for patients to understand that the surgery will not alleviate symptoms from early concomitant osteoarthritis in the knee.

Ligamentous Injury

The knee is comprised of four major ligaments, each of which contributes to stability of the knee joint during movement. The anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL) are located in the intercondylar notch. The ACL mainly stabilizes anterior tibial translation, while the main function of the PCL is to resist posterior tibial translation. The medial collateral ligament (MCL) is located on the medial aspect of the knee and connects the tibia and femur to prevent medial

gapping and medial translation of the tibia. The lateral collateral ligament (LCL) is located on the lateral aspect of the knee and prevents lateral translation of the tibia (Fig. 27.1). All four ligaments also play a role in rotational stability of the joint and work synergistically to control all planes of motion during weight-bearing activities.

Summary of Epidemiology

Ligamentous injury is a common injury to the knee during more demanding activities such as cutting, pivoting, or twisting. The ACL and MCL are the most commonly injured ligaments in the knee. It is estimated that over 200,000 ACL injuries occur annually in the United States, the majority occurring in sports that involve cutting and pivoting movements such as soccer, basketball, skiing, and football. Injuries to the ACL are most prevalent in young and active patients aged 15–45, with females at 2–8 times the risk of their male counterparts for ACL injury due to anatomical and biomechanical differences. MCL injuries also frequently occur in young and active patients who play sports that involve valgus stress, such as soccer, basketball, ice hockey, and football. MCL injuries such as sprains and partial tears are more prevalent than complete ruptures. PCL and LCL injuries can and do occur, but are far less common than ACL and MCL tears, particularly in isolation.

Clinical Presentation

1. *Anterior Cruciate Ligament Injury*

ACL injuries are prevalent among the young and active population and cause pain, swelling, mechanical symptoms, and instability. The majority of ACL injuries occur during a sport or physical activity that involves quick changes of direction. Common sports or activities associated with a high incidence of ACL tears include soccer, basketball, football, skiing, and lacrosse. The mechanism of most ACL injuries is non-contact. Many patients will describe an attempted cut, pivot, or landing from a jump where their knee subsequently "gave out." Some patients may describe a contact mechanism with hyperextension or player contact with the knee bending inward into valgus stress. One of the hallmark descriptions of an ACL tear is a non-contact rotational valgus stress, followed by an audible or felt "pop" in the knee. Most patients have an immediate onset of pain and swelling and are not able to continue activity. Acute ACL injuries will often present to clinic with a large effusion, loss of motion, pain, and anterior instability. Some patients are not able to weight bear at all after an ACL injury, others are able to ambulate with difficulty, and some are able to ambulate but feel overt instability. The mechanism of injury for ACL tears, a rotational force, valgus stress, or hyperextension, can also be associated with other injuries to the menisci or ligaments, particularly the MCL.

2. *Medial Collateral Ligament Injuries*

MCL injuries are another common knee injury and occur more frequently from a contact mechanism. A strong contact force to the outside of the knee that causes the knee to move inward into a valgus position puts stress on the MCL, causing a strain or tear. MCL injuries commonly result in a partial tear or sprain rather than a full-thickness tear or avulsion from the attachment site. A sprain may present with pain localized to the inner aspect of the knee with minimal swelling and no instability. Partial tears may present with moderate to severe pain, a sense of instability, and some swelling. Full-thickness tears often relate to severe pain, instability, loss of range of motion, and a large effusion.

3. *Posterior Cruciate Ligament*

PCL injuries are significantly less common than ACL injuries, and may go unrecognized. Anatomically, the PCL is more robust and stronger then the ACL. The most common mechanism of injury for PCL tears is a strong force to the anterior aspect of the knee while the knee is flexed. For example, a fall on a flexed knee, particularly with the foot plantar flexed, or flexed knee hitting the dashboard in a motor vehicle accident can result in a PCL injury. In more demanding activities such as contact sports, a force on the anterior aspect of the knee with hyperextension can also cause an avulsion injury to the PCL.

4. *Lateral Collateral Ligament*

Injury to the LCL is significantly less common than MCL injuries. The mechanism of injury is an excessive varus stress on the knee, causing the (postero-)lateral structures to strain or tear. Like injuries to the MCL, LCL injuries can range from a sprain to a full-thickness tear. Depending on the severity of the injury, patients may present from localized pain to the outermost aspect of the knee in a sprain or low-grade, partial tear to loss of range of motion and functional limitations in a complete tear.

Physical Examination

1. *Anterior and Posterior Cruciate Ligaments*

A thorough patient history is essential in order to determine what structure has been injured. For ACL injury, a specific incident or event is usually associated with the onset of knee pain. Many patients feel or hear a “pop” in the knee followed by extreme pain, immediate swelling, and inability to continue physical activity.

Inspect both the affected and contralateral knee for bruising or discoloration and obvious swelling. Determine if fluid is present in the suprapatellar pouch or knee joint by performing a ballottement test. Check for point tenderness; an isolated ACL injury may have diffuse tenderness, while an ACL injury combined with a collateral ligament or meniscal injury may be point tender over the medial or lateral joint line.

Test for range of motion on both the contralateral and affected knee to assess a baseline measurement (normal, 0–135°). Patients with a suspected ACL injury may have limitations in extension and flexion with moderate to severe pain.

The Lachman test is the most sensitive clinical examination test for ACL tears and measures the degree of anterior tibial translation. A proper Lachman test is performed with the knee at approximately 15° of flexion and slightly internally rotated. The examiner should stabilize the patient's distal femur with one hand approximately 3–5 cm above the patella and the other hand around the tibia with the thumb placed the proximal tibia and joint line to assess translation (Fig. 27.4a). The examiner should pull the tibia anteriorly while simultaneously stabilizing the femur to assess for anterior tibial translation. The Lachman test should first be performed on the contralateral knee to determine baseline tibial translation. A Lachman is quantified by the amount of tibial translation and the quality of the endpoint. An abnormal finding is highly predictive of a torn ACL.

Another specific test for instability of the knee is the anterior drawer. The anterior drawer is also used to assess for anterior tibial translation. The anterior drawer is performed while the knee is flexed 90° with the examiner's thumbs on the anteromedial and anterolateral joint lines (Fig. 27.4b). The examiner should stress the tibia anteriorly while keeping the thumbs steady on the joint line to determine the amount of translation. The anterior drawer should also be assessed on the contralateral side for a baseline measurement. An anterior drawer resulting in increased

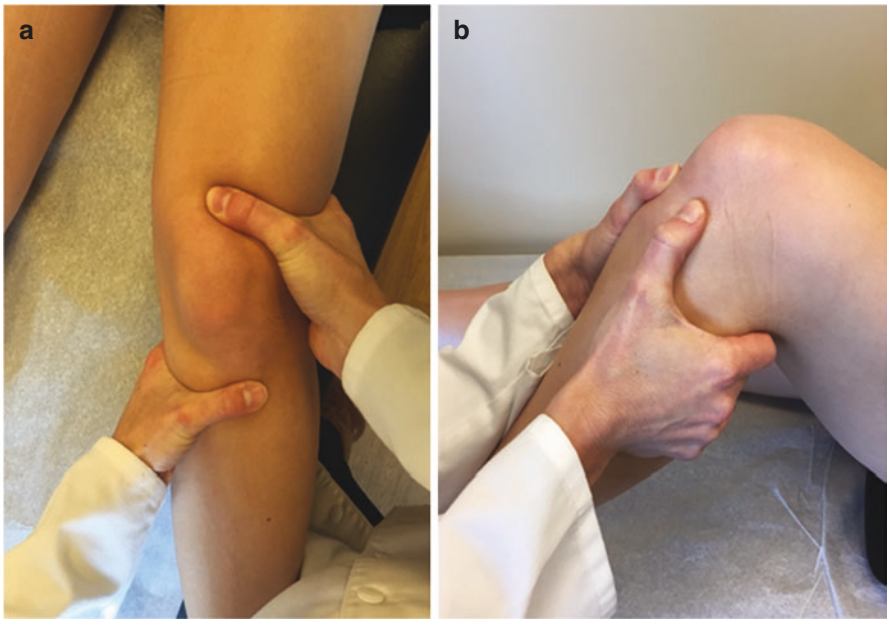

Fig. 27.4 Knee position and examiner hand placement for the Lachman (**a**). Knee position and hand placement for the anterior drawer and posterior drawer (**b**)

anterior tibial translation is also predictive of an ACL tear. Posterolateral and posteromedial structures can also be tested by internally or externally rotating the tibia during this maneuver, respectively. The posterior drawer test assesses posterior tibial translation and is sensitive to PCL injury. In a position similar to the anterior drawer test, the knee is flexed to about 90° with both thumbs placed on the anteromedial and anterolateral joint lines (Fig. 27.4b). The tibia is stressed posteriorly while the examiner feels for any increase in translation compared to the contralateral side. Any increased posterior tibial translation is suggestive of a PCL tear.

2. *Medial and Lateral Collateral Ligaments*

MCL injuries are common after excessive valgus stress on the knee with or without contact to the outside of the knee. An LCL injury can be suspected if a patient describes a varus force to the inside of the knee; by definition, varus mechanisms are less likely to be from direct contact. Inspection, palpation, and range of motion should be performed for every suspected knee injury as described in the anterior and posterior cruciate ligament physical examination section.

The examiner should palpate for point tenderness on the medial or lateral joint line and apply pressure along the native location of the MCL or LCL, from femoral insertion to tibial/fibular insertion. Placing the leg in a "figure four" position can help identify the LCL on the lateral side of the joint. Patients with injury to the MCL or LCL will feel pain and point tenderness along the ligament, not solely confined to the joint line.

The valgus or varus stress test is useful for determining a partial or complete tear of the MCL or LCL, respectively. The patient should lie supine, with their knee flexed approximately 30° to isolate the respective collateral ligament. The examiner should place their fingers over the corresponding joint line while stabilizing the distal femur. A valgus or varus stress is applied to the distal tibia, and the amount of medial or lateral compartment opening or gapping is noted and compared to the contralateral side. The severity of a collateral ligament injury can be quantified as follows: Grade 0, no pain, no gapping; Grade 1, pain, no gapping; Grade 2, gapping with endpoint; and Grade 3, gapping, no endpoint. The valgus/varus stress can be repeated with the knee in full extension. Opening to valgus/varus stress usually indicates a combined injury of the corresponding collateral ligament in addition to damage to the ACL. Also, it is important to remember that the MCL is an extra-articular structure. If a patient with a presumed MCL injury is also carrying an effusion, additional intraarticular injury should be suspected unless the MCL injury is a complete tear involving the joint capsule.

Diagnostic Imaging

Plain radiographs, including standing anterior-posterior, lateral, Merchant, and bilateral posterior-anterior weight-bearing flexion (Rosenberg) views, should be obtained to rule out evidence of fracture or bony pathology. For example, a Segond

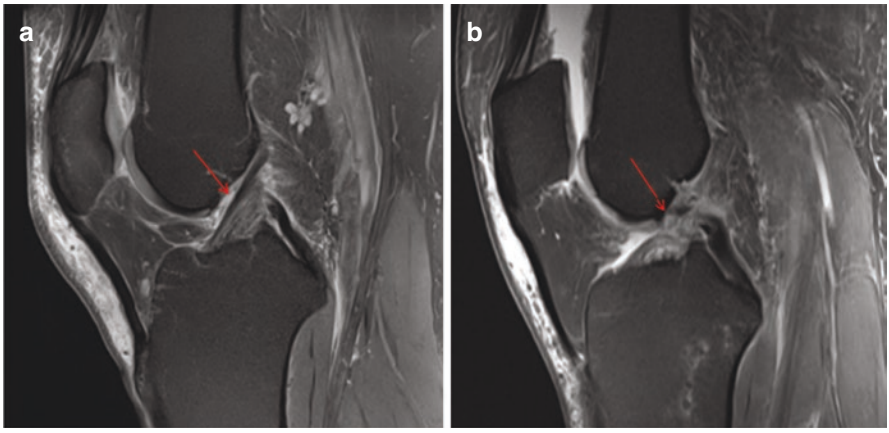

Fig. 27.5 MRI showing a healthy ACL (**a**) and a full-thickness ACL tear (**b**)

fracture is a small avulsion fracture of the lateral aspect of the tibia that infers injury to anterolateral capsular structures that often accompany an ACL injury. If fracture is ruled out and a ligamentous injury is suspected, an MRI should be ordered. MRI is the gold standard for confirming a ligamentous injury, as well as concurrent meniscal and cartilage injuries (Fig. 27.5a, b).

Non-operative Management

1. *Anterior Cruciate Ligament*

Patients with a torn ACL may be managed non-operatively or choose to have an ACL reconstruction. The majority of patients who participate in sports and are younger than the age of 35 opt for surgical reconstruction. However, ACL reconstruction is not for every patient. Patients with moderate-to-severe osteoarthritis are not candidates for ACL reconstruction as it can exacerbate arthritic pain. It is important to convey to the patient the function and purpose of the ACL and that a torn ACL will not impact most straight line movements such as jogging, cycling, or walking. Patients who are not as active and do not regularly participate in cutting and pivoting activities may have success with non-operative management. A formal course of physical therapy to reduce swelling and maximize proximal musculature strength often allows for the desired quality of life. Patients can also be fit for a functional ACL brace that provides added stability in cutting and pivoting activities such as tennis or skiing.

2. *Outcomes of Non-operative Management for ACL Injury*

Several studies have compared the outcomes of operative versus non-operative management for ACL injuries. One recent meta-analysis reviewed 13 publications

encompassing a total of 1246 patients; only two were reports of randomized controlled trials (RCTs). In one of the RCTs, ACL reconstruction was found to yield better functional outcomes than conservative management. The other RCT did not reveal any harm from initial expectant management, but the conservative-to-operative crossover rate in this trial was 51%. The functional outcomes were inconclusive. In six observational studies, knee function was significantly better after surgery; in seven others, it was not. Five out of nine analyses in which knee-joint stability was restored after surgery showed superior functional outcomes after ACL reconstruction compared to nonsurgical management. Three studies in which no satisfactory postoperative knee joint stability was found did not show any functional difference between surgery and conservative management. Overall, there was a trend in observational studies toward better functional outcomes after ACL reconstruction. As an average across studies, conservative treatment failed in 17.5% (±15.5%) of patients. Patients who are ACL deficient may be at risk for future instability episodes and subsequent injuries, causing a progression of osteoarthritis or further damage to the meniscus and cartilaginous structures of the knee. A recent study showed that individuals who were treated non-operatively for an ACL injury had a significantly higher risk of secondary meniscal tear and osteoarthritis. However, it is ultimately the patient's preference in the decision to manage an ACL injury operatively or non-operatively. Younger, adolescent patients and those who wish to participate in cutting and pivoting activities may wish to undergo a reconstruction for knee stabilization in physically demanding activities.

3. *Medial and Lateral Collateral Ligament*

The majority of collateral ligament injuries are sprains and partial tears that should be managed non-operatively. Patients who have an isolated sprain or partial tear of a collateral ligament should be braced if laxity is present and undergo a formal course of physical therapy for soft tissue modalities and functional rehabilitation.

Indications for Surgery

ACL or PCL reconstruction is indicated for young and active individuals who are unable to participate in their desired activities due to knee instability with activities. ACL injuries are often indicated for surgical reconstruction in contrast to the majority of isolated PCL injuries which are managed non-operatively. Knee dislocations resulting in multi-ligament injuries usually necessitate surgical intervention in appropriately demand-matched patients.

Patients with a full-thickness MCL or LCL tear that have recurrent feelings of instability or pain that limits their desired activity level may be candidates for an MCL or LCL reconstruction. While most MCL or LCL tears are treated non-operatively, full-thickness tears that do not improve with non-operative modalities should be considered for surgical reconstruction. Also, distal MCL tears, while less common than tears at the femoral attachment, generally do not heal if the pes anserinus tendons become interposed and require primary repair or reconstruction.

Operative Management

1. *Anterior Cruciate Ligament Reconstruction*

Arthroscopic ACL reconstruction is generally performed through incisions located on the anteromedial and anterolateral aspects of the anterior knee using autograft or allograft tissue. Most common autograft donor sites are the patellar, pes anserinus tendons (semi-tendinosis and gracilis), or quadriceps tendons. An ACL reconstruction is performed by first debriding the torn fibers of the ACL and addressing any other concurrent meniscal or ligamentous injuries (Fig. 27.6a, b). After the graft has been prepared, a reamer is used to create a size-matched tunnel for the graft on both the femoral and tibial sides. The reconstructed ACL graft is introduced into the joint and fixed with an interference screw or cortical fixation device at both the femoral and tibial aperture. PCL, MCL, or LCL reconstructions follow a similar procedure of restoring anatomy with the help of a soft tissue graft fixed into bony sockets corresponding to their native footprint.

Expected Outcomes of Surgical Intervention

Patients undergoing a ligament reconstruction should expect a minimum rehabilitation period of 6 months. The newly reconstructed ligament takes approximately 3 months to heal into the bone, and rehabilitation is controlled until that point. While successful isolated ACL reconstructions do not require postoperative bracing, patients may be managed in a hinged brace and weight-bearing restrictions to protect concomitant procedures during the initial healing phase. After 3 months,

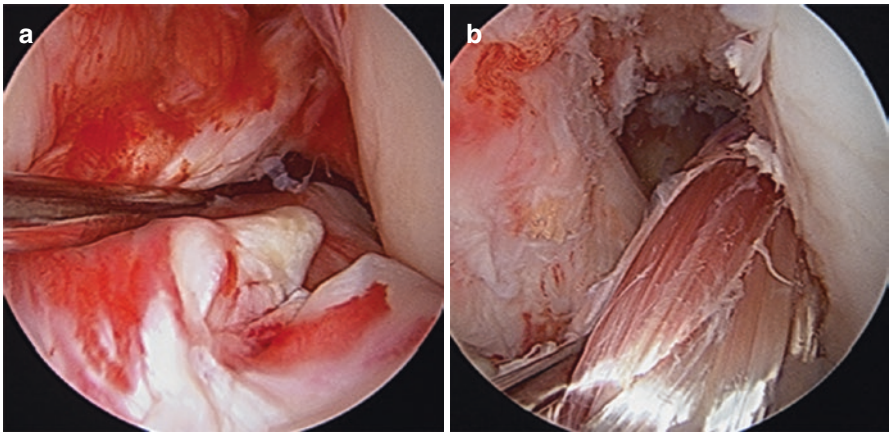

Fig. 27.6 Arthroscopic image of a ruptured ACL in the intercondylar notch (**a**). Arthroscopic image of a newly reconstructed ACL with hamstring autograft (**b**)

patients focus on sport-specific rehabilitation so that they may begin to resume their desired activities at 6 months.

Short-term outcomes from an ACL reconstruction are generally successful, with return to activity and relief of pain and instability symptoms after rehabilitation complete. In the young athlete under 14 years of age, a recent study found that 96% of athletes were able to return to sporting activity and 85% were able to return to sport within 12 months postoperatively. A study examining the rate of return to play in NCAA Division I football athletes showed that 82% of 184 players were able to return to pre-injury level of activity and 94% of starting athletes were able to return to play. Similarly, in a study of 80 NCAA Division I soccer athletes who underwent an ACL reconstruction, the overall return to play rate was 85%. In recreational athletes, a study found that 91.9% of patients (mean age 30) who underwent an ACL reconstruction were able to return to their initial recreational sport level at a medium of 36 months follow-up. Although dependent on level of activity and type of graft utilized, a large study of 17,346 ACL reconstructions found that 95.1% of patients were revision-free 5 years postoperatively.

Unfortunately, ACL reconstructions do not prevent the potential long-term consequences such as the early development of osteoarthritis 10–20 years after the procedure. A study of 135 patients with diagnosed ACL injuries found that the prevalence of osteoarthritis was three times higher in the group treated with a reconstruction at a 14-year follow-up. The group also found that concomitant meniscal resection further increased the risk of OA. A systematic review reported that osteoarthritis occurs in up to 13% of patients with an isolated ACL injury and reconstruction and an occurrence of 21–48% in patients with concomitant injuries. The likelihood of osteoarthritis increases in patients with combined ACL and meniscal injuries, highlighting the importance of both structures in long-term knee function. A recent study showed the development of osteoarthritis progressed faster in mature-aged adults (mean age, 40.2 years) compared to adolescent patients, with significant differences in the presence of OA 5 and 10 years after reconstruction.

Non-operative management of ligamentous injuries can result in good outcomes with appropriate rehabilitation (physical therapy), use of a functional brace as indicated, and limitation of cutting and pivoting at risk activities. The inherent laxity in non-operatively managed knees does increase the risk for additional injury, as recurrent episodes of instability can cause further damage to other ligaments, meniscus, or cartilage, as well as increasing the risk for early osteoarthritis. This, combined with the fact that ACL injuries have become increasingly common, highlights the importance for injury prevention programs to mitigate biomechanical and neuromuscular risk factors to reduce the incidence of ACL injury, particularly in the adolescent population. Clearly, additional research is needed to further optimize both preventive and therapeutic management strategies of ACL injuries (Table 27.1).

Table 27.1 Meniscal and ligamentous injuries of the knee

Clinical entity	Presentation	Physical examination	Conservative management	Indications for surgery	Operative management
Traumatic or non-degenerative meniscal tear	Knee effusion and pain Traumatic mechanism Mechanical symptoms such as locking and catching	Range of motion Joint line tenderness[a] McMurrays test Ballottement test for fluid	Ice and NSAID regimen PT for proximal musculature strengthening	Mechanical symptoms Limitations in daily activities Moderate-to-severe pain	Arthroscopic surgical intervention Depending on size and shape of the tear, meniscal repair vs. debridement
Degenerative meniscal tear	Knee effusion and pain Point tenderness over the medial or lateral joint line Mechanical symptoms such as locking or catching	Range of motion Joint line tenderness[a] McMurray's test Ballottement test for fluid Deep squat test	Ice and NSAID regimen PT for proximal musculature strengthening Corticosteroid injection(s)	Failed conservative management Mechanical symptoms Limitations in daily activity	Arthroscopic surgical intervention Partial meniscectomy and debridement of the torn edges Chondroplasty of cartilage surfaces if indicated
Anterior cruciate ligament tear	Non-contact mechanism involving a rotation or hyperextension of the knee Knee effusion Moderate-to-severe pain Feeling of instability	Range of motion Ballottement test for fluid Lachman[a] Anterior drawer	Ice and NSAID regimen PT for proximal musculature strengthening Functional brace to improve stability	Recurrent feeling of instability with daily activity Unable to achieve desired activity level due to pain or symptoms	Arthroscopic surgical intervention ACL reconstruction with autograft or allograft Address any other injuries
Posterior cruciate ligament tear	Contact mechanism involving an excessive force pushing the anterior aspect of the knee posteriorly Knee effusion Moderate-to-severe pain Feeling of instability	Range of motion Ballottement test for fluid Posterior drawer test[a]	Ice and NSAID regimen PT for proximal musculature strengthening Functional brace to improve stability	Recurrent feeling of instability with daily activities Unable to achieve desired activity level due to pain or symptoms	Arthroscopy surgical intervention PCL reconstruction with autograft or allograft Address any other injuries

(continued)

Table 27.1 (continued)

Clinical entity	Presentation	Physical examination	Conservative management	Indications for surgery	Operative management
Medial collateral ligament tear	Contact mechanism to the outside of the knee causing excessive valgus stress Knee effusion Pain localized to the medial aspect of the knee Moderate to severe pain Feeling of instability	Point tenderness over native MCL Ballottement test for fluid Limited range of motion if complete or high-grade partial tear Valgus stress test[a] MRI to confirm diagnosis	Ice and NSAID regimen PT for proximal musculature strengthening Functional brace to improve stability	Complete tear of the MCL Recurrent feeling of instability Unable to achieve desired activity level due to pain or symptoms	MCL reconstruction with allograft Address any other injuries
Lateral collateral ligament tear	Contact mechanism to the inside of the knee causing excessive varus stress Knee effusion Pain localized to the lateral aspect of the knee Moderate to severe pain Feeling of instability	Point tenderness over the native LCL Ballottement test for fluid Limited range of motion Varus stress test[a]	Ice and NSAID regimen PT for proximal musculature strengthening Functional brace to improve stability	Complete tear of the LCL Recurrent feeling of instability Unable to achieve desired activity level due to pain or symptoms	LCL reconstruction with allograft Address any other concurrent injuries

[a]Indicates most sensitive/specific test

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Chapter 28
Anterior Knee Pain: Diagnosis and Treatment

Natalie A. Lowenstein and Elizabeth G. Matzkin

Anterior knee pain is a common patient complaint yet still presents treatment challenges for physicians. With a broad differential diagnosis, identifying causes of anterior knee pain can be difficult due to both vague physical manifestations and psychosocial contextual overlap, which may skew the patient's symptoms and perception of pain. There are numerous discrete entities that can contribute to anterior knee pain; therefore, obtaining a thorough history and focused physical examination is essential. This chapter outlines the most common causes of anterior knee pain including patellofemoral pain syndrome (PFPS), patellar tendinopathy, quadriceps tendinopathy, and pes anserine bursitis. The relevant anatomical structures are shown in Fig. 28.1.

Patellofemoral Pain

Epidemiology

Despite its high incidence, the etiology of anterior knee pain can often be difficult to pinpoint as many terms are often used interchangeably to describe pain associated with patellofemoral symptoms.

In general, patellofemoral symptoms can reflect pain or instability, with some overlap between the two. Patellar instability, with true subluxation or dislocation, is different from patellofemoral pain and has a completely separate treatment algorithm. Patients complaining of pain, likely have normal patellar mobility, but

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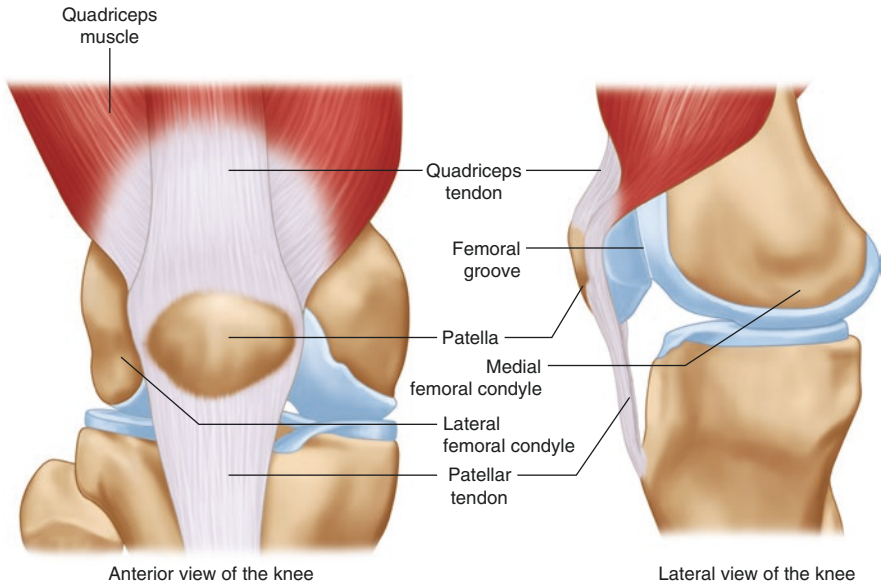

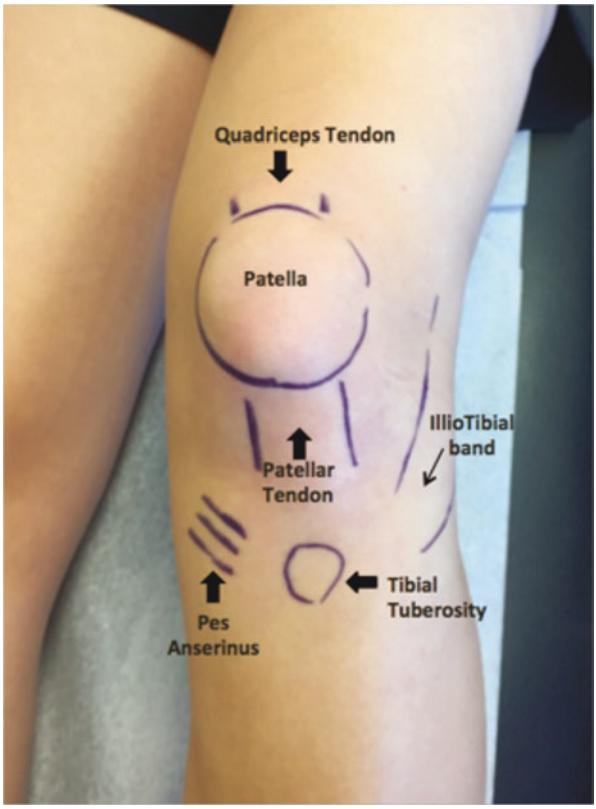

Fig. 28.1 Anatomy of the anterior aspect of the knee

present with symptoms that are aggravated by activity. Patellofemoral pain syndrome (PFPS) is generally classified as anterior or peri-patellar knee pain that occurs with activities that load the patellofemoral articulation, such as ascending or descending stairs. PFPS is more common in females and often follows changes in patients' activity levels, such as an increase in running mileage or adding squats and lunges to a gym workout routine. The increased prevalence in females is the result of biomechanical and anatomical factors that increase joint stress. For females, these factors consist of increased valgus stress, Q-angles, internal hip rotation, decreased external hip rotation, and lower cartilage thickness when compared to males. Some studies have suggested that nearly 15–40% of patients presenting to a sports medicine physician have PFPS; however, there remains little consensus on its etiology or the factors responsible for causing pain.

Clinical Presentation

The patient presenting to the physician with PFPS will often complain of pain along the anterior aspect of the knee with associated pain in the peri-patellar region or directly posterior to the patella. Complaints of swelling, weakness, mechanical symptoms, instability, and functional impairment are also common. These symptoms may be worsened by activities that require deep or prolonged knee flexion such as jumping, stair climbing, running, squatting, or sitting for an extended period of time. Patients may report vague pain with activity, occasional sharp or shooting pain around the anterior aspect of the knee or may complain that their knee feels like it will "buckle" or "give way."

A "buckling" or "giving way" sensation may be secondary to quadriceps inhibition and proximal muscle weakness. This contrasts with true patellar instability when the patella has subluxated or dislocated out of the trochlear groove. True patellar instability is most commonly secondary to a traumatic event and often in patients with underlying patellar malalignment and mal-tracking and/or patellar hypermobility. As a result of weak proximal muscles, the patella will have more direct contact with the trochlear groove, resulting in patellar crepitus, grinding, or pain.

It is important to get a good patient history to determine if the onset of symptoms occurred following a change in an exercise routine or a traumatic event. Patients with PFPS often report anterior knee pain after long periods of sitting. Sharp pain with twisting and pivoting, catching, locking, and recurrent effusions may indicate other pathology besides patellofemoral pain and should alert the physician that further diagnostic imaging with a potential referral to an orthopedist may be necessary. In particular, mechanical symptoms and/or knee effusion can suggest that a full-thickness cartilage defect may be the underlying cause of their anterior knee pain. A pre-patellar effusion could suggest pre-patellar bursitis (septic or aseptic).

Physical examination should begin with a comparison of the symptomatic and contralateral knee, looking for discoloration, bruising, effusion, and appreciable

atrophy. Palpation of the knee may demonstrate pain around the peri-patellar tissues and retinaculum. Patellar mobilization in the medial-lateral and proximal-distal direction can be tested as well as an assessment of patellar tilt and tracking throughout the patient's range of motion (Fig. 28.2a, b). Range of motion should also be assessed by manual flexion and extension of the patient's knee and noting differences between the asymptomatic and symptomatic maximums (Fig. 28.2e, f). When moving the patient's patella in an arc-like manner, the examiner should note any direction of increased laxity, tightness, or apprehension on the part of the patient (Fig. 28.2a, b). The presence of a "J-sign," which is a lateral deviation of the patella as the knee is brought from flexion to terminal extension, can signal an imbalance between the medial (vastus medialis) and lateral (vastus lateralis) muscles. If the vastus medialis activity is significantly less than the vastus lateralis, the imbalance between the two can lead to symptoms of anterior knee pain.

Proximal muscle strength testing is also a critical part of the clinical evaluation as muscular weakness and imbalance can lead to disrupted patellofemoral

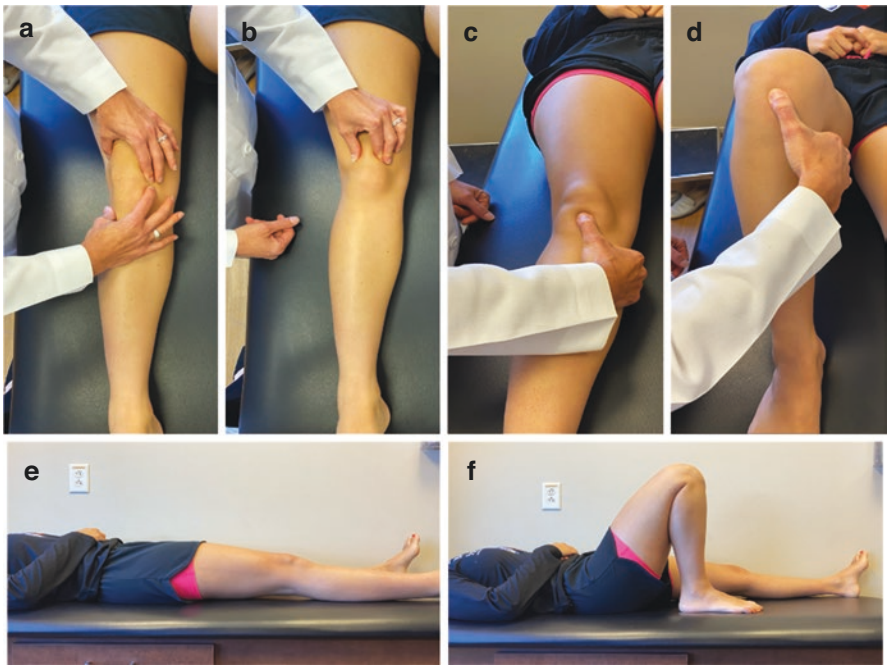

Fig. 28.2 Diagnostic testing. (**a**) Patellar apprehension: lateral pressure to patella results in patient apprehension. (**b**) Patellar compression: pressure on patella in trochlear groove with quadriceps activation. A positive test results in anterior knee pain. (**c**) Bassett's sign (extension): clinician applies pressure to patellar tendon attachment at the distal pole of the patella. Positive test if pain is present. (**d**) Bassett's sign (flexion): clinician applies pressure to patellar tendon attachment at the distal pole of the patella with knee in flexion. Pain is usually less than when in extension. (**e**) Range of motion (extension): patient is lying supine with knee in full extension (0°). (**f**) Range of motion (flexion): patient is lying supine with knee in full flexion (140°)

mechanics (Fig. 28.3). Quadriceps muscle weakness is common in PFPS patients (Fig. 28.3b). The examiner should assess not only quadriceps strength but also hip flexor (Fig. 28.3a), hamstring (Fig. 28.3c), hip abductor (Fig. 28.3d), and abdominal and lumbar core muscles. Weakness of the hip and core muscles can disrupt coronal plane mechanics leading to dynamic patellar mal-tracking.

Differential Diagnosis and Testing

Patellofemoral pain syndrome is a clinical diagnosis; there is no single imaging test or physical exam that establishes the diagnosis with certainty. As such, the clinician should rule out other causes of knee pain to include true patellar instability, patellar tendon or quadriceps tendon pathology, pre-patellar bursitis, chondral pathology, meniscus tear, loose bodies, osteoarthritis, radicular pain, or a systemic cause. The clinician should be wary of a diagnosis other than PFPS in a patient with knee pain and persistent painful mechanical symptoms or evidence of effusion. (Table 28.1).

Initial imaging studies of patients with PFPS would consist of plain radiographs including an AP, PA flexion, 30° flexed lateral, and bilateral merchants/sunrise

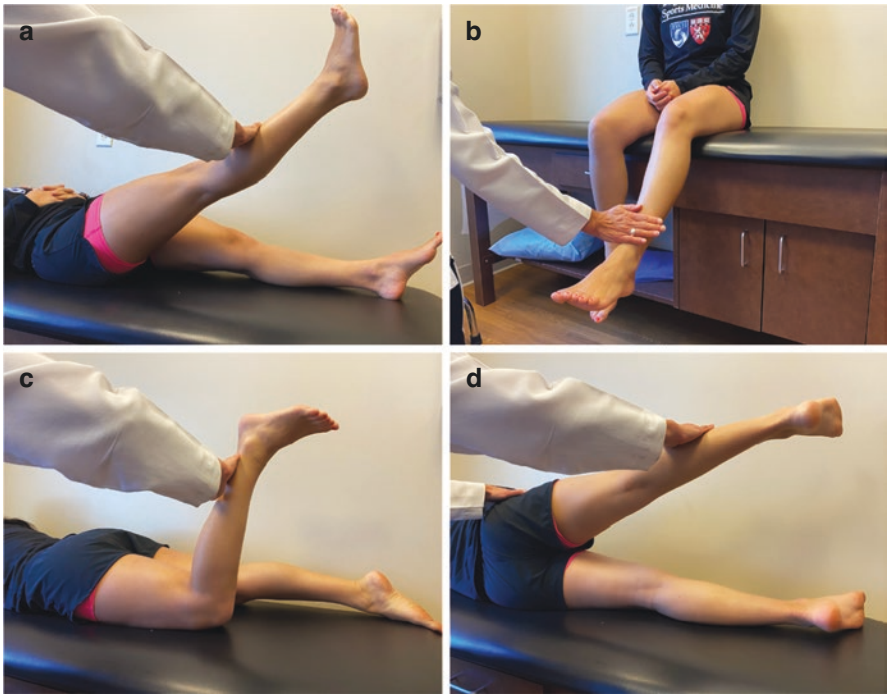

Fig. 28.3 Strength testing. (**a**) Hip flexor strength. (**b**) Quadriceps strength (sitting). (**c**) Hamstring strength. (**d**) Abductor strength

Table 28.1 Differential diagnosis of anterior knee pain

Diagnosis	Presentation	Diagnostic testing	Management	
			Conservative	Operative
Patellofemoral pain syndrome	Peri-patellar pain + "J-sign" Weakness in proximal muscles	Strength testing Plain radiographs (sunrise and lateral views) MRI to evaluate cartilage	NSAIDs, ice, physical therapy	Knee arthroscopy with cartilage debridement Tibial tubercle osteotomy (mal-tracking)
Patellar tendonitis	Tenderness over inferior pole of the patella and along patellar tendon + Bassett's sign	Clinical diagnosis MRI for confirmation	Activity modification, NSAIDs, ice, physical therapy	Tendon debridement, +/− microfracture/ drilling
Quadriceps tendonitis	Tenderness over superior pole of the patella	Clinical diagnosis MRI for confirmation	Activity modification, NSAIDs, ice, physical therapy	Tendon debridement +/− microfracture/ drilling
Pes anserine bursitis	Tenderness and local swelling 3–5 cm below the anterior-medial joint line	Clinical diagnosis	Activity modification, NSAIDs, ice, physical therapy, injection	Removal of bursa
Meniscus tear	Medial/lateral joint line tenderness Effusion + McMurray's Pain with deep flexion/squat	MRI for confirmation	Activity modification, NSAIDs, ice, physical therapy, injection	Knee arthroscopy with partial meniscectomy or meniscal repair
Osteoarthritis	Pain Stiffness Possible effusion	Plain radiographs (AP weight-bearing, sunrise, and lateral views) for confirmation of joint space narrowing, osteophytes, etc.	Activity modification, NSAIDs, ice, weight loss, physical therapy, injection	Total knee arthroplasty
Fracture	Tenderness, possible effusion	Plain radiographs, MRI, CT scan	Non-weight-bearing with crutches Activity modification, NSAIDs, ice, physical therapy	Open reduction and internal fixation

(continued)

Table 28.1 (continued)

Diagnosis	Presentation	Diagnostic testing	Management	
			Conservative	Operative
Patellar/ quadriceps tendon rupture	Palpable defect Effusion Inability to perform straight-leg raise	Clinical diagnosis Plain radiographs for confirmation of patella alta/baja MRI for confirmation of tendon rupture	Only pursue if surgical risks outweigh benefits	Patellar/ quadriceps tendon repair
Medial collateral ligament (MCL) sprain/tear	Tenderness over MCL Pain/laxity with valgus stress Effusion	Clinical diagnosis MRI for confirmation	Activity modification, NSAIDs, ice, hinged knee brace, physical therapy	MCL repair versus reconstruction

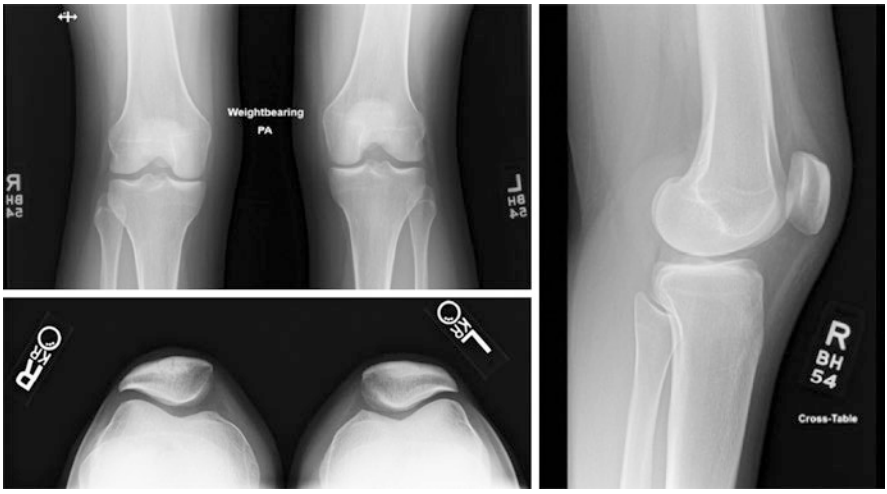

Fig. 28.4 Standard radiographic views of the knee (AP, sunrise/merchant, and lateral)

views (Fig. 28.4). The weight-bearing AP and PA flexion views allow for the assessment of osteochondral lesions and arthritic change in the medial and lateral tibiofemoral compartments. Lateral x-ray can provide important information similar to the coronal views as well as an assessment of patellar height and the presence of trochlear dysplasia.

Bilateral merchants/sunrise views allow for an evaluation of the patellofemoral joint including alignment, patellar tilt, and the presence of arthritis (Fig. 28.4).

Advanced imaging such as computed tomography (CT) and magnetic resonance imaging (MRI) is indicated in patients with PFPS who fail 3–6 months of conservative management. CT is useful for evaluating for any bony pathology as well as patellar height and the tibial tubercle-trochlear groove distance (TT-TG). The

TT-TG is a measurement of the distance between the tibial tubercle and the deepest part of the trochlear groove. A TT-TG of 20 mm or greater in patients signifies that the tibial tubercle is too lateral, and the resultant vector of pull of the extensor mechanism results in mal-tracking of the patella. MRI is valuable for assessing the chondral surfaces and subchondral bone for cartilage abnormalities (chondromalacia) or evidence of arthritic changes. MRI can also be used to quantify TT-TG distance. Both MRI and CT are good studies for analyzing trochlear morphology and dysplasia.

Non-operative Management

For patients experiencing PFPS, a majority will experience symptom improvement through conservative treatment. As a result, the first line of treatment should consist of a comprehensive rehabilitation management strategy including nonsteroidal anti-inflammatories; ice; physical modalities, such as taping and bracing; and physical therapy. Studies have demonstrated that 85% of patients improve with 8 weeks of appropriate physical therapy. The physician must be knowledgeable as to the correct protocols for PFPS rehabilitation as physical therapists differ considerably in the exercises they recommend. The initial goals of rehabilitation should consist of tactics aimed at reducing symptoms: including activity modifications and modalities to improve flexibility and patellar tracking. The restoration of normal knee mechanics with capsular stretching and vastus medialis strengthening has long been the primary focus of rehab protocols in the treatment of patients with PFPS.

Inflexibility of the soft tissues surrounding the knee has also been shown to correlate with anterior knee pain. Alongside strengthening exercises, stretching the gastrocnemius, proximal muscles (hamstrings, glutes, and quadriceps), and iliotibial band muscles is also recommended as part of non-operative treatment to minimize the tension put on the knee joint. As our knowledge has evolved of normal knee kinematics, the importance of the hip and core (abdominal/lumbar) musculature has emerged as another important aspect of treatment focus. Improving not only the strength but also the endurance of the hip and core muscles and correcting biomechanics and neuromuscular control have been shown to better maintain the kinematics of the extensor mechanism. For more active patients, physical therapy should incorporate functional and sport-specific training that allows a gradual increase in knee loading exercises while maximizing core strength and coordination.

Indications for Surgery and Operative Management

Surgical intervention for the treatment of PFPS is rarely indicated as most patients improve with conservative management. Accurate diagnosis is necessary for surgery to be successful. Patients with a cartilage injury on the underside of the patella and chondromalacia, or in the trochlear grove who have persistent pain following non-operative management, can often be treated with arthroscopic debridement consisting of a chondroplasty, microfracture, or cartilage resurfacing procedure. Should the patient fail or plateau after several months of dedicated rehab and exhibit evidence of mal-tracking, then further imaging studies should be performed to assess TT-TG distance and patellar height. Patients with evidence of lateral mal-tracking on exam with TT-TG distance greater than 20 mm are candidates for anteromedialization (AMZ) of the tibial tubercle combined with a possible proximal realignment procedure such as a lateral release or lengthening. In the past, many patients with lateral mal-tracking were treated with an isolated lateral release. This has fallen out of favor as many patients treated with a lateral release continued to have persistent pain and can even develop iatrogenic medial instability.

Current surgical treatment for lateral mal-tracking of the patella includes a tibial tubercle osteotomy, with or without a lateral release or lateral lengthening and vastus medialis advancement depending on glide and tilt, performed through a single anterior incision. This surgery requires a long postoperative rehabilitation. Patients are kept partial weight-bearing with crutches for ambulation until there is healing at the osteotomy site which can take an average of 6–8 weeks. Return to sporting or athletic activities takes a minimum of 6 months.

Expected Outcomes

With some time and effort, 85–90% of patients with anterior knee pain secondary to PFPS will improve with conservative management. With increased flexibility and strengthening of the core, hip, and thigh musculature, improved load transfer and knee kinematics will result in enhanced functional capacity and a decrease in symptoms. In patients who have persistent pain and evidence of mal-tracking, an AMZ tibial tubercle osteotomy can provide significant symptomatic relief as this sufficiently unloads the affected area and improves tracking. Results using an AMZ to treat patients with chondromalacia of the lateral facet and inferior pole have also been met with good to excellent outcomes in the majority of patients.

Patellar Tendinopathy

Epidemiology

Patellar tendinopathy is a common cause of anterior knee pain, especially in the younger population. It is also referred to as "jumper's knee" considering it occurs most commonly in athletes who participate in jumping sports, such as basketball and volleyball. Patellar tendinopathy is not limited to only jumping sports, as any activity that places a repetitive load on the patellar tendon can interfere with normal reparative properties of the tendon and can lead to tendinopathy. Both the proximal and distal patellar tendon attachments are subject to these repetitive loads and are commonly seen in young athletes with open growth plates. It is most frequently diagnosed in athletes from ages 15–30 years and is more common in males than females. The onset of pain is usually insidious. The condition begins with microscopic injury to the tendon, but due to repetitive loading or overuse and resultant delayed healing, patients experience symptoms.

Clinical Presentation

Patients with patellar tendinopathy will localize the pain over the anterior aspect of the knee, at the proximal pole of the patellar tendon (Fig. 28.1). More specifically, the pain is most predominantly confined over its insertion at the inferior pole of the patella. Patients mainly complain of pain and typically do not experience mechanical symptoms, such as locking or catching and swelling. The pain is usually worse with activity (placing an increased load on the tendon) and can be very limiting to an athlete. Going downstairs or sitting for an extending period of time may exacerbate pain.

On physical examination, tenderness to palpation is common over the inferior pole of the patella, although tenderness can occur anywhere along the patellar tendon down to its most distal attachment at the tibial tuberosity. Thickening of the tendon may be appreciated on palpation. Tenderness may be elicited when the knee is either flexed or extended, and in less severe cases, a deep squat or jump may be required to produce symptoms. A "Bassett's sign" is indicative of patellar tendinopathy and occurs when there is increased pain from palpation when the knee is extended and the patellar tendon is relaxed and is not painful or less painful when the knee is flexed (Fig. 28.2c, d). Knee effusion, however, is not common with tendinopathy. If a palpable defect is detected and the patient cannot perform a straight leg raise, this is concerning for a potential extensor mechanism disruption and needs immediate referral to an orthopedic surgeon for further workup and evaluation.

Quadriceps Tendinopathy

Epidemiology

Similar to patellar tendinopathy, quadriceps tendinopathy pain is usually insidious in onset and affects males more often than females. It is also very common in sports or activities that involve jumping which can lead to microscopic injury of the tendon. Quadriceps tendinopathy is significantly less common than patellar tendinopathy given the superior vascularization of the muscle, resulting in faster and more efficient healing.

Clinical Presentation

Quadriceps tendinopathy differs from patellar tendinopathy based on the location of pain. Though pain is also anterior, with quadriceps tendinopathy, the pain is localized over the attachment of the tendon at the superior pole of the patella (Fig. 28.1). Patients usually have increased pain with stairs and increased activity. Sitting for an extended period of time may also exacerbate pain. For patients with quadriceps tendinopathy, knee effusion and mechanical symptoms are not common.

The majority of patients will demonstrate tenderness to palpation just superior to the patella, without palpable defect. There is also frequent pain and/or weakness with resisted leg extension (Fig. 28.3b). Like the patellar tendon, if there is a defect where the tendon should be and the patient cannot perform a straight leg raise, immediate orthopedic evaluation is recommended as this is an indication of a probable extensor mechanism rupture.

Diagnostic Imaging

Patellar and quadriceps tendinopathies are most often diagnosed based on patient history and clinical examination. Plain radiographs are a good first choice of imaging to determine if there is underlying pathology including degenerative changes, calcification in the tendon, or patella mal-tracking that may be contributing to patients' symptoms (Fig. 28.4). MRI should only be pursued when conservative management has failed, there is suspicion of extensor mechanism rupture, or surgical intervention is the next option.

Non-operative Management for Patellar and Quadriceps Tendinopathies

Conservative management is the mainstay of treatment for both patellar and quadriceps tendinopathy, though it may take 3–6 months to fully resolve in some cases. Deficits in quadriceps muscle strength and neuromuscular control can lead to increased symptoms of anterior knee pain. As a result, neuromuscular training and physical therapy, to restore proper mechanics, play an important role in symptom reduction. More specifically, eccentric exercises, introduced once the tendon is not considerably irritable, typically enhance rehabilitation and ultimate return to sporting activity and regular exercise. Stretching and strengthening of the proximal musculature, including hamstrings, quadriceps, hip flexors, and hip abductors, also are important aspects of treatment and should be reevaluated upon follow-up (Fig. 28.3). Nonsteroidal anti-inflammatories (NSAIDs), ice, and activity modification are also beneficial (Table 28.1). Steroid injections are not recommended treatment for patellar and quadriceps tendinopathy. Surgical intervention is indicated rarely.

Operative Management of Patellar and Quadriceps Tendinopathies

Once it has been determined that conservative management has failed, usually after 6+ months with formal physical therapy, operative intervention is an option. At this point, an MRI should be obtained to definitively confirm the diagnosis and to determine if there is any other pathology. Knee arthroscopy can be performed to evaluate the intra-articular structures including the tendon itself and the undersurface of patella. For further operative management, an open patellar tendon/quadriceps tendon debridement with or without drilling/microfracture can be performed. Drilling or microfracture close to the tendon of the patella is performed to stimulate healing with increased vascularization to the area. Depending on the extent of debridement and quality of the tendon tissue itself, a patient may be put in a brace locked in extension for ambulation with weight-bearing as tolerated for the first 2–4 weeks postoperatively. Formal postoperative physical therapy will be beneficial in order for the patient to regain full range of motion and strength and return to all activities anywhere between 3 and 6 months, depending on the extent of the surgery.

Expected Outcomes

Though patellar and quadriceps tendinopathies may linger for many months, the majority of cases will resolve with conservative management as outlined above. Since the majority of patients should experience symptom resolution with

conservative treatment, it is important for the provider to determine that their patient is compliant with non-operative management before proceeding with surgical management. With more risk involved and a long rehabilitation, surgery is usually offered as a last option.

Pes Anserine Bursitis

Epidemiology

The pes anserine is located approximately 3–5 centimeters (cm) below the anteromedial joint line, where the semitendinosus, gracilis, and sartorius tendons attach. A bursa, a small, jelly-like sack that acts as a cushion to help reduce friction, is located at this attachment site, below the tendons. When this bursa becomes irritated and produces excess fluid, inflammation and swelling occur, either due to overuse or direct trauma, and result in pes anserine bursitis.

The exact incidence of pes anserine bursitis is unknown, though it is fairly common among the adult population. The most common causes are overuse injuries, particularly in athletes and runners; however, its etiology is multifactorial. Several studies have shown that overweight females are more at risk than their male counterparts. Patients with diabetes mellitus also have been shown to be at increased risk of developing pes anserine bursitis. Medial knee osteoarthritis is also commonly found in patients with this condition.

Clinical Presentation

On initial examination, the symptomatic knee should be evaluated in full extension. In this position, patients with pes anserine bursitis will localize the pain over the anteromedial aspect of the proximal lower leg, about 3–5 cm below the medial joint line (Fig. 28.1). There will be tenderness to palpation over this area which may extend along the proximal, medial tibial region, and usually, localized swelling will also be present. In some cases, resisted knee flexion at 90° may elicit pain in this area (Fig. 28.3b). Pain may also radiate into the posterior thigh or distal-medial calf. Increasing exercise, sitting with crossed legs, and going up and/or down the stairs may intensify pain to the area as well. A knee effusion may or may not be present in isolated pes anserine bursitis.

Given its location on the medial aspect of the knee, it is important that other causes of medial knee pain are excluded. Other diagnoses that can cause medial knee pain are medial meniscus tear, osteoarthritis, medial collateral ligament pathology, etc. (discussed in separate chapters) (Table 28.1).

Diagnostic Imaging

Pes anserine bursitis is diagnosed based on history and clinical examination. Plain radiographs will not confirm the diagnosis but are a good first choice in imaging in order to determine if there are any bony abnormalities or degenerative changes. MRI is rarely indicated but may be helpful if the diagnosis is uncertain, if there is suspicion of stress fracture, or in determining other soft-tissue pathology of the medial aspect of the knee. Ultrasonography may aid in the diagnosis, especially in cases where there is a significant amount of swelling, and may be used when administering an injection for diagnostic and therapeutic purposes.

Non-operative Management

Conservative therapy is the primary treatment method for pes anserine bursitis. Management options include ice, NSAIDs, activity modification, protective padding over the bursa, and physical therapy. For the long-term resolution of symptoms in the case of deconditioned, diabetic, or obese patients, proximal muscle strengthening and weight loss would also be beneficial. Physical therapy is predominately focused on hamstring stretching and strengthening. Formal physical therapy may also include modalities such as topical corticosteroid treatment (iontophoresis with dexamethasone) with the goal to decrease inflammation and pain. Injection with local anesthetic with or without corticosteroid into the bursa may aid in diagnosis and improve symptoms if there is a limited improvement from physical therapy management. The injection serves as a diagnostic tool in that if pain completely resolves after the injection, it can be determined that it was the sole pain generator. Similarly, if the injection provides no relief, then other causes of pain must be considered (Table 28.1). Non-operative treatment should be successful in the majority of cases, and surgical removal of the bursa is reserved for severe cases that fail to resolve.

Operative Management

Surgical intervention is rarely ever indicated for pes anserine bursitis. If conservative management fails, usually after 6+ months of conservative treatment, then operative treatment may be presented to the patient. The surgical procedure entails an incision over the pes anserine and drainage or removal of the bursa. If there is bone prominence under the bursa, this will also be removed at the time of surgery. Once the soft tissue is healed, patients will usually start a course of physical therapy and return to all activities at about 2–3 months postoperatively.

Expected Outcomes

Nonsurgical management is the mainstay of treatment for pes anserine bursitis and includes ice, NSAIDs, physical therapy, and local injection. Outcomes are typically excellent, especially if patients are compliant and limit activities that incite inflammation while undergoing non-operative treatment. On the rare occasion that conservative therapy fails, surgical intervention may be implemented. Surgical treatment is the last option given as it is associated with more risk. After pursuing surgical management, it is expected that patients will have a resolution of symptoms, and if not, secondary pathology should be considered.

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Part VIII
The Foot and Ankle

Chapter 29
Ankle Arthritis

Eric M. Bluman, Jeremy T. Smith, Christopher P. Chiodo, and Elizabeth A. Martin

Introduction

The tibiotalar joint, or ankle joint, is the major motion segment below the knee. It has an important role in locomotion. In addition to helping propel the body forward during ambulation, it also has an important role in shock absorption during walking and sporting activities.

The anatomy of the ankle joint is complex (Fig. 29.1). It is formed by the tibia, talus, and the fibula. These three bones come together to form the bony mortise. The bony configuration alone does confer some stability to the joint. The ligaments on the medial and lateral aspects of the joint are the most important components of the static stabilization system of the ankle. In addition to the static stabilizers, there are dynamic stabilizers (i.e., requiring motion or applied tension), the most important of which are the peroneal tendons.

Although in a very simple sense the ankle can be thought of as a hinged joint, its motion is much more complex and involves sliding and some translation during normal joint motion.

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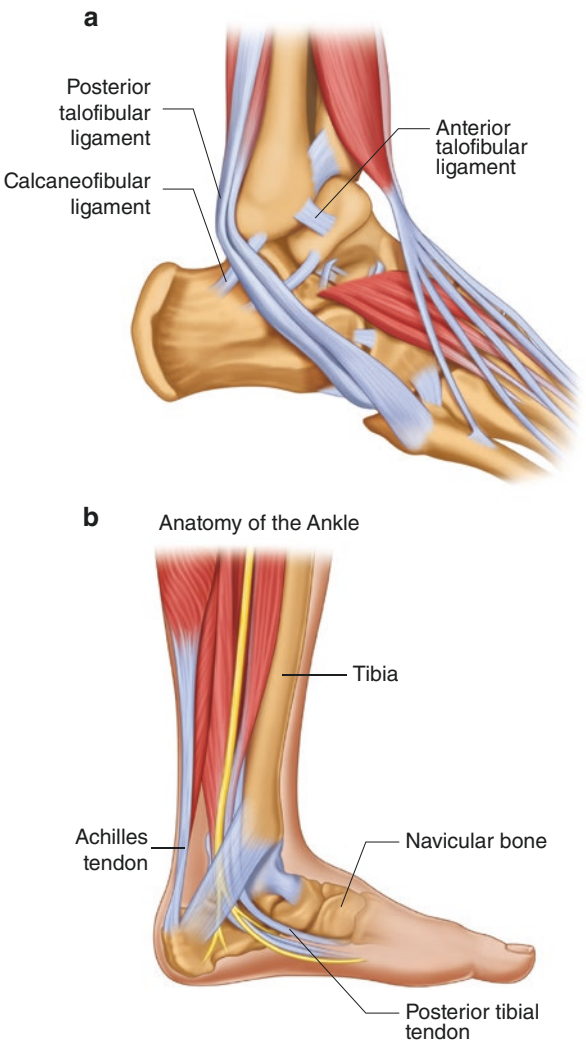

Fig. 29.1 Anatomy of the ankle. Diagram of (**a**) lateral and (**b**) medial ankle anatomy showing the static (joint morphology and ligaments) and dynamic (tendons) stabilizers of the joint

Epidemiology

Overall, the ankle is affected much less frequently by arthritis than the hip and knee joints. While almost 40% of individuals over 60 years of age have radiographic evidence of knee arthritis, in the same age cohort, approximately 5% of individuals have ankle arthritis.

It is not known why the prevalence of knee arthritis is greater than that of the ankle. Biomechanical, histologic, and biochemical reasons have been postulated. The ankle joint is highly constrained when the dynamic and static stabilizers are

considered in aggregate. This large degree of constraint has been suggested as one reason that arthritis is less likely to develop in the ankle. Ankle articular cartilage is thinner and better preserves its tensile stiffness and fracture stress with aging than that of other joints. There are also metabolic differences between ankle and knee articular cartilage that may also help explain the relative rarity of primary ankle osteoarthritis. The cartilage in the ankle has more resilience to biomechanical loading than that within the knee. Biochemical assays have shown that knee and ankle cartilage differ substantially as well. All of these differences may protect the ankle from developing primary osteoarthritis.

Unlike the hip and knee joints, the majority (approx. 70%) of clinically important ankle arthritis is post-traumatic in nature. Those with significant end-stage ankle arthritis usually relate a history of ankle fracture, ankle dislocation, or a single or multiple severe sprains. Rarely, ankle arthritis develops without a history of significant ankle trauma (i.e., inflammatory or idiopathic pathologies).

Clinical Presentation

Patients with ankle arthritis typically complain of pain over the anterior aspect of the ankle. This pain may also feel as if it is deep within the joint. The pain usually develops over a long period of time and is not due to a single recent traumatic event.

Examination of patients may reveal limited joint motion as well as tenderness to palpation along the joint line. One needs to be careful that the ankle joint and not the subtalar joint is not being tested. The ankle joint provides an arc of motion in the sagittal plane, while the subtalar joint provides the majority of motion in the coronal plane. Motion may be limited not only from mechanical blocks from osteophytes but also from contractures of the gastrocnemius and/or soleus musculotendinous units. There may be swelling, erythema, and/or an effusion about the ankle in these patients. In those who have developed arthritis from instability, the joint may show laxity in one or more planes.

Radiographic findings may include diminution of the tibiotalar joint space in a global sense or in a more limited distribution within the joint. Osteophytes can be present throughout the periphery of the joint but most often are seen over the anterior aspect of the joint. There can be subchondral sclerosis of the bone and in some cases even cyst formation. Individuals who have developed arthritis of the ankle secondary to joint instability may show incongruity of the joint either on frontal views or lateral views. Some of the typical radiographic features of ankle arthritis are demonstrated in Fig. 29.2.

The differential diagnosis for ankle arthritis includes ankle joint infections, inflammatory arthritis, osteochondral lesions of the talus, loose bodies within the joint, and ankle joint mechanically induced synovitis. In many of these cases, advanced imaging or lab work including joint aspirates can be very helpful in discerning between possibilities on the differential list.

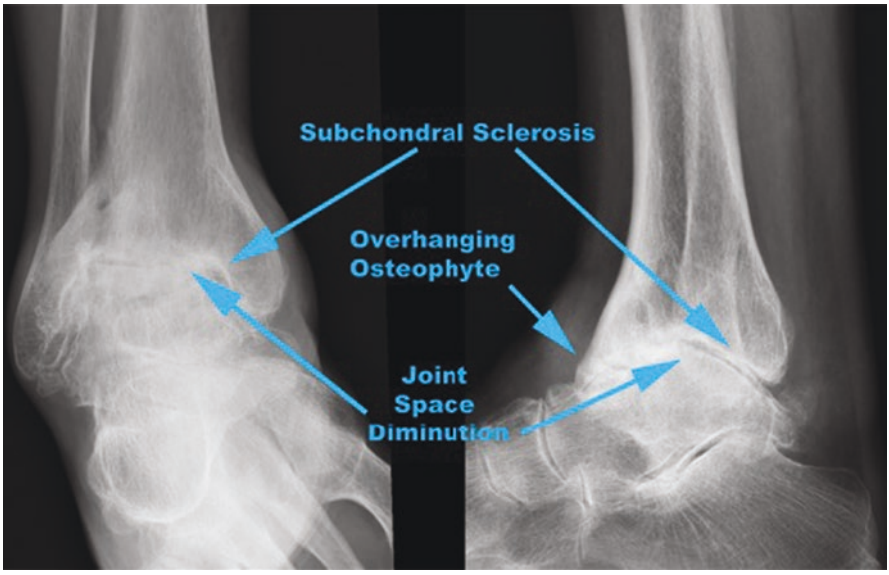

Fig. 29.2 Radiographic features of ankle osteoarthritis. Anteroposterior and lateral roentgenographic views of an ankle showing typical characteristics of ankle joint arthritis

A joint infection may cause swelling, pain, and erythema about the joint. There will likely be a significant limitation in motion, but this will be due to inflammation and pain of the synovium and not to a mechanical block. Patients with ankle infections generally do not want to move the ankle at all and may have fever, chills, nausea, or vomiting associated with the infection. If an ankle joint infection is suspected, an aspirate should be performed and sent for analysis including Gram stain, culture, and antibiotic sensitivity determination.

Inflammatory arthritides may have similar signs and symptoms clinically and joint space diminution radiographically. However, many patients with such pathologies will have polyarticular involvement.

Osteochondral lesions of the talus may cause similar complaints as ankle arthritis, but clinical examination usually will demonstrate a localized point of maximal intensity on the talar dome rather than a more global pain seen with tibiotalar arthritis. Osteochondral lesions are localized softening of the talar bone and overlying articular cartilage. Loose bodies may cause locking and pain within the joint but typically will do so intermittently as the loose body impinges at the articular surface. This intermittent pain and locking is not typical of ankle arthritis. Mechanically induced ankle joint synovitis may have many of the clinical features of ankle arthritis but will not show joint space diminution, osteophytes, or bony changes on standard radiographs.

Non-operative Management

Non-operative management of ankle arthritis can provide substantial relief for years for many patients. Non-operative treatment options include activity modification, cryotherapy, nonsteroidal anti-inflammatory drugs, shoe wear modification, bracing, and injections.

Activities involving impact loading and that require quick accelerations or decelerations tend to exacerbate ankle arthritis. If activities such as these can be minimized or limited, the patient will usually have a substantial decrease in pain.

Shoe wear modification can be added to activity modification to further reduce symptoms. Addition of a rocker bottom to the sole of a shoe can reduce the amount of ankle plantarflexion and dorsiflexion required for walking. In doing so, the amount of pain experienced during ambulation can be greatly diminished. These modifications can be placed by a certified orthotist, or in some cases by a cobbler. The modification is glued onto the bottom of a standard sneaker or shoe and provides a curved surface on the sole to allow the patient to roll through from the heel strike to toe off portions of gait (Fig. 29.3).

Bracing can also be an effective treatment for ankle arthritis. Effective ankle braces range from fabric ankle wraps to custom molded orthoses that provide rigid ankle and hindfoot stability. Each has the goal of limiting the amount of ankle motion by closely applying a rigid or inelastic component to the ankle to prevent or significantly limit the amount of motion through the tibiotalar joint. Fabric ankle wraps have the advantage of being relatively cheap and easy to apply and remove. Custom molded orthotics, such as ankle foot orthoses, provide excellent fit and rigid stability for many patients. Some of these molded orthotics can be covered with soft leather to increase comfort. Carbon fiber ankle braces have the advantages of being lightweight, low profile, and very durable. They can fit easily into most shoes. Figure 29.4 shows examples of fabric, custom molded plastic, and carbon fiber ankle braces.

Lastly, injections of corticosteroids can be placed intra-articularly to combat the pain and inflammation that the arthritis generates. These injections can be both diagnostic and therapeutic. The placement of a corticosteroid admixed with local

Fig. 29.3 Rocker bottom shoe. Rocker bottom shoe that allows the patient to roll through the stance phase of gait so that there is less motion in the ankle joint

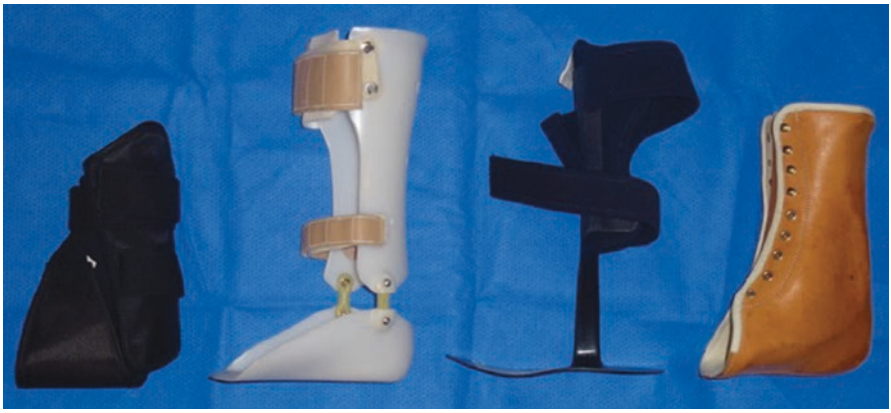

Fig. 29.4 Range of orthotics available for ankle arthritis. Examples of fabric, custom molded plastic, and carbon fiber ankle braces for use in patients with ankle arthritis. The appropriateness of each of these is dependent on a multitude of patient factors

anesthetic can provide immediate feedback in terms of pain relief. The steroid will usually take 48–72 hours to reach a maximal effect. In some cases, injections can be used to manage ankle arthritis conservatively for extended periods of time. These injections should be limited to no more frequently than once every 3 months, to mitigate some of the adverse effects of corticosteroid on the cartilage and the soft tissues surrounding the ankle. Although the corticosteroid has a powerful anti-inflammatory effect, it can also cause some softening of structures containing collagen, including cartilage. It also may lead to thinning and bleaching of the skin at the injection site.

Although individual non-operative treatments can have significant therapeutic effects in isolation, they can be combined for an even greater effect.

Surgical Management

In general, patients should have exhausted non-operative treatment options before considering operative treatment of their ankle arthritis. Surgical options include arthroscopic debridement of osteophytes and loose bodies, cheilectomy to remove osteophytes and increase ankle motion, distraction arthroplasty, ankle fusion, and total ankle arthroplasty.

Arthroscopic debridement of an arthritic ankle joint has the advantage of being a minimally invasive procedure and has a short recovery time. However, in most cases, the effects that are obtained from such a procedure are short-lived (i.e., weeks or months in duration) for those individuals with end-stage ankle arthritis. There may be some beneficial effects that are more durable for those with milder cases of arthritis. Although it may be useful as a bridge procedure in select circumstances, it is generally not an effective method for end-stage arthritis.

Cheilectomy is a procedure in which osteophytes at the anterior margin of the ankle joint are removed. In certain cases, these osteophytes may be "kissing," that is, they may physically abut each other on ankle dorsiflexion. This can cause either bony or soft tissue impingement. If the majority of pain is coming at the end of dorsiflexion range of motion, then a surgical procedure to remove these osteophytes can be very effective. This procedure does not treat the loss of articular cartilage directly. Here again, as with arthroscopic debridement, the procedure is less invasive than other large open procedures and has a fairly short recovery time. In certain individuals, it can be very effective not only in the short run but also in the long run. Studies have shown that about 90% of patients undergoing cheilectomy for the proper indications will have improvement in their symptoms with approximately 60% being pain-free at 2 years post procedure.

Distraction arthroplasty is a technique used to regenerate painless articulating surfaces in the ankle joint. The procedure involves an arthroscopic debridement of the ankle joint followed by an application of an external fixator which creates a distractive force across the ankle joint. Once applied, the patient is allowed to be full weight-bearing on the affected lower extremity. Allowing weight-bearing with the distractor on removes shear forces from the articulating surfaces while maintaining the beneficial effects of fluid pressure to the cartilage. The latter nourishes the cartilage as all of its metabolic needs are obtained through the joint fluid. This therapy has been shown to have durable effects. In fact, studies have shown that the results tend to improve with time after the frame has been removed. This therapy is not for everyone as wearing a frame for 3–4 months is challenging for most. In addition, not all orthopedic surgeons are comfortable with putting on the type of circular fixators required. Some groups have reported that over 90% of patients will have improvement in their pain following ankle distraction arthroplasty.

Fusion has been a very reliable definitive therapy for end-stage ankle arthritis for decades. This procedure causes the tibia and talus to grow together and obliterates the ankle joint. By removing the joint, the arthritis is also eliminated. This therapy requires 6 weeks of non-weight-bearing followed by 6 weeks of weight-bearing in a cast. Following successful fusion, patients experience a reliable reduction, and frequently elimination, of pain in the ankle joint. Once established, the fusion does not degrade and is able to sustain heavy loads placed upon it. Maintenance or monitoring is not required after fusion is established. Ankle fusions can be performed in patients with many different comorbidities and extent of obesity.

As with any therapy, there are side effects to ankle fusion. These are usually not realized for years after the fusion is performed but can arise over the long term. In addition to being a major motion segment for locomotion, the ankle joint also serves as a shock absorber. When this shock absorption is eliminated, the forces of locomotion are transmitted to other joints of the distal lower extremity. The two joints that predominantly absorb these forces are the subtalar and transverse tarsal joints. Years after ankle fusion, these joints become arthritic and may eventually require fusion. When these joints as well as the ankle are fused, the foot becomes extremely stiff and gait changes markedly. Activities such as walking long distance and hiking that were previously relatively easy with an ankle fusion alone become significantly

Table 29.1 Summary of ankle arthritic disorders with synopsis of presentation, diagnostic testing, and suggested management options

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Ankle arthritis	Tenderness to palpation/pain over the anterior ankle joint Diminished ankle joint motion Joint crepitus Antalgic gait	Standing radiographs of ankle Diagnostic injection (to rule out other sites of pathology)	Bracing/ splinting Rocker bottom shoes Steroid injection into joint NSAIDs	Pain refractory to all conservative management	Arthroscopic debridement Distraction arthroplasty Total ankle arthroplasty Ankle fusion

NSAIDs Nonsteroidal anti-inflammatory drugs

more challenging. This is one major reason that ankle arthroplasty remains an option for those with end-stage ankle arthritis.

Total ankle arthroplasty is a procedure that replaces the weight-bearing tibiotalar articulation of the ankle joint. Current implants use durable metal talar and tibial implants with a high-molecular-weight polyethylene components sandwiched between them. Unlike fusion, the tibiotalar articulation is maintained with TAA. The goal is to relieve arthritis pain while maintaining ankle motion and function. In addition to maintaining ankle function, it also aims to prevent hindfoot arthritis that inevitably develops with ankle fusion.

Total ankle arthroplasty has been shown to be effective in properly selected patients in relieving the pain of ankle arthritis while maintaining ankle motion. Ankle replacement surgery when performed properly and for the correct indications has reliably provided pain relief. Historically, over 80% of patients undergoing total ankle arthroplasty had their pain levels reduced to three or less on a visual analog scale. Current designs are more accurately implanted and as a result have more reliable outcomes and greater longevity.

Ankle arthroplasty like any other joint arthroplasty needs to be monitored intermittently once successfully established. This treatment like other joint arthroplasty techniques has complications that can develop throughout the lifetime of the patient; these include osteolysis secondary to wear particle generation, arthrofibrosis, and infection. Long-term survival of total ankle arthroplasty implants is 80–90% 10 years after implantation. Implant failures in patients can be revised in some cases but in other cases require fusion after explant of the implant (Table 29.1).

Suggested Reading

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Chapter 30
Soft Tissue Disorders of the Ankle

Jeremy T. Smith, Eric M. Bluman, Christopher P. Chiodo, and Elizabeth A. Martin

Abbreviations

AFO	Ankle foot orthosis
ATFL	Anterior talofibular ligament
CFL	Calcaneofibular ligament
ECSWT	Extracorporeal shock wave therapy
MRI	Magnetic resonance imaging
ORIF	Open reduction internal fixation
PT	Physical therapy
PTFL	Posterior talofibular ligament
PTT	Posterior tibial tendon
PTTD	Posterior tibial tendon dysfunction
RICE	Rest ice compression elevation

Achilles Tendon Disorders

The Achilles tendon is a confluence of the gastrocnemius and soleus muscles. These muscles, collectively referred to as the triceps surae, consolidate into the Achilles tendon which is the largest and strongest tendon in the body. The Achilles then attaches broadly, with an approximately 2 × 2 cm attachment, to the posterior aspect

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of the calcaneus. During standing and throughout much of the gait cycle, the calf muscle-tendon unit is active and is either lengthening or shortening.

The blood supply to the Achilles comes from both proximally and distally. The proximal portion of the tendon receives its vasculature from intramuscular arterial branches. Distally, the tendon receives its blood supply from interosseous arterioles in the calcaneus. The result of this is that the central portion of the Achilles tendon, roughly 2–6 cm proximal to its insertion, is relatively poorly vascularized. Accordingly, much of the pathology that occurs in the Achilles tendon is at this vascular watershed.

Conditions affecting the Achilles muscle-tendon unit may involve the gastrocnemius or soleus muscle, the non-insertional Achilles tendon which involves the area of tendon typically 2–6 cm above the calcaneus, the insertional Achilles tendon, and the Achilles bursae. Two bursae sit adjacent to the Achilles insertion, one in front (retrocalcaneal bursa) and one behind (Achilles tendon bursa) the tendon insertion.

Achilles Tendinopathy

We advocate using three terms to describe Achilles tendon disorders: *tendinitis*, *tendinosis*, and *tendinopathy*. Tendinitis is an acute inflammatory pathologic process that involves inflammatory changes within either the substance of the tendon or the surrounding tendon layer, the peritenon. Tendinosis is a chronic degenerative process that occurs without inflammation of the peritenon. Tendinosis is characterized by degenerative lesions within the tendon substance with altered microscopic tendon structure. Macroscopically, a tendinotic tendon is thickened and lacks a healthy shiny appearance. Tendinopathy is a general term that includes tendinitis and tendinosis.

Achilles tendon disorders are often described by anatomic location, either at the calcaneal insertion (insertional) or within the substance of the tendon (*non-insertional*). The chronicity of the disorder is also often incorporated into its description with the use of the following terms: *acute* (<2 weeks of symptoms), *subacute* (2–6 weeks of symptoms),or *chronic* (>6 weeks).

Summary of Epidemiology

Tendinopathy of the Achilles tendon is most commonly caused by a combination of intrinsic and extrinsic factors. Intrinsic causes may include aging, foot or ankle deformity that contributes to added tendon stress, and a tight gastrocnemius muscle. Extrinsic factors include poor shoe wear, hard surface conditions, and overuse such as occurs at times with exercise. Achilles tendinitis develops frequently in athletes, often those who run or participate in sports that require frequent bursts of speed (e.g., soccer, basketball, tennis). Tendinitis therefore is seen more often in younger patients. Achilles tendinosis, in contrast, more often develops in older patients and often occurs in the absence of any new or increasingly strenuous activity.

Clinical Presentation

Achilles tendinitis typically presents with the acute onset of pain in the Achilles that is exacerbated by activities that stress the tendon, including walking, stairs, running, and athletics. Additionally, worsened pain with the first steps in the morning is common. This start-up pain is thought to be related to sleeping with the ankle plantarflexed, as most people do, which allows the calf muscle complex to tighten overnight and then the Achilles to stretch painfully with the first morning steps. Erythema or swelling may be present at the site of maximal discomfort. Upon examination, patients often have an antalgic gait in an attempt to limit stress to the tendon. The Achilles is focally tender, and, while it may be somewhat swollen, there are not typically nodules within the substance of the tendon. Palpation should not reveal any gap in the tendon. The Thompson test, which evaluates the continuity of the tendon by squeezing the calf muscle and expecting a plantar flexion response of the ankle, should be intact and symmetric to the non-injured extremity. Ankle range of motion should be carefully assessed, as patients often have a tight gastrocnemius muscle which may be part of the underlying cause of the tendinopathy.

In contrast to Achilles tendinitis, the onset of pain with Achilles tendinosis is often insidious. There is typically no acute event linked to the development of pain. Patients often report an area of swelling with a "knot" or "nodule" within the tendon. This nodule represents the area of greatest tendon degeneration. As with Achilles tendinitis, the tendon itself is often tender to palpation. The distinction between insertional and non-insertional tendinosis, or tendinitis, is based on location of maximal tenderness on physical examination. Whereas non-insertional tendinosis typically presents with a nodule within the tendon, insertional disease is often but not always accompanied by abnormal calcaneal bony morphology. A bony prominence at the posterosuperior calcaneus, just anterior to the tendon insertion, is called a Haglund's lesion (Fig. 30.1). This is distinct from insertional

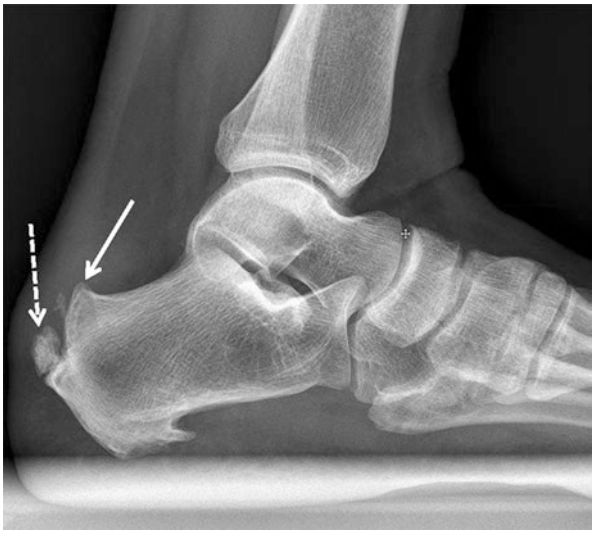

Fig. 30.1 Lateral radiograph with Haglund's lesion (solid arrow) as well as insertional Achilles spur (dashed arrow)

Achilles calcification which can also contribute to pain at the tendon insertion. Insertional tendinopathy may be accompanied by retrocalcaneal or Achilles bursitis.

Fluoroquinolone antibiotics have been associated with disorders of the Achilles, including tendinopathy and rupture. This observation was first published in 1983 and has been documented in many studies since, including in vitro studies showing fluoroquinolone-related damage to collagen and tenocytes. The exact mechanism of fluoroquinolone injury to tendons has not been fully established.

Differential Diagnosis and Suggested Diagnostic Testing

The diagnosis of Achilles tendinopathy is largely based on history and physical examination. Other causes of posterior leg pain include gastrocnemius strain, Achilles tendon rupture, plantaris rupture, chronic exertional compartment syndrome, calcaneal stress fracture, lumbar radiculopathy, and claudication.

Plain radiographs of the ankle are useful in identifying abnormal calcaneal bony morphology. It is important to note that patients may have spurs on the calcaneus that are not associated with any pain or limitation. At times, a calcaneal stress fracture may be appreciated on plain radiograph as a sclerotic line through the calcaneus (Fig. 30.2). Advanced imaging, either MRI or ultrasound, are not often necessary but can be utilized to better clarify the extent of tendon involvement or to assess for another cause of pain should the diagnosis not be certain.

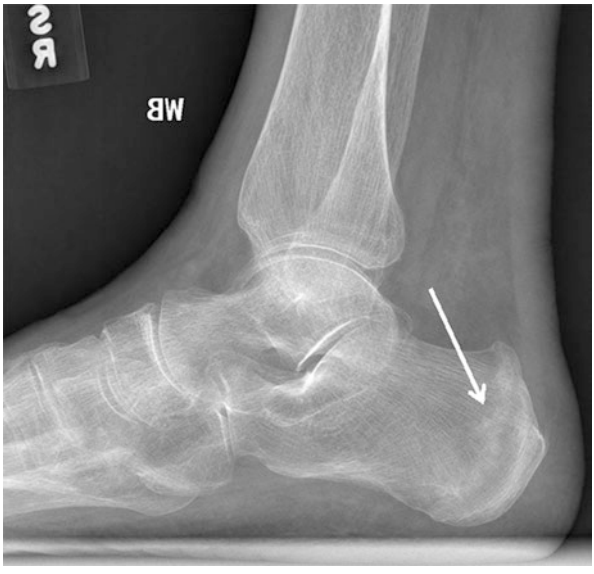

Fig. 30.2 Lateral radiograph showing a calcaneal stress fracture (solid arrow), seen as a sclerotic line through the calcaneus

Non-operative Management

The majority of patients with Achilles tendinopathy can be successfully managed without surgery. For those presenting with tendinitis, particularly with the acute onset of severe pain, the use of a tall walking boot for a few weeks is often helpful. After that, and for those with tendinosis, treatment involves a carefully guided stretching and strengthening program with a physical therapist. Eccentric training, which occurs with firing of the muscle-tendon unit as it is being lengthened, has been shown to be an effective treatment. This program is accompanied by the use of a dorsiflexion night splint, which keeps the tendon stretched during sleep. Additional treatments may include activity modification, anti-inflammatory medications, and the use of a heel lift. For patients with pressure-related pain from shoes rubbing at the posterior heel, often called a "pump bump," the use of a gel heel sleeve to cover this area can be helpful. Corticosteroid injection into the tendon is *not* recommended as this is associated with Achilles rupture.

For those whose pain does not improve with the treatments just outlined, additional non-surgical treatments include the use of a custom ankle foot orthosis (AFO) or extracorporeal shock wave therapy (ECSWT). An AFO shelters the Achilles tendon during ambulation, although often is required for a lengthy period of time. ECSWT, which uses a technology similar to lithotripsy, is performed as an in-office procedure and has shown promising results for patients with recalcitrant Achilles tendinopathy. The mechanism of its effect is not well-understood, although theories include alteration in neural membranes or local vascularity. Lastly, orthobiologics (i.e., platelet-rich plasma, stem cells) may have a role in treating Achilles tendinopathy, although their role remains incompletely understood. This will be discussed in more detail to follow.

Indications for Surgery

Surgical treatment may be indicated for patients who have pain and symptoms that persist despite non-surgical treatment. Most patients have had months of non-surgical management and yet remain limited by pain in the Achilles tendon. Perioperative risk assessment is very important, and for certain patients surgery is not appropriate due to either systemic medical conditions, poor local physiology such as peripheral vascular disease, or an anticipated inability to comply with post-operative restricted weightbearing instructions.

Operative Management

Operative treatment for both insertional and non-insertional tendinopathy has traditionally involved open debridement of the diseased tendon and repair of the remaining tendon. This can at times require removal of a substantial portion of the Achilles, which may necessitate a transfer of one of the other flexor tendons to augment the

remaining Achilles. For insertional tendinopathy, the Haglund's lesion and/or insertional spur is also removed. This involves surgically smoothing down the back of the calcaneus.

Less invasive surgical techniques include endoscopic Haglund's excision and retrocalcaneal bursa debridement for insertional disease, and smaller incision release of adhesions surrounding non-insertional lesions. Gastrocnemius recession, which lengthens the Achilles muscle-tendon unit, has also been shown to be effective in alleviating pain.

Expected Outcome and Predictors of Outcome

Duration of symptoms prior to the onset of treatment has been associated with response to treatment. Those with symptoms for 6 months or longer are more likely to require surgical intervention. Additionally, insertional tendinopathy with a Haglund's lesion or insertional spur is less likely to respond favorably to non-surgical treatment. For those who do require operative intervention, most studies report greater than 80% of patients have substantial pain relief.

Achilles Tendon Ruptures

Summary of Epidemiology

Rupture of the Achilles tendon occurs with an estimated incidence of 18 per 100,000 people. Ruptures occur with rapid loading of a tensed tendon. This condition is seen most commonly in men in the fourth or fifth decade of life and injuries are often sports-related. The majority of patients who sustain a rupture have microscopic and macroscopic alteration in the tendon integrity. Reports in the literature link fluoroquinolone use, certain endocrine abnormalities (hypothyroidism, renal disease), and systemic inflammatory arthritis (rheumatoid arthritis) to Achilles rupture.

Achilles ruptures have a substantial societal cost with many patients requiring an extensive time out of work. Most treatment programs require several months on crutches and then a gradual return to activities.

Clinical Presentation

Patients typically experience the immediate onset of pain and a pop or pull in the Achilles at the time of rupture. Many report the feeling that someone kicked them in the Achilles and then turn around to see that no one was behind them. Patients may be able to ambulate by recruiting the deep flexors of the leg to push off, so ambulation does not exclude this diagnosis. On examination, the most consistent findings include a palpable gap in the Achilles, a lack of plantar flexion of the ankle

when the calf is squeezed (abnormal Thompson test), and increased passive dorsiflexion of the ankle as compared to the non-injured extremity.

The diagnosis of Achilles rupture is missed initially in up to 25% of patients. This is likely because of patients' ability to compensate when walking and swelling making appreciation of the gap difficult. Treatment success relies upon early diagnosis and initiation of treatment.

Differential Diagnosis and Suggested Diagnostic Testing

Plantaris tendon rupture and gastrocnemius strain may also cause a pop or a pull in the back of the calf. Achilles rupture can be distinguished from these diagnoses by palpation of a gap, abnormal Thompson test, and assessment of passive ankle dorsiflexion. As with Achilles tendinopathy, the diagnosis of an Achilles tendon rupture is largely clinical. In many patients, radiographs are not necessary, although can be useful to ensure that there has not been a calcaneal avulsion fracture. MRI and ultrasound may be used to confirm the diagnosis, although are not required if the history and clinical examination is clear.

Non-operative Management

Achilles tendon ruptures can be treated both surgically and non-surgically. Treatment goals include minimizing the risk of surgical wound healing problems or infection, restoring Achilles continuity and push-off strength, and minimizing the risk of re-rupture. Operative treatment carries inherent surgical risks. Non-surgical treatment has historically led to decreased strength or unacceptably high rates of re-rupture. Over the past decade, significant strides have been made to refine both operative and non-operative treatments.

Traditional non-surgical treatment of Achilles tendon ruptures involved cast immobilization of the ankle for 6–8 weeks. With this treatment, rates of re-rupture were reported to occur in roughly 20% of patients. Due to this high re-rupture rate, and based in part upon knowledge that tendons heal better when mobilized, current non-surgical treatment protocols move the ankle early with a program called early functional rehabilitation. A randomized controlled trial published in 2010 with 144 patients randomized to either operative or non-operative treatment using early functional rehabilitation showed a re-rupture rate that was similar between both groups (3% in the surgical group versus 4% in the non-surgical group). In this study, the non-surgical group was treated with immobilization in a splint with the ankle in plantar flexion for 2 weeks, followed by gentle graduated range of motion while protected in a boot. Critical to this treatment is the understanding that non-surgical treatment is not synonymous with non-treatment. The non-surgical approach is a structured program that begins shortly after the injury.

Non-operative treatment of acute Achilles tendon ruptures is a viable and effective method of treatment. To date, it remains unclear whether operative or

non-operative management is superior. Studies suggest that operative treatment carries an increased risk of surgical complications and yet provides benefits in push-off strength and an earlier return to work. The approach at our institution is to thoroughly present both treatment options, evaluate the patient's surgical risk profile, and work with the patient to determine the optimal treatment for him or her.

Indications for Surgery

Success with non-surgical treatment of Achilles ruptures often requires early immobilization with the ankle plantarflexed. This is thought to start the tendon healing by bringing the tendon ends into closer proximity. It is thus thought by many that an initial delay in diagnosis by more than a few weeks is a relative indication for surgical treatment.

Patient comorbidities guide the treatment decision for Achilles ruptures. Systemic or local factors can tilt the risk/benefit scale when deciding between operative and non-operative treatment. Surgical treatment should be approached cautiously in those with diabetes, immunosuppression, peripheral vascular disease, obesity, skin disorders involving the leg, tobacco use, and patients over age 65.

Operative Management

The goal of operative treatment is to re-approximate the ends of the Achilles while minimizing the risk of surgical complications. Open surgical techniques typically approach the Achilles posteriorly and then suture the tendon ends together with tension that matches the contralateral extremity. With careful attention to detail, including gentle handling of the soft tissues, not using a tourniquet, and meticulous closure in layers, the risk of surgical wound complications can be lessened.

In an effort to further minimize wound healing complications, minimally invasive surgical techniques have been developed. Multiple techniques exist, and the general concept is that sutures are shuttled percutaneously through the tendon ends and then tied together through a small incision overlying the site of the rupture. Minimally invasive techniques have been shown to have very low surgical wound complication rates and importantly have functional outcomes that are similar to more traditional open surgical repair techniques. These smaller incision techniques mitigate some of the risk associated with traditional open surgical approaches.

Expected Outcome and Predictors of Outcome

Recovery from an Achilles tendon rupture takes many months. Patients are counseled that it can take up to 1 year to regain near-normal strength and that mild weakness in the injured extremity may persist. With appropriate treatment, either surgical or non-surgical, good function can be achieved in the vast majority of patients.

Peroneal Tendon Disorders

The peroneus brevis and peroneus longus muscle-tendon units run along the lateral aspect of the leg and cross the ankle posterolaterally, behind the fibula. The peroneus brevis tendon continues to its insertion at the base of the fifth metatarsal, and the peroneus longus crosses under the foot to attach at the plantar surface of the medial cuneiform and first metatarsal (Fig. 30.3). The peroneal tendons are adjacent to one another as they pass behind the fibula and then diverge at the peroneal tubercle, a bony prominence on the lateral wall of the calcaneus. The peroneus brevis tendon is thinner and more ribbon-shaped and is more prone to injury.

Peroneal tendon injuries include tenosynovitis, tendon tears, and dislocation from their groove behind the fibula. Tears of the tendon are seen most commonly behind the fibula but can also occur at other sites such as the peroneal tubercle.

Peroneal Tendinitis/Tendon Tears

Summary of Epidemiology

Pathology of the peroneal tendons is uncommon without either a traumatic event such as an ankle inversion injury, a predisposing mechanical abnormality, or a systemic inflammatory condition. Chronic ankle instability, resulting in frequent inversion ankle injuries, can lead to injury of the peroneal tendons. Similarly, varus hindfoot alignment, which tilts the ankle and hindfoot inward and places stress on the structures at the lateral ankle and foot, can lead to peroneal tendinopathy.

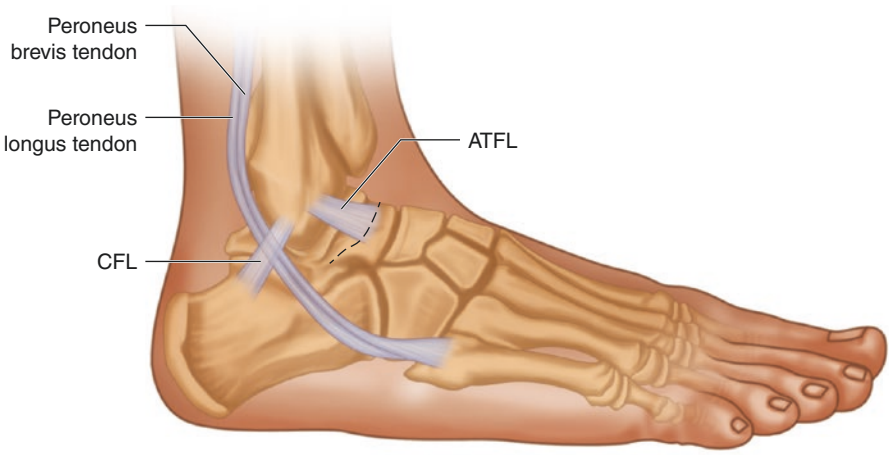

Fig. 30.3 Lateral ankle illustration showing ankle ligaments and peroneal tendons. The peroneus brevis and peroneus longus pass posterior to the fibula

Clinical Presentation

Tenosynovitis of the peroneal tendons typically presents with pain along the lateral and posterolateral aspects of the ankle. Patients often report activity-related pain with stair climbing and walking on irregular ground. This discomfort may be accompanied by swelling along the tendons. Recent shoe wear changes or alternation in activity level is common. Peroneal tendon tears, which frequently accompany tenosynovitis, are typically longitudinal split tears, and therefore near-normal tendon strength is preserved.

Peroneal tendon subluxation, which occurs when the tendons dislocate laterally from behind the fibula, often occurs traumatically. Patients may be able to replicate the tendon instability by rotating the ankle in a large circle (circumduction). Tenderness just behind the fibula is common. With time, the pain from an acute injury may improve, but the tendons often remain unstable which predisposes to tears. Clicking of the tendons may be due to subluxation from behind the fibula or intra-tendinous subluxation where one of the tendons clicks in and out of a tear of the adjacent tendon.

Physical examination should include assessment of gait, hindfoot and foot alignment to assess for a varus hindfoot, site of maximal tenderness, peroneal tendon strength, ankle circumduction to test for peroneal subluxation, and ankle stability testing.

Differential Diagnosis and Suggested Diagnostic Testing

Additional causes of posterolateral ankle pain and lateral hindfoot pain include injury to the fibula, calcaneus, or sural nerve, sinus tarsi syndrome, tumor, and radiculopathy. Plain radiographs evaluate bony changes as well as assess radiographic alignment, specifically looking for varus of the ankle or hindfoot. An enlarged peroneal tubercle may be appreciated by plain X-ray. Additional studies, such as MRI and ultrasound, can be very helpful in defining the extent of tendon involvement. In the setting of peroneal tendon instability, MRI enables measurement of the depth of the fibular groove. A shallow groove is associated with tendon subluxation and may necessitate surgery to deepen the groove. Ultrasound assessment of the tendons can be very useful due to the dynamic nature of this modality, allowing for real-time assessment of the tendons. If the source of the patient's pain remains unclear, a diagnostic local anesthetic injection administered into the peroneal tendon sheath may help determine if the tendons are the source of the pain.

Non-operative Management

Peroneal tendon pain without tendon instability is most often treated with immobilization for 4–6 weeks. This can be with a walking boot for more severe pain or a softer ankle brace for more mild symptoms. This period of rest is typically followed

by physical therapy. Ice, anti-inflammatory medications, and activity modification may be helpful. For those with a high arch or varus hindfoot, orthotics can be obtained that take pressure of the lateral hindfoot and peroneal tendons. As with Achilles tendon disorders, corticosteroid injections into the tendons are not recommended due to risk of rupture.

Peroneal subluxation or dislocation can also be treated non-surgically, although this requires six weeks of cast immobilization with the ankle in plantar flexion. It is logistically difficult to ensure that the tendons remain reduced during this casting period, and tendon instability is successfully treated non-surgically in only about 50% of cases.

Indications for Surgery

Surgery for peroneal tendinitis or tendon tears is considered for patients who have persistent symptoms despite extensive non-surgical treatment. Surgery is more commonly recommended early for those with peroneal tendon instability.

Operative Management

The traditional approach for peroneal tendinitis or tendon tears is an open surgical procedure that exposes the tendons and enables debridement of inflamed tenosynovium and repair of tears. Some tears are amenable to repair and others require removal of the torn portion of tendon. With extensive involvement, a tendon transfer may be necessary. Attention is also given to the underlying cause of the problem, which may be addressed simultaneously, for example, ligament repair for chronic ankle instability, correction of a varus hindfoot with osteotomies, or removal of an enlarged peroneal tubercle.

Recently developed arthroscopic techniques enable less invasive procedures and a quicker recovery. Tendoscopy allows for excellent visualization of the tendons and debridement of tissue from within the tendon sheath (Fig. 30.4).

Peroneal tendon subluxation is treated with an open repair of the peroneal retinaculum. The tendons are exposed to address any associated tendon tears and the fibular groove may be deepened with an osteotomy. This procedure, as with most open peroneal tendon surgery, requires a period of 6 weeks of postoperative restricted weightbearing on crutches.

Expected Outcome and Predictors of Outcome

Due in part to the length of recovery required for healing after open peroneal tendon surgery, patients are counseled to expect a recovery period of 3–6 months. Clinical outcomes studies report good results with peroneal tendon procedures.

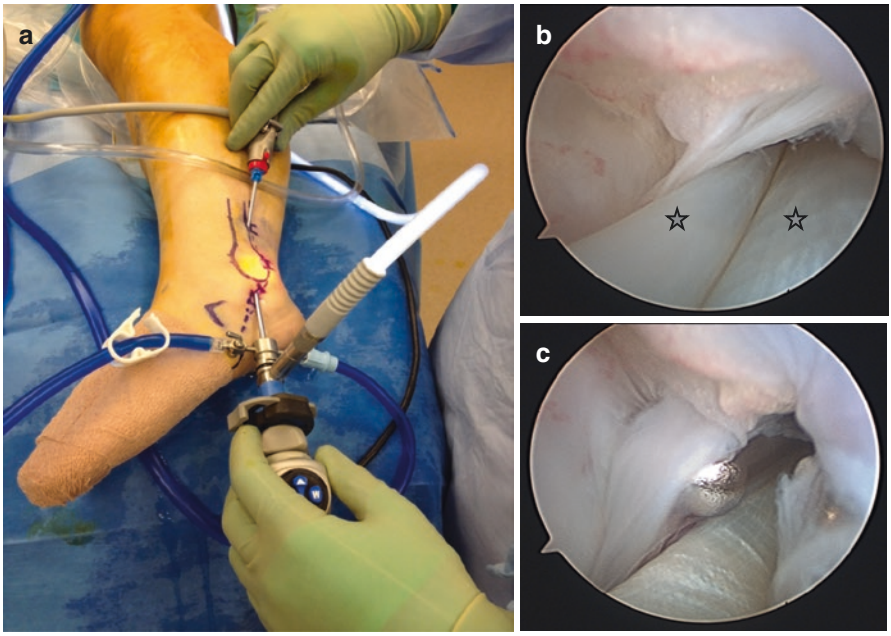

Fig. 30.4 Photograph of a patient undergoing peroneal tendoscopy (**a**). Arthroscopic photographs (**b**) show the peroneal tendons (stars) and a shaver device utilized to remove scar tissue (**c**)

Posterior Tibial Tendon Disorders

The posterior tibialis muscle originates at the posterior aspect of the tibia, fibula, and interosseous membrane. The posterior tibial tendon (PTT) then passes behind the medial malleolus at the ankle and inserts broadly at the medial midfoot. The navicular bone is the primary site of attachment for the PTT. This muscle-tendon unit initiates push-off when walking and helps to maintain the arch of the foot.

Several anatomic features predispose the PTT to injury. As with the Achilles tendon, injury to the PTT often occurs at the site of poor tendon vascularity at the level of the medial malleolus. Secondly, the tendon has an excursion of only about 2 cm, meaning that the tendon travels a relatively small distance within its sheath as the muscle contracts and lengthens. Thus, even minor injuries that lengthen the tendon impact its function.

Posterior Tibial Tendon Dysfunction

Summary of Epidemiology

Posterior tibial tendon dysfunction (PTTD) occurs most commonly in middle age and most often results from attritional wear of the tendon. Posterior tibial tendon pathology typically begins with inflammation without lengthening or loss of

function of the tendon. With chronic inflammation, the tendon can then attenuate, lengthen, and then progressively deteriorate and weaken. As it deteriorates, it loses its ability to maintain the arch of the foot. As the foot begins to collapse, the hindfoot drifts laterally into valgus and the midfoot begins to sag. Correspondingly, the Achilles tendon unit tightens as its working length shortens. This pathophysiologic process can then become cyclical, as the resultant flat shape of the foot puts added stress on the PTT and can lead to further injury.

The etiology of PTTD is often multifactorial. Causes may include repetitive microtrauma, anatomic predisposition to tendon injury resultant from congenital pes planus alignment or an accessory navicular bone, systemic inflammatory conditions such as seronegative spondyloarthropathy, vascular insufficiency, and obesity. Posterior tibial tendinitis also occurs in younger patients, although more commonly after traumatic events such as an ankle eversion sprain, fracture, or repetitive sports-related injury.

Clinical Presentation

Posterior tibial tendon dysfunction is classified into four stages and if untreated often progresses through the stages with increasing severity. The first stage of PTTD is tendinitis without deformity. This typically causes pain and localized swelling along the course of the PTT. Examination should always include a standing analysis of alignment, which in Stage I disease will reveal a neutrally aligned hindfoot. Patients may have difficulty initiating a single-leg heel rise due to pain along the PTT. Stage II PTTD occurs when the hindfoot has drifted into valgus (Fig. 30.5) and yet the deformity remains flexible. The tendon is often tender and swollen. With manipulation, the alignment of the foot may be corrected, which distinguishes Stage II from Stage III. The rigid deformity in Stage III disease often is caused by hindfoot arthritic changes. As the deformity progresses, the site of pain may shift as well. If the posterior tibial tendon completely tears, this may alleviate pain at the medial ankle as the inflamed tendon has now released. Pain may occur laterally due to impingement of the calcaneus against the lateral soft tissues and the fibula. As the condition worsens, the long-standing valgus deformity can lead to injury to the deltoid ligament, which is the primary medial ankle ligamentous support. As this occurs, the ankle joint can drift into valgus alignment, which is Stage IV PTTD.

Differential Diagnosis and Suggested Diagnostic Testing

Additional causes of medial ankle pain include bony injury such as stress fracture of the medial malleolus or navicular, tendinitis of the flexors to the toes, and tarsal tunnel syndrome.

Examination should begin with a barefoot standing examination to assess alignment. Localized swelling along the PTT may be appreciated, and range of motion testing will often reveal a contracture of the gastrocnemius muscle. A single-leg heel rise assesses the competence of the posterior tibial tendon, as patients with

Fig. 30.5 Photograph of a patient with a left valgus hindfoot. On the uninvolved right side, the hindfoot alignment is neutral

posterior tibial tendon dysfunction will likely experience pain with this maneuver or be unable to do so. Plain radiography of the foot and ankle is indicated to evaluate the source of the pain and to assess alignment. To evaluate alignment, it is critical that the radiographs be *weightbearing*. MRI is a useful study to assess the degree of injury to the PTT, although is not required if the diagnosis is clear.

Non-operative Management

Many patients with PTTD can be effectively managed without surgery. The approach to treating this disorder is to rest the tendon, train the tendon with physical therapy, and then protect the tendon with an orthotic. Immobilization is accomplished with a tall walking boot. Patients with milder symptoms or who are unsteady on their feet and therefore not safe in a boot may be treated with a smaller ankle brace. The duration of immobilization is typically 4–6 weeks, and then patients transition from the boot into orthotics that support the medial hindfoot. Over the counter orthotics are much less expensive and for more mild disease can be adequate. Alternatively, custom molded orthotics can be made. If orthotics are not sufficient, larger braces such as an ankle foot orthosis may be considered. Physical therapy begins after boot

immobilization and concentrates on training the tendon and stretching the tight gastrocnemius muscle. Corticosteroid injection is not recommended as it can further attenuate the PTT.

Indications for Surgery

Surgery may be considered for patients who have persistent or worsening symptoms despite the treatments outlined above. The specifics of surgery depend upon the stage of disease, age, and functional level. Larger reconstructive procedures often require 6 weeks of non-weightbearing after surgery, and thus patients must be able to manage this challenging restriction.

Operative Management

The classification scheme outlined previously guides treatment. Stage I disease (inflammation without deformity) is typically treated with tenosynovectomy. This can be done either in an open fashion or tendoscopically. Stage II PTTD (flexible deformity) is most commonly managed with a combination of procedures that reshape the foot while removing the painful tendon. Often this involves lengthening the tight gastrocnemius muscle, removing the diseased PTT, transferring another tendon into its place (most commonly the flexor to the lesser toes), and correcting the bony alignment with osteotomies. Since Stage II disease is flexible and joints of the hindfoot are typically non-arthritic, the joints are preserved. In Stage III PTTD (fixed deformity), corrective osteotomies and tendon transfers are not powerful enough, and thus treatment often involves fusions of the joints of the hindfoot. When the ankle is involved in Stage IV disease (valgus ankle deformity), procedures typically address both the hindfoot and ankle deformity. This may preserve the ankle joint as with a deltoid ligament reconstruction or sacrifice the ankle with an ankle replacement or fusion.

Expected Outcome and Predictors of Outcome

As PTTD progresses, the surgical treatment increases in complexity. While surgical intervention for all stages of disease has been shown to be successful, the more complex procedures carry increasing risk, and it is therefore optimal to interrupt the disease progression early. Pain relief along the medial ankle is a good indicator of successful non-operative treatment, so if a pain-free state can be achieved with the use of an orthotic, for example, then progressive deterioration of the tendon is unlikely to occur.

Ankle Ligament Injuries

Ankle Sprains

Summary of Epidemiology

Ankle sprains are the most common musculoskeletal injury, and it has been estimated that one ankle sprain occurs every second in the United States. The majority (85%) of these injuries involve the lateral ankle ligaments, with the remaining injuries occurring either to the medial ankle ligaments or the syndesmotic ligaments (the ligaments that connect the tibia to the fibula). Ankle sprains have been reported to account for 30% of all sports-related injuries and are more common in contact sports such as basketball and soccer. Up to 30% of ankle sprains can lead to chronic symptoms. These resultant problems may include chronic ankle instability, osteochondral lesions, ankle impingement syndromes, and peroneal tendinopathy.

Lateral ankle sprains most commonly cause injury to the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and/or the posterior talofibular ligament (PTFL) (Fig. 30.6). Medial ankle sprains cause injury to the deltoid ligament. And the syndesmotic ligament complex is what is injured with a *high ankle sprain*.

Clinical Presentation

The majority of patients presenting with an ankle sprain report a twisting injury to the ankle. Lateral ligament injuries often occur from supination (rolling inward), medial ligament injuries from pronation (rolling outward), and syndesmotic

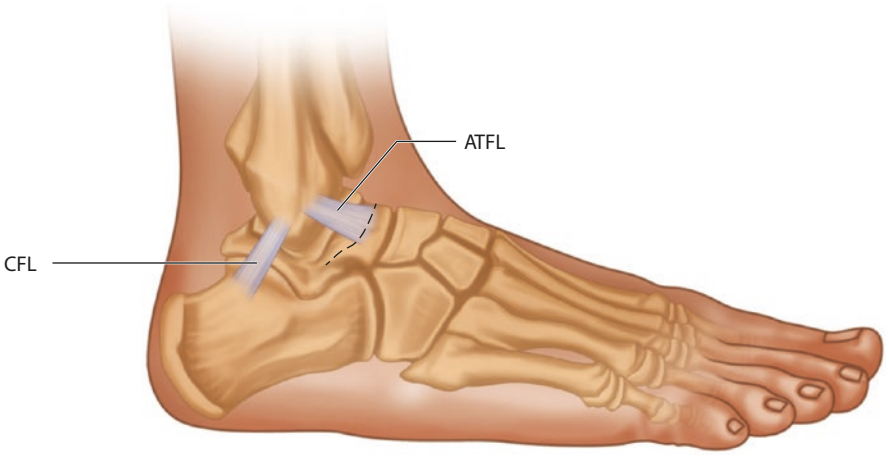

Fig. 30.6 Lateral ankle illustration showing anterior talofibular (ATFL) and calcaneofibular ligament (CFL)

ligament injuries from external rotation while the foot is planted and fixed to the ground. Patients report the immediate onset of pain and swelling and may hear or feel a pop. Many patients have difficulty weightbearing after the injury.

The site of maximal tenderness often indicates which ligament(s) were injured. Medial ankle pain may accompany a lateral ligament injury as the talus abuts the medial tibia during an inversion injury. With syndesmotic sprains, discomfort may radiate up the syndesmosis along the anterolateral leg. In the acute setting, specific tests to assess ligamentous instability are not feasible due to patient discomfort. As the acute pain subsides, an anterior drawer test is used to assess the degree of anterior shift of the talus relative to the tibia. Alignment should be evaluated as well, best done by examining the foot and ankle with the patient standing. Varus alignment of the hindfoot predisposes the ankle to rolling inward.

Ankle sprains are graded depending upon the severity of the injury. Grade I ankle sprains occur with minimal ligament injury, minimal swelling and tenderness, and minimal pain with weightbearing. Grade II injuries occur when the ligaments have been stretched but remain in continuity. These patients have moderate swelling, tenderness, and pain with weightbearing. Grade III sprains occur with complete rupture of the ligaments and cause significant pain and swelling.

Differential Diagnosis and Suggested Diagnostic Testing

In addition to lateral ligament sprain, supination injuries may cause ankle fracture, lateral process talus fracture, anterior process calcaneus fracture, peroneal tendon injury, osteochondral lesion of the talus, and stretch injury to the superficial peroneal nerve. For this reason, we routinely obtain weightbearing radiographs of the ankle to ensure that there is no bony injury or malalignment through the ankle joint. Pronation injuries similarly can cause medial ligament, bone, and/or tendon injury.

Acute surgical intervention is rare when the ankle joint remains properly aligned, whereas surgery is often necessary in cases with altered alignment. While disruption of normal alignment is very uncommon with lateral or medial ankle sprains, in part since the talus is nestled within the confines of the lateral and medial malleoli, syndesmotic injuries more commonly cause disruption of the ankle alignment. With high-grade injury to the syndesmotic ligaments, the fibula may shift laterally, allowing the talus to follow. This is detrimental to the long-term function of the ankle due to abnormal loading of the thin cartilage of the ankle joint and the development of post-traumatic arthritis. It is for this reason that we stress the importance of weightbearing ankle radiographs to assess alignment, with imaging of the contralateral and presumably normal ankle for comparison as needed.

Additional imaging is not typically necessary when radiographs demonstrate a well-aligned ankle. If symptoms persist a few months after an ankle sprain, MRI may be indicated. Additionally, stress radiographs can quantify laxity at the tibiofibular or tibiotalar joints.

Non-operative Management

Ankle sprains are treated with rest, ice, compression, and elevation (RICE). The severity of the ligament injury guides treatment. In minor injuries where weightbearing, walking, or even sporting activities can be performed immediately following the injury, the use of a soft ankle brace and ankle strengthening exercises may be sufficient. These exercises concentrate of range of motion, proprioception, and peroneal strengthening. With more significant injuries, immobilization in either a restrictive ankle brace or a walking boot may be necessary. For many patients, a few weeks of immobilization is followed by physical therapy. Activity can then be gradually resumed, although care should be taken with cutting sports or when on irregular ground so as not to sustain a reinjury. Patients are instructed to wear a soft supportive ankle brace for up to 6 months following an ankle sprain. Similarly, patients are instructed to avoid high heels as this can increase the likelihood of recurrent injury.

Medial ankle sprains and high ankle sprains take considerably longer to recover from than lateral ankle sprains. In these injuries, boot immobilization is typical for 1 month, followed by physical therapy.

Indications for Surgery

Weightbearing ankle radiographs after an acute ankle sprain evaluate the alignment of the ankle joint. If there is no malalignment of the ankle, which is the case in the vast majority of patients, then surgery is rarely indicated. If malalignment of the ankle has developed as a result of the injury, which most commonly is at the level of the syndesmosis, then acute surgical intervention is often recommended to reduce and stabilize the ankle.

Surgical treatment after ankle sprains is more commonly performed as a result of persistent symptoms months after the injury. Indications for surgery typically include a failure of non-surgical treatment and objective pathologic findings, such as documented mechanical ankle instability or an osteochondral lesion seen on MRI.

Operative Management

Ligament reconstruction procedures can address lateral or medial ankle instability. These procedures either utilize native local tissue, reroute tendons, or use allograft (cadaver) tendon to provide stability to the joint. More than 50 lateral ligament reconstruction procedures have been described. The most commonly used procedure is the modified Broström procedure, which involves tightening the lateral ankle ligaments while also incorporating part of the extensor retinaculum into the repair. Most ligament reconstruction procedures require a lengthy recovery.

Associated injuries may require operative treatment as well. Osteochondral lesions may be treated with open or arthroscopic cartilage procedures that stimulate

healing or replace injured cartilage. Ankle arthroscopy can be effective in treating loose bodies or ankle impingement syndromes, which cause pain with ankle range of motion due to chronically inflamed tissue within the ankle joint. And peroneal tendon injuries, as mentioned previously, may require operative intervention.

Expected Outcome and Predictors of Outcome

The majority (more than 80%) of patients with ankle sprains are effectively treated without surgery. In those requiring surgery, studies report that roughly 80% of patients experience substantial pain relief and a sense of ankle stability. Surgical treatment is less effective, and should be utilized infrequently, in patients with pain after an ankle sprain yet without objective findings on clinical examination or imaging. Surgical outcomes also become less predictable in cases of revision surgery.

New Techniques and Treatments

The goal of treatment for soft tissue disorders of the foot and ankle is to optimize function while minimizing risk. If a non-surgical treatment can accomplish a good outcome, then this is typically the preferred treatment. When interventions such as injections or surgery are necessary, great care is given to minimize risks. Recent developments in intervention techniques for some conditions further minimize risk and yet accomplish equivalent or superior outcomes. These include minimally invasive surgical techniques, which address pathology through smaller approaches, and the use of orthobiologics to assist with healing. The evolution of these techniques has refined treatment for certain conditions.

Minimally Invasive Surgery

Arthroscopy, which uses a small camera and small tools to work within a joint, has been used for decades. Recently, the advent of smaller cameras and tools has enabled surgeons to work within tighter spaces, such as within the joints of the ankle and foot. While certain conditions remain better managed with traditional open approaches, osteochondral lesions, cartilage injury, and loose bodies – to name a few – are frequently treated with arthroscopic techniques. Certain soft tissue conditions also can be addressed with the use of the arthroscope. For example, peroneal tendoscopy is a useful technique for diagnosing and treating some peroneal tendon problems. Similarly, tendoscopy of the Achilles tendon can have a role in treating Achilles tendinitis. Bone spurs, particularly around the Achilles tendon insertion, can be removed with endoscopic techniques (Fig. 30.7). And lastly, newer surgical

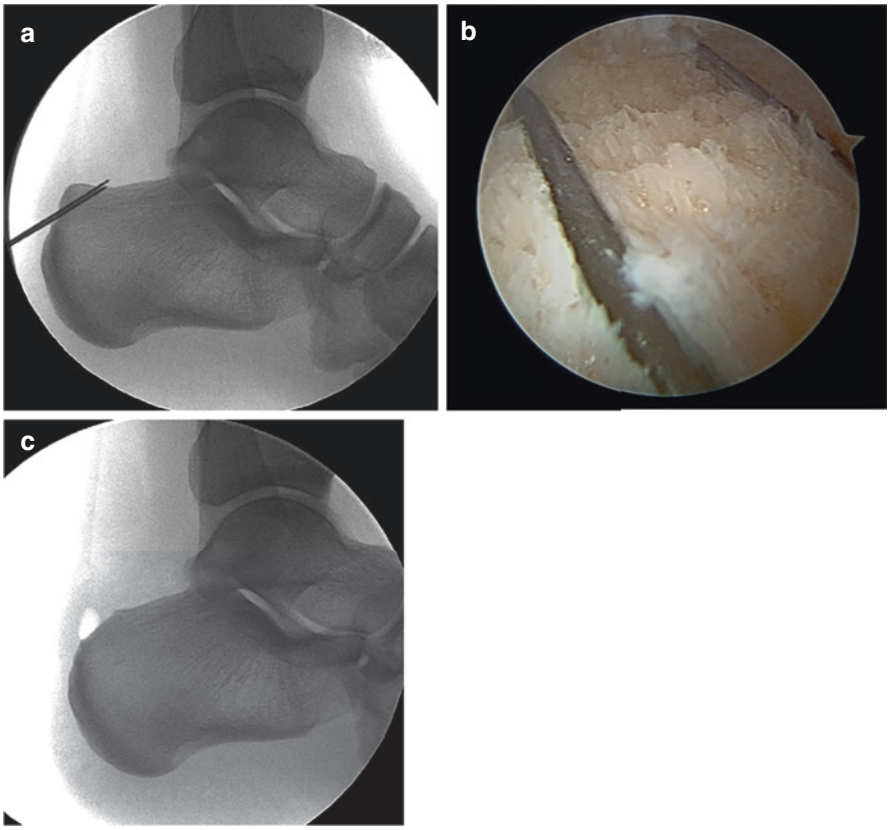

Fig. 30.7 Intraoperative X-ray of Haglund's lesion before removal with wires marking level of planned excision (**a**). Endoscopic removal of Haglund's lesion with the bone shaved down to level of metal wires (**b**). X-ray after removal of bony prominence and wires (**c**)

shaving tools that run at low speed and high torque facilitate safe removal of bone spurs around the ankle and foot through small incisions.

Orthobiologics

The past decade has seen a growth in the interest and use of biologic agents to treat orthopedic problems. These include platelet-derived growth factor (PDGF), which are released in response to tissue injury by macrophages and platelets. These factors recruit additional cells to the site of injury and are thought to promote healing by increasing angiogenesis, in addition to other mechanisms. PDGFs have been studied in the foot and ankle with regard to bone healing. The impact of PDGF on soft tissue injuries, such as Achilles rupture, remains an area of great interest. Platelet-rich plasma (PRP) is another area of interest for biologic treatment of foot and ankle

conditions. PRP contains a concentrated volume of platelets and is derived from autologous blood. Platelets from this concentrate have been shown to release growth factors that augment healing. In addition to other conditions, PRP has been investigated as a treatment of Achilles tendinopathy and plantar fasciitis. As of yet, there is no conclusive evidence supporting the use of PRP for these conditions. Nevertheless, PRP and other biologic compounds remain an area of great potential within musculoskeletal care (Table 30.1).

Table 30.1 Ten common soft tissue disorders of the ankle with associated typical presentation, diagnostic testing, and treatment

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Achilles tendinosis	TTP midportion of Achilles, posterior heel Achilles pain with active plantar flexion (single-leg raise)	Thompson test to rule out rupture Radiograph to evaluate for Haglund's lesion or insertional spur -Ultrasound or MRI for confirmation of tendinosis	PT: eccentric strengthening, ankle/foot intrinsic strengthening Heel lift Dorsiflexion night splint	Pain refractory to conservative management	Tendon debridement Removal of Haglund's lesion Gastrocnemius recession
Achilles tendon rupture	Acute onset pain and weakness in calf May hear or feel a pop	Thompson test Plantar flexion strength Passive ankle dorsiflexion	Non-surgical treatment can be effective treatment but must follow specific protocol	Healthy patient who elects for operative intervention	Tendon repair using either open or minimally invasive technique
Gastrocnemius strain	Acute onset of pain in upper calf May hear or feel a pop	Thompson test to rule out Achilles rupture Palpation for site of maximal tenderness	Immobilization in tall walking boot PT: strengthening and stretching to begin when pain improved	Very uncommon to treat surgically	NA
Peroneal tendinopathy	Pain at posterolateral ankle May have clicking or tendon instability	Identify site of maximal tenderness Assess hindfoot alignment Test ankle ligament stability Circumduction testing for peroneal instability MRI	Immobilization in tall walking boot PT: strengthening and range of motion	Persistent symptoms despite non-surgical care Peroneal tendon instability	Open tendon exploration and repair Tendoscopy

(continued)

Table 30.1 (continued)

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Posterior tibial tendon dysfunction	Pain at medial ankle Deformity may be present	Standing evaluation of alignment Plain weightbearing X-rays MRI	Immobilization in tall walking boot PT: stretching of calf and training of tendon Orthotic to off-load tendon	Persistent symptoms despite non-surgical care	Depends upon stage of disease May include tenosynovectomy, deformity correction, hindfoot fusions
Ankle sprain	Acute onset pain after twisting injury Swelling and ecchymosis present	Weightbearing plain ankle X-rays Palpation for site of maximal tenderness	Boot immobilization followed by PT and return to activities	Altered ankle alignment on X-rays (rare) Chronic associated injury with persistent symptoms	ORIF if altered alignment (rare) Ligament reconstruction for chronic symptoms Ankle arthroscopy
Ankle instability	Recurrent ankle sprains	Weightbearing plain ankle X-rays MRI	PT: strengthening, proprioception, peroneal strengthening	Persistent limiting symptoms despite PT	Ligament reconstruction Ankle arthroscopy
Ankle impingement syndrome	Pain along ankle joint line May follow injury	Identify site of maximal tenderness Plain X-rays MRI	Immobilization in tall walking boot PT Intra-articular corticosteroid injection	Persistent pain or mechanical symptoms of locking or catching	Ankle arthroscopy
Tarsal tunnel syndrome	Pain at posteromedial ankle Sensory changes at plantar foot	Tenderness along tibial nerve Replicated symptoms with tibial nerve testing (Tinel's sign)	Immobilization in tall walking boot Orthotic if associated valgus hindfoot Anti-inflammatory medication Local corticosteroid injection	Persistent symptoms despite non-surgical care	Tarsal tunnel release
Flexor hallucis longus tenosynovitis	Pain at posterior ankle Pain exacerbated with great toe flexion	Weightbearing plain ankle X-rays MRI	Immobilization in tall walking boot PT	Persistent pain despite non-surgical care	FHL tenosynovectomy (open or arthroscopic)

Suggested Reading

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Chapter 31
Midfoot Arthritis and Disorders of the Hallux

Christopher P. Chiodo, Jeremy T. Smith, Elizabeth A. Martin, and Eric M. Bluman

I. *Hallux Rigidus and Hallux Valgus.* The hallux, or great toe, has an important role in normal foot function. Biomechanically, it contributes to foot strength, stability, and balance. It is comprised of two bones, the proximal and distal phalanges (Fig. 31.1). These bones are relatively large when compared to the bones of the other toes. The base of the proximal phalanx articulates with the head of the first metatarsal at the first metatarsal-phalangeal (MP) joint. The two most common conditions that affect the great toe are hallux rigidus and hallux valgus.

   A. *Hallux Rigidus.* Osteoarthritis of the first MP joint is referred to as hallux rigidus, which is Latin for "stiff big toe." Many patients with hallux rigidus are minimally symptomatic. In these instances, the disease results only in decreased motion that often goes unnoticed by the patient. The frequent absence of pain in the face of radiographic changes and reduced motion make *stiffness* typical of this disease, as reflected in the name "hallux rigidus." As the disease progresses, large osteophytes form on the dorsal aspect of the MP joint (Fig. 31.2). These may cause pain by irritating adjacent soft-tissue structures or by impinging with terminal dorsiflexion of the joint.

      (i) *Summary of epidemiology.* Hallux rigidus generally affects middle-aged and elderly individuals. In a minority of cases, it may be caused by trauma or inflammatory disease. In most cases, however, the etiology is unknown. Some have speculated that it may be secondary to subtle dorsiflexion malalignment of the first metatarsal, contracture of the flexor hallucis brevis tendon, or chondral injury.

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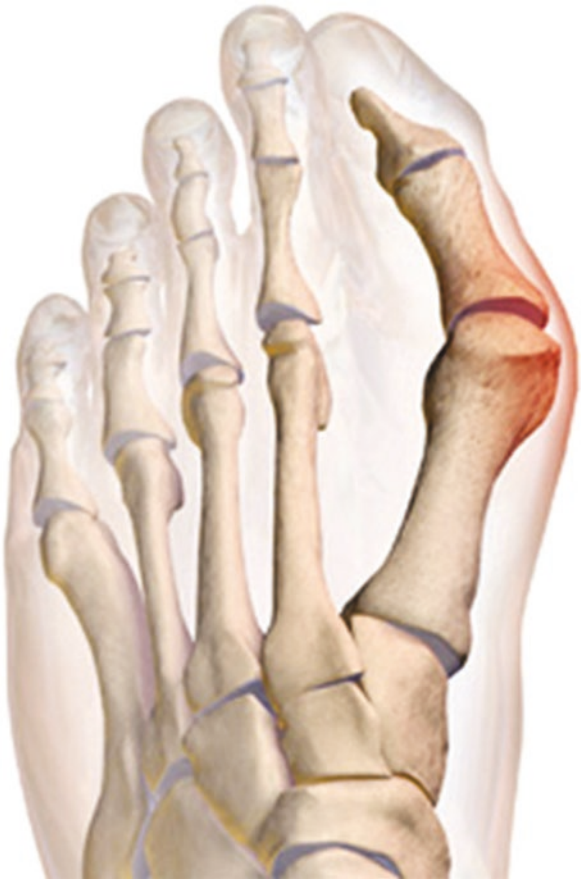

Fig. 31.1 Line drawing of a moderate hallux valgus deformity. Note the lateral deviation of the hallux as well as the medial angulation of the first metatarsal. Additionally the sesamoid bones are displaced lateral to the center of the MP joint

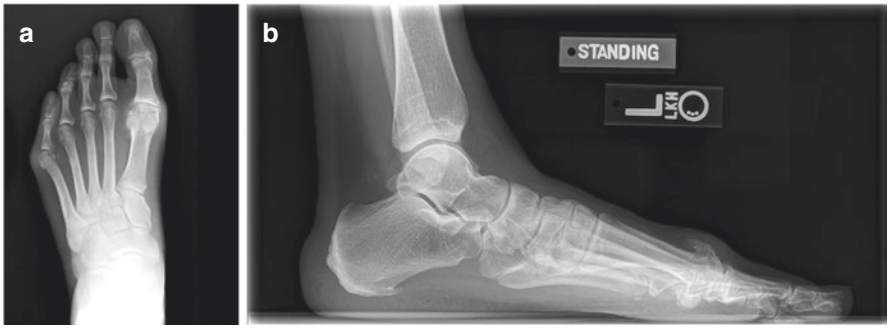

Fig. 31.2 Anteroposterior (**a**) and lateral (**b**) radiographs of a patient with hallux rigidus. Note the dorsal osteophyte formation as well as the loss of joint space at the metatarsal/phalangeal joint

(ii) *Clinical presentation.* Patients with hallux rigidus typically complain of pain on the dorsal aspect of the joint, centrally within the joint, or both. This contrasts with medial pain, which is more typical of a bunion, or plantar pain, which usually indicates sesamoid pathology. Two pertinent factors in the patient's history are (1) pain that is exacerbated by the joint rubbing against shoes and (2) pain that occurs when the affected foot pushes off. These indicate that the dorsal osteophytes are the primary pain generator. If, on the other hand, symptoms persist throughout the gait cycle or in the absence of shoes rubbing on the joint (e.g., going barefoot), then it is more likely that the underlying arthritis of the MP joint is the main pain generator.

(iii) *Differential diagnosis and suggested diagnostic testing.* The differential diagnosis for central and dorsal MP joint pain includes gout, stress fracture, and osteochondral injury. Gout will typically present more acutely and with a greater degree of inflammation. Meanwhile, pain and tenderness from a stress fracture will typically be localized more proximally at the metatarsal neck. Finally, an osteochondral injury will usually not be associated with dorsal spur formation.

Weight-bearing radiographs should be obtained in all patients with suspected hallux rigidus. Dorsal osteophyte formation and joint space narrowing will be apparent in most cases. Beyond radiographs, advanced imaging is usually not indicated. One exception would be the use of magnetic resonance imaging to rule out a stress fracture or sesamoid pathology. Similarly, laboratory studies are generally not needed, unless ruling out gout or infection. In these instances, however, joint aspiration is more specific and accurate.

(iv) *Non-operative management.* There are several non-operative treatment options available. Shoewear modification, specifically choosing shoes with a deep toe box, makes room for the dorsal osteophytes that are typically present. Wider shoes may be also helpful in this regard. Unfortunately, some patients report that while wider and deeper shoes provide relief for the hallux, such shoes are ill fitting in other regions of the foot. This may lead to excessive motion, shear, and blister formation.

Shoes with a stiff sole may also be helpful. The stiff sole will decrease dorsiflexion through the MP joint, effectively "stress-shielding" it. Both custom-made and commercially available rocker bottom shoes may also stress-shield the joint by creating a biomechanical "rocker" that decreases motion at the joint.

Finally, shoe stretching may also help. Stretching the toe box in the region of the osteophytes may minimize or eliminate spur pain. There are several available devices that accomplish this; however, in our experience a "ball-and-ring" shoe stretcher is the most effective. In contrast to other shoe stretching devices, this device stretches the shoe material locally, over the enlarged joint. As such, it avoids problems

with shear and improper shoe fitting elsewhere in the foot that may occur when using other devices.

With regard to custom orthoses, these devices are often expensive, not covered by insurance, and ineffective in the treatment of hallux rigidus. Their poor efficacy is due to the fact that, with hallux rigidus, the dorsal spurs normally crowd the shoe. Placing a custom orthosis in the shoe only further exacerbates this crowding.

Instead of orthotics, many providers have begun using carbon fiber inserts to treat hallux rigidus. These are non-custom devices that are very thin and as such do not "overstuff" the shoe. However, they are relatively stiff and thus still stress-shield the MP joint. When prescribing these devices, the phrase "Morton's extension" is used to specify that the device extends to the tip of the hallux.

Finally, cortisone injections are another reasonable non-operative treatment option for hallux rigidus. The accuracy of these procedures likely increases when performed under fluoroscopic or ultrasound guidance. Repeated injections, however, may have a harmful effect on the remaining cartilage. As such, only one or two injections are considered prior to surgery. In one clinical study, two-thirds of patients with mild disease who underwent injection combined with gentle manipulation were able to avoid surgery.

The clinical literature with regard to the non-operative treatment of hallux rigidus is sparse. Nevertheless, in the authors' experience, many—if not most—patients with hallux rigidus will enjoy sufficient relief and avoid surgery with one or more of the measures listed above.

(v) *Indications for surgery.* Patients with hallux rigidus are considered appropriate surgical candidates if they have regular pain that has been present for at least 3 months, is refractory to non-operative measures, and interferes with activities of daily living. This functional criterion includes the ability to wear reasonable shoes.

(vi) *Operative management.* The surgical management of hallux rigidus may be divided into joint-sparing and joint-sacrificing procedures. Traditionally, joint-sparing procedures are reserved for patients in whom there is some cartilage remaining or in whom pain is caused primarily by irritation or impingement of the dorsal osteophytes, rather than by the osteoarthritic process within the joint. Such patients will typically complain of shoe pain and pain as the affected foot pushes off during the gait cycle. The most commonly performed joint-sparing procedure for hallux rigidus is a cheilectomy, in which the offending dorsal osteophytes are resected. Recently, some surgeons are now using minimally invasive techniques to perform this procedure. Less commonly, distal metatarsal and proximal phalangeal osteotomies have been described to "decompress" the joint. Finally, procedures that replace the entire joint or half of the joint have also been described.

These have not enjoyed widespread popularity, perhaps due to concerns about the longevity of the implants.

Arthrodesis is indicated when there is pain throughout the gait cycle and not just at terminal push-off. With arthrodesis, the first metatarsal and proximal phalanx are surgically fused. The joint is eradicated and the bones are fixed together with a plate and/or screws. Of note, the hallux is positioned in slight valgus to facilitate shoewear. It is also fused in slight dorsiflexion. With this adjustment, the tip of the toe will not touch the ground. This positioning creates a plantar rocker to allow for more efficient ambulation, and patients should be reassured that this is planned and advantageous.

(vii) *Expected outcome and predictors of outcome.* Most patients undergoing treatment for hallux rigidus are able to return to the activity level they enjoyed prior to the onset of symptoms. In one recent study examining arthrodesis, high levels of function in both everyday life and recreational activities were noted postoperatively. For instance, 92% of patients could hike, 75% of patients could return to jogging, and 80% of patients could resume golfing. Nearly all patients could return to work.

B. *Hallux Valgus.* A hallux valgus deformity, or bunion, is characterized by lateral deviation of the great toe. This leads to an angular deformity at the MP joint and the development of a secondary bony prominence (Fig. 31.2). While in some patients the medial eminence of the distal first metatarsal may be slightly enlarged, it is important to note that the abnormal physical appearance of the hallux is due primarily to angular deformity.

(i) *Summary of epidemiology.* Bunions are one of the most common conditions treated by foot and ankle specialists. They affect over 30% of adults, occur primarily in shoe-wearing societies, and are more prevalent in females compared to males. The narrow toe box of many shoes, especially women's shoes, applies a laterally directed force on the hallux. This may contribute to the development of a bunion or irritate an existing deformity due to rubbing of the shoe. Other factors that have been implicated in the etiology of bunions include joint instability, muscle imbalance, hindfoot pronation, skeletal abnormalities, and hereditary predisposition. Symptomatic bunions occur in patients of all ages, although the prevalence is higher in middle-aged and elderly individuals.

(ii) *Clinical presentation.* Many patients with mild bunion deformities have little to no pain. Patients who do have symptoms typically will note pain localized to the medial aspect of the first MP joint. This is an important clinical finding, as dorsal or plantar pain should alert the clinician to another pathology, such as hallux rigidus or sesamoid pain. Symptoms are typically aggravated by shoes, and especially fashionable shoes with a narrow toe box. Pain may also be exacerbated by

repetitive joint motion from prolonged walking, running, and sports. It may occasionally radiate proximally due to irritation of the dorsal medial cutaneous nerve as it courses over the MP joint.

(iii) *Differential diagnosis and suggested diagnostic testing.* Two radiographic parameters are particularly important when assessing hallux valgus. The first is the hallux valgus angle, formed by the intersection of the long axes of the hallux and first metatarsal. This is useful in grading deformities as mild (15–30°), moderate (30–40°), or severe (>40°). Meanwhile, the intermetatarsal angle measures the divergence between the first and second metatarsals. This angle is useful in determining the type of procedure that will be necessary if surgical correction is performed.

(iv) *Non-operative management.* The non-operative management of hallux valgus includes shoe stretching as well as local pads and braces. As with hallux rigidus, a ball-and-ring shoe stretcher is able to focally stretch the toe box over the deformity while preserving the contour of the remaining shoe. Numerous braces, pads, and spacers are available commercially. Soft, low-profile devices are tolerated best. One appealing option is a forefoot neoprene sleeve with a seam along the medial side of the device. This both cushions the bony prominence and also draws the hallux into more anatomic alignment.

Custom orthoses are usually not effective in the treatment of hallux valgus. The one exception is in the case of advanced hindfoot pronation that results in increased pressure on the medial hallux.

(v) *Indications for surgery.* Patients are considered appropriate surgical candidates if they have substantial, chronic pain that is not alleviated by reasonable shoewear or shoe stretching. It is important to note that cosmesis and the desire to wear high heels or fashionable shoes are generally not considered indications for surgery. Patients who inquire about bunion surgery should be carefully counseled that cosmesis is not considered an indication for surgery. Patients must also be cognizant of—and be able to comply with—a non-weight-bearing period of up to 6 weeks postoperatively, depending on the nature of the procedure performed.

(vi) *Operative management.* Numerous procedures have been described for the treatment of hallux valgus. More mild deformities may be addressed with a “modified McBride” procedure, in which the contracted lateral structures of the MP joint are released while, medially, the metatarsal eminence is shaved and the joint capsule is tightened. This realigns the hallux at the MP joint and to some degree may correct the elevated intermetatarsal angle. Alternatively, a “chevron” osteotomy of the distal first metatarsal may also be performed to shift the metatarsal head laterally.

When the intermetatarsal angle is substantially elevated (e.g., greater than 14°), a proximal procedure may also be necessary. Most

surgeons will perform some type of metatarsal osteotomy to address the malalignment of this bone. Other options include arthrodesis of the first TMT joint or even suture-button fixation.

Advanced arthritis or instability of either the first MP or TMT joint is an indication for surgical arthrodesis (fusion). With this, the deformity is corrected through the joint, which is then fused in more anatomic alignment.

(vii) *Expected outcome and predictors of outcome.* Approximately 90% of patients undergoing surgery for hallux valgus are satisfied and have lasting relief. One common complication of surgery, however, is recurrent deformity. This occurs in less than 15% of patients and is likely due to residual soft-tissue contractures as well as changes in the bony anatomy on the plantar aspect of the metatarsal. In our experience, though, pain relief and patient satisfaction usually persist despite recurrent deformity. Another complication associated with hallux valgus correction is decreased motion at the first MP joint. This is particularly noticeable for patients who attempt to wear high heels postoperatively. Finally, residual sesamoid malalignment on postoperative x-rays has been associated with recurrent deformity. This is likely due to the persistent eccentric pull of the flexor hallucis brevis tendon, within which the sesamoids are located.

II. *Midfoot Arthritis.* The midfoot is composed of the navicular, cuboid, and cuneiform bones. It connects the hindfoot to the metatarsals and allows for efficient force transmission and propulsion, which are critical for ambulation. Osteoarthritis often affects the joints of the midfoot and, as with other foot arthritides, can be quite debilitating.

A. *Summary of epidemiology.* Symptomatic midfoot arthritis affects both middle-aged and elderly individuals and is present in approximately 10% of individuals over 50 years of age. There may be a slightly higher prevalence in females, and both obesity and occupation have been cited as risk factors. While usually idiopathic, in some cases, the condition may be secondary to a prior midfoot sprain or Lisfranc fracture dislocation.

B. *Clinical presentation.* Patients typically complain of insidious aching pain localized to the midfoot. There is usually "start-up" pain, which occurs when standing after sitting for a long period of time or when first arising in the morning. A tight shoe counter may irritate dorsal osteophytes. Deformity may also be noted, specifically, abduction of the forefoot. This, in turn, may compromise the medial longitudinal arch and lead to the development of an acquired flatfoot.

C. *Differential diagnosis and suggested diagnostic testing.* Two important items in the differential diagnosis for midfoot arthritis are tendon pathology and stress reaction. The posterior tibial tendon is the main dynamic stabilizer of the medial longitudinal arch. It inserts primarily on the medial pole of the navicular. Associated pain and swelling is usually more medial and extends

proximally into the hindfoot and medial ankle. Meanwhile, a navicular stress fracture can present with symptoms similar to midfoot arthritis. The pain associated with this diagnosis, however, usually has a more acute onset and is of greater intensity. A period of strict non-weight-bearing is integral to healing navicular stress fractures, and, as such, provider awareness is critical.

Weight-bearing x-rays of the foot are usually sufficient to establish the diagnosis of midfoot arthritis. Computed tomography (CT) is helpful in further specifying which joints are involved and the extent of disease present. CT is also important for surgical planning. Magnetic resonance imaging is helpful in differentiating midfoot arthritis from stress fractures and tendinopathy and should be considered if the diagnosis is in question.

Inflammatory arthritis is also on the differential diagnosis. If the patient reports pain in other joints and/or morning stiffness, screening laboratories should be considered, especially in the setting of bilateral disease, a relatively young patient age, or a positive family history.

D. *Non-operative management.* The non-operative treatment of midfoot arthritis includes nonsteroidal anti-inflammatory medications, shoe modification, inserts, and cortisone injections. As with hallux rigidus, the use of a rocker bottom shoe and carbon fiber baseplate decreases bending moments at the joints of the midfoot. In the absence of significant dorsal spur formation, this often provides substantial relief. Cortisone injections also play a valuable role in the treatment of midfoot arthritis. Some patients have long-term relief with a single cortisone injection into the diseased joint(s), while others require serial injections every 4–6 months. Of note, the joints of the midfoot are small and often occluded by dorsal osteophytes. As such, the accuracy and efficacy of injections is significantly enhanced by either fluoroscopic or ultrasound guidance.

E. *Indications for surgery.* Surgery is indicated for those patients who have chronic, recalcitrant pain that interferes with daily living. Those patients who require arthrodesis must also be willing and able to comply with a postoperative protocol that may entail up to 3 months of protected weight-bearing. Contraindications for fusion include active infection as well as insufficient perfusion and soft-tissue coverage. Finally, many orthopedists insist on smoking cessation given the negative impact smoking has on fusion rates.

F. *Operative management.* The surgical management of midfoot arthritis generally entails either exostectomy or arthrodesis. An exostectomy—i.e., removal of painful osteophytes—is indicated when patients have pain that is felt to be caused by irritation of the soft-tissue structures that overlie a prominent osteophyte. Patients must understand that with an exostectomy the arthritic joint is still present and could potentially result in persistent pain. Arthrodesis better addresses this concern and is indicated for patients who have pain both with and without shoes and in whom the primary pain generator is felt to be the arthritic joint itself.

G. *Expected outcome and predictors of outcome.* Most patients with mild disease respond to non-operative measures as delineated above. For more advanced disease in which fusion is indicated, modern success rates have ranged from 92% to 100%. Risk factors for nonunion include diabetes, smoking, poor nutrition, compromised bone stock, and a history of trauma with advanced soft-tissue stripping. A low vitamin D level may also predispose to nonunion.

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Chapter 32
Plantar Fasciitis

James P. Ioli

Plantar Fasciitis

Summary of Epidemiology

Plantar fasciitis accounts for about one million patient visits per year in the USA. It affects 10% of the general population and makes up 10% of runner-related injuries. It is estimated that between $192 and $376 million dollars is spent annually on treatments for this condition. It usually affects adults of all ages and peaks between 40 and 60 years of age. Adult women present twice as often as men, while in younger patients men and women are affected equally. There is no association with race or ethnicity. One third of the patients experience plantar fasciitis bilaterally.

Clinical Presentation

The fascia is a long thick ligament-like structure that is located on the plantar aspect of the foot (Fig. 32.1). It extends from the medial and lateral calcaneal tubercles distally toward the toes. The fascia consists of a medial, central, and lateral band. The plantar medial heel attachment is the most common area of pain and discomfort. The pain is usually acute when the patient first steps out of bed or first moves after a period of inactivity. The heel pain usually subsides after a few minutes of activity. At times, the pain will worsen as the day progresses. Some patients may also complain of burning, tingling, and sharp pain in the heel. The discomfort can also be felt in the medial arch. Factors that can influence or trigger the onset of pain

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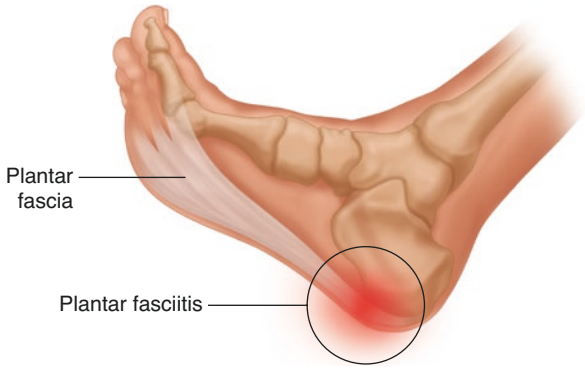

Fig. 32.1 Depiction of the plantar fascia and the most common area of pain at the plantar medial heel attachment

are foot structure (pes cavus, pes planus); overpronation, an excessive inward roll and collapse of the medial arch and foot; tightness and/or weakness of the gastrocnemius, soleus, or Achilles tendon; weight; occupation, such as factory workers, teachers, and postal workers; worn-out or poorly fitting shoes; and sudden increase in activity or excessive training. Some studies report that plantar fasciitis can linger for 12–18 months. It is possible for the condition to resolve spontaneously, and 80% of cases resolve in 1 year. About 5% of patients for whom conservative therapy is ineffective choose surgery in an attempt to resolve their symptoms. Some patients are concerned when they see a plantar heel spur on a radiograph. Advise them not to be concerned. Many people have heel spurs and have no heel pain.

Differential Diagnosis and Suggested Diagnostic Testing

When investigating the origin of inferior heel pain, one should think of the musculoskeletal, vascular, dermatological, and neurological systems. As mentioned previously, the musculoskeletal system is usually the primary cause of heel pain (plantar fasciitis). If the pain worsens, and does not respond to treatment, it is important to rule out a stress fracture, bone cyst, bone tumor, bone contusion, osteomyelitis, Paget's disease, rupture of the plantar fascia, apophysitis (Sever's disease – pain in the growth plate of the heel), sarcoidosis, and inflammatory arthropathy. Conditions of the vascular system, such as peripheral arterial disease and vascular insufficiency, can cause heel pain. Plantar verrucae, porokeratoses, ulcers, and foreign bodies fall into the dermatological system. Neurological causes of heel pain can be tarsal tunnel syndrome, medial or lateral plantar nerve neuritis, peripheral neuropathies, entrapment, or neuroma. S1 radiculopathy should also be considered. A proper and thorough physical exam will aid in the assessment of all four systems.

Recalcitrant heel pain requires further diagnostic assessment, which may include some of the following studies: x-rays to look for bony lesions; MRI to rule out soft tissue or bony lesions; electromyography (EMG) for tarsal tunnel syndrome; three-phase bone scan for stress fracture or bone infection; computed tomography (CT)

for subtalar arthritis, calcaneal cysts, and stress fractures; ankle-brachial index/pulse volume recording (ABI/PVR) for peripheral arterial disease; ultrasound to rule out soft tissue pathology; or bone scan. If there is a suspicion of an inflammatory arthropathy, laboratory studies may include a complete metabolic panel, a complete blood count (CBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), a rheumatoid factor, and HLA-B27 (to evaluate for spondyloarthropathy).

Non-operative Management

If a patient presents to your office and complains of symptoms which you determine to be consistent with plantar fasciitis, consider furnishing a handout with information about plantar fasciitis, including an explanation of its origin and general treatment regimens. Pictures and/or drawings of the anatomy and of stretching/strengthening exercises are extremely helpful to patients, so that they can develop insight into the problem (Fig. 32.2). Initial recommendations would also

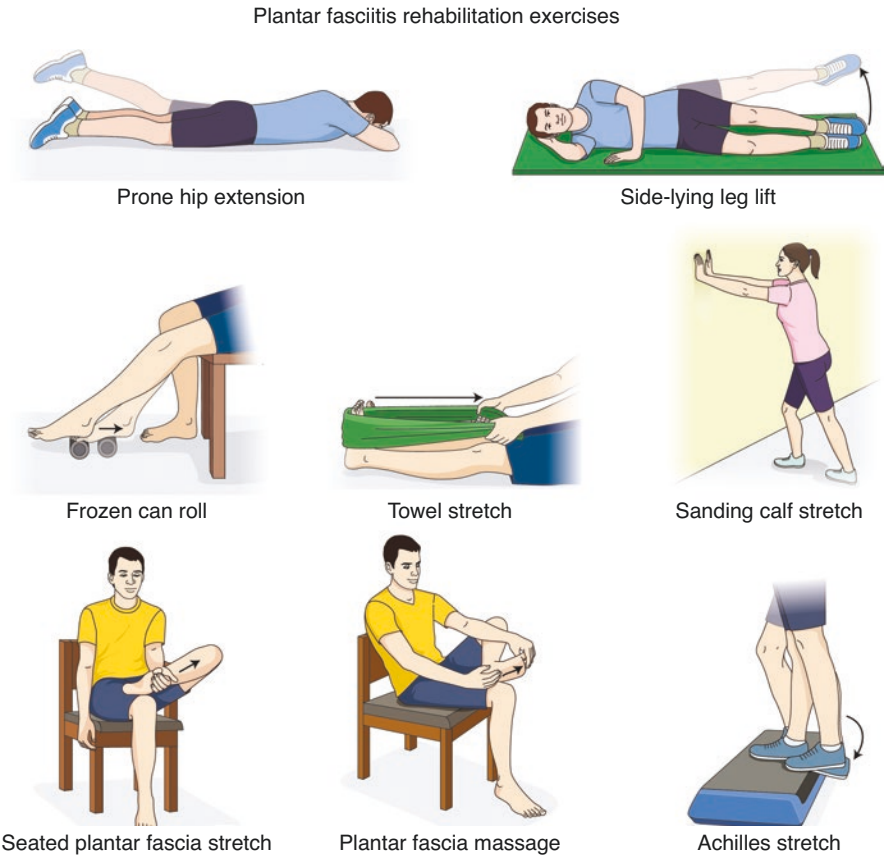

Fig. 32.2 Recommended plantar fasciitis rehabilitation exercises

include an evaluation for new supportive shoes, silicone heel inserts, and over the counter orthotics; custom orthotics may be considered if there is no or minimal progress after 3 months of treatment. OTC and custom orthotics are used to provide support and a better alignment of the foot in order to decrease the mechanical forces that aggravate the condition. Initially a home exercise program is usually recommended. It has been shown that plantar fascia and intrinsic foot muscles stretching techniques have reduced pain associated with plantar fascia. As illustrated later in this article, a towel stretch, plantar fascial stretching and massage, and Achilles stretch are all helpful in decreasing pain and discomfort. If this fails, then physical therapy would be recommended. There is no evidence that a formal PT program is better than the home exercise program. There is inconsistent or limited-quality patient-oriented evidence that when used with other conservative therapies, nonsteroidal anti-inflammatory drugs can provide short-term improvement in plantar fascial pain. However, OTC NSAIDs could also be recommended. A night splint is usually prescribed if the patient complains of pain on the first step out of bed. Keeping the foot and ankle in a neutral 90° position, the night splint prevents ankle plantar flexion (and associated tightening of the heel cord) during sleep. There is evidence that the night splint helps; however patients may complain of foot discomfort and interrupted sleep. Patients should don supportive shoes after removing the night splint and avoid walking barefoot or in non-supportive slippers.

Typically, a follow-up appointment is scheduled for 6–8 weeks following the initial visit in which plantar fasciitis was diagnosed. When the patient returns, the initial treatment regimen is reassessed. If there has been improvement with formal physical therapy, the prescription is usually renewed. A physical therapy program that focuses on lower extremity stretching and strengthening can be beneficial and provide reinforcement for present and future patient compliance. It is also important to review the home exercise program, which supplements the formal PT program, use of the night splint, and OTC NSAIDs. The new shoes and inserts should also be evaluated. If the pain has worsened, then review the differential diagnosis. If there is moderate pain, a prescription for NSAIDs can be considered. If there is severe pain from plantar fasciitis, then immobilization in a removable, below-the-knee walking boot would be recommended at that visit. It is important to review with the patient as well as remind him or her about activities that can aggravate or exacerbate the condition. It is especially important to advise patients, particularly those who run or participate in strenuous athletic activities, to avoid such activities while treatment is in progress. Encourage the patient to cross-train and limit long periods of standing and walking barefoot on hard surfaces. If the patient has a high BMI, a frank discussion about nutrition and weight loss is paramount.

If, at the time of the patient's next appointment (generally within 6–8 weeks), the patient still complains of plantar fasciitis pain, a steroid injection could be considered. The patient should be advised that there can be a weakening of the plantar fascia and possible rupture of the plantar fascia, especially from multiple steroid

injections. Many primary care providers will have little experience with plantar fascial injections; it would be reasonable to refer these patients to foot specialists (e.g., podiatrists, foot and ankle surgeons) for an injection.

Extracorporeal shock wave therapy may be another conservative option for recalcitrant plantar fasciitis. A recent study of the effectiveness of ECSWT demonstrated success rates between 50% and 65% as compared with 34.5% with placebo. This study involved 250 subjects in a prospective, multicenter, double-blind, randomized, and placebo-controlled US Food and Drug Administration trial. If, after 12 months of conservative treatment, the patient is still in severe pain, surgery may be an option.

Indications for Surgery

Surgery is only an option if all conservative treatment has failed and the patient is in severe pain.

Operative Management

Fasciotomy: Part or all of the fascia is sectioned. This can be accomplished by an endoscopic or open procedure.

Others: There are certain other procedures, such as percutaneous partial fasciotomy, cryosurgery, bipolar radiofrequency microdebridement, and Strayer procedure (a gastrocnemius recession procedure to increase ankle dorsiflexion), which have initially appeared promising in small studies. If these treatments prove to be successful in larger studies, they will likely become more common.

Expected Outcome and Predictors of Outcome

The reported success rate for fasciotomy ranges from 70% to 90%. Postoperatively most patients begin weight-bearing to tolerance after 24 h. After suture removal at 10 days, an athletic shoe can be worn as tolerated. Post-op course for open plantar fasciotomy can vary. One study showed that those patients who wore a below-knee walking cast for 2 weeks required less time to obtain 80% pain relief, need less time to return to full activities, and had fewer complications. Both open and endoscopic fasciotomy has been associated with instability complications such as lateral foot pain (calcaneocuboid and metatarsal-cuboid joints), overload, and medial foot pain. A decrease in the longitudinal arch, numbness in the heel, medial arch pain, and strain are potential complications.

Summary Table for Plantar Fasciitis Chapter

Table 32.1 is a summary of the presentation, diagnostic testing, and conservative and operative management of plantar fasciitis.

Table 32.1 Plantar fasciitis

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Plantar fasciitis	Pain in heel after stepping down out of bed or after a period of inactivity Tender to palpation plantar medial heel Nature of pain: sharp, achy, burning	Physical exam X-ray MRI or ultrasound for recalcitrant cases Laboratory studies	Weight loss New shoes with support OTC heel cups, orthotics RICE (rest, ice, compression, elevation) OTC/Rx ibuprofen, naproxen Night splints Physical therapy Stretching and strengthening of plantar fascia and gastrocnemius soleus before and after activity Respite from sport or activity that aggravated the condition Steroid injection Limit prolonged standing and walking barefoot on hard surfaces Cross-training Extracorporeal shock wave therapy	Pain refractory to all conservative management	Plantar fascia release Percutaneous partial fasciotomy Cryosurgery Bipolar radiofrequency microdebridement Strayer procedure

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Chapter 33
Foot and Ankle Injuries

Elizabeth A. Martin, Eric M. Bluman, Christopher P. Chiodo, and Jeremy T. Smith

Abbreviations

AP	Anteroposterior
CT	Computed tomography
EHL	Extensor hallucis longus
FHL	Flexor hallucis longus
IP	Interphalangeal
MRI	Magnetic resonance imaging
MTP	Metatarsophalangeal
NWB	Non-weightbearing
ORIF	Open reduction internal fixation
PT	Physical therapy
TMT	Tarsometatarsal
WBAT	Weightbearing as tolerated

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Toe Fractures

Summary of Epidemiology

Toe fractures are common injuries seen in primary care practice. They are the most common fractures in the foot and comprise 3.6 percent of all fractures. The most common mechanisms are a direct axial load, such as from kicking a stationary object, or a crush injury, such as dropping a heavy object on the toes. The toes, particularly the 5th toe and great toe, can become caught and suffer an abduction force leading to fracture or dislocation.

Clinical Presentation

The patient typically presents with pain, swelling, and ecchymosis in the affected toe. Paresthesias and neuritic pain can sometimes occur, secondary to swelling, traction or crush to the nerve, or a combination. Physical examination of the patient with suspected toe fracture should focus on evaluation of the skin for open wounds, including nail bed trauma, and inspection of toe and foot alignment. Both feet should be evaluated to review and compare toe alignment side by side. Malalignment can be seen as shortening, malrotation, or angulation compared to the surrounding toes and the contralateral corresponding toe. If a wound or malalignment is present, radiographs should be performed urgently with referral to orthopedics or the emergency department. Weightbearing films are preferable to evaluate alignment.

Differential Diagnosis and Suggested Diagnostic Testing

Differential diagnosis of a toe fracture includes IP or MTP dislocation, capsular or ligamentous injury, soft tissue contusion, or metatarsal fracture. AP, lateral, and oblique views of the foot are ordered to evaluate for fracture or malalignment. Weightbearing films are ordered if the patient is able to place weight on the affected foot (Fig. 33.1a, b). Toe x-rays are not typically ordered. Advanced imaging, either MRI or CT scan, is not often necessary but can be utilized to better clarify the extent of soft tissue involvement or to assess for occult fracture or another cause of pain should the diagnosis not be certain on radiographs.

Nonoperative Management

Most lesser toe fractures are treated nonsurgically. Provided the toe is clinically aligned well and there is no dislocation or open injury, buddy taping to the adjacent toe(s) can be performed for comfort (Fig. 33.1c), and a stiff-soled open-toed shoe is used for immobilization. Patients are allowed to bear weight as tolerated.

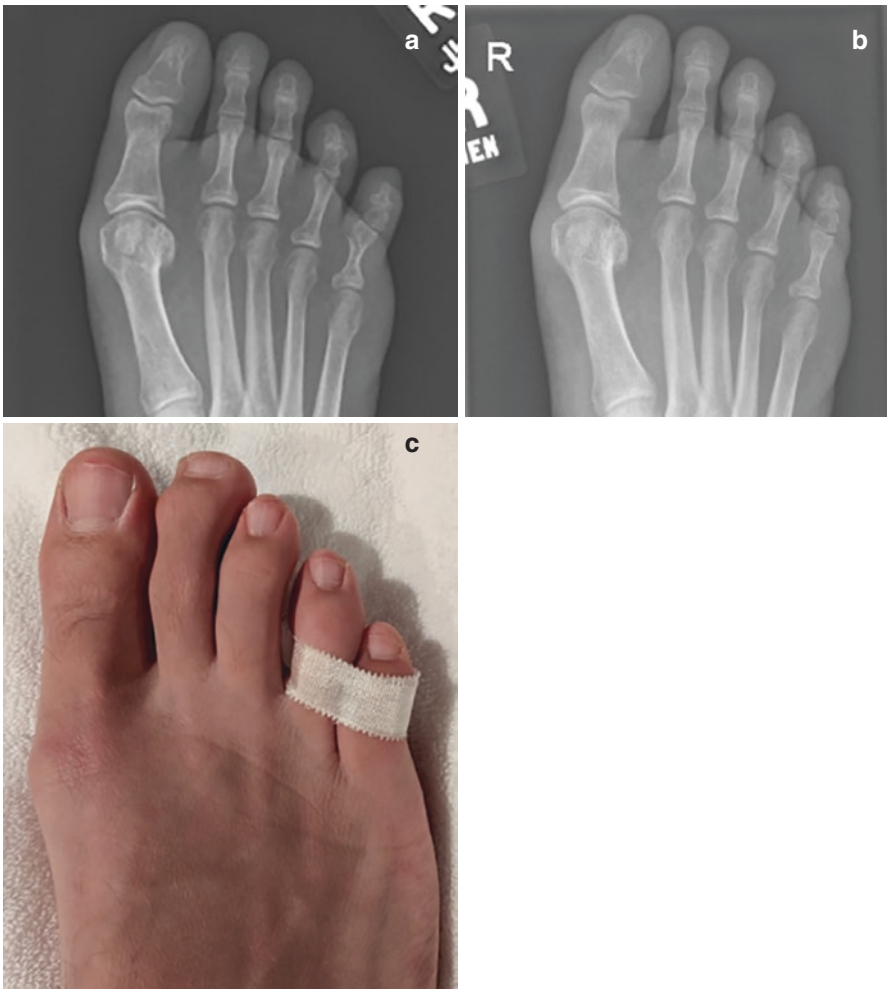

Fig. 33.1 An angulated, displaced 5th toe fracture (**a**) treated with buddy taping to the 4th toe (**b**). Buddy taping should be performed in the opposite direction of the displacement and can be performed with surgical tape (silk tape ½" preferred) or a commercially available Velcro strap (**c**)

When the toe is maligned clinically, buddy taping can be used to correct deformity and support the toe. This is commonly seen after injuring the 5th toe. Occasionally, a reduction maneuver may be needed to realign the toe. This is most commonly needed for a dislocated IP joint or a transverse displaced fracture. To perform the reduction, a digital block is performed and the injury is recreated. Axial traction can be placed manually or with a finger trap and the deformity is exaggerated and reduced. A pencil can be placed between the toes as a fulcrum to facilitate reduction. The reduction can then be supported by buddy taping.

If the MTP or IP joint is dislocated, reduction should be performed as soon as possible. Open fractures should be sent to the emergency department for antibiotics and debridement.

Great toe fractures are often treated nonoperatively; however there is less tolerance for malalignment and displacement than lesser toes. The great toe has stronger potential deforming forces, such as the abductor and adductor hallucis muscles, the plantar plate on the proximal phalanx, and the EHL and FHL muscles on the distal phalanx.

Operative Management

Operative treatment of toe fractures is uncommon. Surgery is indicated if reduction cannot be obtained or maintained with closed methods, or if there is an open fracture that requires irrigation and debridement. As opposed to open fractures of the hand, open fractures of the foot are generally treated with operative irrigation and debridement due to bacteria present in shoewear and the potential for debris. Nail lacerations with underlying fracture are also important to diagnose and treat as open fractures.

Expected Outcome and Predictors of Outcome

Toe fractures typically heal well and rarely go on to nonunion. Discomfort can sometimes take months to fully resolve; swelling can persist as well. Intra-articular fractures can go on to eventual arthritis. Mild malalignment is typically tolerated well. Late surgical intervention can be considered for painful deformity or arthritis.

Turf Toe and Sesamoid Injuries

Summary of Epidemiology

Turf toe and sesamoid injuries occur after a hyperextension of the great toe, commonly seen in athletes. Sesamoid fractures can also occur from blunt direct force to the plantar foot, or more chronically from overuse. The 1st MTP joint is a rather shallow articulation supported by joint capsule and ligaments. The plantar plate is a tough band of tissue that supports the plantar aspect of this articulation, extending loosely from the metatarsal neck to the sesamoids below the metatarsal head to the proximal phalanx base. The sesamoids sit within the distal aspect of the flexor hallucis brevis tendons and articulate with the plantar aspect of the metatarsal head. The collaterals, abductor and adductor hallucis, insert more medially and laterally to offer further support. Turf toe is a spectrum of injuries to the capsuloligamentous structures supporting the 1st MTP joint, ranging from minor sprain of the plantar 1st MTP ligaments to frank dislocation of the great toe and retraction of the sesamoids. Sand toe is a variation where the toe is hyperplantarflexed and the dorsal structures are injured.

Clinical Presentation

Since turf toe is a spectrum of injuries to the plantar great toe, presentation can vary based on the severity. Grade I is a minor sprain with mild pain, grade II a partial tear, and grade III a complete tear with progressively more limitation and associated injuries. The patient typically presents with pain about the 1st MTP, focused plantarly. Swelling and ecchymosis are present acutely in the affected toe. In a less severe injury, symptoms can be more mild. Physical examination of the patient with turf toe will reveal tenderness plantarly at the great toe in the area of the sesamoids and extending distally to the proximal phalanx base. The patient may have pain or apprehension with passive 1st MTP motion, particularly dorsiflexion. Alignment of the great toe should be examined and compared to the contralateral side. Although generally alignment is not affected, subtle malrotation or MTP angulation can be present. Gross malalignment can be secondary to joint dislocation or subluxation, or rarely a traumatic bunion. Traumatic claw toe indicates a severe injury. If new deformity or malalignment is present, radiographs should be performed urgently with referral to orthopedics or the emergency department. Weightbearing films are preferable to evaluate alignment.

Differential Diagnosis and Suggested Diagnostic Testing

Differential diagnosis of turf toe includes sesamoid fracture, MTP dislocation, MTP osteochondral lesion, soft tissue contusion, and metatarsal fracture. It is important to note that these items on the differential may also be associated injuries with turf toe. AP, lateral, and oblique views of the foot are ordered to evaluate for fracture or malalignment. Imaging of the contralateral foot may provide comparison to evaluate for sesamoid malposition or deformity. Sesamoid views can be performed to evaluate for sesamoid fracture or malposition. Weightbearing films are ordered if the patient is able to place weight on the affected foot. Toe x-rays are not typically ordered. A stress view with the MTP in extension may provide further information about joint and sesamoid stability. Advanced imaging, generally MRI, can be utilized to better define the extent of soft tissue injury or to assess for occult fracture or osteochondral injury.

Bipartite or tripartite sesamoid, when the ossification centers of the sesamoid do not fuse during adolescence, can be confused radiographically with a sesamoid fracture. In the setting of plantar 1st MTP pain and a multipartite sesamoid without pre-injury radiographs, MRI and CT scan can help to distinguish acute fracture from sesamoiditis or plantar plate injury. This imaging is often unnecessary acutely as initial treatment is generally identical in a stable joint. MRI can also be helpful in chronic sesamoid pain to distinguish sesamoiditis from more difficult to treat avascular necrosis of the sesamoid.

Nonoperative Management

Most turf toe injuries are treated nonsurgically. In an acute injury, plantarflexion taping (Fig. 33.2) or casting of the great toe is performed in addition to RICE. Gentle range of motion is initiated after a few days as tolerated to minimize stiffness. Depending on symptoms and injury severity, the injury can be treated with a walking boot, stiff-soled shoe or carbon fiber shoe orthosis. Patients are allowed to bear weight as tolerated provided dorsiflexion motion is restricted. Taping is continued as activity increases to prevent recurrent hyperdorsiflexion until stability returns and symptoms resolve.

Sesamoid fractures are treated in a walking boot or cast with RICE and protected weightbearing once pain allows – acute sesamoid fractures are often kept non-weightbearing for 4–6 weeks. An orthotic with sesamoid relief or a metatarsal pad can be used to off-load the injured sesamoid in the boot and then in a sneaker throughout the healing process. A carbon fiber plate can be considered to restrict MTP dorsiflexion. The sesamoids are off-loaded using the same approach in acute or chronic sesamoiditis.

Indications for Surgery

Operative treatment of turf toe and sesamoid injuries is uncommon. Sesamoid fractures can be repaired acutely if markedly displaced and is considered in a high-level athlete. High-grade turf toe injuries with retraction of the sesamoids are generally treated operatively as are traumatic bunions. Delayed reconstruction of these injuries is difficult.

Delayed surgical treatment can be considered for persistent pain after sesamoid injury – typically with partial or complete excision of the sesamoid. This is generally delayed until failing 3–6 months of conservative treatment.

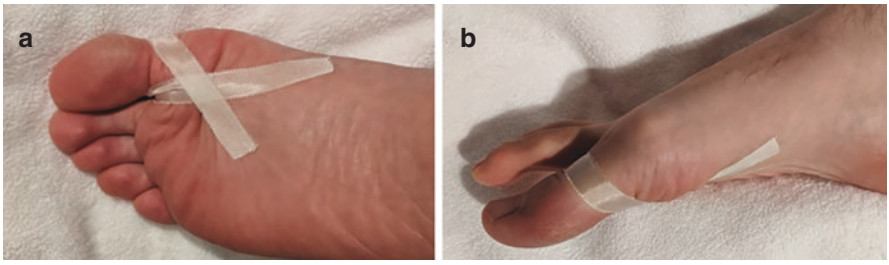

Fig. 33.2 An acute turf toe injury can be treated with plantarflexion taping (**a**, **b**)

Expected Outcome and Predictors of Outcome

There is limited literature on long-term outcomes of these injuries. Low-grade injuries tend to do quite well, and patients can return to activities within a few weeks, although may have residual stiffness. High-grade injuries and sesamoid injuries tend to demonstrate prolonged healing and can be greater than 6 months. Great toe arthritis (hallux rigidus) and development of toe deformities such as bunion and claw toe can occur.

Metatarsal Fractures

Summary of Epidemiology

Metatarsal fractures are common injuries that can occur via numerous mechanisms. Many result from a twisting event. They can be secondary to crush injuries to the foot such as dropping a heavy object on the forefoot, or an axial load such as a motor vehicle collision. They are also seen after repetitive injury or occasionally, generally, in osteopenic bone, as a stress injury with no clear mechanism. The treatment of these injuries depends on the location and nature of the fracture, the displacement and the number of metatarsals involved.

Clinical Presentation

Typically a patient with a metatarsal fracture presents with forefoot and/or midfoot pain and swelling after a crush, twisting, flexion, or repetitive stress injury. Inability to weight bear is common. Bony point tenderness may be present, although in an acute injury, this may be more diffuse. Physical exam should include skin inspection for lacerations or threatened areas, neurovascular exam, and active range of motion. Comparison to the contralateral extremity can be used to evaluate for deformity or malalignment of the foot or digits.

Differential Diagnosis and Suggested Diagnostic Testing

Differential includes soft tissue injury, ligamentous injury such as a Lisfranc injury (see below), and bone contusion. AP, lateral, and oblique foot images, weightbearing if the patient is able, are the next step in diagnosis (Fig. 33.3). In the case of stress fracture, the differential is broad and includes other causes of metatarsalgia such as

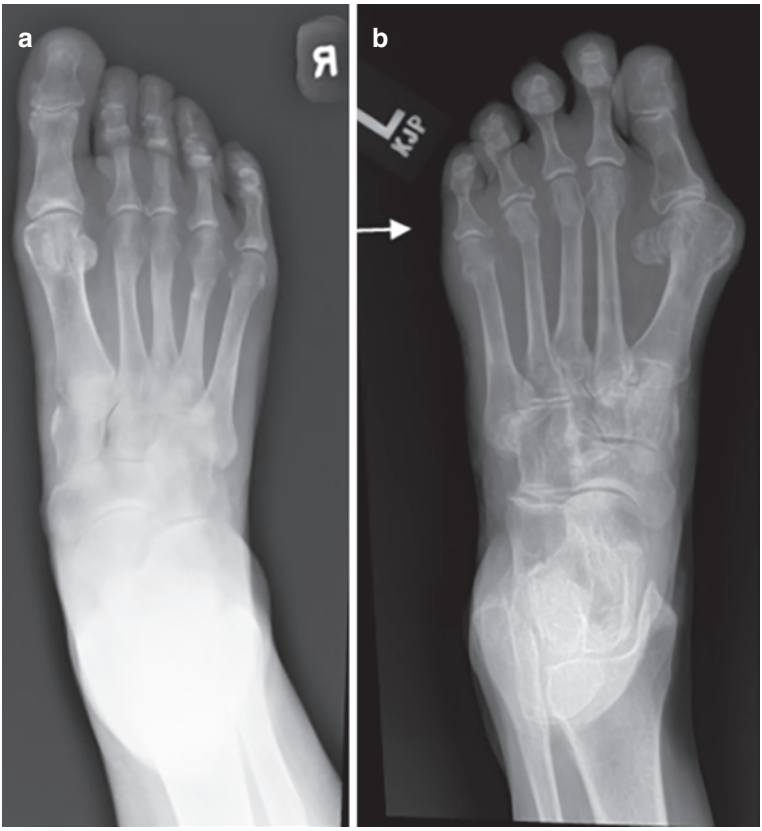

Fig. 33.3 A fourth metatarsal stress fracture (**a**) and multiple (3rd, 4th, 5th) mildly displaced metatarsal fractures (**b**) treated nonoperatively

MTP synovitis, intermetatarsal bursitis, transfer metatarsalgia, neuroma, and stress reaction. Physical exam and location of tenderness can help to differentiate between a metatarsal neck stress fracture and these other sources of pain. X-rays may be negative, particularly in the 1st 3–4 weeks after the development of pain. MRI may be useful if stress fracture is suspected but repeat imaging after 3–4 weeks is negative.

Nonoperative Management

Nonoperative management is appropriate for most isolated metatarsal fractures. Protected weightbearing in a stiff-soled shoe or boot is recommended until clinical and radiographic evidence of healing is noted, typically 6–8 weeks. Metatarsal base fractures and intra-articular injuries will generally benefit from a period of non-weightbearing in either a cast or boot.

Indications for Surgery

Surgical management is considered in markedly displaced fractures, multiple metatarsal fractures, intra-articular fractures, and some 5th metatarsal base fractures. 1st metatarsal fractures are also surgically repaired more often due to the nature of the foot as a tripod and importance of the 1st ray in hallux alignment and weightbearing. If a metatarsal fracture causes a rotational or angular deformity of the toe, surgery may also be discussed. A plantarflexed or dorsiflexed metatarsal fracture may cause late weightbearing pain, metatarsalgia, or toe deformity and may also benefit from fixation. Having multiple metatarsal fractures increases the tendency for displacement and instability, and surgical management is considered for these as well. Open fractures and dislocations are treated as a surgical emergency.

Expected Outcome and Predictors of Outcome

Metatarsal shaft and neck fractures tend to heal well, typically within 8–12 weeks. 5th metatarsal fractures typically take longer to heal due to the blood supply as well as the weightbearing and extrinsic forces along the length of the bone. Fractures that heal with shortening may cause toe deformity and transfer pain to the adjacent metatarsals. Plantar translation and angulation causing prominence of the metatarsal head may cause painful metatarsalgia.

5th Metatarsal Fractures

Fractures of the 5th metatarsal are the most common metatarsal fracture, have different outcomes, and are treated differently than fractures of the other metatarsal bones. Tuberosity fractures, exiting proximal to the articulation between the 4th and 5th metatarsal, generally heal well without surgical intervention. Some authors advocate for fixation if these are displaced, but evidence also supports similar outcomes despite displacement.

Fractures that exit into the 4th–5th metatarsal articulation, also called Jones fractures, have a higher propensity for nonunion and delayed union and are often treated more aggressively (Fig. 33.4). These fractures are treated with a period of NWB or protected weightbearing. Surgical treatment is considered in active individuals as it can potentially speed recovery time.

Fractures of the 5th metatarsal diaphysis are particularly prone to nonunion and are often associated with chronic stress overload of the 5th metatarsal. These injuries are treated with a period of non-weightbearing, and surgery is considered, especially if sclerotic bone consistent with a stress fracture is noted on imaging.

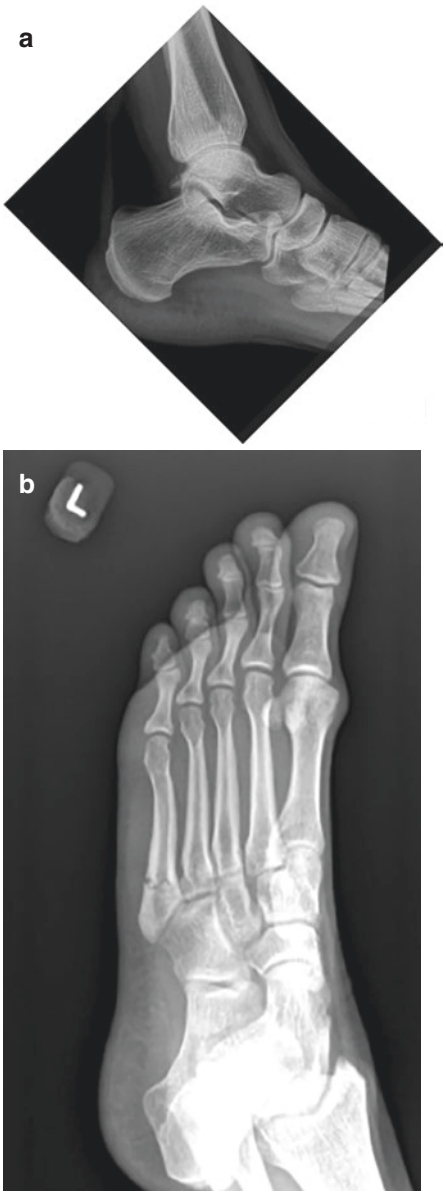

Fig. 33.4 Appearance of a Jones fracture on a lateral ankle x-ray (**a**) and on an oblique foot x-ray (**b**).

Bony injuries to the distal metatarsal, so called dancer's fractures, are oblique bony injuries that tend to heal well without surgery, even if displaced (Fig. 33.5). Surgery is considered for marked shortening, plantar, or dorsal angulation that may cause metatarsalgia or toe deformity, displacement resulting in minimal cortical contact, and rotational or angular deformity of the 5th toe.

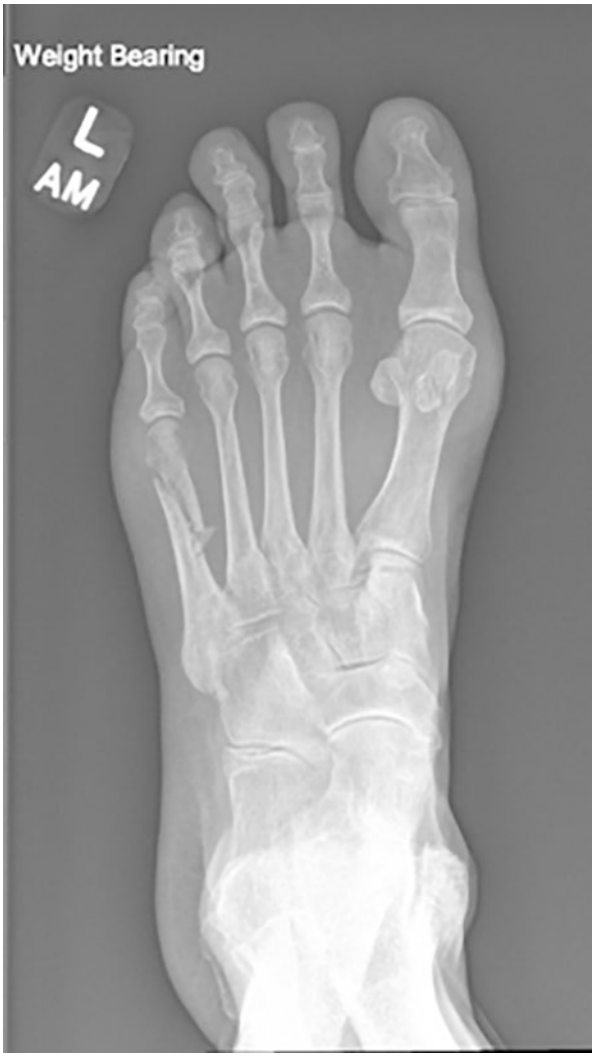

Fig. 33.5 A displaced distal 5th metatarsal Dancer's fracture

Lisfranc Injuries

Summary of Epidemiology

Injuries to the Lisfranc complex include injuries to the metatarsal bases and the cuneiforms and cuboid articulation. The Lisfranc ligament itself is a tough band of three segments of tissue that run between the base of the 2nd metatarsal and the medial cuneiform. There are tough intermetatarsal ligaments that run between the bases of the 2nd–5th metatarsals, but no ligamentous structures connect the bases of the 1st and 2nd metatarsals. The 2nd metatarsal base is described as the keystone of the arch and if injured can cause instability and deformity of the midfoot and arch.

These injuries, while relatively uncommon, can be easily missed and can cause significant pain and deformity due to disruption of the arch integrity. Although often caused by high-energy trauma such as motor vehicle collisions, approximately one-third of injuries are lower energy and more likely to be seen in the primary care setting. The most common mechanism is an axial load on a plantarflexed foot with extended toes.

Clinical Presentation

A patient who has suffered an injury to the Lisfranc complex will present with diffuse swelling to the midfoot and limited ability to bear weight. Plantar ecchymosis often develops within a few days and is regarded as pathognomonic for a Lisfranc injury. The foot should be examined for open areas, deformity, or neurovascular injury.

Differential Diagnosis and Suggested Diagnostic Testing

Differential includes a stable midfoot sprain or ligamentous injury, metatarsal base, or cuneiform fractures. It can include Charcot arthropathy in a neuropathic patient. Initial diagnostic testing consists of AP, lateral, and oblique imaging, weightbearing if able, with bilateral weightbearing AP images on the same cassette if needed for more subtle injuries to evaluate for differences in the midfoot alignment. The exact measurement signifying instability is debated, but a diastasis of 2 mm is thought to be unstable (Fig. 33.6). Any incongruity noted at the TMT joints on a weightbearing lateral x-ray is also indicative of an unstable injury. An avulsion fracture at the base of the 2nd metatarsal, known as a fleck sign, can also signify an unstable Lisfranc injury. CT scan can be helpful in better visualizing midfoot fractures, or in diagnosis of the patient with moderate to severe midfoot swelling following a midfoot injury with negative x-rays. The emerging modality of weightbearing CT can be valuable in detecting subtle instability. MRI can be considered to evaluate the Lisfranc ligament in the absence of fracture, but it does not itself detect instability. A manual stress x-ray can be performed in equivocal cases.

Nonoperative Management

Initial management consists of immobilization in a well-padded splint or tall pneumatic boot with a period of non-weightbearing. A stable Lisfranc injury or bony Lisfranc with anatomic alignment can be treated nonoperatively with a 6–8-week period of non-weightbearing and close clinical and radiographic follow-up.

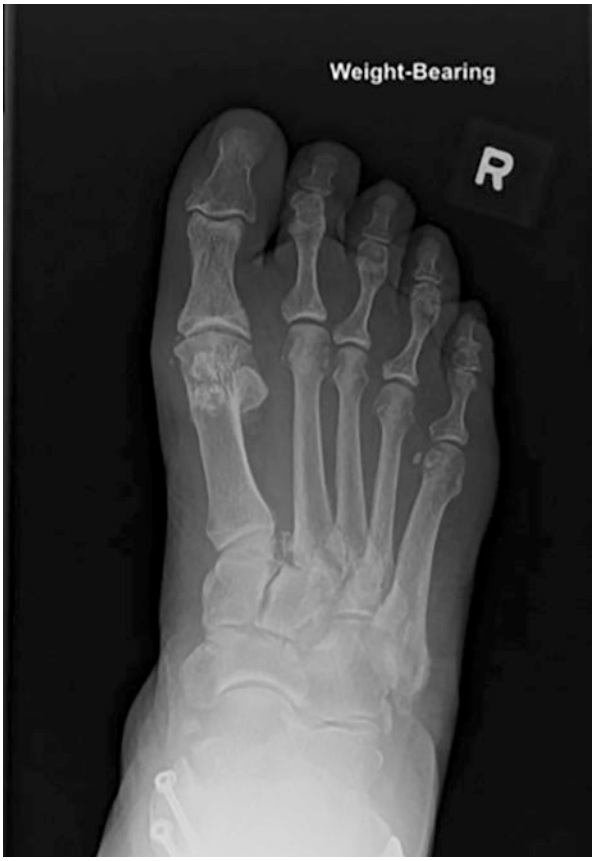

Fig. 33.6 A subtle displaced Lisfranc injury. Note the step-off between the second metatarsal and middle cuneiform, as well as the fleck sign denoting possible Lisfranc injury

Indications for Surgery

Surgical management is indicated in an unstable Lisfranc injury, generally defined as more than 2 mm of diastasis at the 1–2 intermetatarsal space, a side-to-side alignment difference or subluxation of the joints on a weightbearing view. Comminution and malalignment at the joint level also is a consideration for surgery. Surgical management can consist of either anatomic reduction and fixation of the injury or anatomic reduction and fusion of the midfoot. The exact indications for each procedure are currently debated in the foot and ankle literature, but anatomic reduction is paramount to restore the arch.

Expected Outcome and Predictors of Outcome

Recovering from Lisfranc injuries is a difficult process. Outcomes are largely based on the severity of injury as well as the quality of the reduction, although many patients have persistent disability, deformity, progressive arthritis, and pain.

Hindfoot Fractures

Summary of Epidemiology

Fractures of the talus, calcaneus, navicular, and cuboid can lead to long-term pain and functional difficulty even if nondisplaced or repaired in anatomic fashion. Most commonly, these injuries result from a high-energy mechanism, such as a fall from height or motor vehicle collision. Less severe fractures such as avulsion injuries and fractures to the lateral or posterior process of the talus and anterior process of the calcaneus can be caused by a low-energy twisting or angular injury. Talar and calcaneal process fractures are a common source of persistent pain after an ankle sprain.

In the primary care setting, avulsion fractures, talar lateral and posterior process fractures, stress fractures, and anterior process of the calcaneus fractures are more frequently encountered.

Clinical Presentation

Hindfoot fractures tend to cause marked swelling and ecchymosis through the hindfoot extending into the foot and the ankle. Even in low-energy injuries and avulsion fractures, these patients typically have a difficult time bearing weight. Hindfoot stress fractures tend to have a lesser degree of swelling with a more insidious onset. Examination should include assessment of any deformity, tenderness, skin evaluation for areas of skin compromise and neurovascular exam. Active ankle, hindfoot, and digit range of motion is assessed.

Differential Diagnosis and Suggested Diagnostic Testing

Other conditions that present similarly to hindfoot fractures can include ankle fractures and sprains, tendon injuries at the level of the hindfoot, and plantar fascial tear. Non-weightbearing images including three views of both the ankle and the foot should be obtained for a suspected hindfoot fracture. In certain injuries, such as a lateral process of the talus fracture, the injury may be best viewed on the mortise or AP ankle images. If a calcaneus fracture is suspected, a calcaneal axial (Harris) view should be obtained if able. CT scan can better delineate and diagnose hindfoot fractures, and MRI can be utilized to better clarify the extent of soft tissue involvement or to assess for occult fracture should the diagnosis not be certain on radiographs.

Nonoperative Management

Nonsurgical management is considered if a hindfoot fracture is minimally displaced or is an avulsion or stress injury. Nonsurgical management consists of initial immobilization in cast, splint, or tall boot and early active range of motion. Avulsion fractures, talar lateral and posterior process fractures, and injuries to the anterior process of the calcaneus can typically allow the patient to bear weight as tolerated. Patients with stress fractures cab bear weight as soon as pain allows. Articular injuries are typically kept non-weightbearing for 6 weeks or greater depending on radiographic healing.

Urgent orthopedic referral is indicated for fractures displaced more than 2 mm and articular injuries.

In addition to open fractures, emergent referral is needed for talar neck fractures and joint subluxation and dislocation, and to prevent skin compromise and necrosis in calcaneal beak fractures (Fig. 33.7).

Indications for Surgery

Surgical management is indicated for displaced fractures, fractures causing hindfoot malalignment, large displaced lateral process of the talus, and anterior process of the calcaneus fractures and is considered for stress fracture resistant to nonoperative treatment or in athletes. Talar neck fractures are often fixed even if minimally displaced secondary to the tenuous blood supply of the talus.

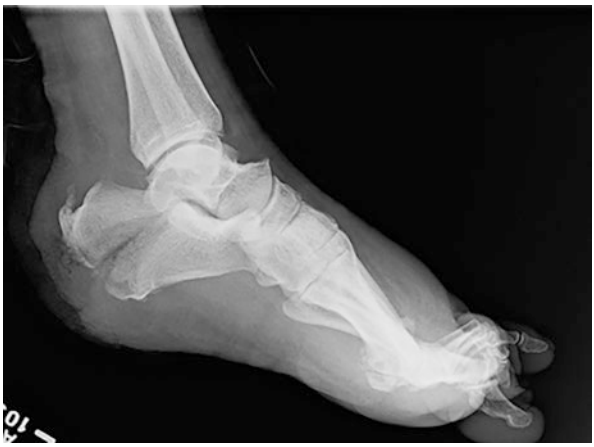

Fig. 33.7 Calcaneal beak fracture. This is a surgical emergency as the skin is compressed by the fracture fragment

Fixation of calcaneus fractures is considered, but is debated even in the case of displaced fractures, as some literature shows similar short-term outcomes in operative and nonoperative treatment.

Expected Outcome and Predictors of Outcome

Patients with hindfoot avulsion fractures typically fare quite well. Displaced talar and calcaneal process fractures may progress to a symptomatic nonunion, in most cases treated with excision of the fracture fragment. More severe articular fractures of the talus, navicular, and calcaneus have more mixed outcomes, with high rates of subtalar, talonavicular, and tibiotalar arthritis following these injuries.

Ankle Fractures

Summary of Epidemiology

The ankle joint is a complex hinge joint consisting of the distal tibia, fibula, and talus surrounded by stabilizing ligaments including the syndesmosis superiorly, the deltoid medially, and the talofibular ligaments laterally. Ankle fractures include fractures to the three malleoli – medial, lateral (distal fibula), and posterior. These injuries are quite common and encompass a spectrum from an avulsion fracture that behaves more like a sprain to a severe unstable fracture-dislocation. Fractures involving the tibial joint surface are known as plafond or pilon fractures and are a separate, more severe injury that is beyond the scope of this chapter. Avulsion fractures in many cases are stable but can point to a more severe injury such as a peroneal tendon dislocation or deltoid ligament injury.

Clinical Presentation

Ankle fractures typically present after a rotational injury with some degree of axial load. The fracture pattern is determined by the position of the foot and direction of the rotation and injury forces as well as the amount of force applied and quality of the bone. Typically, the patient is unable to bear weight or has difficulty doing so. Following an ankle injury, examination should include assessment of any deformity, skin evaluation for areas of skin compromise, palpation for point tenderness, and neurovascular exam. Although patients typically have bony tenderness, more diffuse tenderness may be present in the acute setting. Active ankle and digit range of motion is assessed. Palpation distally in the foot and proximally along the tibial and

fibular length are important to look for associated injuries such as hindfoot fractures, metatarsal fractures, and proximal fibula fractures.

Differential Diagnosis and Suggested Diagnostic Testing

Differential includes hindfoot and distal tibia fractures, osteochondral injury, ankle sprain, periarticular tendon injury, and syndesmotic or deltoid injury. Initial diagnostic testing should evaluate for bony tenderness about the malleoli followed by non-weightbearing AP, mortise, and lateral ankle radiographs. AP and lateral x-rays of the tibia and fibula should be added if concern for more proximal injury, and three-view foot x-rays are regularly performed to evaluate for concomitant foot injury. CT scan is recommended if concern for articular injury, or occasionally for surgical planning. Some authors recommend MRI if there is clinical or radiographic concern for syndesmotic or deltoid ligament disruption.

Nonoperative Management

The management of an ankle fracture depends on fracture displacement but also importantly on the stability of the joint. If multiple malleoli are injured, or there is a combination of a malleolar and ligamentous injury, the congruity of the joint may be compromised. As little as 1 mm shift of the talus in the plafond can change the contact forces in the joint and lead to early arthritic change. If instability is visualized on injury films, or if more than one malleolus is involved, surgical management is considered.

If there is injury to a single malleolus and instability is not seen on the injury images, congruity can be assessed with stress radiographs – either a manual external rotation stress, gravity stress, or weightbearing stress. Translation or angulation of the talus within the ankle mortise with applied stress points to instability.

If less than 2 mm of fracture displacement is noted with a stable ankle joint, nonsurgical treatment is typically recommended (Fig. 33.8a, b). More displacement is accepted in avulsion fractures. If the ankle fracture is stable and nonsurgical management is selected, the patient is placed into a tall pneumatic boot or brace and allowed to bear weight as tolerated.

Nonsurgical management of unstable injuries can be considered as well, particularly in patients with comorbidities, consisting of closed reduction and casting with a period of protected weightbearing.

Malleolar avulsion fractures tend to be stable injuries with a few exceptions. A medial malleolar avulsion can be indicative of an unstable deltoid ligament injury and should be worked up further with stress imaging or MRI. A distal fibular avulsion fracture from the lateral surface of the bone, rather than the typical injury at the distal tip, may denote an injury to the superior peroneal retinaculum and portend peroneal tendon injury or instability.

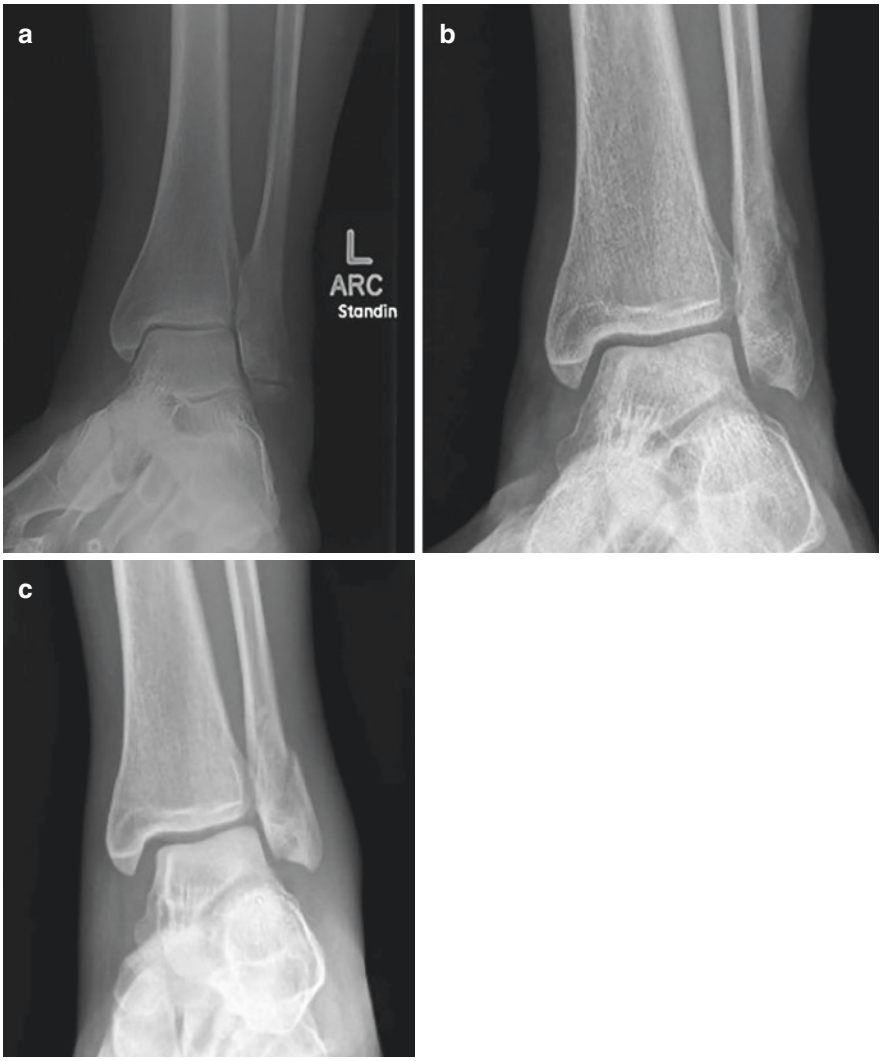

Fig. 33.8 Stable avulsion fracture (**a**), stable distal fibula fracture (**b**), and unstable distal fibula fracture with increased medial clear space noted on stress exam (**c**)

Indications for Surgery

Surgery is considered if more than one malleolus is fractured, if there is ankle joint instability noted on injury or stress radiographs, or if fractures are displaced more than 2 mm (Fig. 33.8c). Open fractures are managed surgically. Surgery typically consists of open reduction and anatomic fixation.

Expected Outcome and Predictors of Outcome

Outcomes after malleolar ankle fractures are typically good, with worse outcomes in several subgroups. Patients with dislocations, syndesmotic injuries, and more severe articular injuries tend toward poorer outcomes.

Charcot Arthropathy

Summary of Epidemiology

Charcot arthropathy is a progressive, destructive process involving the joints in patients with neuropathy. The incidence ranges broadly in the literature from 1% to almost 40% of diabetic patients and is most commonly seen in the foot and ankle. This process is commonly misdiagnosed; a high level of suspicion for Charcot arthropathy is critical in the neuropathic patient.

Charcot is thought to be caused by a combination of factors. Two theories have been developed to explain neuropathic arthropathy: neurovascular and neurotraumatic. The neurovascular theory proposes that autonomic neuropathy causes an increase in blood flow, bone resorption, and proinflammatory cytokines that contribute to destruction. The neurotraumatic theory purports that the patient experiences an injury and continues to traumatize the area due to impaired protective sensation. In reality, both factors contribute.

Charcot progresses over a period of months through three stages at various speeds, often unpredictable but typically slower to consolidate in more proximal locations in the foot and ankle. Stage I is the acute phase, also called fragmentation, in which patients have swelling, warmth, and erythema and develop malalignment and joint fragmentation on radiographs. This phase clinically appears similar to infection. Stage II is the subacute or coalescence phase, where clinically the swelling and warmth subside and radiographically the fragmentation stabilizes and bony healing is noted. Stage III is the chronic or consolidation phase, signified by healing and consolidation of the joints. Charcot is classified according to location, with several classification schemes developed to help determine treatment.

Clinical Presentation

The patient with acute neuropathic arthropathy typically presents with a swollen, erythematous extremity that closely resembles cellulitis or infection. A history of trauma may exist, or more likely may be atraumatic or with unknown trauma. Often Charcot is painless, although this is not always the case. The patient may also note a sensation of instability. The erythema associated with Charcot will often vastly

improve when the extremity is elevated. Patients may have a low-grade fever and elevated inflammatory markers, further complicating the diagnostic process.

Differential Diagnosis and Suggested Diagnostic Testing

Charcot neuroarthropathy is most commonly mistaken for infection. The most important initial diagnostic test is to order an x-ray, weightbearing if possible, in any neuropathic patient who presents with a swollen or erythematous extremity in addition to infectious workup as appropriate. Early on, x-ray findings may be subtle. If clinical suspicion for Charcot arthropathy is high and x-rays are equivocal, the patient can be immobilized with close 1–2-week clinical follow-up and repeat radiographs. MRI is sensitive for detection of early Charcot arthropathy and can be used if x-ray findings are negative. Also on the differential is fracture or ligamentous injury without neuroarthropathy.

Nonoperative Management

Most cases of Charcot arthropathy are initially treated nonsurgically with the goal of maintaining a plantigrade foot. Ideally, once the joints have consolidated, the patient's foot will fit into a shoe without bony prominences prone to ulceration. It is important to immobilize these patients early to prevent collapse and deformity as the joints dissolve and consolidate. Boot immobilization versus total contact casting is debated, as is the need for NWB versus WBAT. The timing from acute Charcot to consolidation is variable and can span months. The duration of immobilization is debated as well, with the consensus being to immobilize until the inflammation has resolved and imaging shows evidence of consolidation. Once consolidated, the patient can ambulate in a diabetic insert or brace depending on the degree of deformity.

Indications for Surgery

The risks of surgery in this patient population need to be carefully weighed against the need for joint stabilization. Surgery is considered in ankle and hindfoot instability, for deformity that precludes shoewear or bracing, if the patient's skin appears threatened, or in cases of ulceration or infection. Timing of surgery for deformity is debated; many authors prefer to allow for consolidation prior to surgery, in the absence of frank instability or ulceration, to decrease the risk of failure of fixation, while others recommend earlier fixation to decrease potential healing time and prevent further deformity. Surgery can include a combination of internal fixation and external fixation as needed.

Expected Outcome and Predictors of Outcome

Outcomes vary widely depending on many factors including timing of diagnosis, degree of deformity, diabetic control, compliance with treatment regimen, level of deformity, precondition functional status, presence of ulceration, and infection. Counseling patients about the prolonged time to consolidation, importance of diabetic control, and potential for deformity following healing is crucial (Table 33.1).

Table 33.1 Common foot and ankle injuries seen in the primary care setting with associated typical presentation, diagnostic testing, and treatment

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Toe fracture	Bony tenderness on the toe after an injury	Foot radiographs, weightbearing if able	Stiff-soled shoe Buddy taping WBAT	Open fracture Unstable or irreducible deformity	Open or closed reduction and pinning
Turf toe	Plantar pain in the 1st MTP after a hyperextension injury	Foot radiographs weightbearing if able Consider contralateral foot x-ray to compare Consider MRI	Immobilization in tall walking boot Plantarflexion taping Carbon footplate	Severe grade III injury Instability Traumatic bunion or claw toe	Plantar plate and capsular repair
Sesamoid injury	Plantar pain in the 1st MTP after an axial load or hyperextension	Foot radiographs, weightbearing if able Sesamoid view Consider MRI	Immobilization in tall walking boot Metatarsal pad or orthotic with sesamoid relief Carbon footplate	Widely displaced fracture Elite athlete Failure of conservative management	ORIF versus partial or complete sesamoid resection
Metatarsal fracture	Swelling and bony tenderness in the midfoot after crush, twisting, axial load, or repetitive stress	Foot radiographs, weightbearing if able Consider CT	Immobilization in tall walking boot	Open fracture Toe deformity Shortening Marked displacement Multiple fractures	ORIF versus closed reduction and percutaneous pinning
Lisfranc injury	Swelling and bony tenderness in the midfoot after crush or axial load Plantar ecchymosis	Foot radiographs, weightbearing if able Consider contralateral foot x-ray to compare Consider CT	Immobilization in cast or tall walking boot if stable injury	Open fracture Unstable injury Joint dislocation or subluxation	ORIF versus primary fusion of the midfoot

(continued)

Table 33.1 (continued)

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Lateral talar process fracture	Lateral pain after twisting injury Swelling and ecchymosis Maximal tenderness slightly distal and anterior to fibula	Foot and ankle x-rays	Tall boot immobilization or ankle brace	Large fragment with more than 2 mm displacement or symptomatic nonunion	ORIF versus fragment excision
Posterior talar process fracture	Acute deep or posterior pain Swelling and ecchymosis Pain with ankle plantarflexion	Foot and ankle x-rays	Tall boot immobilization or ankle brace	Large fragment with more than 2 mm displacement or symptomatic nonunion	ORIF versus open or arthroscopic fragment excision
Anterior calcaneal process fracture	Lateral pain after twisting injury Swelling and ecchymosis Maximal tenderness about a cm distal and a few cm anterior to the distal fibula	Foot and ankle x-rays	Tall boot immobilization or ankle brace	Large fragment with more than 2 mm displacement or symptomatic nonunion	ORIF versus fragment excision
Ankle avulsion fracture	Acute onset ankle pain and bony tenderness after twisting injury Swelling and ecchymosis present	Foot and ankle x-rays Consider tibia and fibula x-rays Manual or weightbearing stress images to determine stability	Tall boot immobilization or ankle brace if stable	Medial malleolar avulsion with instability Avulsion from lateral fibula at peroneal retinaculum	Ankle ORIF or deltoid repair Peroneal retinacular repair
Ankle malleolar fracture	Acute onset ankle pain and bony tenderness after twisting injury Swelling and ecchymosis present	Foot and ankle x-rays Consider tibia and fibula x-rays Manual or weightbearing stress images to determine stability	Tall boot immobilization or ankle brace if stable	Open fracture Ankle joint instability Fracture displacement	ORIF or external fixation
Charcot arthropathy	Swelling and erythema in the neuropathic foot and/or ankle Often atraumatic	Weightbearing foot and ankle x-rays	Total contact cast or pneumatic boot immobilization Diabetic control	Ankle or hindfoot Charcot with instability Deformity with nonplantigrade foot Infection	ORIF, external fixation, exostectomy

Suggested Reading

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Part IX
Cost and Quality in Musculoskeletal Care

Chapter 34
Managing Cost and Quality in Musculoskeletal Care

Cameron R. Egan, Adam E. Roy, and Richard Iorio

Introduction

The musculoskeletal system consists mainly of bone, muscle, tendon, ligament, and cartilage that together support and protect the body while also providing the foundation for movement [1]. Musculoskeletal disorders (MSD) affect one to multiple above mentioned musculoskeletal system components, occur in people of all ages, and result from both acute and chronic processes [2]. These disorders are extremely common and will only increase as our population continues to age.

In the 2015 National Health Interview Survey (NHIS), 124.1 million adults (persons age 18 and over) in the United States reported a MSD. This number is staggering, equating to roughly one in two adults [3]. Moreover, the rate of chronic MSD in adults is greater than the rates of both chronic circulatory and respiratory disorders, and the associated costs are substantial and have far-reaching implications on societal burden. Total MSD-related costs are divided into direct and indirect costs. Direct costs include those incurred in the diagnosis and treatment phases of care, such as hospital services (emergency, inpatient, and outpatient), physician and advanced practice provider outpatient services, prescription costs, and administrative costs. Indirect costs include those incurred through productivity loss as a result of disability or death [3, 4]. Between 2012 and 2014, the average total cost to treat MSD in the United States was $322 billion per year [3].

Through education and research, the quality of musculoskeletal care improves and associated cost decreases. Unfortunately, research funding for MSD is severely

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lacking despite the clear healthcare impact. In terms of NIH funding, between 2012 and 2016, MSD research received $7.9 billion, while heart and circulatory disorders research received $23.1 billion [3]. For the long-term sustainability of our healthcare system, we must advocate for increased MSD research funding. In the short term, education, preventative strategies, and interdisciplinary collaborative efforts are key components to increasing quality while decreasing costs. In this chapter, we will highlight these key components as we discuss common MSD and the recommended diagnoses and treatment pathways. The information provided is intended to help streamline the care of patients suffering from MSD and improve the cost-effectiveness of care delivery.

Prevalence

According to the 2015 NHIS, roughly one in two adults living in the United States reported a MSD [3]. As the national death rate continues to decline, people will live longer, and the prevalence of MSD will only increase. In 2015, in the United States, life expectancy was 79.8 years, and by 2050 estimates project life expectancy to be as high as 85.9 and 93.3 years for men and women, respectively [3].

In 2012, arthritis, chronic joint pain, and low back pain were all listed in the top five of medical conditions reported. These MSD result in direct costs to the healthcare system but also in indirect costs due to the activity limitation and disability that commonly results [3]. Accordingly, of the 1.225 billion medical diagnoses made in 2013, 235.1 million, or 19.2 percent, were made for a MSD [3]. In addition, 10–20 percent of primary care visits occur for evaluation of musculoskeletal complaints [4]. In order to provide quality care and reduce unnecessary costs, it is important for primary care, emergency medicine, and hospitalist providers to be comfortable with appropriate diagnoses and treatment of MSD. In the remaining sections, we will outline appropriate steps in prevention, diagnosis, and treatment of three of the most common MSD: low back pain, osteoarthritis, and osteoporosis and fracture.

Low Back Pain

Back pain presents in many forms that can be differentiated based on location of pain, chronicity of symptoms, and cause. Low back pain is typically defined as back pain in the region distal to the 12th rib and proximal to the inferior gluteal folds [4, 5]. Back pain lasting less than 6 weeks is typically described as acute, between 6 and 12 weeks as subacute, and longer than 12 weeks as chronic [4]. On occasion, a specific pathoanatomic cause for pain is identified, but unfortunately, back pain is nonspecific in 90 percent of patients [4]. Patients with low back pain generally fit into one of three categories: nonspecific low back pain, back pain with radiculopathy, and specific back pain, including patients with associated neurologic deficits [6].

Most people will experience back pain at some point in life. In 2015, 72.3 million adults in the United States reported chronic back pain [3]. In addition, a 2002 survey revealed roughly 2 percent of all physician visits occurred due to low back pain [7]. It is important for frontline providers to identify patients at risk for back pain, to categorize patients appropriately, specifically looking out for red flag presentations and symptoms, and to subsequently initiate the appropriate diagnoses and treatment pathways.

Prevention

As with most disease processes, prevention typically results in reduced costs in comparison to disease treatment. Preventative measures focus on lifestyle modifications, which reduce comorbid conditions that frequently complicate and exacerbate MSD. Low back pain has been associated with excessive weight/obesity and smoking history [8, 9]. Shiri et al. conducted meta-analyses to evaluate the association between obesity and low back pain, and also smoking and low back pain. Analyses revealed that overweight/obesity increased the risk of low back pain while also having the strongest association with seeking care. In addition, analysis revealed a higher incidence and prevalence of low back pain in current and former smokers [8, 9]. Interestingly, in the meta-analysis conducted by Steffens et al., education and exercise were found to be effective in preventing low back pain. As a society, it is important that we focus on preventative measures, such as education, lifestyle modifications, ergonomics, and exercise, as a means to reduce both the incidence and prevalence of MSD [10]. Furthermore, the connection between comorbid disease processes is not always overly apparent to patients, and must be emphasized.

Diagnosis

Patients with low back pain generally fit into one of the three categories: nonspecific low back pain, back pain with radiculopathy, and specific back pain. Specific back pain is rare, identified in roughly 5 percent of patients with low back pain [6]. However, frontline providers must be aware of red flag presentations and/or symptoms that warrant expedited, advanced work-up to rule out conditions such as fracture, tumor, infection, or cauda equina syndrome. Red flag presentations and symptoms include major trauma (or minor trauma in the elderly), night pain associated with unexplained weight loss, unrelenting pain associated with recent fevers or chills, bacterial infection, intravenous drug use, saddle numbness, urinary retention or incontinence, and severe, progressive lower extremity neurologic deficit [11].

In patients with nonspecific low back pain without red flag presentations and/or symptoms, the conservative approach to diagnosis and treatment is preferred [6]. The natural history suggests improvement over time; however, recurrence is

common, with a lifetime recurrence rate of roughly 85 percent, with 5–10 percent of patients developing chronic symptoms [4, 11]. A thorough history and physical exam is essential in order to properly categorize patients and determine appropriate treatment. X-rays are typically obtained as part of the initial work-up; however, there is a lack of evidence to prove that obtaining X-rays improves patient outcomes [6, 12]. Deyo et al. conducted a prospective study to assess the effects of omitting spine X-rays in patients presenting with low back pain. 101 patients were randomized into one of the two groups: initial spine X-rays or education with subsequent spine X-rays with failure to improve. Analysis revealed no serious diagnoses missed and similar symptom resolution and functional improvement between groups. Furthermore, radiology costs were substantially decreased in the education group [12].

Treatment

For patients with nonspecific low back pain, conservative treatment is preferred. Conservative treatment options are vast and include education, activity modification, exercise therapy, manipulation, bracing, medications, and injections [11]. However, little is known in regard to the efficacy of these treatment options. Lin et al. conducted a systematic review assessing the cost-effectiveness of treatments for low back pain. For subacute and chronic low back pain, interdisciplinary rehabilitation, exercise, acupuncture, spinal manipulation, and cognitive behavioral therapy were found to be cost-effective, while massage alone was unlikely to be cost-effective [13].

When treating back pain, the primary objective of the frontline provider must be to determine if the patient has red flag presentations and/or symptoms based on history and physical exam. If so, expedited, advanced work-up must occur, with involvement of the subspecialist if surgical intervention is warranted. If nonspecific back pain, or back pain with radiculopathy, is identified, conservative treatment can be pursued. Patients must first be educated on the natural history of low back pain. Additionally, X-rays are likely not needed initially, as forgoing initial imaging does not lead to serious missed diagnoses or delayed functional improvement.

Osteoarthritis

Osteoarthritis (OA) is the most common joint disorder, primarily affecting the hips, knees, and hands, and is a leading cause of disability in the United States [14–19]. OA is a progressive degenerative joint disease affecting the joint cartilage, synovium, and subchondral bone [15, 20]. Although the primary etiology remains unclear, there is a common endpoint of joint pain and stiffness, leading to functional limitations and disability [19, 20]. A recent systematic review reported the overall

prevalence of OA in hand at 43%, knee at 23.9%, and hip at 10.9% [21]. The prevalence of clinically symptomatic OA, when both joint pain symptoms and radiographic OA are present, increases with age and is predicted to rise from 40 million by 2030 to 78 million by 2040, as the US population continues to age [22, 23]. OA also varies by sex, as females comprise 78% of adults with OA [3, 24]. The high prevalence of OA results in a significant economic burden through both direct and indirect costs.

In 2013, OA was diagnosed in 2.4% of ambulatory visits and 10% of all hospitalizations for any cause [3]. Direct medical costs totaled 65.5 billion dollars annually in 2013, while indirect earning costs have been estimated at 71.3 billion dollars in the same year [3]. The increasing prevalence and concomitant high cost of OA treatment and resulting disability underscore the importance for frontline providers to identify at-risk patients and to have a sound treatment framework that emphasizes prevention and early intervention.

Prevention

The etiology of OA is multifactorial, with non-modifiable risk factors of age and sex being the strongest predictors of disease development. The propensity for older women to have increased incidence of OA after the age of 65 is thought to be related to hormonal changes affecting the volume of cartilage [18, 24]. Previous joint injury is also a strong non-modifiable risk factor, with some studies reporting a fourfold increase in post-traumatic arthritis [25].

The most established modifiable risk factor for onset and progression of OA in the hip and knee is obesity [26–29]. Obesity has been shown to increase the risk for knee OA by threefold [30]. In addition to the increased mechanical load transferred through the joint, there is evidence that an inflammatory process mediated by adipokines may also play a role in OA onset and progression [31]. Furthermore, the risk of OA development has been shown to be proportional to the number of years spent at high BMI, highlighting the importance of disease prevention through weight loss [32].

Diagnosis

OA should be suspected in older patients with pain related to specific joint usage. The pain is typically worse with weight-bearing and can present as stiffness after a period of immobility that resolves within minutes [32]. The radiographic features of osteoarthritis include joint space narrowing, osteophyte formation, and subchondral sclerosis and cysts [15]. It is important to rule out other potentially red flag causes of perceived joint pain, such as septic arthritis, septic bursitis, crystalline arthropathy, inflammatory arthropathy, or bone pathology [33]. Additional diagnostic

imaging with ultrasound, computed tomography, and magnetic resonance imaging are not necessary in the initial work-up of a patient with osteoarthritis; however, these imaging techniques can help rule out red flag causes of joint pain if they are suspected [32].

Treatment

Three treatment modalities exist for osteoarthritis: non-pharmacologic, pharmacologic, and surgical. Regardless of treatment modality, all recommendations should be patient-centered through shared decision-making. Non-pharmacologic treatment recommendations by both the American Academy of Orthopaedic Surgeons (AAOS) and the American College of Rheumatology/Arthritis (ACR) in 2019 strongly recommend exercise for all patients with hand, hip, and knee arthritis [34, 35]. Self-management programs for OA treatment have been found to be the most effective interventions for managing OA over the long term. These programs combine risk factor optimization, wellness, pain coping, and exercise options, which aid in arthritis care [36].

As previously mentioned, obesity is a risk factor for the development and progression of OA. Further studies have shown a dose response for weight loss and functional improvement in OA symptoms with weight loss of 11% improving OA symptoms by 50% [37]. The use of assistive devices, such as a cane for hip and knee OA, tibiofemoral knee braces for knee OA, and hand orthoses for hand OA, is generally cost-effective and allows for improved daily function and limitation of disability [34, 35].

Pharmacologic treatments of OA are used in combination with non-pharmacologic treatments. As a first-line treatment, the AAOS and ACR both recommend the use of oral nonsteroidal anti-inflammatory drugs (NSAIDs) [34, 35]. If the use of oral NSAIDs is not recommended due to concerns over gatrointestinal toxicity or concurrent coritcosteroid or anticogulant use, then the AAOS recommends acetominophen, topical NSAIDs with the addition of a gastroprotective agent, or a COX-2 specific agent [15, 35]. The use of glucocorticoid intra-articular injections is widely used for knee OA. However, a recent Cochrane review outlines the short-term efficacy of such treatment, revealing only slight benefit for 1–6 weeks [38]. The ACR recommends intra-articular glucocorticoids for hip OA and knee OA, while the AAOS only recommends intra-articular glucocorticoids for hip OA [34, 35, 39]. Alternative intra-articular injections with hyaluronic acid have also been studied with a recent meta-analysis of data from only double-blinded placebo-controlled trials showing no clinically important difference [40]. These treatments are often costly to the patients and lack clinical benefit. Finally, opioids are often used by the frontline provider given the chronic pain that accompanies OA [41]. However, the current evidence-based guidelines from the AAOS and ACR do not recommend opioids for treatment of symptomatic OA of the hip, knee, or hand [34, 35]. Lastly, referral to an orthopedic surgeon to consider surgical intervention for end-stage OA

with a total joint arthroplasty should be reserved for patients who have exhausted the abovementioned conservative treatment modalities and who have optimized their health status by improving modifiable risk factors that impact arthritis progression and total joint replacement success (i.e., obesity, diabetes, smoking) [42].

Osteoporosis and Fracture

Osteoporosis is a skeletal disorder characterized by microarchitectural degradation leading to decreased bone strength predisposing to increased fracture risk [43, 44]. The National Osteoporosis Foundation (NOF) estimates that nearly 10 million US adults have osteoporosis and 43 million have low bone density [45]. While 50 percent of women are likely to experience a fracture related to osteoporosis in their lifetime, fragility fractures also occur in 20 percent of men [46]. Osteoporotic fracture in long bones most commonly occurs in the spine, proximal femur, and distal forearm and portends future fragility fractures. In contrast, fracture of fingers, toes, skull, and face is not associated with underlying bone strength [43, 47]. The impact of these fractures on patient quality of life ranges from full recovery to disability and death [47]. Moreover, a single fragility fracture of the hip or spine increases the risk of a future fragility fracture by 2.5-fold and 2-fold, respectively [47, 48]. Hip fracture has been shown to increase all-cause mortality in both sexes, with an almost twofold increase in mortality persisting greater than 8 years after the injury, even when controlling for comorbidities and lifestyle factors [49].

The public health burden of osteoporotic fractures becomes more clear in the context of a recent review of hospitalizations for osteoporotic fractures in postmenopausal women. The review found that these admissions are more common than stroke, myocardial infarction, and breast cancer [50]. Osteoporotic fractures primarily occur in older populations who often rely on Medicare for their insurance coverage. An analysis of the financial burden of osteoporosis in the United States found that Medicare pays for approximately 80 percent of the annual 432,000 hospitalizations, 2.5 million office visits, and 180,000 nursing home admissions [46, 47]. Furthermore, the estimated cost of osteoporotic fracture care is expected to reach $25 billion in 2025 [51]. Given both the medical and economic burden of osteoporotic fractures, it is important for frontline providers to have an understanding of risk factors, diagnostic variables, and treatment strategies for this common disease.

Risk Factors and Diagnosis

Patients are commonly asymptomatic prior to the index fracture event, which makes proper screening a critical step in the diagnosis and prevention of osteoporotic fracture. The NOF recommends that all postmenopausal women and men aged over

50 years should be screened for osteoporosis [47]. Screening at a younger age is recommended for patients with risk factors such as low body weight, early menopause (age less than 45 years old), and family history; comorbidities such as rheumatoid arthritis, inflammatory bowel disease, and chronic obstructive pulmonary disease; as well as long-term use of medications such as glucocorticoids, proton pump inhibitors, and selective serotonin reuptake inhibitors [43, 46, 47]. Patients with previous osteoporotic fragility fractures are also at high risk for future fragility fracture, with some studies reporting 31 percent of patients will have an additional fragility fracture within 5 years [48, 52].

The screening process involves calculating a T-score, which is the standard deviation of one's bone density compared with the average bone density of a 30-year-old healthy adult. The diagnosis of osteoporosis can be made with a T-score of less than or equal to −2.5 based on radiographs of the lumbar spine, femoral neck, hip, or distal radius [53]. The Fracture Risk Assessment Tool (FRAX®) is a 12-question risk calculator combining variables such as age, sex, T-score, and other risk factors to predict an individual patient's risk of osteoporotic fracture within the following 10 years [54]. The FRAX® is important as it helps the frontline provider determine the indicated treatment based on an individual's overall risk.

Treatment

The primary purpose of treating osteoporosis is to avoid fracture through maintenance of bone integrity. Non-pharmacologic treatments include weight-bearing exercise for at least 30–40 min three times per week, in addition to calcium and vitamin D supplementation to maintain serum 25-hydroxyvitamin D greater than 30 ng/mL [43, 53]. The NOF and the American College of Endocrinology (ACE) recommend initiation of pharmacologic therapy for all individuals with osteoporosis based on their T-score or presence of fragility fracture. The NOF and ACE also recommend pharmacologic treatment in patients with a T-score of −1.0 to −2.5 in combination with a risk for hip fracture and major osteoporotic event greater than or equal to 3 and 20 percent, respectively, in the next 10 years based on the FRAX® calculation [43, 53]. The FDA has approved two classes of medications for the treatment of osteoporosis, bisphosphonates, and denosumab, which have been shown to reduce osteoporotic fractures of the spine, hip, and nonvertebral fractures [43, 53]. The dose and frequency of these medications vary based on their route of entry, but all require continued monitoring of potential side effects with regular blood work, and specifically drug holidays for patients being treated with bisphosphonates [55]. While these treatments have been shown to greatly reduce the risk of future fracture, many patients remain untreated in the year after their index fragility fracture, which emphasizes the importance of patient education and shared decision-making [56].

The consequences of osteoporotic fracture on overall patient health in both the short and long term can be devastating. As the population ages, fractures attributed to osteoporosis will increase, as will the economic impact. The frontline provider

should be aware of the risk factors, diagnostic criteria, and treatment options for osteoporosis in order to mitigate the burden of disease on the individual and healthcare system.

Summary

Musculoskeletal disorders (MSD) are the norm, not the exception. The incidence and prevalence of MSD will continue to increase as our population ages, with life expectancy projections reaching as high as 80–90 plus years by 2050. Additionally, risk factors such as obesity and smoking, if not modified, will further increase the number of patients affected.

As a society, the sheer number of people with MSD should be alarming, as the associated direct and indirect costs, in terms of diagnosis, treatment, and resultant disability, could overwhelm the healthcare system. Healthcare providers must take control and start preemptively thinking about MSD in a proactive manner, as has been achieved in other common medical conditions such as heart disease and diabetes.

Prevention of the development of MSD through education and counseling is key, as patients are often unaware of the associations between MSD and comorbid conditions. Additionally, initial conservative management of common MSD is almost always the correct option once red flag diagnoses are ruled out. Conservative treatment pathways have been shown to reduce costs while maintaining quality of care. Information included in this chapter provides the data and clinical principles necessary to prevent, diagnose, and treat common MSD successfully.

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