A Case-Based Approach to Hip Pain

A Pocket Guide to Pathology, Diagnosis and Management

Jasmine H. Harris · Grant Cooper · Joseph E. Herrera · Ana Bracilovic · Arth Patel *Editors*

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To my daughter, Zahra, who was born during this project—I hope to always inspire and make you proud.
Jasmine H. Harris
For Ana, Mila, Lara, Luka, Twinkle, and Lili.
Grant Cooper
For Grant, Mila, Lara and Luka.
Ana Bracilovic
For Apoorva, Diya, Manoj, Alka, and Ben.
Arth Patel

Preface

Hip pain is a common musculoskeletal presentation among patients at all ages and activity levels. Although the hip is stable as the largest weight-bearing joint in the human body, the intra- and extra-articular structures have been at risk for injury and disease ever since humans were able to walk upright on two legs. This unique clinical text is a concise practical guide for physicians and practitioners with varied clinical experiences to understand hip anatomy, accurately diagnose, and provide evidence-based treatment for hip injuries and diseases in the pediatric and adult populations.

Jasmine H. Harris

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Joseph E. Herrera, DO, FAAPMR

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Arth Patel

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Hip Anatomy

1

Adedeji Olusanya
and Aditya Raghunandan

Introduction

The osseous layer (Layer 1) is composed of the femur, acetabulum, and a pair of innominate bones that form the pelvis. This provides joint congruence while allowing four planes of motions: abduction, adduction, flexion, and extension in a normal hip [1].

The femur is the heaviest, strongest, and the longest bone in the human body. The femoral head is encased within the acetabulum cavity and is convex in shape [2]. The femoral head stabilizes the hip joint during gait and allows weight bearing for the upper body [3]. It is the reliable shape of the femur that allows it to play a role in both the movement of the hip and knee joint and serves as the site of origin and insertion for many muscles and ligaments that play a role in the continuum of the lower extremity kinetic

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chain [3]. The femur can be divided into three separate sections: proximal, shaft, and the distal bone. In this chapter, the proximal part of the femur and its role in the hip joint will be the focus of discussion. The femoral head has an anterior, medial, and superior surface that serves as insertion sites for multiple tendons [3]. To accommodate the full range of motion of the hip joint, the femoral head is overlayed with an articular cartilage [3]. Hyaline cartilage covers approximately 60–70% of the femoral head [3]. The rest of the area not covered by hyaline cartilage is called the fovea capitis and is located at the center area of the femoral head [3] which serves as the insertion site of the ligamentum teres [3].

The femoral neck supports the femoral head by playing a vital role in the connection between the femoral head and shaft [4]. The neck shaft angle (NSA), measures to 125 ± 5, in a normal adult [3]. This angle is also termed the angle of inclination, which is the angle measured from the medial side of the femoral shaft to the femoral neck [3]. If the NSA were to exceed 130°, this condition is termed coxa valga [3], if less than 120°, this condition is termed coxa vara [3], and outside the range of 120–130°, it would decrease the range of motion of the hip joint [3]. The NSA is measured at 150° at birth, but because of the loaded stresses at the femoral neck (due to increased weight bearing and walking during adolescents and childhood), the NSA will decline to 125° by adulthood [3].

Is the measured angle located between the mediolateral line through the anterior knee and a line through the femoral shaft and head [3]. Femoral anteversion creates an anterior rotation oriented in the coronal plane and is measured to be 15–20° [3]. Pelvic parameters are necessary to measure in order to characterize the position of the lumbar spine and the effects of the pelvic tilt in the sagittal plane [5]. The pelvic parameters consist of pelvic incidence (PI), pelvic tilt (PT), and the sacral slope (SS). PI is defined as the relative position of the sacral plate in relation to the femoral heads [5]. Patients with a low PI have a small anterior posterior (AP) diameter [5]. This is also termed a vertical pelvis, which is narrow horizontally and strong vertically [5]. A pelvis with a high PI has a large AP diameter and is large in the horizontal plane [5]. The pelvic incidence is the sum of

the sacral slope and the pelvic tilt [5]. The pelvic tilt is the angle between a vertical line of the two sacral endplates and indicates the spatial orientation of the pelvis [5]. The sacral slope is the angle of the sacral plateau to the horizontal plane [5]. For a given pelvic incidence, a specific pelvic tilt will be associated with sacral slope [5].

There are important radiographic parameters a physiatrist should be familiar with to accurately assess and manage various hip morphologies. The alpha angle is the angle measured that connects the femoral head to the distance from the center of the head to the point where the head exceeds the radius of the femoral head [6]. The alpha angle is used to measure the femoral head sphericity and is the measurement used by clinicians to diagnose CAM lesion [6]. The Tonnis angle is the measurement that quantifies the weight-bearing portion of the acetabular roof. It is seen on AP pelvis radiographs and is the line formed between the horizontal line and the tangential line that connects the medial edge to the lateral edge of the sourcil [6]. Clinically, the Tonnis angle has been correlated with subluxation of the hip, femoroacetabular impingement (FAI), and developmental dysplasia of the hip (DDH) [6]. The lateral center edge angle (LCEA) is the X-ray assessment of the lateral acetabular coverage in the frontal plane [6]. This measurement is used to classify hip dysplasia or characterize acetabular under coverage. A LCEA value of 20–25° is considered normal in an adult. Hips with a value of LCEA of less than 15° (in children) and less than 20° (in adults) are considered to have developmental hip dysplasia. If the LCEA shows over coverage of the femur head on radiographic imaging, FAI can be diagnosed [6]. The anterior center edge angle (ACEA) is the angle formed by the line passing through the center of the femoral head to the most anterior part of the acetabular sourcil [6]. An ACEA as low as 20° can indicate hip pathology such as acetabular dysplasia or FAI [7]. Low ACEA is correlated with anterolateral acetabular deficiency [6].

The greater and less trochanter, which projects superolateral and posteromedial, respectively, sit at the base of the femoral neck, where the periarticular muscles are inserted [4]. The trochanters' bony prominences significantly impact the muscle

groups that are attached to them [3]. Three main groups supply the arterial blood to the proximal femur which are the extracapsular arterial ring, ascending cervical branches of the extracapsular arterial ring on the surface of the femoral neck, and the arteries that supply the round ligament [3]. Injury to the retinacular arteries can be seen in any fracture of the femoral neck [3].

The acetabulum is a cup-shaped cavity, created by the innominate bones (ilium, ischium, and the pubis bones) [3]. Before the age of 14, these bones are skeletally immature and are divided by the triradiate cartilage [3]. Between the ages 14 and 16, these bones start to fuse together and are completed by the age of 23 [3]. The acetabulum is a Y-shaped triradiate cartilage that covers 70% of the femoral head [8]. The acetabulum is 40% made from the ilium, 40% from the ischium, and 20% from the pubic bone [3]. Acetabulum is a cavity located outside the pelvis where the ball shaped femoral head sits in and is encompassed by the acetabular edge which is a circular bone flap [4]. The lunate surface is the most peripheral portion of the acetabulum, and the acetabular surface is the most central portion [4]. The lunate contains a central inferior acetabular fossa that engulfs fat tissue, and the origin of the ligamentum teres [3]. These two surfaces are used during the articulation of the femoral head and acetabulum. The acetabular pit contains the round ligament which is surrounded by vascular anatomy and adipose tissue [4].

Within the osseous layer, structural pathologies may exist which are classified as dynamic or developmental [9]. There are three groups of dysfunctions, which are identified as static overload, dynamic impingement, or dynamic instability [1]. Dynamic pathologies related to the hip joint include trochanteric impingement, sub-spine impingement, delamination, and femoroacetabular impingement [9]. Pathologies that fall within the developmental category include femoral inclination, acetabular protrusion/profunda, dysplasia, acetabular version, femoral version [9]. Dynamic pathology also includes loss of the femoral head sphericity due to increase in age leading to impingements, labral tears or delamination [9].

The inert layer (Layer 2) is composed of the labrum, capsule, ligamentous complex, ligamentum teres, capsule-ligamentous

structures, and the synovial joint. The purpose of the inert layer is to distribute stability to the hip joint [9].

The labrum has many functions of the hip which includes shock absorption, joint lubrication, and pressure distribution [8]. It forms a fibrocartilaginous extension of the acetabulum which increases the containment of the femoral head [10]. The fibrocartilage characteristic of the labrum creates a negative intra-articular pressure within the hip joint [11]. The labrum works to hinder the fluid flow in and out of the hip joint, creating a "seal effect" that creates distraction of the femoral head from the acetabular pocket [10]. The seal limits lateral motion of the femoral head creating joint stability and preserves the quality of the hip joint [12]. Studies suggest that the labrum helps to center the femoral head in the cavity of the acetabulum, maintaining the thickness of the fluid layer between the bony surfaces [10]. The seal effect also functions to increase joint stability, and distribute compressive loads to the articular surfaces, which in return decreases the stress on the hip during weight-bearing action [10]. Ferguson et al. studies suggest that the labrum maintains joint stability in the most extreme range of motion, especially in flexion, due to the inconsistent nature of the femoroacetabular joint [10]. The labrum circumferentially encircles the acetabulum's perimeter from the base of the fovea capitis to the transverse acetabular ligament anteriorly and posteriorly [2]. During loading in the gait cycle, the labrum acts as a tension band that limits the expansion during the motion between the anterior and posterior columns [13]. If this tension is lost, due to labral tears, the joint will be more susceptible to increased load forces and repetitive trauma [2]. Increasing the concavity of the acetabular fossa, as well as increasing the contact area with the femoral head; the labrum plays a significant role in the stability of the hip joint [4]. In the absence of stability, the hip joint is vulnerable to many pathologies. The surface of the labrum has limited vascularity and minimum synovial coverage, making the labrum more susceptible to tears and low healing probability [2]. Blood vessels that supply the labrum are found in the outmost layer in the capsular surface, thus explaining why the labrum remains avascular [2]. Labral tears clinically manifest as hip instability more than pain, due to

free nerve endings located in the superficial layer of the labrum tissue [2]. The labrum is innervated by the branches of the nerve to the quadratus femoris and the obturator nerve [3]. The anterior labrum is more prone to tears due to the anterior section of the labrum being thinner while the posterior portion is thicker [2]. Labral tears can arise due to repetitive strain to the anterior section during abduction or external rotation in flexion or extension [2].

The capsule is a strong fibrous tissue that enforces hip stability established by the labrum [3]. The capsule fibers that encircle the femoral neck (zona orbicularis) help counteract the stresses placed on the acetabular labrum [3]. The capsule, along with the labrum, plays a significant role in bearing weight load in the flexion range of motion, thus explaining instability of the hip joint if capsular laxity is present [13]. The capsule is part of capsular ligamentous complex system that is made of the deep and superficial layers of the longitudinal ligaments [2]. The function of the ligamentous complex is to reinforce stability to the hip joint [2]. Repetitive tears of the capsule tissue can create a rotational instability of the hip joint, which is also associated with deficiency of labral tissue [2]. The capsule is the primary location that receives multiple innervations at the hip joint [3]. The posterior and inferior regions of the capsule are innervated by the posterior articular nerve, which is a branch of the nerve to the quadratus femoris [3]. The superior gluteal nerve innervates the superior regions of the capsule, while the branch of the femoral nerve innervates the anterior region of the capsule [3]. The anteroinferior and anteromedial regions of the capsule are innervated by the medial articular nerve which is a branch of the obturator nerve [3].

The function of the ligamentum teres remains to be unclear. Due to the existence of arteries around the ligamentum, this suggests that it plays a role in the blood supply to a developing hip [14]. The ligamentum teres tightens in hip flexion, adduction and external rotation [2]. It has been hypothesized that the ligamentum teres aids in hip stabilization and can resist dislocation forces placed on the femoral head [15]. The innervation of the ligamentum teres is by the posterior branch of the obturator nerve [3].

Ligaments involved in the attachment of the pelvis and femur to reinforce the capsule of the hips are the iliofemoral, pubofemoral, and ischiofemoral ligaments [3]. In the motion of hip extension, these ligaments are noted to be tight [3]. They are most slack in the motion of flexion, abduction, and external rotation [3]. Of the three ligaments, the iliofemoral ligament is the strongest, as it extends from the anterior inferior iliac spine to the anterior intertrochanteric line in an inverted Y shape [3]. The primary function of the iliofemoral ligament, also known as the ligament of Bigelow, is to help prevent hyperextension of the hip joint [3]. The pubofemoral ligament attaches to the superior pubic ramus proximally and the inferior femoral neck distally [3]. The key role of the pubofemoral ligament is to prevent hyperabduction of the hip. The ischiofemoral ligament, the thinnest extracapsular ligament, will attach the ischial rim of the acetabulum to the femoral neck crossing the posterior aspect of the hip joint [3]. The primary function of this ligament is to stabilize the joint in the extension motion. The innervation to the ischiofemoral ligament is the posterior articular nerve [3].

The synovial fluid in the hip joint is also part of the inert layer. The synovial joint lines the inner surface of the joint capsule and will form a sheath that covers the joint of the round ligament [4].

The osseous layer (Layer 1) and the inert layer (Layer 2) have a consequential relationship where if there is damage to either layer, it will affect the other [1]. For example, mechanical stress that influences the hip joint due to underlying abnormalities in the osseous layer lead to certain pathologies of the inert layer [1]. These pathologies can include labral tears and ligamentous tears, capsular irritation, hip instability or adhesive capsulitis [1].

Layer III, as known as the contractile layer, consists of all the muscular tissue that initiates, controls, and supports specific movement about the hip joint [9]. The contractile layer includes trunk stabilizers and the pelvic floor musculature. The main purpose of this layer is to provide dynamic stability to the hip, pelvis, and trunk [9]. Abnormal mechanics between the osseous (Layer 1) and inert layer (Layer 2) may lead to instability and increased tension to the lower spine, pubic symphysis, and SI joint. This tension therefore creates secondary stress to the muscles attached

to the same pelvic structures. When examining the muscles and tendons of the hip joint, it is great practice to examine the muscles by anatomical position: the anterior, lateral, medial, and posterior muscle groups.

The Iliopsoas group consists of the iliacus, psoas major, and minor muscle. These muscles originate from the lateral surface of the spine between the 12th thoracic vertebra and the upper region of the fifth lumbar vertebrae, the iliac crest, and the sacrum [3]. The muscles then insert on to the lesser trochanter of the femur. As the muscle begins to enter the thigh region and passes under the inguinal ligament, it becomes thinner leaving the muscle more susceptible to injury [4]. The psoas muscle joins the inguinal ligament to form the iliopsoas muscle [4]. Innervated by the femoral nerve, the iliopsoas muscle is the major hip flexor of the hip joint [3]. The medial femoral circumflex and iliolumbar arteries act as the main blood supply of the muscle [3]. The iliacus muscle originates from the upper portion of the iliac fossa and the anterior inferior iliac spine (AIIS) [3]. The rectus femoris muscle originates from the AIIS and the iliac crest while inserting on the superior patella [3]. This muscle also plays a significant role in flexion of the hip joint and is innervated by the femoral nerve, specifically the L2–L4 nerve root [3]. The blood supply of the rectus femoris muscle is the lateral femoral circumflex artery of the descending branch [3].

Structural pathology (Layer 1) of the hip joint is related to muscular tissue pathology (Layer 3). Mechanism of such pathology can be acute, traumatic, developmental avulsions, or overuse tendinosis [1]. Hip internal snapping of the psoas muscle can be seen when the muscle is displaced laterally with flexion and medially with hip extension [9]. When making a diagnosis of the anterior muscles of the hip, certain enthesopathy pathologies should be considered when making a differential diagnosis. Hip flexor strains, psoas impingement, and sub-spine impingement syndrome can be seen on physical examination of patients with hip flexor pathology [1]. Psoas tendonitis and rectus femoris avulsion pathologies may be associated with patients complaining of groin pain [16]. When the psoas and iliacus muscle merge to become a common tendon, it is called iliopsoas musculotendinous junction

[17]. It is at this junction that the iliopsoas tendon is susceptible to pathologic conditions such as iliopsoas bursitis, distal iliopsoas strain, partial or complete tears, and snapping of the iliopsoas tendon [17]. Snapping iliopsoas syndrome, defined as pain or discomfort occurring in the groin due to the snapping motion of the psoas tendon [17], was thought to be caused by the impingement of the psoas tendon against the iliopubic bone [17]. Recent studies under sonography have shown that it is due to the distal iliopsoas tendon that causes iliopsoas snapping syndrome, as the iliopubic eminence remained medial to the psoas tendon during dynamic and static evaluations [17]. It is during the motion of flexion, abduction, and external rotation that the psoas major tendon glides around the medial part of the iliacus muscle, which then can become entrapped between the tendon and the superior pubic ramus, therefore causing the snapping sensation [17]. Internal snapping of the psoas muscle could occur over either the femoral head or the iliopectineal eminence [9]. During the physical exam, the psoas muscle will be noted to be displaced laterally with flexion and medially with hip extension [9]. Pain will be localized over the anterior part of the groin when diagnosing internal snapping of the hip [18].

The gluteus maximus, medius, minimus, and the tensor fascia lata are considered to be the lateral muscles of the hip joint and the main function is abduction of the hip joint. The gluteus maximus originates from the posterior part of the ilium, dorsal sacral surface, and coccyx [3]. The muscle then inserts onto the posterior iliotibial tract and the gluteal tuberosity [3]. The gluteus maximus, innervated by the inferior gluteal nerve (L5, S1, S2), is considered the main hip extensor, but also plays a role in external rotation, abduction, and adduction [3]. The muscle receives its blood supply from both the superior and inferior gluteal artery [3]. The gluteus medius originates from the anterior gluteal line and inserts onto the lateral surface of the greater trochanter [3]. The gluteus minimus also inserts onto the lateral surface of the greater trochanter but originates from the outer cortex of the ilium [3]. Both muscles are involved in internal rotation and abduction of the hip joint. The muscles are also innervated by the superior gluteal nerve and receive their blood supply from the superior gluteal

artery [3]. The tensor fascia lata takes its origin from the anterior superior iliac spine and is inserted onto the iliotibial tract [3]. This muscle plays a role in hip abduction and internal rotation, but also part takes in hip flexion and external rotation and receives its blood supply from the ascending branch of the lateral circumflex femoral artery [3]. The piriformis muscle also originates from the inner surface of the sacrum and inserts onto the greater trochanter [3]. The piriformis muscle will aid in hip extension and external rotation. The muscle receives its blood supply from a branch of the inferior gluteal artery [3].

As a physiatrist, it is important to recognize lateral enthesopathies when examining the lateral muscle groups of the hip. These pathological conditions may involve the peri-trochanteric space and injuries to the gluteus minimus and medius tendons [1]. Tears of the gluteus medius and minimus muscle can clinically result in a positive Trendelenburg sign and weakness when patients are ascending stairs [9]. Tears of the muscle were also found in approximately 20% of hip fractures [9]. The tensor fascia lata can show decreased activation during hip flexion in patients diagnosed with femoroacetabular impingement syndrome (FAI) [1]. One must also consider deep gluteal syndrome, which is a syndrome that involves posterior soft tissue injury and irritation or compression of the sciatic nerve [1]. This can cause a constellation of pain patterns that involve the hamstrings but also the gluteal muscles as well [1]. Tears of the hip abductors may present as a musculoskeletal condition called the greater trochanter pain syndrome (GTPS), in which the gluteus medius and minimus become irritated at their insertion (greater trochanter) [19]. Patients may complain of lateral sided hip pain that is dull and aching and is exacerbated by weight bearing and resisted hip abduction motion [19]. Gluteus medius tears are seen more than gluteus minimus tears in GTPS due to the medius muscle having three anatomical attachments (central, posterior, and anterior) to the greater trochanter compared to the gluteus minimus which only has two insertions. Therefore, the gluteus medius is more prone to tears and degeneration during the gait cycle [19]. Hip snapping, specifically external type, can be caused by snapping of the posterior border of the iliotibial band or the anterior border of

the gluteus maximus over the greater trochanter when the hip is seen in flexion from an extended position [18].

Besides the gluteus maximus, the extensor muscles of the hip joint are the semitendinosus, semimembranosus, and bicep femoris muscles [3]. All three muscles originate from the ischial tuberosity, while having different insertion sites [3]. The semitendinosus inserts onto the anteromedial tibial plateau, while the semimembranosus inserts onto the posterolateral tibial plateau [3]. The bicep femoris inserts onto the fibular head of the fibula and the posterolateral tibial plateau as well [3]. All three muscles are innervated by the tibial branch of the sciatic nerve (L5, S1, and S2) [3]. The inferior gluteal and the perforating arteries are the blood supply to the semitendinosus muscle. The blood supply of the semimembranosus muscle is the profunda femoris and gluteal arteries [3]. Bicep femoris (long and short head) are supplied by the inferior gluteal, popliteal arteries, and the perforating branches from the inferior gluteal and profunda femoris arteries [3].

The semitendinosus, semimembranosus, and bicep femoris muscles are considered the posterior muscle group of the hip joint. Pathologies that can be seen are posterior enthesopathy, and proximal hamstring strains [9]. Proximal hamstring tendonitis is a differential diagnosis with patients that present with groin pain [16].

The external rotators of the hip include the piriformis, obturator internus and externus, quadratus femoris, superior and inferior gemellus muscles. These muscles run in the horizontal plan of the hip below the piriformis muscle [3]. All muscles play a role in hip adduction and external rotation; these muscles are also innervated by the sacral plexus branches L5–S1 [3]. The obturator internus muscle originates from the inner surface of the obturator membrane and inserts onto the medial greater trochanter, receiving innervation from the nerve to the obturator internus (L5–S1) [3]. The blood supply of the obturator internus muscle is the superior gluteal artery [3]. The obturator externus muscle originates from the outer surface of the obturator membrane (including the pubic ramus and the ischium bone) and inserts onto the trochanteric fossa. The innervation to the obturator externus is the obturator nerve (L3, L4), while receiving blood from the anterior branch of

the obturator artery and the medial circumflex femoral artery [3]. The superior and inferior gemellus muscle originate from the ischial spine and the ischial tuberosity, respectively [3]. Both muscles insert onto the posterior greater trochanter and are supplied by the branches of the inferior gluteal artery [3]. The superior gemellus is innervated by the nerve to the obturator internus (L5, S1), while the inferior gemellus is innervated by the nerve to quadratus femoris [3]. The piriformis muscle originates from both the anterior surface of the sacrum and sacrotuberous ligament, while inserting into the posterosuperior trochanter [3]. The ventral rami of S1 and S2 acts as the nerve supply to the piriformis muscle [3]. The last muscle of the external rotator group is the quadratus femoris muscle. This muscle originates from the lateral border of the ischial tuberosity while inserting into the quadratus tubercle [3]. The innervation to the quadratus femoris muscle is the nerve to the quadratus femoris [3].

The medial muscles of the hip include the adductor longus and brevis, adductor magnus, adductor minimus, gracilis, and the pectineus muscles [4]. All the medial muscles of the hip function to adduct the hip joint [3]. The adductor longus originates from the superior pubic ramus and inserts into the middle third of the linea aspera [3]. Not only does this muscle help with hip adduction but can also flex the hip up to 70° [3]. The adductor brevis originates from the inferior ramus and body of the pubis and inserts onto the proximal linea aspera and the pectineal line [3]. The adductor magnus muscle originates from the inferior pubic ramus, ischial ramus, and the ischial tuberosity. While inserting into the gluteal tuberosity, and the adductor tubercle of the medial femur [3]. This muscle not only functions in hip adduction but also contributes to hip extension and external rotation [3]. The adductor minimus originate from the inferior pubic ramus and insert into the medial portion of the linea aspera. This muscle will contribute with hip extension along with adduction [3]. The gracilis muscle originates from the inferior pubic ramus and inserts onto the distal knee [3]. The obturator nerve (L2–L4) is the innervation to the adductors of the hip [3]. The adductor magnus also receives innervation from

the sciatic nerve, while the pectineus muscle receives innervation from the femoral nerve [3].

Medial enthesopathies are the pathological patterns seen with medial muscles of the hip. These can include adductor rectus tendinopathies which are also called athletic pubalgia or sports hernias [1]. This can be caused due to overactivity of the adductor muscles. For example, the adductor muscles are seen to be overactive especially when the abductor muscles are weak [9]. This forces the adductor longus to act as a hip stabilizer in the frontal plane, causing repetitive motion and overactivity, leading to a tendinosis of the adductor muscles [9]. The adductor muscles work synergistically with the abdominal muscles to stabilize the pelvis during motion [16]. When there is injury to the adductors, patients will most commonly complain of groin pain and would not be able to take part in athletic activity [16]. During the physical exam, if there is tenderness to palpation at the adductor enthesis, with pain on passive adductor stretching and resisted adduction, adductor pathologic injury should be highly considered [16].

Lumbosacral muscles can be further distinguished by their function related to the hip. The rectus abdominus, external and internal abdominal oblique serve as the core muscles. The transversus abdominus and the multifidus muscle function as the stabilizers/deep core muscles. The erectors (ILS) include the interspinales, iliocostalis lumborum, and longissimus lumborum muscles.

Due to the attachments of the lumbosacral muscles to the pelvis, this can often affect the abdominal wall architecture correlating to the prevalence of sports hernias, and femoroacetabular impingement syndrome (FAI) seen in clinic [9]. When examining layer III, the physiatrist must consider postural adaptive changes that can lead to the Lower Crossed Syndrome. The Lower Crossed Syndrome is defined as an imbalance of muscle strength between the abdominal and gluteal muscles, iliopsoas, and thoracolumbar extensor muscles thus creating an anterior tilt of the pelvis [9]. Rectus abdominis tendinopathies are often seen with overuse of the rectus abdominal tendon and can lead to athletic pubalgia aka sports hernia. Sports hernias can be illustrated as occult hernias

that are caused by a tear or weakness of the posterior inguinal wall without a visible hernia on physical exam [16]. Other etiologies of sports hernias can be due to reduced force of conjoined tendons, injury at the insertion of the rectus abdominis muscle, tearing of the internal oblique muscle at the pubic tubercle, tearing within the internal oblique musculature, or abnormality seen in the external oblique aponeurosis [16]. Gilmore's groin pathology is seen when there is a tear seen in the external oblique aponeurosis [16]. Often athletic pubalgia can be co-diagnosed with FAI during a physical exam. This can be explained due to restriction in flexion and internal rotation at the hip joint resulting in abnormal motion in the pelvis [16]. The abnormal motion is responsible for the injury to the ligaments and musculature associated with sports hernia and FAI [16]. When athletes have weak abdominal muscles but relatively strong adductor muscles, this can result in trunk hyperextension and thigh hyperabduction therefore increasing the risk of sports hernias [16]. On physical exam, pain can be reproducible with resisted hip adduction or Valsalva maneuver [16].

The neuromechanical layer, also known as layer IV, is the theoretical layer that is composed of physiological processes, anatomical formation, and kinematic changes that occur throughout the pelvic area that drive proprioception and pain generators through the hip joint [9]. The neuro-kinetic layer serves as the neuromuscular link between the spine and the lower extremity as it functions within its environment [1]. Layer IV can be viewed either locally or globally. Locally, this layer contains the neuro-vascular structures, mechanoreceptors, and nociceptors at the site of the hip [9]. From the global view, layer IV refers to the posture and position of the pelvis over the femur bone [9]. In this section, we will discuss the neuro-vascular anatomy of the hip, regional mechanical receptors, the thoracolumbar mechanic and its relation to the hip, and the lower extremity mechanics and the compensation response from the hip. This section will also aim to illustrate how the neuromechanical layer entails examining the functional movement patterns and how it effects the dynamic movement of the pelvis over the femur, or femur under the pelvis [9]. The compensatory injuries that are seen within this layer are pain syndromes, nerve compressions, neuromuscular dysfunction, and spinal referral patterns [1].

The hip joint is innervated by multiple nerves specifically pertaining to the capsule of the hip. The inferior region of the hip and the ischiofemoral ligament is innervated by the posterior auricular nerve which is a branch of the nerve to the quadratus femoris [3]. The hip capsule is also innervated by the superior gluteal nerve which supplies the superior aspect of the capsule [3]. The branch of the femoral nerve supplies the innervation to the anterior capsule [3]. Peripheral nerve disorders are commonly seen in hip pathology. This includes femoral neuropathy, lateral femoral cutaneous neuropathy (Meralgia taresthetica), sciatic neuropathy (aka piriformis syndrome), obturator neuropathy, pudendal neuropathy, ilioinguinal, ilio-hypogastric, genitofemoral, superior and inferior gluteal neuropathies [1].

The hip receives its blood supply from several different arteries [3]. The medial and lateral circumflex femoral arteries are the main blood supply to the hip joint [9]. These arteries originate from the profunda femoral artery, which is the deep artery of the thigh, but there are variations where the arteries arise from the femoral artery [9]. The femoral head is also supplied by a small branch of the posterior division of the obturator artery which passes through the ligamentum teres [9]. The gluteal arteries and the femoral or the deep artery of the thigh are the main components of the gluteal and trochanteric anastomosis of the hip [9]. The acetabulum receives its blood supply from the obturator, superior, and inferior gluteal arteries [3]. The foveal artery, which is a branch of the posterior division of the obturator artery, runs along the ligamentum teres to supply the fovea centralis of the femur bone [3]. The proximal end of the femur bone is supplied by the extracapsular arterial ring (located at the femoral neck composed of the medial and lateral femoral circumflex, and the superior and inferior gluteal arteries), the ascending cervical branches of the arterial ring, and the arteries of the round ligament [3]. Overall, the labrum is not well supplied with blood, reasons why there is a slow healing process after the labrum is torn [9]. Even though it has scarce blood supply, the labrum does receive blood from a combination of arteries which include the inferior and superior gluteal arteries, and the obturator artery [9].

Nerve endings can be further categorized by mechanoreceptors and free nerve endings. The four types of mechanoreceptors are Ruffini endings, Pacinian corpuscles, and Golgi tendon organs [9]. Free nerve endings are also known as intra-articular nociceptive receptors [9]. Joint mechanoreceptors can be further subcategorized in type I, type II, type III, and type IV receptors. Type I receptors are found in tendons, ligaments, periosteum, and the joint capsule [9]. These receptors are responsible for proprioception and are also known as the Ruffini endings [9]. Type II receptors are located in the fat pads and the deep joint capsule [9] These receptors are responsible for kinesthesia and are also called Pacinian corpuscles [9]. Type III receptors are found in the extrinsic and intrinsic ligaments [9]. They are also known as the Golgi tendons and are responsible for proprioception [9]. Type IV receptors, free nerve endings, are responsible for pain sensation and are found in the blood vessels, fat pads, joint capsule, ligaments, and tendons [9]. Type II receptors are receptors that have a low threshold and are rapidly adapting [9]. These receptors are inactive at rest but are active during point position and movement [9]. Type I receptors, when compared to type II, have a higher threshold and are slow adapting [9]. These receptors are located in the inferior joint capsule of the hip and are less notable in the peripheral joints [9]. Type I play a significant role in the hip joint by being able to respond to stress and stretch in the hip joint capsule [9]. Past studies have shown that there is a minimum number of mechanoreceptors found in the hip joint capsule, further explaining the lack proprioception response when injury occurs [9]. This explains the lack of local reflexive response during injury, the increased vulnerability to neuromuscular inhibition, and dysfunction in the hip joint compared to peripheral joints [9].

The lumbopelvic rhythm explains how the lumbar spine and surrounding muscles move with the pelvis. It is the motion relationship between the hip joints and the lumbar spine in the sagittal plane. The first part of the lumbosacral rhythm involves the lumbar and sacral spine in flexion while the pelvis has an anterior tilt orientation at the hip joint. When a subject is returning to the erect posture, the rhythm is then reversed. This motion is commenced by the posterior tilting of the pelvis at the hip joint, which is fol-

lowed by extension of the lumbar spine. Existing hip or spinal pathology can affect the muscles of the hip which may change the mechanics of the lumbar pelvic rhythm [9]. Low back pain, especially amongst athletes, is usually associated with injury at L5–S1 vertebrae. There is a local response to the lumbosacral muscle due to this injury, which is inhibition to the segmental stabilizers, multifidus, and transversus abdominus [9]. This injury will also affect the myotomal distribution of the hip abductors, extensors, external rotators, knee flexors, peroneal, dorsiflexor, and plantar flexors [9]. Thus, understanding the spine and hip neuromuscular relationship is essential for thorough examination and successful treatment of hip pathology.

The neuromechanical layer can also explain lower extremity mechanics and the effect it has on the hip. There is an existent kinetic chain of adaptive reactions that can cause changing movement patterns from the ankle and foot to the hip [9]. These adaptive reactions can either occur from the foot to the pelvis or from the pelvis to the foot [9]. Past studies have shown evidence that neuromuscular patterns and reflexive relationships exist. For example, patients with chronic ankle sprains will demonstrate delayed activation of the gluteus maximus muscle during hip extension in a prone position [9]. The muscles of the gluteus maximus will have a change in the firing pattern at the hip (in hip extension) due to the sprained ankle [9]. Lower extremity pathology can significantly affect the arthrokinematics, muscle timing, and performance around the hip [9]. Other examples can be seen with external or internal foot rotations. Patients with a progressing external foot walking gait can indicate signs of trauma, effusion to the hip, femoral retroversion or FAI [1]. Patients with internal rotation of the foot can indicate increased femoral anteversion or acetabular retroversion pathology [1]. Treatment of the hip starts with addressing the kinetic chain in layer IV. It is important for physiatrists to examine the foot and how it hits the ground while patients are walking. The walking mechanics can affect the muscles (myotome patterns) of the hip and pelvis.

Furthermore, it is important and essential for physiatrists to communicate the correct pelvic girdle physical therapy prescriptions to the therapist while a patient is completing rehabilitation.

A "one for all" type of treatment approach regarding pelvic rehabilitation will likely fail [20]. Depending on the type of injury, different treatment approaches should be taken into consideration. When dealing with static overload type of injuries, rehabilitation should be focused on strengthening the hip abductors and extensors [20]. Exercises that should be included are squatting, lunging, and hip hinging with body weight. Optimization of the pelvic tilt should be completed in order for the acetabulum to cover the femoral head [20]. Patients should not participate in aggressive soft tissue exercises and avoid worsening pain in the soft tissue surrounding the stabilizers of the hip [20]. To avoid instability, exercises that focus on flexibility, such as barre and yoga, should be avoided as well [20]. As it pertains to patients that suffer from dynamic overload pathology, exercises that strengthen hip abductors, extensors, and flexors should be the focus of pelvic rehabilitation. The exercises should also include squats, lunges, and hip hinging but without aggressive end range stretching to avoid impingement syndrome. Physical therapists should allow slight variations of the foot and knee angles during these exercises to allow muscle strengthening optimization [11]. Pelvic tilt should be optimized to avoid hip impingement.

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Bursitis and Tendonitis

2

Naldine Isaac, Valerie Bresier, and Debbie Lee

Introduction

Synovial bursae are small fluid-filled sacs that serve as cushioning to decrease friction between bones, muscles, and tendons. Once irritated, the result is an inflammatory condition called "bursitis." Tendonitis refers to pain, inflammation, or dysfunction of the tendons. Bursitis and tendonitis are discussed together because of the close proximity of tendons and bursae, and also because bursitis is a common complication of tendonitis so they can present simultaneously. This chapter will focus on three common conditions in the hip: trochanteric bursitis,

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iliopsoas bursitis, and ischial bursitis, which occur in the lateral, anterior, and posterior hip regions, respectively (Fig. 2.1).

Greater Trochanteric Pain Syndrome (Trochanteric Bursitis)

Pathology

Trochanteric bursae are located between the abductor muscles of the hip (the gluteus muscles and iliotibial band (ITB)) [1]. Previously known as trochanteric bursitis, greater trochanteric pain syndrome (GTPS) is a more general and preferred term referring to localized lateral hip pain with focal tenderness over the greater trochanter that is a result of injury and/or irritation to the gluteal tendons and bursa [2, 3].

In order to better understand the pathology of GTPS, it is helpful to stratify the different bursae and tendons that are involved (Fig. 2.2):

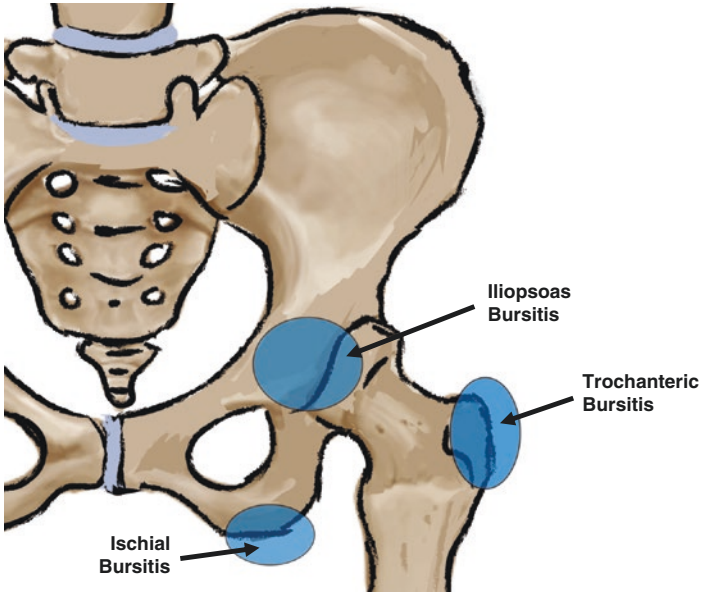

Fig. 2.1 Common locations of tendonitis/bursitis in the hip

- Subgluteus maximus bursae: located deep to the gluteus maximus muscle and ITB that crosses superficial to the gluteus medius. This is the largest bursae of the greater trochanter region and usually the bursa that is referred to when the term greater trochanteric bursa is used.

- Subgluteus medius bursae: located deep to the gluteus medius where it attaches to the lateral and superoposterior facets of the greater trochanter of the femur.
- Subgluteus minimus bursae: located deep to the gluteus minimus as it attaches to the anterior facet of the greater trochanter of the femur [4, 5].

While this pain has previously been thought to originate from trochanteric bursae alone, recent studies have shown that this is less common and often a secondary symptom. More common is an injury to the tendons of the gluteus medius and gluteus minimus muscles resulting in GTPS. There are also various mechanisms that lead to bursitis contributing to this pain syndrome. Bursitis can be triggered by direct trauma to the lateral hip such as a fall. Repetitive movements leading to increased friction between the gluteal tendons and the bursae can cause microtrauma to the area. The bursae can also become irritated due to compression by the adjacent mus-

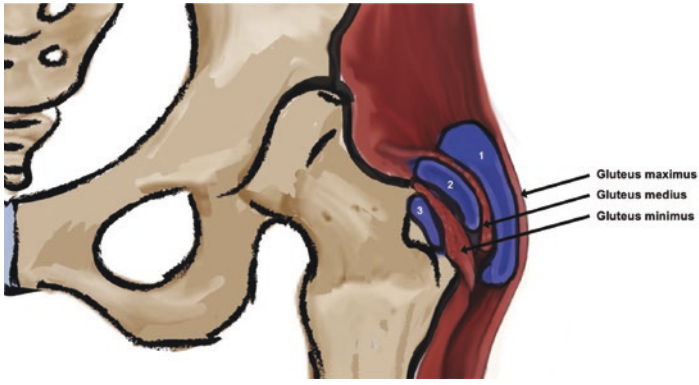

Fig. 2.2 Gluteus bursae. (1) Subgluteus maximus bursa; (2) subgluteus medius bursa; (3) subgluteus minimus bursa

cles. For example, a tightened ITB or tensor fascia lata, piriformis, or gluteus muscle compresses the bursae and causes pain. Additionally, positioning that requires hip flexion for an extended period of time such as sitting with legs crossed can lead to GTPS. A rarer cause is infection leading to septic trochanteric bursitis [2, 3].

While this condition is present in all demographics, the prevalence of this condition is the highest amongst women over the age of 50. It is estimated that unilateral GTPS affects about 15% of this group, and when the condition is bilateral it can affect 8.5% of women over age 50. In men over the age of 50, the prevalence was 6.6% and 1.8%, respectively [6].

In addition to gender and age, other risk factors for GTPS include obesity, arthritis, and/or a history of knee or lower back pain. Additionally, it may be found in those with leg-length discrepancies, osteoarthritis, or iliotibial band thickening [3].

Clinical Presentation

Symptoms of tendonitis and bursitis of the lateral hip can vary. Typically, one will present with unilateral hip pain that gradually worsens in severity. This is in contrast to a gluteal muscle or tendon tear which has similar symptoms but develops suddenly. Some other common complaints are pain that increases with activities such as walking, standing, or running. Patients may report lateral hip pain when sitting with their legs crossed or tenderness when lying on the affected side [7].

In the rarer case of septic trochanteric bursitis, patients can present with warmth, erythema, swelling, and pain over the lateral hip. They may also show more systemic symptoms like fevers or chills.

Physical Exam

Physical exam is the gold standard for the diagnosis of trochanteric bursitis or GTPS. On exam, there is tenderness just above the greater trochanter of the lateral hip; however, this test has low specificity. There can also be associated tenderness to palpation along the ITB.

According to one study, reproducing the lateral hip pain during a Flexion Abduction External Rotation (FABER) test on the affected side is the most sensitive and specific test for GTPS when combined with palpation of the greater trochanter [3]. Pain can also be reproducible with passive adduction as well as passive or active resisted abduction of the affected side. Sudden release of resistance during this abduction maneuver causes a significant increase in pain [5].

Positive Trendelenburg test has a low sensitivity and high specificity for GTPS. The patient stands on the affected leg, and the opposite side of the pelvis will drop indicating gluteal weakness on the affected side. This can also be positive in gluteal tendinopathy.

Diagnostic Exam

Imaging studies are often of little value in the diagnosis of trochanteric bursitis but may be useful to identify the specific cause and involved bursa of GTPS.

Ultrasound has several advantages including lower cost, patient comfort, and the ability to examine areas of tenderness in real time. Ultrasound can reveal distension specifically at three of the possible bursal spaces (subgluteus maximus bursa, subgluteus medius bursa, or subgluteus minimus bursa) consistent with bursitis. It can also demonstrate thickened gluteal tendons with loss of the typically smooth fibrillar pattern to suggest chronic tendinosis [8] (Fig. 2.3).

Plain radiographic films may reveal calcification of the bursae or tendons indicating chronic inflammation.

Magnetic resonance imaging (MRI) can show tendinopathy of the gluteus medius and/or minimus, including partial or complete tendon rupture. There may be associated edematous changes at the enthesis, where the tendon attaches to the greater trochanter [10].

If infection is a suspected cause, laboratory blood testing can be helpful to detect elevated White Blood Cell count (WBC) and Erythrocyte Sedimentation Rate (ESR). If there is sufficient bursal distention, fluid analysis can be done.

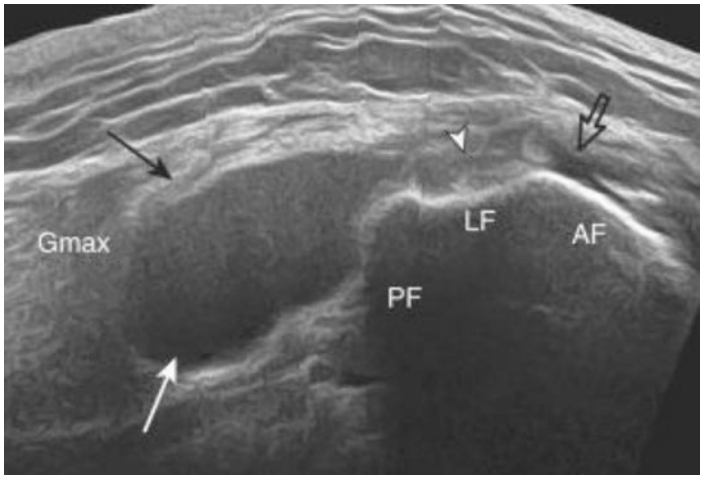

Fig. 2.3 Ultrasound image in short axis to the femur showing distension of the subgluteus maximus bursa (white and black arrow); tendinosis of the gluteus medius (arrowhead); tear of the gluteus minimus (open arrow). *Gmax* gluteus maximus, *AF* anterior facet of the greater trochanter, *LF* lateral facet, *PF* posterior facet. (Adapted from Jacobson, J 2018 [9])

Treatment

There is no defined protocol for the treatment of GTPS. Most patients improve with conservative measures such as rest, ice, non-steroidal anti-inflammatory drugs (NSAIDS), and physical therapy. Behavior modification can also be beneficial [8]. For example, if GTPS is due to how a patient is walking or running, then shoes that provide more support and prevent over inversion may be helpful. If patients lean on one hip when they stand, they should be counseled to distribute their weight evenly.

When patients are very active and develop GTPS from squatting, adjustments can be made to replace these exercises with isometric wall sits for example. If the cause is muscle tightening, then massaging the area surrounding the bursae can relax the muscle and take some pressure off of the bursae.

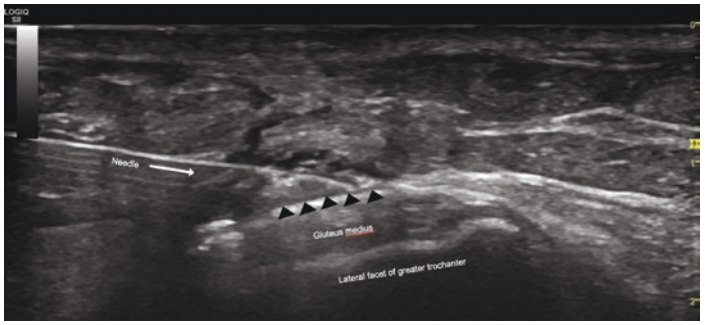

Fig. 2.4 Example of corticosteroid injection into subgluteus maximus bursa under ultrasound guidance. (Arrowheads) outline the distended subgluteus maximus bursa

Corticosteroid injections into the bursae under ultrasound guidance can be considered if pain persists despite initial conservative measures, or if the pain is so severe that patients may benefit from an injection to better tolerate exercises (Fig. 2.4).

Another more controversial option is platelet-rich plasma (PRP) injection. Most of the studies exploring this option are either inconclusive or have outcomes that cannot be compared to one another because of the different PRP formulations used. One study demonstrated that after 12 weeks, injection of platelet-rich plasma into the affected tendon resulted in greater clinical improvement than a glucocorticoid injection as determined by a pain and function assessment [11].

In cases of septic trochanteric bursitis, antibiotic therapy should be initiated.

It may take 6–9 months or sometimes longer to fully recover from greater trochanteric pain syndrome, or trochanteric bursitis. While GTPS is usually self-limiting, under rare circumstances, it can persist. These refractory cases may require surgical bursectomy, ITB lengthening or gluteal tendon repair [12].

Iliopsoas Bursitis

Pathology

The iliopsoas bursa, also known as the iliopectineal bursa, is the largest synovial bursa in the body. It is located between the iliopsoas muscle/tendon and the ilium or the anterior capsule of the hip. Its role is to reduce tendon friction over the hip joint during iliopsoas muscle activation and joint movement [13]. When irritation in this region occurs, the result is inflammation and distention of the iliopsoas bursa. Due to the bursa's close proximity to the tendon of the iliopsoas muscle, tendonitis of the area often coincides with the bursitis and is known as iliopsoas syndrome [13]. Iliopsoas syndrome is typically caused by trauma or overuse of the area. Some suggest extensive hip flexion and extension (especially in the setting of tight iliopsoas muscle) may be the most likely cause of the bursitis [14].

This condition is sometimes referred to as "internal" snapping hip syndrome due to the sensation of snapping or audible snapping noise while the iliopsoas tendon moves across the ilium or anterior hip capsule during activity. This should be distinguished from "external" snapping hip syndrome that occurs in the lateral hip, with snapping sensation occurring between the ITB and greater trochanter [15].

Iliopsoas syndrome is common particularly amongst female dancers, as approximately 9.2% of dancers may report a painful or painless snapping or clicking sensation upon flexion or extension of the hip compared to 3.2% of male dancers [16]. This often occurs due to the repetitive flexion of the externally rotated hip motion that is commonly involved in dance movements such as the "passé developpé" [16].

Risk factors include histories of osteoarthritis or rheumatological conditions (i.e., Gout or rheumatoid arthritis), as these can directly cause irritation at the iliopsoas tendon attachment site or bursa.

Clinical Presentation

Clinical presentation may be variable. While some may be asymptomatic, patients often present with anterior groin pain. Patients may also experience referred pain down the anterior thigh or even experience swelling of the lower extremity. Though typically gradual in onset due to overuse, this can occur more acutely after sports injuries or work-related trauma. Activities such as running uphill, track and field, and strength training may precipitate symptoms such as groin pain which may indicate iliopsoas bursitis [14].

Physical Exam

On exam, there can be tenderness to palpation in the anterior hip region below the inguinal ligament and lateral to the femoral artery [14]. Though rare, enlarged bursa can sometimes be palpated. Pain may also be provoked by both active and passive hip motions, especially during semicircular hip motion while the hip is flexed [13].

In rare cases where the bursal distention enlarges to compress the adjacent femoral nerve, there may be decreased sensation of the anteromedial thigh and medial lower leg as well as weakened and atrophic quadriceps muscles in later stages of development. The patellar reflex may also be lost [14]. However, if a patient presents with these symptoms, further investigation of lumbar spine pathology should be done first.

Diagnostic Exam

Diagnostic imaging has limited utility. However, utilization of ultrasound may be an efficient method to identify bursal distention (Figs. 2.4 and 2.5). Ultrasound can also detect snapping motion of

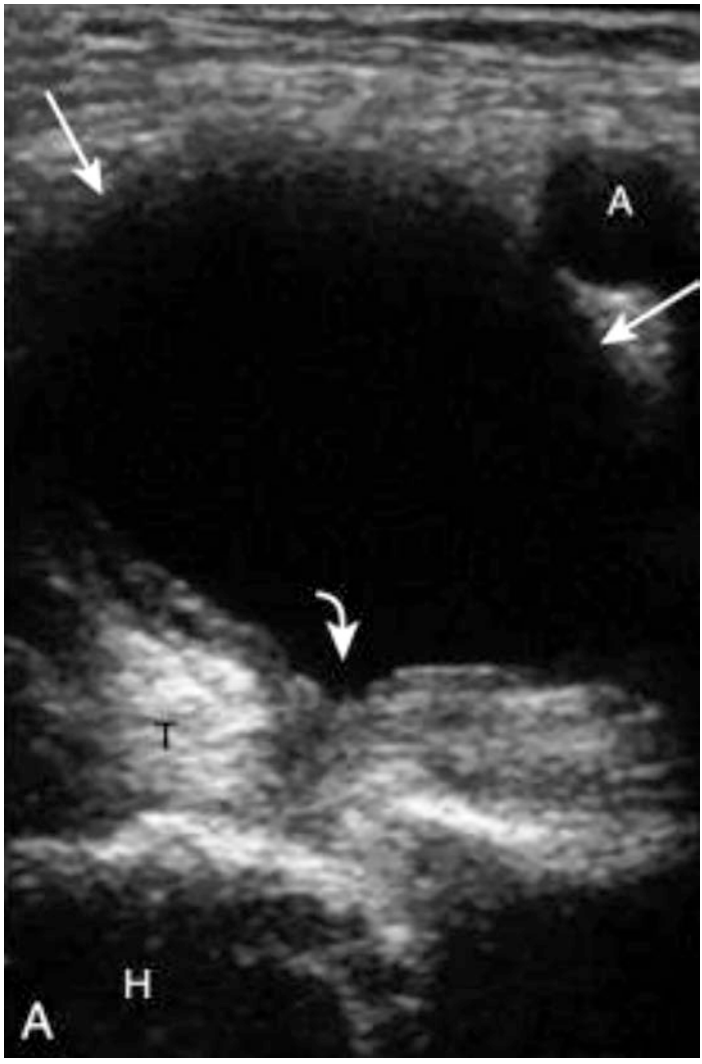

Fig. 2.5 Ultrasound image shows anechoic distention of the iliopsoas bursa as shown by the arrows. H = transverse of femoral head, T = psoas major tendon, A = femoral artery. (Adapted from Jacobson, J 2018 [9])

iliopsoas tendon with dynamic hip motion in the case of internal snapping hip syndrome [17]. Diagnostic ultrasound-guided injection of the iliopsoas bursa with local anesthetic may be useful [18].

Plain radiograph can be helpful to assess for advanced arthritic changes of the hip or other bony changes that may contribute to irritation of the iliopsoas tendon/bursa.

MRI of the pelvis and hip are reserved for more complex presentations [19]. It can be helpful mostly to rule out other pathologies such as tumors, femoral hernia, femoral artery aneurysm, or lymphadenopathy. However, in the case of iliopsoas syndrome that involves significant enlargement of the bursa, MRI may demonstrate retroperitoneal expansion, abdominal, or pelvic mass [14]. Melamad et al. proposed the triad of palpable mass, extrinsic pressure on adjacent structures, and radiographic changes of advanced arthritis of the hip for more complicated cases of iliopsoas bursitis involving retroperitoneal extension [14].

Treatment

Like trochanteric bursitis, treatment is typically conservative and does not require surgery unless the patient is not responsive to other non-invasive methods. This includes avoidance of aggravating activities, hip extension stretches for 6–8 weeks, and hip rotation strengthening exercises. Referral to injection therapy or surgery may follow if initial non-operative plans fail to show improvement of symptoms.

Johnston et al. proposed a specific rehabilitation program for iliopsoas syndrome which includes up to 2 weeks of hip rotation exercises with knees flexed, 2–4 weeks of external rotation and abduction exercises with knees flexed, then 4 weeks or more of mini squats, external rotation of the leg and gait retraining [15]. Johnston states that the rehab program targets both internal and external rotator muscle groups in the initial portion of the program. These exercises are performed while sitting with an elastic resistance band. If strength on the initial exam is observed to be weak in one direction in comparison to their sides, exercises were

prescribed in a 3–2 ratio with the weaker direction doing more exercises to regain strength over time [20].

It typically takes between 6 and 8 weeks with the aforementioned exercises though some patients may experience prolonged weakness in the hip rotator muscles which may lead to a decrease in hip stability.

Other treatment options include the use of ultrasound-guided iliopsoas peritendinous corticosteroid injection [21]. More recently, regenerative medicine techniques including injection of platelet-rich plasma, mesenchymal stem cells, and other products derived from amniotic tissue which are actively involved in the healing process have been used in treatment of iliopsoas bursitis [22].

Though rarely indicated, if refractory bursitis persists after 3 months of conservative treatment, surgical intervention may be introduced involving bursectomy and closure of the hip capsule [14]. Such procedures can also involve releasing or lengthening the iliopsoas tendon and show variable degrees of success in alleviating the pain associated with internal snapping hip [23].

Ischial Bursitis

Pathology

Ischial bursitis refers to the inflammation of the bursa which sits in between the ischial tuberosity and the hamstring tendons (semimembranosus tendon, and the conjoined tendons of semitendinosus and biceps femoris muscles). This may be one singular bursa or multiple segmented inflamed bursa.

This type of bursitis is often caused by chronic irritation of the leg in those who lead a more sedentary lifestyle. It may also occur as a result of sitting on hard surfaces such as bike riding or horseback riding for an extended period of time. Other risk factors include histories of rheumatological conditions including but not limited to gout, rheumatoid arthritis, systemic lupus erythematosus, ankylosing spondylitis, or Reiter's syndrome [24, 25].

Clinical Presentation

Pain related to ischial bursitis is often localized to the ischial tuberosity but may be referred down the posterior thigh. Pain can be provoked during hip or knee extension, or during sitting due to direct pressure on the bursa. Pain can also increase at night as patients frequently report difficulty sleeping on the affected side and report a sharp painful sensation on hip flexion and extension when waking up in the morning [24].

Physical Exam

On exam, pain may be provoked upon passive straight leg raising and active resistance of extension of the affected side due to activation or stretching of the involved hamstring muscles/tendons. There may also be an increase in pain upon release of the affected leg from active extension [24]. On inspection, the lower buttock region may appear erythematous and swollen over the ischial tuberosity in the case of septic bursitis though it may be too small to appreciate these findings [25].

Diagnostic Exam

As with the previously mentioned conditions, diagnostic imaging is not necessary. However, ultrasound can be useful to detect hypoechoic bursal distension surrounding the iliopsoas tendon at its attachment site on the ischial tuberosity.

Ultrasound and plain radiographs may also show calcification surrounding the hamstring tendons in chronic cases (Fig. 2.6) [24].

MRI may show irritation of the hamstring muscles, including partial or complete tendon rupture. There may be associated edematous changes at the enthesis, where the tendon attaches to the ischial tuberosity. It can also demonstrate associated bursal distention.

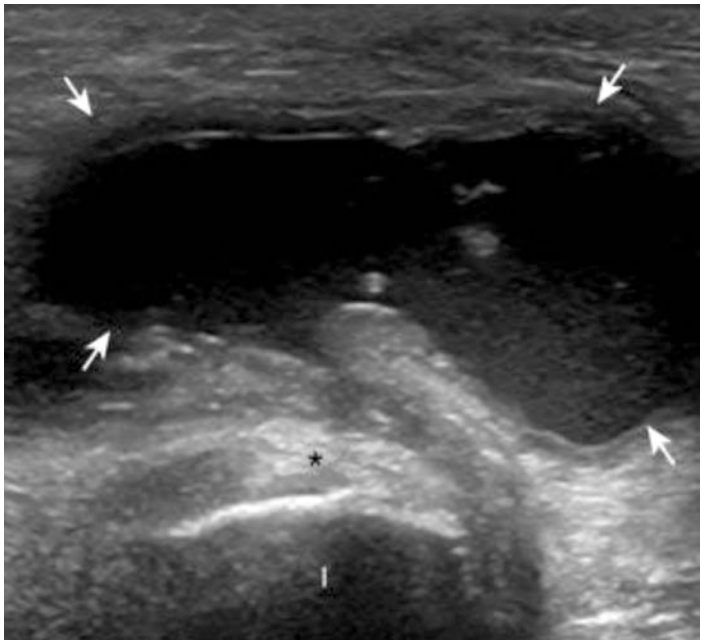

Fig. 2.6 Ultrasound image arrows show the transverse plane of the ischium with hypoechoic complex bursa distention predominantly. *I* = ischium. (Adapted from Jacobson, J 2018 [9])

Computed tomography (CT) or radionuclide bone scanning may be indicated in refractory cases or if there is concern for more severe disease such as cancer metastasis or occult fracture in the hip and pelvic region [24].

Treatment

As with other bursitis of the hip, treatment of ischial bursitis is often conservative consisting of initial rest, NSAIDS, physical therapy, and other modalities such as cold pack application as needed. In the acute phase, isometric hamstring exercises are encouraged with gradual progression to eccentric and concentric

activation exercises. Such exercises may involve the use of a treadmill in which the patient is instructed to face backwards, hold the rails with both hands, and place the stationary limb off of the belt. The patient will then extend the hip of the injured limb while extending the knee with the foot placed on the belt behind the patient and resist the force of the belt as it pulls the limb forward while maintaining proper pelvic posture [26].

It is also important to change lifestyle factors to prevent refractory cases of ischial bursitis. For example, if the patient has a history of a sedentary lifestyle, it would be beneficial to incorporate more physical activity after fully healing [24].

For refractory tendonitis, tenotomy with local anesthetic injection may be considered to promote blood flow and healing. For refractory bursitis, corticosteroid injection with ultrasound guidance may be helpful [27] (Fig. 2.7).

As with other cases of hip bursitis and tendonitis, regenerative medicine can also be applied for ischial bursitis. Platelet-rich plasma may be helpful in providing longer term pain relief or reducing rates of recurrence [28].

Overall, patients may improve within days to weeks without treatment with the exact timing being dependent on the severity of the bursitis [29].

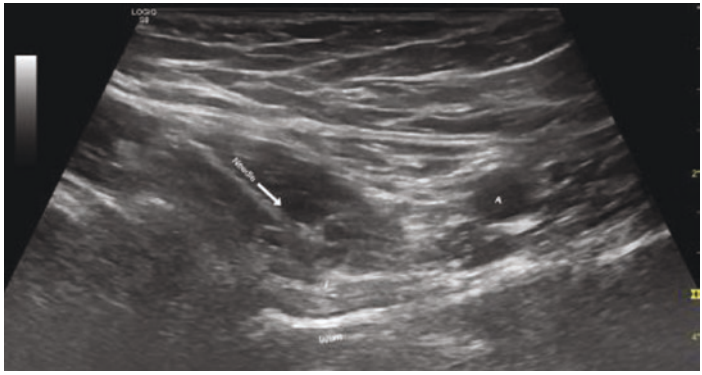

Fig. 2.7 Diagnostic iliopsoas bursa injection under ultrasound guidance. I = iliopsoas tendon; A = femoral artery

Conclusion

Bursitis and tendonitis are common pathologies of the hip that often occur simultaneously.

In three common pathologies of the hip, Greater Trochanteric Pain Syndrome, Iliopsoas Bursitis, and Ischial Bursitis, there is irritation or inflammation to surrounding tendons, muscles, and bursae that result in different manifestations of pain.

Diagnosis is made through patient history and physical exam maneuvers, while imaging such as US, CT, and MRI can provide supporting evidence.

Conservative measures along with behavior modifications and rehabilitation exercises are standard treatments. There are various exercises that target the areas of dysfunction that work to provide relief.

In severe or refractory cases, corticosteroid injections, platelet-rich plasma injections, or surgical intervention are considered. As the effect of these techniques continue to be studied, it is possible that these modalities will become more common practice for the management of bursitis and tendonitis of the hip.

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Labral Tears

3

Fabienne Saint-Preux and Tochi J. Nworu

Pathology

The acetabular labrum is a 2–3 mm thick fibrocartilaginous structure which attaches to the rim of the acetabulum. One of its primary functions is to provide additional stability by deepening the acetabulum [1]. It also functions in shock absorption, pressure distribution, and joint lubrication. It has been reported that the prevalence of labral injuries is anywhere from 22% to 55%, occurring more in females than males [2]. The labrum can be torn secondary to high forces at the hip joint, chondral degeneration, or repetitive microtrauma associated with repeated twisting and pivoting [3]. Isolated lesions occurring from trauma tend to occur more frequently in younger patients while tears secondary to degenerative joint changes are more common in older individuals. Injury is commonly associated with sports where repetitive external rotation occurs such as in ballet, golf, hockey, and soccer [2]. In addition to external rotation, end range motions of hip hyperextension, hyperflexion, and hyperabduction are thought to contribute to injury as the labrum plays a role in pressure distribution and weight bearing [4]. Other factors associated with labral injury

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include femoral acetabular impingement (FAI), laxity/hypermobility, and hip dysplasia [5].

There are two forms of FAI: pincer and cam impingement. Pincer impingement occurs when the acetabulum creates excessive coverage over the femoral head. This can lead to impaired hip mechanics, thus leading to posteroinferior chondral lesions [5]. These lesions can be localized or circumferential. Movement at the hip can result in pinching of the labrum between the acetabular rim and the femoral neck, resulting in degeneration and bony apposition of the adjacent rim [1].

Cam impingement occurs when there is abnormal contact between the femoral head and the acetabulum due to an unusually large femoral head radius. This also leads to dysfunction of normal joint mechanics inadvertently causing anterosuperior labral and chondral lesions [4, 5]. The labrum gets caught between the abnormal femur and the normal acetabular rim, especially during flexion and internal rotation [1].

Capsular laxity and hypermobility decrease hip stability thus increasing the chance of injury. Joint hypermobility can occur in patients with dysplasia due to the shallow acetabular socket resulting in decreased coverage of the femur anteriorly and laterally. This can lead to compression of the labrum, resulting in tears most often occurring at the anterior labrum [5].

Positioning of the femoral head within the acetabulum is compromised in patients with hip dysplasia resulting in joint incongruity or decreased joint surface area. Consequently, this increases stress on the acetabulum and the labrum, potentially causing detachment of the labrum from the acetabulum. Patients with hip dysplasia tend to have hypertrophic labrums occurring in the anterior portion. The hypertrophy likely contributes to the impingement of the labrum between the acetabulum and femoral head [1].

Clinical Presentation

The presence of a labral tear is often associated with complaints of anterior hip or groin pain, locking, clicking, catching, instability, and giving away; about 61% reported an insidious onset [1]. In some

cases, patients report buttock pain as well as posterior and/or lateral hip pain. This can be indicative of a posterior labral tear, although pain in these locations is more commonly associated with lumbosacral spine pathology, posterior hip musculature injuries, trochanteric bursitis, and/or iliotibial band syndrome [5]. Apart from pain, which 90% of patients complain of, clicking is one of the most common symptoms associated with labral tears [6]. Typically, patients report constant dull pain with intermittent sharp pain brought on or worsened by activity [1]. Symptoms can be exacerbated with pivoting, squatting and hip movements in various directions [6]. After provocation, pain typically persists even after rest [1]. In many cases, the duration of symptoms reported by a patient prior to appropriate diagnosis averages about 2 years. This may be due to the difficult nature of identifying labral tears as the source of hip pain [7]. In the case of labral tears associated with FAI, a common symptom includes anterior pinching pain with sitting. Medial thigh pain and morning stiffness is typical of injury secondary to degenerative changes. Patients with capsular laxity often report pain with instability [8].

Physical Exam

The diagnosis of acetabular labral injury can be difficult to make as patient history and physical exam maneuvers lack specificity [6]. Accurate diagnosis is further challenging due to the plethora of hip pathology presenting with similar characteristics. Although this is true, a thorough history and physical exam accompanied with specific provocative maneuvers can help identify a labral tear. Anterior hip impingement tests and femoral acetabular grind maneuvers are most consistent with provoking symptoms of labral tear [6]. Other tests to consider when assessing for labral pathology include FABER, FADIR, hip scour test, and resisted straight leg test, which are described in detail in Table 3.1. One needs to bear in mind that these tests assess the presence of intra-articular lesions. With that in mind, a positive or negative finding alone does not rule a labral pathology in or out. Some provocative maneuvers, such as the scour and resisted straight leg raise may also strain the lumbosacral region helping to identify the location

Table 3.1 Provocative tests to assess for hip pathology

Name of test	Position	Maneuver	Positive finding	Differential diagnosis
Labral anterior impingement test	Supine	Examiner brings the patient's hip to 90° of flexion, 20–25° of hip adduction and maximal internal rotation	The test is considered positive if there is reproduction of anterior or lateral hip pain	FAI, labral impingement, anterior superior labral tear
Labral posterior impingement test	Supine	Start with the patient's hip in full flexion and adduction, then bring the patient's hip into extension, abduction, and lateral rotation	The test is considered positive if patient exhibits sharp catching pain with or without a "click"	Anterior hip instability, posterior inferior hip impingement, posterior labral tear
FABER	Supine	Patient's hip is brought into flexion, abduction, and external rotation (leg position looks like the number 4) with the lateral ankle resting on the contralateral thigh, proximal to the knee	Reproduction of pain in the Sacroiliac Joint (SIJ), groin, or posterior hip	Low back pain; SIJ dysfunction. Groin pain: FAI, labral tear, loose bodies, chondral lesion or hip OA. Posterior hip pain; posterior hip impingement
FADIR	Supine	Patient's knee and hip are brought to 90° of flexion with adduction and internal rotation at the hip	The test is considered positive if there is reproduction of patient's pain	FAI, labral tear, loose bodies, chondral lesion, or hip OA

Table 3.1 (continued)

Name of test	Position	Maneuver	Positive finding	Differential diagnosis
Hip Scour test	Supine	Patient's hip is brought into 90° of flexion and the knee in full flexion. The examiner applies a downward force through the femur while adducting and externally rotating at the hip. The same is done with abduction and internal rotation of the hip	The test is considered positive if there is reproduction of patient's pain	Labral pathology, osteochondral defects, acetabular defects, OA, and femoral acetabular impingement syndrome
Resisted leg test	Supine	Patient actively flexes hip to 30° with knee extended against resistance	The test is considered positive if there is reproduction of the patient's pain and or weakness with resistance	Athletic pubalgia, slipped capital femoral epiphysis, FAI

Lewis CL, Sahrmann SA (2006) Acetabular Labral tears. Physical Therapy 86:110–121; Martin RRL, Enseki KR, Draovitch P, Trapuzzano T, Philippon MJ (2006) Acetabular Labral tears of the hip: Examination and diagnostic challenges. Journal of Orthopaedic & Sports Physical Therapy 36:503–515

of symptoms. In doing so, one can better identify if the etiology originates from the hip or lumbosacral region. If there is high suspicion for a labral tear, associated factors such as FAI, capsular laxity, and articular cartilage degeneration should also be assessed [1].

Diagnostic Studies

Diagnosis of acetabular labral pathology can be challenging due to the lack of specificity in the clinical history and physical exam in addition to overlapping symptoms with similarly presenting intra-articular hip disorders such as snapping hip syndrome and/or femoroacetabular impingement (FAI). Imaging serves as a supplemental tool to help confirm the appropriate diagnosis. Imaging work up should include initial plain radiographs of the hip joint to rule out other sources of disease such as osteoarthritis, dysplasia, deformity, fracture, loose bodies [9], and to look for underlying structural abnormalities that may be the cause of the labral tear. Pertinent views include standing AP pelvis, AP hip, frog-leg lateral, Dunn, cross-table lateral and false profile view. These views allow for specific detection of subtle developmental dysplasia of the hip (DDH) or FAI. MRI can be used for diagnostic evaluation of labral and chondral lesions however offers false positive rates and low sensitivity; furthermore, MRI may underestimate labral pathology. If MRI is utilized, irregular labrum shape, a non-triangular labrum, a thickened labrum with no labral recessus, a labrum with increased signal intensities on T1 images, and a labrum that has detached from the acetabulum are suggestive of labral tear [10]. Definitive diagnosis of labral injuries is by direct visualization with arthroscopy and has four morphological classifications: radial flap, radial fibrillated, longitudinal peripheral and unstable (Table 3.2).

Table 3.2 Arthroscopic morphological classification of labral tears

Classification	Morphologic finding
Radial flap	Disruption of the free margin of the labrum with consequent formation of a discrete flap
Radial fibrillated	Appearance of a shaving brush Hairy appearance at the free margin of the labrum
Longitudinal peripheral	Variable length along the acetabular insertion of the labrum
Unstable	Reflection of abnormal labrum function rather than shape; subluxing acetabular labrum

Lage, Lafayette A., Jig V. Patel, and Richard N. Villar. The Acetabular Labral Tear: An Arthroscopic Classification. *Arthroscopy: The Journal of Arthroscopic & Related Surgery* 12.3 (1996): 269–72. Web [17]

Magnetic resonance arthrography (MRA) with intra-articular contrast, however, is the imaging modality of choice when labral pathology and/or chondral injury is suspected due to its high specificity and sensitivity when compared to plain MRI [11, 12]. Most labral tears occur at the anterior superior labrum with MRA showing a linear hyperintense T2 signal intensity contacting the labral surface with associated findings of subchondral bone marrow edema and/or cystic changes, osseous fragmentation at the superior acetabulum and paralabral cysts which by themselves are a highly specific finding for labral injury [9]. A classification system based on MRA findings is provided by Czerny et al. [13] (Table 3.3). In addition to MRA findings, diagnostic intra-articular anesthetic or steroid injection can be used to confirm intra-articular etiology of hip pain if reduction in pain is correlated with MRA findings [12].

Table 3.3 MRA classification of labral tears

Stage	MRA finding
0	Homogeneous low signal intensity, a triangular shape, and a continuous attachment to the lateral margin of the acetabulum without a notch or a sulcus; labra had a recess between the joint capsule and the labrum (labral recessus) that consisted of a linear collection of contrast material extending between the cranial margin of the acetabular labrum and joint capsule
1A	Area of increased signal intensity within the center of the labrum that did not extend to the margin of the labrum, a triangular shape, and a continuous attachment to the lateral margin of the acetabulum without a sulcus and revealed the labral recessus
1B	Similar to stage IA labra, but the labrum was thickened and no labral recessus was present
2A	Extension of contrast material into the labrum without detachment from the acetabulum, labrum were triangular, and had a labral recessus
2B	Same as stage 2A except the labrum was thickened and a labral recessus was not present
3A	Labra were detached from the acetabulum but were of triangular shape
3B	Labra were thickened and detached from the acetabulum

Czerny, C et al. Lesions of the acetabular labrum: accuracy of MR imaging and MR arthrography in detection and staging. *Radiology* vol. 200(1) (1996): 225–30. https://doi.org/10.1148/radiology.200.1.8657916

If diagnostic intra-articular injection is being considered to confirm labrochondral pathology, imaging guidance with ultrasound or fluoroscopy is recommended due to high miss rate of non-image-guided procedures [12]. Table 3.4 summarizes the diagnostic imaging workup for acetabular labral tears.

Table 3.4 Diagnostic modalities

Imaging modality	Views	Findings[a]	Utility
Plain radiograph	• AP pelvis • Cross table lateral • ± Frog lateral view • False profile view	• Usually normal	• Evaluates underlying structural abnormalities of hip and pelvis • Rules out other causes of pain
MRI	• Standard without contrast	• Irregular labrum shape • Non-triangular labrum • Thickened labrum with no labral recessus • Labrum with increased signal intensities on the T1 images • Labrum that has detached from the acetabulum	• Evaluate labral and chondral pathology • Low sensitivity
MRA	• + Gadolinium/ contrast	• Contrast extending into the labrum • Contrast extending into acetabular/labral interface • Blunted appearance of labrum • Displacement/ detachment from underlying bone	• Modality of choice • Rules out other hip abnormalities • Great for anterior labral tears but not posterior and lateral tears

Table 3.4 (continued)

Imaging modality	Views	Findings[a]	Utility
Arthroscopy	• Direct surgical visualization	• Radial flap • Radial fibrillated • Longitudinal peripheral • Unstable[b]	• Gold standard • Allows for comprehensive evaluation of labral anatomy • Diagnostic and therapeutic
Intra-articular steroid injection (image guided)			• Confirms intra-articular pathology • Prognosticates response to future surgery if needed • Sensitive and specific for intra-articular pathology

[a] Findings that are specific to labral tears

[b] Lage, Lafayette A., Jig V. Patel, and Richard N. Villar. The Acetabular Labral Tear: An Arthroscopic Classification. *Arthroscopy: The Journal of Arthroscopic & Related Surgery* 12.3 (1996): 269–72. Web [17]

Treatment

Non-operative Management

Pharmacologic

Conservative management of labral tears is first line and is guided by pain, mobility, and functional limitation. Treatment usually consists of a 2–4 week course of non-steroidal anti-inflammatory medications (NSAIDs) however, hepatic, gastrointestinal, cardiac, and renal side effects of NSAIDs limit its prolonged use and must be considered on an individual basis. Chronic NSAID use is not recommended. Relative rest, activity modification, and offloading the joint as well as therapeutic exercise aimed at improving femoral head motion within the joint and improving biomechanics around the hip should accompany the initial treatment plan.

Physical Therapy

Therapeutic exercise consists of a 4–12 week focused formal physical therapy (PT) program after which the patient should be reassessed for functional improvement and reduction in pain [1]. The PT program should optimize the alignment of the hip joint and precision of joint motion by avoiding excessive force to the anterior hip joint, correcting movement patterns during gait and exercise and minimizing pivoting motions [14]. A comprehensive therapeutic exercise regimen seeks to restore range of motion (ROM) and strengthen the hip flexors, abductors, external rotators, and extensors. Special emphasis on the correction of knee and hip hyperextension during stance phase is especially important to combat faulty gait patterns and improper gait mechanics. Functional lifestyle modifications should include avoiding sitting in excessive hip flexion, sitting cross-legged, sitting with hip rotated, sitting on the edge of a seat with hip flexors contracted, and avoidance of quadriceps and hamstring weight training [14].

Interventional and Regenerative Medicine

Aside from the diagnostic utility of intra-articular anesthetic injection and prognostic utility of intra-articular steroid injection, intra-articular steroid injection can be used as a therapeutic modality for temporary pain relief if chondrosis or wearing of articular cartilage is present [12].

Indications for regenerative treatments include mild-moderate joint effusion/synovitis, hyaline cartilage degeneration, and fibrocartilage tears/degeneration [15]. The acetabular labrum is a ring of fibrocartilage that lines the border of the acetabulum and thus would benefit from regenerative treatments specific to fibrocartilage including lipoaspirate positive (LA+) platelet-rich plasma (PRP), amniotic membrane, bone marrow concentrate (BMC), and dextrose prolotherapy [15]. Large, detached labral tears are the least likely to respond to regenerative therapy due to vascular supply and limited healing capacity and thus should be referred for surgical evaluation.

1. Platelet-rich plasma

   PRP delivers a supraphysiological concentration of platelet-derived bioactive factors that are capable of modulating

inflammation and promoting tissue healing. PRP has been injected, clinically, into tendons, ligaments, and joints to improve pain and function with variable results and is currently used "off label" due to no FDA-approved indications [15]. Lipoaspirate positive (LA+) denotes the process by which adipose tissue is removed from the body and used to perform fat grafting or lipotransfer to another part of the body, in this case to a torn labrum. Whole LA serves as a structural scaffold for large fibrocartilage tears or hyaline defects thus supporting the healing defect of a partial labral tear. PRP can be leukocyte rich (LR) or leukocyte poor (LP); LP PRP creates a stronger fibrin matrix, LR-PRP stimulates greater inflammation and vascular proliferation. LP-PRP is preferred for intra-articular use because it induces greater chondrocyte proliferation and has lower counts of inflammatory cytokines [15]. Although either can be used for labral tear, theoretically, LP-PRP may confer more benefit.

2. Amniotic membrane

   While there is no literature to support the use of amniotic membrane for labral tears, it contains numerous growth factors and collagen scaffolding that theoretically support its use to "fill in" the labral defect [15].

3. Bone marrow concentrate

   BMC is a concentrated form of bone marrow aspirate containing mesenchymal stem cells (MSCs) and bioactive substances obtained via aspirating bone marrow from the pelvic bone. MSCs inhibit apoptosis/cell death secondary to ischemia, inhibit scar formation, secrete large amounts of growth factor and promote angiogenesis which stimulates MSCs to continue on in a cascade that ultimately leads to tissue regeneration and healing [15]. BMC, however, does not contain an extracellular matrix which is needed to best fill labral defects; this should be taken into consideration when choosing an injectate as it may affect treatment response.

4. Prolotherapy

   This modality involves injecting irritant solutions, usually dextrose, into the labrum/labral defect with the hope of stimulating a low grade inflammatory reaction to promote tissue proliferation and healing.

According to Malanga and Ibrahim, an injectate containing LP-PRP, LR-PRP, adipose-derived mesenchymal stem cells (ADSC), BMC, amniotic membrane (AMG), or a combination of these can be injected into the acetabular labrum using ultrasound guidance. This can be followed by offloading of the hip with crutches for 2–5 days and/or a hip unloading brace for 4–6 weeks post injection; however, there is no literature to support a difference in outcomes with non-weight bearing after injection [15].

Operative Management

Referral to an orthopedic surgeon for surgical management is reserved for the patient who has failed to reach adequate pain control and optimal function with conservative management. Surgical intervention includes hip arthroscopy for labral debridement and/or labral repair. While the current literature supports the superiority of acetabular labral repair compared to debridement in terms of decreased rate of osteoarthritic progression and positive patient outcomes, there is no significant difference in the rate of progression to total hip arthroplasty up to 10 years later [16]. Labral repair, however, remains the gold standard for surgical treatment of labral tears. A good prognosis is anticipated with a positive response to intra-articular steroid injection; a higher degree of chondrosis and/or articular cartilage wearing is associated with poor prognosis after surgical intervention [9].

Return to Activities

Return to activities and/or play depends on the healing and location of the tear in addition to whether or not it responds to conservative vs. surgical intervention. With conservative management (i.e. a short course of NSAIDs and physical therapy) return to activity can occur as early as whenever pain is reduced and function is restored. However, because a labral tear is a mechanical issue, PT may result in only temporary resolution of symptoms with return of symptoms with full activity. Blood vessels supply only the peripheral one-

third of the acetabulum, the inner two-thirds are avascular resulting in poor healing potential based on location. In the case of poor healing, surgical management will likely be required for successful outcome especially for return to play in athletes [14]. In the case of surgical intervention, return to play can occur anywhere between 6 and 12 weeks depending on sport (cutting and running sports) or greater than 12 weeks if intervention for bony abnormalities occurred [9].

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Osteoarthritis

4

Nnaemeka Nweke, Vishal Bansal,
and Emanuel Narcis Husu

Hip Osteoarthritis

Osteoarthritis (OA) is considered a degenerative disease of one or more joints and its associated ligaments and can be observed in weight-bearing and non-weight-bearing joints. It goes beyond the simple wear and tear definition and can be viewed as the final stages of chronic imbalance between breakdown and repair of bone.

It is most seen in the hands and knees but also frequently noted in the hips and spine [1]. A multitude of risk factors exist for the development of hip osteoarthritis and can be categorized based on factors involved at the local joint structure or generalized systemic factors [2].

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At the joint level, risk factors include and are not limited to:

Acetabular dysplasia: A smaller than normal acetabulum can lead to development of hip osteoarthritis. This is proposed to be due to altered biomechanical forces within the joint [3].

Slipped capital femoral epiphysis: A commonly occurring condition in 9–16-year-olds where the femoral head is displaced posterior and inferior leading to a varus, extension, and external rotation deformity [4]. Studies have shown that if insufficient remodeling occurs, the impingement can lead to early osteoarthritis.

Femoro-acetabular impingement (FAI) syndrome: Femoro-acetabular impingement syndrome is classified into two types. They are Cam-type and Pincer-type. *Cam-type*: Non-spherical morphology of femoral neck over femoral head. *Pincer-type*: Excessive arching of acetabulum over femoral head. FAI leads to abnormal contact between joint surfaces and increasing the likelihood of hip degeneration [1].

Perthe disease (Legg-Calves-Perthes disease): A condition of idiopathic avascular necrosis most observed in those less than 10 years of age [1]. This has been linked to the development of osteoarthritis in later years [5].

Developmental dysplasia of hip (DDH): A condition of abnormal hip joint architecture, described as the acetabulum being shallow and improper fit of the femur. This can lead to altered biomechanical forces leading to hip osteoarthritis [6].

At the individual level, risk factors include and are not limited to:

Age: Advancing age past 60 has been known to be a risk factor in the development of radiographic OA [3, 7]. This is hypothesized to be due to chondrocalcinosis, an age-related matrix change, that promotes production of pro-inflammatory markers that have been linked to the development of OA.

Obesity: Directly plays a role in weight-bearing joints such as the hip, and thus promotes osteoarthritis. Furthermore, in addition to the direct effect, there is also an indirect effect from obesity and its

connection with metabolic syndrome. Studies have shown that diabetes, dyslipidemia, hypertension, and/or cytokines from adipose tissue (adipokines) may play a role in the development of joint damage due to a chronic low-grade inflammatory state [2, 8].

Ethnicity: Caucasians have higher hip OA prevalence, ranging from 3% to 6%, compared to Asians, African-Americans, East Indians, or Native Americans who have 1% or less. Different races and genetic differences have variations in hip morphology which may account for these observed patterns [1].

Occupational risk factors: In a systematic review by Sulsky et al., the literature suggested a possible causal relationship with repetitive, long-term heavy lifting (10–25 kg loads), and the development of hip OA [1, 9]. Another study that looked at male athletes that represented Finland in international events (competitive sports) from 1920 to 1965 concluded they were at a slightly higher risk of requiring hospital care due to osteoarthritis of the hip, knee, or ankle, but at an older age for the endurance athletes [10].

Metabolic factors: *Estrogen*—Women have a higher prevalence of OA after the age of 50, whereas men under the age of 50 have a higher prevalence. Some studies have hypothesized this may be due to postmenopausal changes, an indication of protective effects from estrogen replacement therapy with respect to hip OA [2]. *Vitamin D*—It is involved with homeostasis of calcium and regulation of bone metabolism, and a deficiency may lead to impaired bone deposition [1]. Vitamin D deficiency has been associated with OA prevalence, incidence, and progression but primarily in the knee [1].

Gender: Females are known to have stronger hip joints compared to males and likely explains the case for higher symptomatic osteoarthritis in males compared to females.

Osteoarthritis involves and is not limited to the hips, shoulders, spine, feet, and hands. As a clinician, when evaluating hip osteoarthritis, it is advised to be cognizant of the different risk factors discussed and how one or more plays a role in the development or can predispose your patient to the development of hip OA.

Pathological Process

A general overview of hip osteoarthritis can be described as degradation of cartilage, alterations in subchondral bone, meniscus degeneration, synovial inflammatory response, and the associated periarticular bone response [1]. At the cellular level, this can be further described as disorganized breakdown and repair. Osteoblasts are cells involved in generating, remodeling, and regulating bone architecture and matrix mineralization through the production of extracellular matrix proteins and by signaling osteoclastogenesis. In individuals with osteoarthritis, there is an alteration in the functionality of these cells [11].

Clinical Presentation

The general presentation when a chief complaint of hip pain is made can often be challenging to pinpoint as some patients have difficulty in describing their pain. Hip pain descriptions include groin pain, thigh or buttock pain, low back pain, or ipsilateral knee pain [12]. The more specific description of groin pain can often point the examiner towards hip OA, but it is important to note that every patient describes their hip pain differently and some have reservations when it comes to describing pain [12, 13]. Thus, a comprehensive evaluation is vital; the physical examination and the diagnostic workup are important to rule out systemic manifestations as well as other musculoskeletal causes of the hip pain. Depending on the setting, ruling out recent infection, fever, or injury can help narrow down the differential and ensure other possible preceding causes for hip pain are not overlooked.

Physical Exam

An important part of the physical exam is understanding the patient's chief complaint.

Evaluation begins as the patient is walking through the clinic or the hospital setting with gait assessment. Patients have a tendency to shy away from the location of pain by tilting, applying more weight to the unaffected side, which is known as the Trendelenburg sign. It is due to weakness of the hip abductors and slowing down the glide of the affected limb with each stride [14–16].

When physically examining the hip, it is important to perform examinations that rule out lumbar spine or the sacroiliac joint as the source of the hip pain. It's common to have hip pain that stems from the low back radiating to the buttocks and down the leg. It's important to start with palpation but prior to this you should ask the patient where their maximum point of pain is. Next, focus your palpation of the hip accordingly, dividing into anterior, posterior, lateral, and medial regions [14] (Fig. 4.1). As an examiner, it is important to save the region of maximum pain for last to maintain and build patient trust.

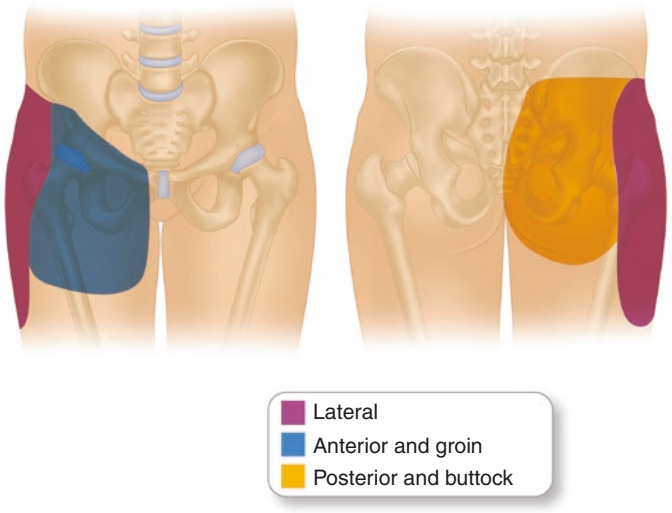

Fig. 4.1 Left: anterior hip and right: posterior hip. (Wilson JJ, Furukawa M (2014) Evaluation of the patient with hip pain. American Family Physician. https://www.aafp.org/afp/2014/0101/p27.html. Accessed 9 Jan 2022)

Lumbar spine range of motion (ROM) such as forward flexion, back extension, lateral flexion, and rotation can be assessed to see if any pain is generated [17]. The examiner will be watching for structural or pain-limited limitations with ranging. It is important to always listen to the patient throughout this process [13]. In addition, to delineate whether it is a true low back pain as the cause for hip pain vs. sacroiliac joint pain, the FABER test (Flexion Abduction External Rotation) of the hip is helpful as studies have shown it to have 91.4% positivity in diagnosing SI joint dysfunction [18] (Fig. 4.2).

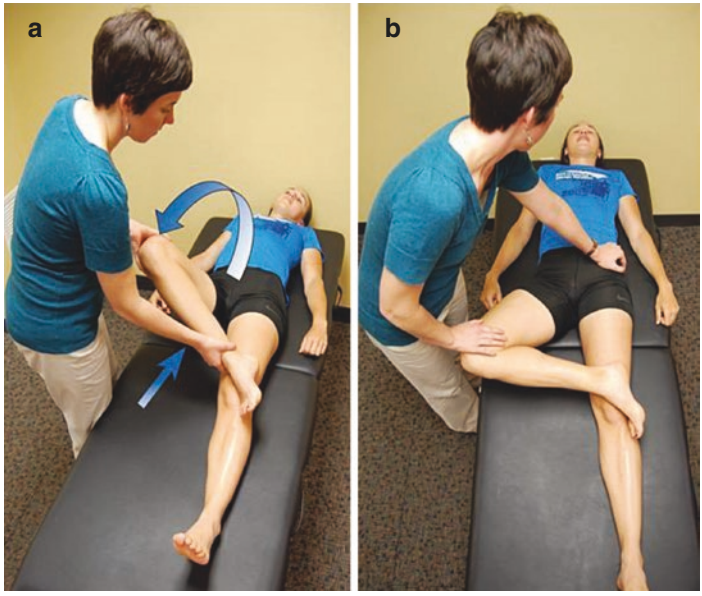

Fig. 4.2 FABER test (flexion, abduction, external rotation; Patrick test). The examiner moves the leg into 45° of flexion, then (**a**) externally rotates and (**b**) abducts the leg so that the ankle rests proximal to the knee of the contralateral leg. (Wilson JJ, Furukawa M (2014) Evaluation of the patient with hip pain. American Family Physician. https://www.aafp.org/afp/2014/0101/p27.html. Accessed 9 Jan 2022)

After ruling those out, before concluding it is hip OA or other hip pathologies, further examination is needed. Active and passive ROM of the hip, performed while sitting and supine, should be assessed with exception of hip abduction which can be best assessed in the lateral position [14] (Fig. 4.3). You should be looking for range reduction especially with internal rotation. A reduction greater than or equal to 15° with pain in hip internal rotation

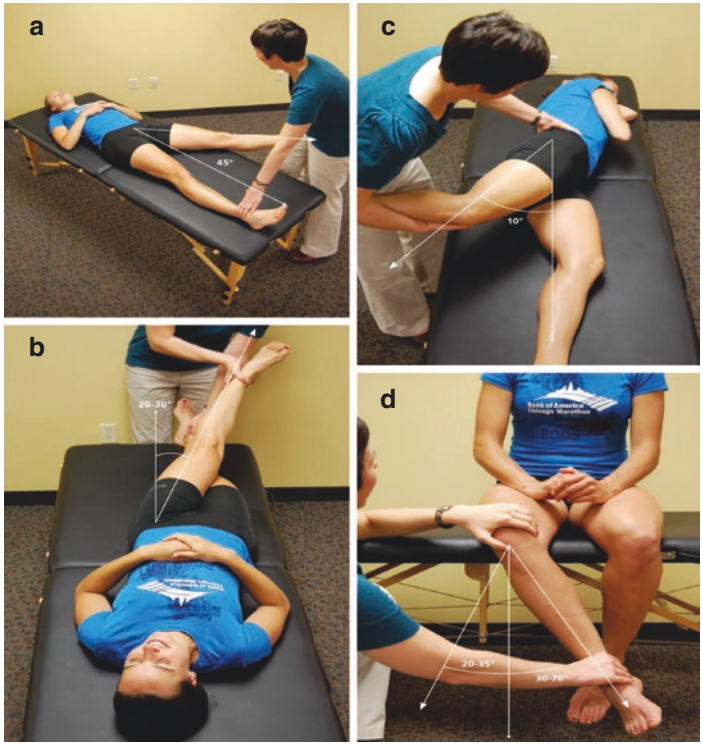

Fig. 4.3 Hip range-of-motion testing (photos demonstrate normal range of motion). (**a**) Abduction. (**b**) Adduction. (**c**) Extension. (**d**) Internal and external rotation. (Wilson JJ, Furukawa M (2014) Evaluation of the patient with hip pain. American Family Physician. https://www.aafp.org/afp/2014/0101/p27.html. Accessed 9 Jan 2022)

is a part of the criteria used in ruling in hip OA, which was noted in a clinical study of 201 patients with complaints of hip pain [19]. Other tests to note is the FADIR test, log roll test, straight leg test, and OBER test which all can be performed and used to further delineate other causes of hip pain such as labral tears, femoroacetabular impingement, and greater trochanteric pain syndrome (Table 4.1).

Table 4.1 Physical examination tests for the evaluation of hip pain

Test	*Other names*	*Positioning*	*Positive findings*	*Differential diagnosis*
Gait testing (C sign, Figure 1A; gait analysis, Figure 1B)	—	Standing	Antalgic gait, Trendelenburg gait, pelvic wink (rotation of more than 40 degrees in the axial plane toward the affected hip when terminally extending the hip), excessive pronation or supination of the ankles, and limps caused by differing leg lengths	Hip labral tear, transient synovitis, Legg-Calvé-Perthes disease, SCFE
Modified Trendelenburg test (Figure 1C)	Single leg stance phase	Standing	2-cm drop in the level of the iliac crest, indicating weakness on the contralateral side	Hip labral tear, transient synovitis, Legg-Calvé-Perthes disease, SCFE
ROM testing (Figure 2)	—	Supine, lateral,or sitting	Pain with passive ROM, limited ROM	Pain with passive ROM:Transient synovitis, septic arthritis Limited ROM: Loose bodies, chondral lesions, osteoarthritis, Legg-Calvé-Perthes disease, osteonecrosis
FABER test (Figure 3)	Patrick test	Supine	Posterior pain localized to the sacroiliac joint, lumbar spine, or posterior hip; groin pain with the test is sensitive for intra-articular pathology	Hip labral tear, loose bodies, chondral lesions, femoral acetabular impingement, osteoarthritis, sacroiliac joint dysfunction, iliopsoas bursitis
FADIR test (Figure 4)	Impingement test	Supine	Pain	Hip labral tear, loose bodies, chondral lesions, femoral acetabular impingement
Log roll test (Figure 5)	Passive supine rotation, Freiberg test	Supine	Restricted movement, pain	Piriformis syndrome, SCFE
Straight leg raise against resistance test (Figure 6)	Stinchfield test	Supine	Weakness to resistance, pain	Athletic pubalgia (sports hernia), SCFE, femoral acetabular impingement
Ober test (eFigure B)	Passive adduction	Lateral	Passive adduction past midline cannot be achieved	External snapping hip, greater trochanteric pain syndrome

FABER = flexion, abduction, external rotation; FADIR = flexion, adduction, internal rotation; ROM = range of motion; SCFE = slipped capital femoral epiphysis.

Wilson JJ, Furukawa M (2014) Evaluation of the patient with hip pain. American Family Physician. https://www.aafp.org/afp/2014/0101/p27.html. Accessed 9 Jan 2022

Diagnostic Workup

Understanding the morphological changes seen in an arthritic joint is predicated on understanding the structure and physiology of the joint. Hence why the hip joint, which is used for weight bearing, is best assessed radiographically during weight bearing. Osteoarthritis is a pain generator mainly during articulation of that joint. Hip OA early presentation is generally a result of any shape-modifying pathologies that lead to poor articulation, causing dysfunction within the joint and ultimately early degeneration [20].

Radiographic Study

A poor detector of early OA, radiographic film is excellent for assessing the progression of the disease [20]. The hip joint is best assessed in the anterior and posterior view looking for joint space narrowing, osteophytes, subchondral cysts, and sclerosis [20]. Joint space narrowing, a result of articular cartilage loss, can be typically seen and analyzed as symmetrical versus asymmetrical (Fig. 4.4). Symmetrical changes are more indicative of inflammatory arthritis versus asymmetrical changes, which are more significantly seen in OA [21]. Several bony changes can be seen with OA on radiographic studies such as osteophytes (represents new bone formation to replace cartilage loss), enthesophytes, subchondral sclerosis, and cysts [20]. In late stages of OA, it is not uncommon to see joint malalignment [20].

CT Study

Computed tomography is less commonly used in diagnosing OA, but it is better than a radiographic study in recognizing subchondral bone (Fig. 4.5) [20, 21]. In addition, CT arthrography can be used to detect soft tissue injuries seen in many inflammatory states, which can be seen as a sign of early osteoarthritis [20].

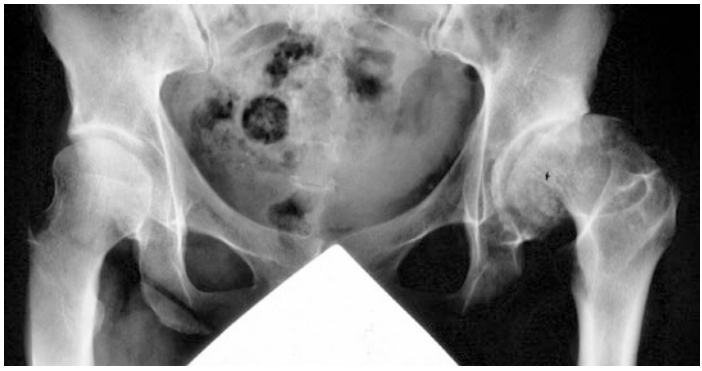

Fig. 4.4 Frontal view left hip—severe osteoarthritis; narrowing of joint space, subchondral sclerosis and cystic changes. (Courtesy of Wikimedia Commons)

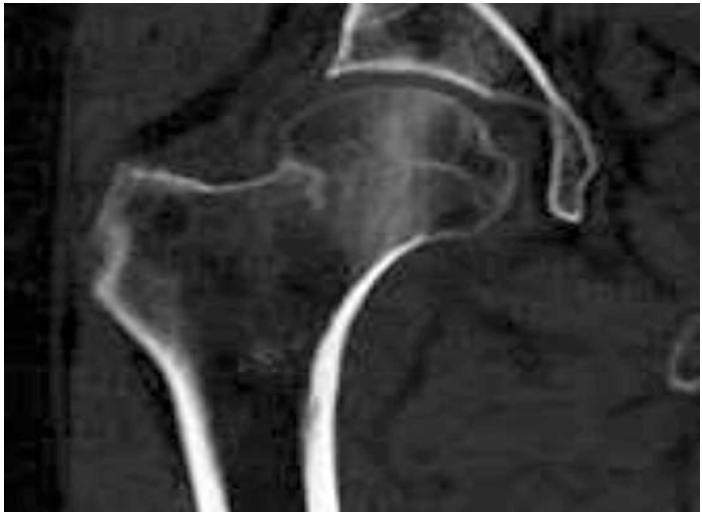

Fig. 4.5 Frontal view of right hip on CT. (Courtesy of Wikimedia Commons)

MRI Study

Magnetic resonance imaging (MRI) is also excellent in detecting soft tissue swelling and subchondral cartilage damage (Fig. 4.6) [20, 21]. When it comes to diagnosing OA, MRI is not conventionally used initially. It serves well in identifying synovitis, osteophytes, and joint effusion [20].

Ultrasound Study

Ultrasound evaluation in the clinical setting is very excellent in evaluating soft tissue swelling and is comparable to MRI (Fig. 4.7) [20]. One of the other advantages of it is that it is readily available. Ultrasound is disadvantageous in identifying subchondral cysts, sclerosis, and bone inflammation, but it is advantageous in assessing cortical changes such as osteophyte formation, erosions, and enthesophytes [20].

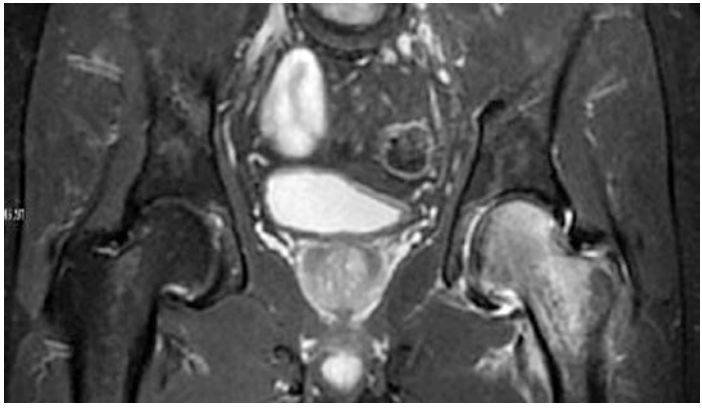

Fig. 4.6 MRI view of bilateral hips. (Courtesy of Wikimedia Commons)

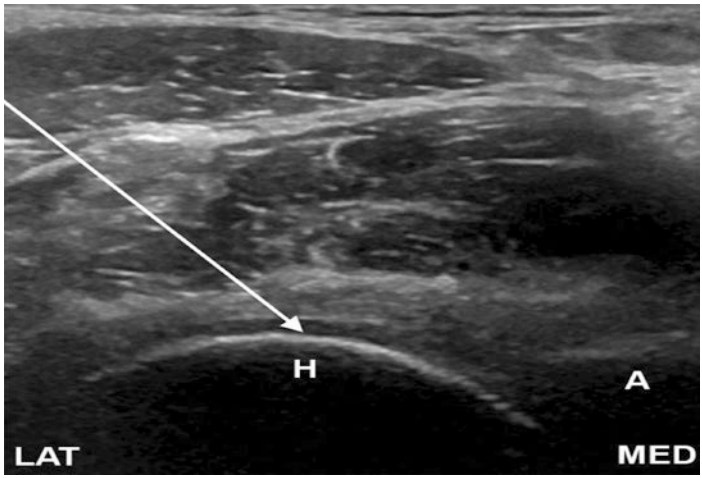

Fig. 4.7 Ultrasound view of the hip. (Courtesy of Wikimedia Commons)

Laboratory Test

It is not generally indicated for diagnosing osteoarthritis but can be important in ruling out other systemic causes. If fever is present, ruling out infectious causes is necessary. A complete blood count (CBC) with differential, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) can be helpful. If infection is of concern, arthrocentesis of the joint can be performed to further evaluate the synovial fluid cells, and a Gram stain performed. If negative for infection, ruling out inflammatory causes such as rheumatoid arthritis can be achieved with rheumatoid factor and human leukocyte antigen B27 tests, with consideration of rheumatology referral [22].

Treatment

Osteoarthritis can be managed through a holistic approach, and both pharmacologically and/or with surgery.

Holistic Approach

Efforts should be prioritized to starting off with non-pharmacologic management when approaching hip osteoarthritis. Additionally, these modalities can provide a synergistic effect with the medications and/or surgery after conservative management fails. A holistic approach to osteoarthritis includes and is not limited to:

Patient education: Educate about illness, the nature of pain, and how the two are interconnected in hip osteoarthritis and your patient.

Weight management: Reductions in weight (in those with obesity) will assist with limiting force on weight-bearing joints [1, 2, 7, 23]. A referral to a dietician to address maladaptive eating patterns leading to obesity can be helpful. A referral to a psychiatrist can also be considered to address underlying psychiatric disorders that contribute to a sedentary lifestyle and maladaptive eating patterns and social activities (smoking, illicit drugs, excessive alcohol intake).

Exercise: Aerobic exercise, ROM, weightlifting/strengthening, and tai chi can be helpful [23]. Aquatic therapy limits the weight-bearing forces through its buoyancy effect, and thus minimizes barriers to participation (i.e., pain, fatigue). A Cochrane review by Bartels et al. concluded there was a small, short-term clinically relevant effect with an aquatic-based exercise program [24]. The review reported improvement in pain, disability, and quality of life in individuals with knee and hip OA [24].

Physical and occupational therapy: Refer patients to obtain a personalized exercise program to help address weakness and reduced ROM and address functional deficits. Modalities can include transcutaneous electrical nerve stimulation, thermal therapy, cryotherapy, and ultrasound therapy.

Massage therapy: Caution is advised to avoid excess ROM/stretching that could impact the articulating surface.

Other useful tools: Bracing/taping, acupuncture, and orthotics

Pharmacological Treatment

The approach for medical management of osteoarthritis involves the use of the following common medications:

- *Acetaminophen*: Trialed as first-line therapy, indicated for mild to moderate pain levels:
  - Dosing: 325–650 milligrams (mg) every 4–6 h as needed. Can consider increasing to 1 gram (g) every 6 h [25, 26]. Maximum daily dose of 4 g.
  - Warning: Risk of hepatotoxicity in large doses > 4 g. Not advised to be used in patients with pre-existing severe active liver disease or significant hepatic impairment [25].
- *Non-steroidal anti-inflammatory drugs (NSAIDS)*: Diclofenac has been found to be the most efficacious for hip OA pain [2, 27].
  - *Diclofenac potassium or sodium formulations*

    Dosing: 100–150 mg a day, can be prescribed in 2–4 divided doses (immediate or delayed release). If extended release is used, then it is prescribed once daily. Maximum daily dose of 150 mg.

    Warning: Risk of gastrointestinal, cardiovascular, and renal side effects. Risk of hepatic damage with an increase in liver transaminase levels.
- *Topical agents*: Capsaicin cream, topical NSAIDS, lidocaine. It can be used as an adjunct to acetaminophen and/or NSAIDS. Variable responses in patients who use topical medications for hip osteoarthritis due to the depth between the skin surface and the joint compared to other areas such as the hand and knee.
- *Opioids*: Can be considered when acetaminophen and/or NSAIDS have not provided appreciable relief for the patient. Caution advised due to potential interactions with alcohol, benzodiazepines, and other depressants, their addiction potential, and their side-effect profile (especially in the elderly).
- *Selective serotonin and norepinephrine reuptake inhibitors (e.g., duloxetine):* can consider when chronic widespread pain or pain sensitization are present.

Interventional Procedures

Intra-articular steroid injections are widely used for the management of osteoarthritis and have been shown to be effective in improving pain and function in the short term. But concerns arise with frequent use, which increase the risk of infection and further cartilage breakdown [28]. Because of the risk of infection and cartilage breakdown, another option to offer your patient is an intra-articular ketorolac injection. Studies have investigated intra-articular ketorolac injection as an alternative and concluded that it is just as effective in pain management [28]. In conclusion, both present as great options for the management of hip OA.

Radiofrequency ablation is another alternative to managing pain from hip OA, especially in patients with late-stage OA, who have contraindication to undergoing total hip arthroplasty (THA). This procedure targets the articular branches of the obturator nerve and femoral nerve that innervate the hip with the use of high frequency alternating current to lesion the nerves. Rivera et al. conducted a prospective study of 18 patients with contraindications for THA and concluded that cooled radiofrequency (CRF) treatment can be an effective method to reduce chronic hip pain. Significant reduction in hip pain and an increase in functionality were noted 6 months after the CRF ablation procedure [29].

Surgical intervention: Depending on the severity and medical stability, good candidates often undergo total hip arthroplasty. Other options include femoral head resurfacing, hip fusion, and femoral head resection.

Regenerative Medicine

Hyaluronic acid (HA): FDA approved in 1997, hyaluronic acid is thought to be effective by restoring elastic and viscous properties of synovial fluid and hyaluronan synthesis by synoviocytes. Although not approved for hip OA, there is evidence that it is just as effective for the hip as it is for the knee. The benefits can be seen in its possibility of delaying the need for hip replacement surgery.

Platelet-rich plasma (PRP): It involves using the patient's own cells to stimulate the release of growth factors involved in tissue healing and disrupting catabolic processes. A study by Sanchez et al. involved 40 patients with unilateral hip OA and found statistically significant reductions in hip pain with leukocyte-poor PRP. A meta-analysis by Ye et al. examined randomized controlled trials on PRP compared to HA in patients with hip OA and concluded higher reductions in pain at 2 months compared to HA but no significantly better outcomes at 6 and 12 months. Much controversy still exists over the use of PRP, and it can be attributed to inconsistencies in PRP sample preparation methods (leukocyte-rich versus leukocyte-poor) and the lack of standardization in techniques.

Stem cells (SC): Although evidence of its effectiveness in hip OA is lacking, studies have shown its ability to reduce pain. A recent study conducted by Shapiro et al. [30] on 25 patients with bilateral knee OA in which one group received bone marrow aspirate concentration (BMAC) plus PRP in one knee and the other saline placebo demonstrated reduction of pain, but no significant difference was noted between the two. Another study conducted by Rodriquez-Fontan et al. [31] evaluating BMAC for early hip and knee OA concluded that intra-articular injection of BMAC for early OA treatment was safe and satisfactory in 63.2% of patients. Still, there is limited evidence of stem cell use in the treatment of hip OA [32].

Supplements

Glucosamine and chondroitin are oral supplements that have been hypothesized to be taken up by articular cartilage leading to its widespread use. However, more recent studies with no funding association demonstrated no statistically significant clinical effect and were no longer recommended for OA treatment [1, 2].

Compliance

Most importantly in the treatment of OA is developing good rapport with the patient with the use of clear patient-friendly language that considers the patient's description of their pain, which, in turn, forms a congruent relationship with the caregiver. This has been shown to improve patient compliance with the treatment [13].

Prevention

Obesity—numerous studies have established the connection between obesity and lower limb OA. Although not as strong as the association between obesity and knee osteoarthritis, the association between obesity and hip OA still exists [1, 8]. The connection between the two goes beyond the excessive force put on weight-bearing joints and is also thought to be due to additional metabolic factors that accompany obesity and associated conditions (metabolic syndrome) [2, 8]. Addressing obesity involves using a multimodal approach with your patient and examining their diet, physical activity, and lifestyle. Looking at the patient's psychiatric well-being can also help provide additional insight into how certain decisions and choices are made that can be contributing to obesity.

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5 Impingement Syndrome/ Femoroacetabular Impingement Syndrome

Katherine Caldwell

Pathology

Femoroacetabular impingement (FAI) refers to abnormal contact between the acetabulum and femoral head-neck junction during range of motion of the hip joint, especially extreme flexion and rotation [1–3]. This can eventually lead to pain and degeneration of the joint. "Femoroacetabular impingement" is a relatively new term, first coined by Ganz et al. in their 2003 article "Femoroacetabular Impingement, A Cause for Osteoarthritis of the Hip." In this article, they noted that, while axial overload of the hip does cause many cases of osteoarthritis (OA), it does not account for OA in many young patients. They found that FAI is common in these young patients with seemingly idiopathic OA [2].

The three skeletal morphologies of FAI are cam, pincer, and mixed. Cam morphology refers to an abnormal, aspherical shape of the femoral head. Pincer morphology is an over-coverage of the femoral head-neck junction by the rim of the acetabulum. Mixed

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refers to a combination of both cam and pincer deformities. These skeletal morphologies cause increased mechanical stress between the bones, especially in hip flexion and rotation [1–5].

FAI, especially that caused by cam morphology, may lead to hip osteoarthritis over time. This is currently thought to be the primary cause of "idiopathic" hip OA. With repetitive wear and tear on the soft tissues, it can also lead to labral tears and cartilage defects. When FAI progresses to cause symptoms, it is referred to as femoroacetabular impingement syndrome (FAI syndrome or FAIS) [3, 6, 7].

Clinical Presentation

FAIS is more common in young, active adults [1, 3, 6–8]. Although the exact prevalence has been difficult to determine, studies have shown that cam deformity is present in over a third, and pincer deformity is present in roughly two-thirds of FAIS. Males are more likely to have cam morphology, while females are more likely to have pincer morphology [6]. Athletes are three times as likely as the general population to have a cam deformity [6–8]. It is thought that high-impact sports participation, especially during youth while the growth plate is open, may contribute to the development of a cam morphology via adaptive changes to the bones [4, 6, 8]. Sibling studies have also suggested a genetic predisposition to developing a cam deformity. Less is known about the etiology of pincer deformity, at this time studies have not linked it with either activity or genetics [3, 7].

The typical clinical presentation of FAIS is a young patient with pain in the hip or groin which is brought on by activity or certain positioning [3]. The pain pattern can be variable and may radiate to the lateral hip, buttock, or thigh. Some patients endorse mechanical symptoms, such as stiffness, clicking, and locking during movement. Pain associated with FAIS is usually insidious and progressive over time. Many patients do not present for workup or treatment until the pain has been present for months to years and has become severe [1, 3, 7].

Physical Examination

There is no single exam maneuver to diagnose FAIS. The purpose of the exam when this diagnosis is suspected is to help rule out other causes of groin pain and correlate positive exam findings with the history and radiographic findings.

As with any report of hip pain, the exam should include assessments for asymmetry and pain with sitting, standing, ambulation, and squatting. Some studies have shown that patients with FAIS have reduced strength and proprioception on single-leg balance and squat [7]. With the patient supine, the clinician should inspect for abnormalities in limb length, pelvic asymmetry, and hip rotation at rest.

Range of motion of both hips should be evaluated. Restricted internal rotation would be expected in hip impingement. One of the most common tests used to evaluate for FAIS is FADIR, in which the patient is lying supine and the hip is brought into flexion, adduction, and internal rotation by the examiner. This test is positive if there is reproduction of pain in the groin and reduced internal rotation. A positive FADIR is sensitive but not specific for FAIS [1, 3].

Diagnostic Studies

The Warwick Agreement on FAI syndrome, which is an international consensus statement on FAIS, was published in 2016. This consensus statement recommended three criteria in the diagnosis of FAIS: hip and/or groin pain, physical exam findings consistent with FAI, and imaging findings of pincer and/or cam deformities [3]. It is important to note that impingement seen on imaging in the absence of pain or physical exam findings does not constitute a diagnosis of FAIS.

Initial imaging should begin with an anteroposterior (AP) X-ray of the pelvis in an upright position centered on the patient's midline. A lateral view of the proximal femur and femoral head [1, 3, 6] should be obtained on initial imaging as well. There are

multiple lateral views that can be obtained, but modified Dunn and frog-leg are used most frequently in the evaluation of FAIS. Of note, the sensitivity of plain radiographs is not high enough to rule out the diagnosis. If X-rays are unremarkable, further imaging with magnetic resonance imaging (MRI) is warranted. A benefit of utilizing MR imaging during workup of FAIS is the option to use MR arthrography to simultaneously detect a labral tear.

Some studies have proposed specific measurements on imaging to diagnose a cam or pincer deformity; however, because FAIS is a dynamic process and the culmination of multiple factors, there is no currently agreed-upon criteria [3, 7]. FAI does tend to be bilateral; however, symptoms often do not match up, so it may be beneficial to assess both hips when this diagnosis is being considered [3, 6].

Treatment

Treatment of FAIS broadly falls into two categories: conservative measures or operative intervention. As with many musculoskeletal conditions, the treatment is largely dependent on the patient's clinical presentation, lifestyle, and goals. Unlike some of the other hip conditions, FAIS is relatively new in the literature; therefore, the evidence comparing different treatment options is lacking.

From a conservative standpoint, treatment consists of pain relief with medications or injections and/or a physical therapy program. A systematic review and meta-analysis from 2019 showed that both physical therapy and intra-articular corticosteroid injections improve pain and function in patients with FAIS, but did note that there are not many high-quality publications on this topic [5]. There is currently insufficient evidence to discuss treatment of FAIS with orthobiologics [8].

A more aggressive approach to the treatment of FAIS is surgical correction of the bony deformities, via either open or closed surgery. An additional benefit of surgical intervention is the opportunity to simultaneously address an associated issue such as a labral tear or cartilage defect. In a meta-analysis from 2020

comparing therapy versus arthroscopic surgery, data was analyzed from 644 patients within three randomized controlled trials. From these studies, the authors concluded that surgery is an effective treatment of FAIS and may be superior to therapy [9]. Multiple studies have shown that predictors of positive outcomes from surgery are younger age, lower BMI, male sex, and pain relief from intra-articular hip injections [10, 11].

Because FAI is thought to predispose patients to developing OA and soft tissue damage later on, there has been much debate about prophylactically correcting the impingement with surgery. There is not enough evidence currently to support surgically correcting an asymptomatic cam or pincer deformity [3, 7, 8].

Patient education and shared decision-making are strongly encouraged so that the patient understands the indications, risks, and benefits of each treatment option. In general, it is recommended to begin with nonoperative management prior to surgery [3, 7, 8].

Return to Activities

Recommendations on returning patients to normal activities depend somewhat on the patient's lifestyle and goals. For example, a professional athlete may be more inclined to return to sport as soon as possible, while a "weekend warrior" may be okay with resting for a longer period of time.

There have been reports of returning to high-level sport at 3 months post-arthroplasty; however, the average return is around 6 months. A recent clinical trial showed that reduction in pain and improvement in functional outcomes plateaued at 6 months, thus patients can be educated that the majority of their improvement will likely occur within this timeframe. This article also noted that, while the majority of patients are able to return to sport or recreational activities, less than 20% of patients are able to return to pre-injury levels of performance [12]. Negative predictors of returning to high-level athletics following surgery include higher BMI, greater than 2 years of symptoms, presence

of chondral damage, hip OA, mental health concerns, increased age, chronic opioid use, and moderate to severe hip dysplasia. The severity of hip OA correlates most strongly with the ability to return to high-level performance, with more severe OA indicating worse outcomes [7].

Regardless of the management, pain is generally the factor that guides the timeline. Patients should be instructed to resume activities gradually and avoid positions or movements that cause pain.

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Pediatric Hip Pain

6

Kayla Williams, Onyinyechi Chidomere, and Stephanie Tow

Introduction

The hip is a complex joint, able to move in all planes of motion while supporting the entire weight of the body. It is a relatively stable joint, composed of bony architecture, strong ligaments, and supportive muscles. The pediatric hip is uniquely fascinating in that it achieves this while simultaneously developing in its own architecture and functioning through the development of bipedal mobility. When this process is interrupted in children either by congenital or extrinsic factors, it causes pathology leading to pain and dysfunction, which varies depending on age and differs greatly from common etiologies of hip pain in adults [1, 2].

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Physical Exam and Diagnostic Studies

Physical exam components including inspection, palpation, hip range of motion (ROM), provocative testing, muscular strength testing, gait analysis, and functional testing can help elucidate a child's potential pathology.

Assessing hip ROM, specifically external and internal rotation, should be performed both supine (with the hip flexed and knee flexed) and prone (with the hip in neutral and knee flexed) to assess for femoral version (Fig. 6.1) [3]. Dynamic testing of hip ROM can also be useful. Torsional or angular deformity of the femur may be an indication of joint laxity, muscular strength imbalance, or malalignment. The Barlow and Ortolani tests can be used to screen for hip instability in infants. In the supine infant with hips flexed and adducted, the Barlow test is performed by applying gentle pressure in a posterior and lateral direction to attempt to provoke dislocation of the femoral head from the acetabulum. A normal test reveals no motion in this direction. A positive test is characterized by a distinct "clunk" palpated as the femoral head dislocates out of the acetabulum and is concerning for hip dislocation. In the supine infant, the Ortolani test is

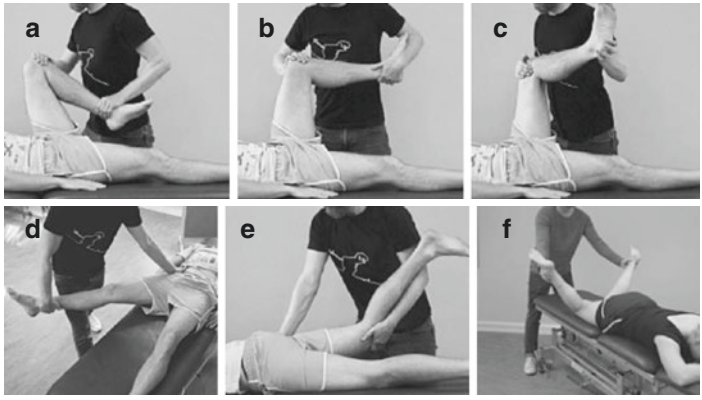

Fig. 6.1 (**a**–**f**) Passive hip ROM in flexion (**a**), internal rotation with 90° of hip flexion (**b**), external rotation with 90° of hip flexion (**c**), abduction (**d**), extension (**e**), and internal rotation with a neutral hip position (**f**) [3]

performed by abducting the hips and applying gentle pressure from the posterior proximal thigh in the anterior direction. This test is positive if a "clunk" is palpated as a dislocated femoral head is reduced back into the acetabulum. The Galeazzi test is performed with the child supine and hips and knees flexed to position the feet flat on the table. In unilateral hip dislocation, the affected knee will appear more proximal as compared to the contralateral side. Asymmetry of the height of the knees may indicate hip dislocation or leg length discrepancy [1]. The Klisic test is performed with the child in supine and the examiners index finger positioned over the anterior superior iliac spine and middle finger on the greater trochanter. An imaginary line connecting these points should point to or superior to the umbilicus; with hip dislocation, this line passes inferior to the umbilicus [1].

Variations in gait and ability to bear weight can also be seen due to hip pathology. An antalgic gait, or limp, is associated with a shortened stance phase due to ipsilateral pain. A positive Trendelenburg sign, or Trendelenburg gait, occurs when standing on one lower extremity, the pelvis on the contralateral side drops while in the swing phase due to weak hip abductors on the ipsilateral side. Out-toeing is often benign, but when asymmetric may be an indication of hip pathology, external tibial torsion, or femoral retroversion. In-toeing, more commonly seen than out-toeing, can be caused by internal tibial torsion, femoral anteversion, and metatarsus adducts [4, 5].

Specialized provocative maneuvers can be helpful in differentiating between intra-articular and extra-articular pathologies. Log roll can be performed in the supine position with the hip in neutral by applying passive internal and external rotational movements of the thigh and hip. Limited range of motion or pain reproduction with log roll testing is often concerning for intra-articular pathologies but can also provoke pain with piriformis syndrome or other tight musculature. It is therefore important to attempt to delineate the location of pain during this maneuver to help differentiate. The Ober test is performed in the side-lying position with the bottom hip and knee flexed and with upper leg extended. The upper leg is passively abducted and extended then slowly lowered until the leg no longer requires support of the examiner.

This is useful to assess for iliotibial band tightness. Pain or limited range of motion with the hip flexion, abduction, and external rotation (FABER) test may raise suspicion for sacroiliac syndrome, lumbar spondylolysis/spondylolisthesis, iliopsoas strain or bursitis, posterior femoroacetabular impingement (FAI), or other intra-articular hip pathologies (e.g., labral tears). Pain or restricted range of motion with the hip flexion, adduction, and internal rotation (FADIR) test is associated with anterior FAI, especially if there is a loss of associated internal rotation, and also can be seen in other intra-articular hip pathologies such as labral tears or chondral injuries. Both FADIR and FABER tests may elicit pain with associated tight musculature, so it is important to distinguish the specific location of pain reproduced. The posterior impingement test is performed in the supine position with the affected hip unsupported by the table. The affected leg is placed in hip extension and external rotation and overpressure applied into extension. A positive test provokes anterior hip pain [6]. The fulcrum test is positive when gentle pressure on the dorsal knee in the seated position with lower legs dangling produces increased discomfort in the thigh and is suspicious for stress fracture. The hop test in positive when one-legged hop produces pain at the suspected fracture site [7]. Hip instability is another source of hip pain and should be suspected with signs of hypermobility which include anterior hip pain with the hip externally rotated in extension or an elevated Beighton Score [8]. It is important to note that back or spine pain can be referred to the hips and should be evaluated as a potential etiology of hip pain as well.

In young children, children with serious cognitive impairment or children with behavioral problems, evaluation of strength is largely observational, assessing the ease of performance and symmetry of movement. This can be evaluated by watching the child explore their environment and encouraged by parent participation, incorporating play and strategic placement of toys to assess squatting, rising from seated, crawling, walking, or climbing. After school age, children are typically able to cooperate with standard manual muscle testing [9].

Imaging of the infant hip involves ultrasound up to 6 months of age when the hip is primarily composed of cartilage and soft

tissue. Imaging after ossification occurs around 3–6 months of age typically starts with plain radiographs. Common radiograph views of the pelvis may include an anteroposterior (AP) view, a lateral view, a false profile view, a frog-leg view, or a Dunn view depending on the differential diagnosis. Advanced imaging may be useful for more detailed examination of bony and soft tissue structures and for preoperative planning. MRI is useful to visualize soft tissues, bony edema, osteochondral injuries, and osteonecrosis. When compared with radiographs, computed tomography (CT) scan with three-dimensional (3D) reconstructions can achieve improved bony anatomy visualization but should be completed with a low-dose protocol to limit radiation exposure [8].

Developmental Conditions of the Hip

Developmental Dysplasia of the Hip

What Is the Pathology?

Developmental dysplasia of the hip (DDH) describes a spectrum of disorders involving abnormal development of the femoral head and acetabulum. It is present from birth and is a gradual and progressive disorder with distinct anatomic changes which initially are largely reversible. Incidence estimates range from 1 per 1000 to 3.4 per 100 live births depending on the source. DDH is four times more common in females than males and occurs twice as often in the left hip as compared to the right. Predisposing factors include ligamentous laxity, prenatal breech position (especially footling), postnatal positioning (hips swaddled in extension), and racial predilection (particularly Navajo Native American and Whites). It is also associated with intrauterine crowding as seen in first-born children or pregnancies complicated by oligohydramnios. These children may also have other postural abnormalities including torticollis or metatarsus adductus. Children with neuromuscular disorders are also at higher risk of DDH due to muscle imbalance and variation in weight-bearing abilities [10, 11].

DDH is commonly caused by excessive laxity in the hip capsule resulting in inability to maintain the femoral head in the

acetabulum. Subtle increases in muscle tone or imbalance, most notably involving the hip adductors, can also contribute to deformities and deficient positioning of the femur in the acetabulum. The femur can be partially or fully displaced, often resulting in poor development of acetabular coverage. Subluxation describes partial displacement of the hip with the femoral head not fully covered by the acetabulum. Dislocation describes complete displacement of the hip with the femoral head completely uncovered and out of the acetabulum. Dysplasia refers to the deficient development of the acetabulum and can be present with or without subluxation or dislocation [10].

Clinical Presentation

DDH is diagnosed by a combination of clinical presentation and imaging studies. In the infant, the most reliable finding is decreased hip abduction. Asymmetric thigh folds can be seen although this is not specific for DDH and can be a normal variant. Hip dysplasia may present in older adolescent children with or without a history of DDH, and typically presents as aching pain in the groin or lateral hip, worsened with prolonged walking or standing, and an antalgic gait [1].

Teratologic dislocation of the hip (also known as antenatal dislocation of the hip) is a distinct classification that describes a fixed dislocation at birth with limited range of motion and the inability to reduce the hip. This is usually associated with neuromuscular conditions including arthrogryposis, myelomeningocele, chromosomal abnormalities, diastrophic dwarfism, and lumbosacral agenesis [1].

Physical Exam

Infants may present with a positive Galeazzi sign, a positive Barlow or a positive Ortolani sign, although the reliability of these exams relies heavily on examiner experience. The Klisic test can be used and will be positive in a dislocated hip at any age. In the ambulatory child, an exam is suspicious for dislocation when the hip is positioned in slight flexion, adduction (due to shortened adductors), and internal rotation. Other concerning physical exam findings include leg length discrepancy or an abnormal gait. Gait deviations may include ipsilateral toe walking, antalgic gait, Trendelenburg gait, or increased lumbar lordosis due to hip flex-

ion contracture. The hip may also demonstrate excessive internal or external rotation [10].

All infants should undergo screening for hip instability with physical exam [1]. The American Academy of Pediatrics recommends imaging prior to 6 months of age for all infants who were breech in the third trimester or have symptoms that raise parental concern, inconclusive or positive finding on physical exam as described above, family history of DDH, or history of being swaddled improperly [12].

Diagnostic Studies

Ultrasonography is used primarily in the neonate and young infants and plain radiographs thereafter [1]. When using ultrasonography, the Graf technique is the primary tool for stratification of risk [12]. Angles created by anatomic structures of the hip are used to classify the acetabular morphology and femoral head coverage and guide the appropriate treatment approach [13, 14]. With plain radiographs, there are several classic lines that can be helpful in evaluating the position of the immature hip (Fig. 6.2) [15].

Radiographic studies in the adolescent with hip dysplasia commonly reveal degenerative changes in the form of bony sclerosis, cyst formation, and joint space narrowing [1]. MRI can provide detailed anatomic visualization although is not routinely used due to relative expense and the need for sedation in young children.

Treatment(s)

Treatment goals are to obtain and maintain positioning of the femur within the acetabulum, avoid damage to the femoral head, and prevent degenerative and functional complications. Long-term complications of untreated DDH can include early arthrosis, osteoarthritis, labral tears, gait abnormality, and pain. In the infant under 6 months of age, the preferred method is using a Pavlik harness. The Pavlik harness positions the hips between 90 and 120° of flexion while preserving the ability to spontaneously abduct and adduct. In the infant over 6 months of age, a dynamic hip abduction orthosis (Visser orthosis) can be used to position the hips in an abducted and flexed position and allows for some mobility. Once a child reaches 1 year of age, a Hilgenreiner orthosis may be used which similarly optimizes positioning of the hips

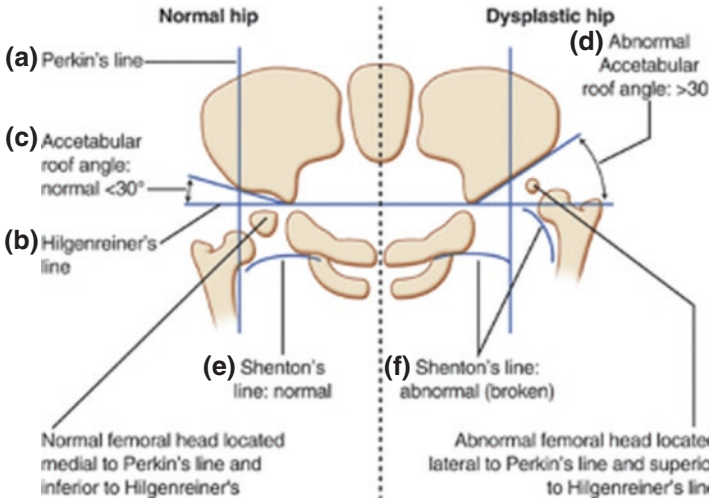

Fig. 6.2 (**a**) Perkins line is a vertical line drawn from the most lateral margin of the roof of the acetabulum. (**b**) Hilgenreiner line is a horizontal line drawn through the upper margin of the triradiate cartilage and perpendicular to the Perkins line. In an intact hip, the lesser trochanter lies medial to the Perkins line and inferior to the Hilgenreiner line. In the subluxed hip, the lesser trochanter lies lateral to the Perkins line and in the dislocated hip, the lesser trochanter lies lateral to the Perkins line and superior to the Hilgenreiner line. (**c**) The acetabular index is the angle formed by the bisection of a line along the acetabular roof and the Hilgenreiner line, which in the typical hip should be less than 30°, and in the dysplastic hip (**d**) will be greater than 30°. (**e**) In the intact hip, Shenton's line is a smooth, continuous arc between the inferior border of the superior pubic ramus and the inferomedial border of the femoral neck; (**f**) disruption suggests hip dysplasia including hip subluxation or dislocation [11, 15]

but also allows the child to walk [10]. Complications associated with bracing include failure to reduce the hip, avascular necrosis (AVN), and femoral nerve palsy. Increased rates of complications are associated with forced positioning, especially wide abduction and internal rotation and these should be avoided. Regular clinical and radiographic assessment should be used for ongoing assessment of reduction and stability. If the hip remains reduced, the child is serially monitored at 6 months of age, 1 year old and annually to biennially to the point of skeletal maturity to ensure no further complications occurs [1].

By 6 months of age, 80–90% of children treated with the Pavlik harness or hip abduction orthosis have successful repositioning. Those who do not may require closed reduction. If this is unsuccessful, open reduction, sometimes accompanied by femoral shortening or acetabular reorienting innominate osteotomy, may be necessary [10]. Surgical interventions are typically not performed past 9–10 years of age for unilateral dislocation and 8 years of age for bilateral dislocation. If the hip is reduced before 4 years of age, the acetabulum gradually remodels, most hips fail to have complete development and the acetabulum remains shallow. Dysplastic and subluxed hips usually become painful due to inherent instability with rapid progression of degenerative changes as early as adolescence and into adulthood [1].

Treatment options of dysplasia in the adolescent include a variety of reconstructive pelvic osteotomies, but if the hip is asymptomatic or not causing significant impairment, surgery may not be considered to preserve functional outcome. Untreated unilateral hip dislocations can lead to limb length discrepancy, impaired range of motion, ipsilateral valgus knee deformity, gait abnormality, and postural scoliosis. Bilateral hip dislocations can cause significant back pain due to excessing lumbar lordosis [1].

Treatment of most children with teratologic dysplasia includes reduction (open vs closed). Prior to surgical intervention, functional level and prognosis for ambulation should be considered. If the child has low functional levels or high paralytic levels, these may best be left untreated [1].

Inflammatory and Infectious Conditions of the Hip

Transient Synovitis

What Is the Pathology?

Transient synovitis is the most common cause of acute hip pain in children aged 3–10 years. Boys are affected two to three times more frequently than girls, and the right hip more frequently than the left. It sometimes follows a recent viral upper respiratory infection, though the exact cause remains unknown [12].

Clinical Presentation

Transient synovitis is characterized by a rapid onset pain in the groin, which may radiate to the thigh or knee, and irregular gait pattern with refusal to weight bear. About 15% of the time, pain is only in the thigh or knee. It may be accompanied by fever and there is often no history of preceding injury [10, 16].

Physical Exam

The hip may present in a preferred flexed, abducted, externally rotated position. On physical exam, there is a positive log roll test and limited range of motion, particularly internal rotation [10].

Diagnostic Studies

Diagnosis can be made by a combination of clinical presentation, laboratory evaluation, and imaging to exclude other etiologies with similar presentation, specifically septic arthritis, osteomyelitis, avulsion fracture around the pelvis, or trauma. Plain radiographs and ultrasonographic images may show variable joint effusion and joint space widening [16]. In contrast to septic arthritis as detailed below, there is no bone marrow edema. Lab values include a slightly elevated ESR/CRP and negative joint aspiration although this must be performed if there is any suspicion for septic arthritis (Table 6.1) [10, 12].

Table 6.1 Transient synovitis and septic arthritis

	Transient synovitis	Septic arthritis
Age	3–10 years old	Younger than 3 years old
Presentation	Atraumatic acute hip pain with refusal to bear weight, limited ROM	Atraumatic acute hip pain, fever; frequently associated with underlying osteomyelitis
Laboratory investigations	Slightly elevated ESR/CRP, normal to slightly elevate serum WBC count	ESR at least 40 mm/h, elevated CRP, serum WBC count >12,000/mm^3
Imaging	Plain radiographs/MRI—variable effusion/joint space widening	Plain radiographs—normal MRI—signal intensity in bone marrow
Treatment	Self-limited, treated symptomatically with NSAIDs	IV antibiotics, surgical irrigation/debridement

Treatment(s)

The condition is self-limiting with resolution of symptoms within 2 weeks with bed rest followed by gradual increase in activity as tolerated. Non-steroidal anti-inflammatory drugs (NSAIDs) can be used for symptomatic relief [16]. Unilateral transient synovitis often self-resolves without long-term sequelae. Recurrence occurs in 4–20% of cases depending on the source and course remains benign without progression to chronic orthopedic condition [17]. It is important to note that transient synovitis can present similarly to septic arthritis which requires prompt treatment to avoid irreversible damage.

Septic Arthritis

What Is the Pathology?

Septic arthritis describes a painful infection within the synovial fluid and tissues of the hip joint. This can be due to direct inoculation (traumatic or post-surgical), hematogenous seeding, or extension from adjacent osteomyelitis. The majority have no organism identified. When identified, the most common causative organisms are *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Staphylococcus epidermidis*, and *Kingella kingae* [18]. *Group B streptococcus* is prevalent in the neonatal period and *Neisseria gonorrhea* in the adolescent population [19].

Clinical Presentation

Septic arthritis usually presents as an atraumatic, acute hip pain in children younger than 3 years of age and is often associated with underlying osteomyelitis. These children classically present with a sudden onset fever and limp or refusal to bear weight [18].

Physical Exam

On physical exam, the hip rests in a flexed, abducted, and externally rotated position. There may be localized swelling, effusion, tenderness, and warmth. There is severe pain with log roll and passive ROM [18].

Diagnostic Studies

The four main clinical predictors of septic arthritis are fever, refusal to bear weight, elevated ESR at least 40 mm/h and elevated serum WBC count more than 12,000/mm^3. It is important to note that the value of these predictors diminishes with the decreasing prevalence which varies among clinical practice setting (primary referral general hospital vs tertiary referral center). CRP can also be helpful for early diagnosis within 6–8 h as the levels rise more quickly than ESR [18]. Nonetheless, fever is the most significant and reliable predictor despite clinical setting [19].

Plain radiographs are usually normal. MRI may reveal a variably sized effusion, synovial thickening/enhancement, and signal changes in bone marrow and pericapsular soft tissues. Intra-articular aspiration may be used to isolate the causative organism to guide treatment [12].

Treatment(s)

Treatment includes intravenous antibiotics in addition to operative irrigation and debridement [18]. Septic arthritis must be diagnosed and treated quickly to avoid lytic destruction of the articular cartilage causing irreversible damage [12].

Osteomyelitis

What Is the Pathology?

Acute hematogenous osteomyelitis most commonly affects long bones but can occur in the pelvis in about 9% of children. It affect boys more frequently than girls, typically around 9 years old [20].

Clinical Presentation

It can be a challenge to diagnose due to its nonspecific clinical presentation. It is extremely painful and is associated with low-grade fever, and normal to slight elevation in serum WBC and elevated ESR/CRP [10]. Children often present with refusal to walk [20].

Physical Exam

Children will prefer to hold their hip in a fixed position. There may be a positive log roll and decreased active and passive range of motion with external rotation and full extension particularly painful [20].

Diagnostic Studies

Bone or joint aspiration and blood cultures can be performed with most common causative organism being *Staphylococcal aureus* although there have also been reports of *Group A streptococcus*, *Streptococcus pneumoniae*, *Fusobacterium,* or *Salmonella.* Plain radiographs may be normal and remain normal up to 1 month but are helpful in initial evaluation to rule out traumatic or malignant lesions. Early use of technetium bone scintigraphy is a very effective tool for identifying the exact location of a lesion and MRI can be helpful in assessing the extent and quality of inflammation [20].

Treatment(s)

Treatment is with antibiotic therapy and in severe cases may require surgical debridement and irrigation [20].

Juvenile Idiopathic Arthritis

What Is the Pathology?

Juvenile idiopathic arthritis (JIA) includes all forms of arthritis of one or more joints that develop before the age of 16 years and persists for at least 6 weeks without an identified cause [12, 16]. Incidence is 1.6–23 cases per 100,000 per year. There is a bimodal distribution of age of onset between 1 and 3 years and between 9 and 11 years. It is more common in girls. Etiology is multifactorial with contributions from immune-mediated processes, hereditary involvement, and postulated postinfectious or posttraumatic considerations [16]. It is a degenerative process beginning with synovial inflammation and hypertrophy evolving to effusion, synovitis, tendinitis, and bursitis which can progress to varying

degrees of osseocartilaginous destruction, degenerative changes, ankylosis, or malalignment. It can be classified into three types—oligoarticular, polyarticular, and systemic [12].

Clinical Presentation

Generally, presentation is characterized by morning stiffness with symptoms decreasing throughout the day. In the oligoarticular subtype, less than five joints are affected, which is accompanied by persistent low-grade inflammation of involved joints and a slight decrease in range of motion. Involvement of the hip is rare. Remission is common and the overall course is generally benign compared to other subtypes. It may be accompanied by uveitis or iritis in which most cases are asymptomatic, so referral and early evaluation by ophthalmology is essential. In the polyarticular subtype, there are five or more joints involved within the first 6 months. Large joints of the lower extremity, including the hip, are commonly involved and overall has varying prognosis, with RF positivity associated with poorer prognosis [16]. The systemic subtype presents with pronounced febrile illness and characteristic macular rash. There is early involvement of the knee, ankle, and elbow with late hip involvement in 20–40% of cases. Classically, systemic JIA was associated with very poor prognosis although the advent of targeted biologic pharmacologic interventions has made long-term remission possible [12, 21].

Physical Exam

Presentation is variable depending on the specific diagnosis as above. It will often involve several joints and be marked by the presence of joint swelling and erythema and tenderness with restriction in ROM [22].

Diagnostic Studies

JIA is a diagnosis of exclusion with no specific confirmatory laboratory tests. Joint fluid analysis is useful to exclude infection [16]. Ultrasound can be useful to detect effusion, synovitis, tenosynovitis, cartilage thickness, erosion and guide intra-articular injections. MRI with contrast is the most sensitive for detecting synovitis [12].

Treatment(s)

Treatment is best achieved with a multidisciplinary team involving rheumatology, orthopedic surgery, ophthalmology, physical therapy, occupational therapy, nursing, and social work. Specific treatment varies depending on subtype. Pharmacologic intervention begins with NSAIDs for an anti-inflammatory effect and can progress to intra-articular corticosteroid injection, DMARDs, or biologics as appropriate. Systemic corticosteroid can be used for acute, life-threatening flairs of disease, but are not used as long-term therapy to avoid iatrogenic Cushing syndrome. Physical and occupational therapy are vital for both conditioning training and to reduce pain, increase range of motion, and improve muscular coordination. In an acute flair, isometric exercises are preferred to maintain muscle strength. When not in an acute flair, aerobic and low-impact activities should be encouraged and isotonic exercises are helpful to improve muscle strength. In the case of severe contracture, surgical intervention may be warranted for synovectomy, contracture release, or total joint arthroplasty [16].

Chondrolysis

What Is the Pathology?

Idiopathic chondrolysis is marked by the progressive loss of articular cartilage in the hip. This is most seen in preadolescents and adolescents, with females more frequently affected than males. Etiology remains unclear, but it is likely immune-mediated [12]. Acquired chondrolysis can be secondary to slipped capital femoral epiphysis, juvenile idiopathic arthritis, infection, trauma, or prolonged spica cast immobilization [10].

Clinical Presentation

Adolescents typically present with restriction of motion in all directions and a painful, stiff hip. They may also have an antalgic gait [12].

Physical Exam

Pain is generally in the anteromedial hip and may also be in the groin, inguinal region, thigh, or knee with no obvious inflammation or tenderness on palpation. There is significant reduction in range of motion in all directions [10].

Diagnostic Studies

Plain radiographs of the hip can show joint space narrowing without osteophytes, blurring of the subchondral line, osteopenia, protrusion acetabula, subchondral cyst, growth plate narrowing, or premature growth plate closure [12].

Treatment(s)

Treatment includes restricted weight bearing, anti-inflammatory and analgesics, range of motion, and surgery if recalcitrant. Surgical intervention may involve a subtotal capsulectomy with muscle release or hip arthroplasty although results are variable [10, 12].

Vascular Conditions of the Hip

Avascular Necrosis

What Is the Pathology?

Avascular necrosis occurs when there is diminished vascular supply to the femur leading to collapse and degeneration of the underlying bone causing hip pain. Peak incidence is 5–6 years old with boys more commonly affected than girls. It can be caused by traumatic hip dislocation, treatment of developmental dysplasia of the hip, sequelae of slipped capital femoral epiphysis, septic arthritis, hemoglobinopathies including sickle cell disease, leukemias, steroid medication, or idiopathic (Legg-Calve-Perthes disease) [23, 24]. It is characterized by four stages: necrotic (avascular) stage, fragmentation stage, re-ossification (revascularization) stage and the reparative (remodeling) stage [12].

Clinical Presentation

It is typically unilateral, however about 15% of cases have asynchronous bilateral involvement [12]. Children present with an antalgic gait and pain and stiffness which may radiate to the groin and is worsened by activity and relieved by rest [12].

Physical Exam

Physical exam findings are often nonspecific. There may be hip or knee pain with associated global restriction in range of motion and an antalgic gait [12].

Diagnostic Studies

Initial plain radiographs may be normal. Early in the clinical course, a bone scan may show decreased perfusion to the femoral head and an MRI may show marrow changes or necrosis involving the central subchondral epiphysis, and less commonly the medial or lateral epiphysis. Later in the clinical course, radiographs may show early sclerosis of the femoral head followed by collapse and/or subchondral fracture leading to crescentic lucency ("crescent sign"), then subsequent fragmentation. Healing may look normal, or lead to coxa magna (wide head) or coxa breva (short neck) [12].

Treatment(s)

Of those with avascular necrosis, 60–70% heal spontaneously without functional impairment. The goal of treatment is to preserve normal morphology of the femoral head and prevent irreversible damage, thus decreasing risk of early development of osteoarthrosis. This includes use of anti-inflammatory medications and physical therapy for strengthening and gait mechanics. In severe cases, surgical intervention may be considered. Loss of containment of the femoral head within the acetabulum or femoral collapse portend poor prognosis [12].

Legg-Calve-Perthes Disease

What Is the Pathology?

Legg-Calve-Perthes disease is a form of idiopathic avascular necrosis in the pediatric hip, where the blood supply to the femoral head is disrupted resulting in variable degree of necrosis and disruption of bone growth. It can occur in children from 18 months through 14 years of age, is four to five times more prevalent between the ages 4 and 8 and is more common in boys than girls. Etiology remains unclear; many theories exist involving systemic susceptibility, trauma, coagulopathy, collagenopathy, and the double infarction theory with less emphasis on hereditary or environmental factors. There is an increase in serum inflammatory cytokines due to ischemia and necrosis which leads to a chronic inflammatory process. Ischemic necrosis causes trabecular fracture and microdamage which contributes to an imbalance in bone resorption and formation and overall decreased mechanical strength leading to the development of femoral head deformity [24].

Clinical Presentation

Children complain of mild to moderate pain in the groin which may radiate to the thigh or knee which is exacerbated by activity and relieved by rest [10]. In about 15% of cases, initial presentation may be isolated to the thigh or knee pain [24].

Physical Exam

Physical exam is notable for an antalgic gait (sometimes with positive Trendelenburg sign), decreased range of motion, and a positive log roll test. There may be a varying degree of gluteus, quadricep, and hamstring atrophy depending on the severity and chronicity of symptoms and range of motion impairment as the disease progresses. Hip synovitis is common in the initial stages, contributing to pain and loss of range of motion and can persist up to 1 year [24].

Diagnostic Studies

Waldenstrom created a widely used classification system of disease progression based on radiographic changes organized into four stages [24]. Additionally, there are several classification systems used to describe the severity including the Catterall classification, Salter and Thomas classification, and lateral pillar classification systems [10]. MRI with perfusion/diffusion modalities can provide early reliable diagnosis due to enhanced visualization of the femoral head and acetabulum. CT may be useful in the later stages of disease to evaluate mechanical symptoms and differentiate between incomplete reossification and true osteochondrotic lesions [24].

Treatment(s)

Most approaches, both nonoperative and operative, are based on the concept of containment of the femoral head within the acetabulum to influence remodeling of the femoral head in a functionally favorable manner. Management begins with symptomatic treatment involving avoidance of high-impact activities, relative rest, and short-term use of NSAIDs. It may also include abduction bracing, Petrie casting, femoral osteotomy, or innominate osteotomy and acetabular shelf procedure [24]. However, treatment options are controversial due to unclear differences in clinical outcomes between children treated with an abduction orthoses, physical therapy, or surgery [10].

Prognosis is influenced by age of disease onset and duration of symptoms. Children less than 6 years of age generally have a favorable prognosis despite severity due to increased time for remodeling during growth. After 8 years of age, prognosis decreases drastically. Involvement of less than 50% of the femoral head portends a good prognosis. There is a higher susceptibility to future development of hip pain and loss of hip mobility due to FAI and acetabular dysplasia with instability [24]. Long term, this results in early arthrosis and minimal to severe deformation [10]. The most important factor for prediction of long-term outcome is the degree of femoral head reshaping and its congruency with the acetabulum [24].

Musculoskeletal Conditions of the Hip

Slipped Capital Femoral Epiphysis

What Is the Pathology?

Slipped capital femoral epiphysis (SCFE) commonly occurs in adolescents, frequently corresponding to the upswing in growth velocity. Boys are affected twice as frequently as girls. Obesity is a primary risk factor for the disorder, with approximately 80% of affected children having BMI above the 95th percentile. SCFE occurs when stress applied to the femoral head exceeds the strength of the capital femoral physis, causing the capital femoral epiphysis to displace through the physis from the femoral neck (growth plate). The common path of displacement of the proximal femoral metaphysis is anterolateral and superior, resulting in the epiphysis appearing to be displaced posteriorly and inferiorly. In actuality, the epiphysis remains in a normal position within the acetabulum, making the name "slipped capital femoral epiphysis" a bit of a misnomer [25].

Clinical Presentation

Children with SCFE classically present with a unilateral, non-radiating, dull ache located deep in the groin or along the distal medial thigh and knee that is accentuated by walking, running, jumping, or pivoting activities. This is accompanied by a classic external rotation of the leg and alteration of gait including an antalgic gait or Trendelenburg gait [25]. Children with stable SCFE will present with the ability to bear weight on the affected extremity, with or without crutches, whereas those with unstable SCFE are unable to bear weight on the affected side. Bilateral hip pain associated with SCFE rarely occurs. SCFE should be especially considered in a child younger than 10 years old or with metabolic abnormalities [26].

Physical Exam

Observation of gait in the ambulatory child is a useful aspect of clinical examination [27]. A loss of internal rotation of the affected

hip is usually seen and attempts to bring the hip into internal rotation causes pain. This is most accurately assessed with the patient in the prone position. When compared to the contralateral side (if unaffected), internal rotation may be decreased, and in some cases, absent. Diminished hip flexion is often observed relative to the contralateral side, and typically produces pain before full flexion is achieved. When examining hip flexion, most SCFEs result in some degree of obligate external rotation especially with attempts to passively flex the hip. Decreased hip abduction, apparent shortening of the affected extremity, or flexion contracture of the hip can also be seen [25, 27]. If symptoms have been longstanding, atrophy of the thigh can also be observed [25]. Any adolescent with decreased hip internal rotation and out-toeing (especially if asymmetric), with or without pain, should be presumed to have an SCFE until proven otherwise [27].

Diagnostic Studies

At minimum, plain radiographs with an AP view of the pelvis and a frog lateral view of both hips within one film should be obtained. Imaging may reveal a widened growth plate with maligned epiphysis and the epiphysis slipping posteriorly along the anterior neck [28].

SCFE can be classified in terms of duration of symptoms (temporal classification) and percentage of the displacement (SCFE grading system), stability based on weightbearing status (Loder Classification), or by the epiphyseal-diaphyseal angle (Southwick Slip Angle Classification) [29]. The duration classification may be acute (<3 weeks) or chronic (>3 weeks) and may have a clear inciting event [26]. According to the SCFE grading system, mild slips demonstrate displacement up to one third of the metaphyseal width, moderate slips occupy from one third to one half, and severe slips reveal a slippage of greater than one half of the metaphyseal width [29]. By Loder Classification, SCFE with the ability to bear weight with or without crutches are classified as stable; complete inability to bear weight classifies the SCFE as unstable and is associated with a high risk of osteonecrosis (10–60%) [26]. By the Southwick Slip Angle Classification, an epiphyseal-diaphyseal

angle of less than 30° is considered mild, 30–60° is considered moderate, and greater than 60° is considered severe [30].

To standardize the assessment of slip severity in chronic slips, CT may be useful; however, it has not been shown to be superior to plain radiographs [31, 32]. MRI is more sensitive in detecting early, radiographically occult but symptomatic pre-slips [33]. Ultrasonography can also demonstrate characteristic findings [28].

Treatment(s)

Stabilization of chronic or stable slips is commonly treated with the use of a single cannulated screw placed in the center of the epiphysis. A single screw decreases the risk of osteonecrosis. In unstable slips, a gentle reduction is achieved followed by placement of two screws in the epiphysis [28]. Contralateral fixation is considered for patients at high risk for slippage, including those with age younger than 10 years, unreliable follow-up, metabolic abnormality, or extreme obesity [26]. Severe, chronic slips may be treated with proximal femoral osteotomies, hip capsule decompression, or epiphyseal reduction with pinning [26].

Return to Activities

Following surgery, gentle range of motion can be initiated within a few days, followed by protected weight bearing for 6–8 weeks. Stable slips can bear weight after fixation whereas unstable slips are typically maintained at touch-down weightbearing status. This is followed by gradual progressive strengthening and functional exercises [34]. The time frame for return to play is variable and established by the orthopedic surgeon. In general, return to advanced activities including sports can be achieved once full strength is regained and children can participate and perform all functional skills required for their particular sport without pain [34].

Avulsion Fracture

What Is the Pathology?

Avulsion injuries of the pelvis occur most often in the adolescent athlete. Due to the inherent weakness across the unfused apophysis of the skeletally immature child, the unfused apophysis is displaced

at the site of tendon attachment. Injuries typically occur from a violent forceful contraction of the muscle. Chronic repetitive stress or direct trauma may also place the growth plate of a apophysis at risk for an acute injury [35]. The most common location for apophyseal avulsion injury in the pelvis is the ischial tuberosity. This injury usually results from a forceful contraction of the hamstring muscles, most commonly during sprinting or with excessive passive lengthening of the hamstring muscles. Injury to the anterior superior iliac spine and anterior inferior iliac spine typically occurs during forceful extension of the hip with the knee in flexion. The majority of iliac crest avulsions result from repetitive trauma but may also result from direct trauma to the iliac crest. Avulsion injuries at the pubic symphysis are rarely from acute injuries and can be seen with chronic repetitive stress from the hip adductors or from turning movements of the abdomen and excessive twisting, commonly seen in tennis, soccer, and ice hockey. Avulsion injury of the lesser trochanter arise from forceful contraction of the iliopsoas muscle while the thigh is fixed in an extended position [35].

Clinical Presentation

In general, injuries are associated with point tenderness, focal swelling, and difficulty walking. Injuries to the ischial tuberosity may demonstrate a popping sensation at the time of injury and typically manifest with severe posterior thigh or gluteal pain. With injury to the anterior superior iliac spine, anterior inferior iliac spine or iliac crest, point tenderness, and swelling directly overlying the site of avulsion is present [35]. Children tend to prefer a position which relieves the tension of the involved muscle group and decreases their pain [36].

Physical Exam

Physical examination reveals local tenderness with palpation at the site of injury and possibly edema and ecchymosis. The pain at the apophysis may worsen with resisted muscle contraction or passive stretching of the muscle attached to the apophysis [8].

Diagnostic Studies

Plain radiographs are usually sufficient to diagnose an apophyseal avulsion injury [35]. In the early setting, if radiographs are inde-

terminate, MRI can be useful in detecting minimally displaced or even nondisplaced apophyseal injuries. Fluid or edema can be identified within the cartilage growth plate of the apophysis, and edema is often visualized along the proximal tendon in proximity to the level of attachment. MRI of a chronic lesion may reveal a prominent osseous attachment site, thickening of the tendon at the level of attachment, and callus or heterotopic bone formation. Ultrasound can also be used to identify avulsion injuries of the anterior superior and anterior inferior iliac spine [35].

Treatment(s)

The majority of apophyseal avulsion injuries are treated with a conservative approach of rest of the involved muscle and ice for symptomatic relief. Surgery is rarely required but has been reported in competitive athletes to prevent functional disability. Some advocate for reduction and fixation if the avulsed fracture fragment is greater than 2 cm in diameter. The optimal timing of surgical intervention remains controversial [35].

Return to Activities

Return to full weight bearing and passive range of motion occurs gradually. Full strength, full range of motion, and the ability to perform all functional skills related to the athlete's sport without pain should be regained before full competitive activity is resumed [35].

Femoroacetabular Impingement

What Is Pathology?

Femoroacetabular impingement (FAI) is a condition where variant morphology predisposes the hip to premature joint degeneration. This morphology is classified as cam, pincer, or mixed. Cam morphology describes a loss of sphericity of the femoral head, pincer describes an acetabulum with excessive coverage of the femoral head, and mixed morphology is a combination of the two deformities. These hip morphologies cause the femoral neck to abut against the acetabular rim during functional range of motion

with resultant damage to the labrum, delamination of the adjacent acetabular cartilage, and over time, secondary osteoarthritis [37].

Insufficiency of the femoral-neck concavity is the hallmark of cam impingement. The eccentric part slides into the anterosuperior acetabulum during flexion, causing compression and shear stresses at the junction between the labrum and the cartilage and at the subchondral tidemark. The labrum and cartilage are separated as the labrum is stretched and pushed outwards and the cartilage is compressed and pushed centrally. In pincer impingement, the dominant feature is a deep socket, which limits the range of movement of the hip due to the overcovering acetabular rim. The narrow band of labrum along the acetabular rim receives the force, and repeated microtrauma induces bone growth at the base of the labrum, which subsequently ossifies [38].

Clinical Presentation

The primary symptom of FAI syndrome is pain, varying in its location, nature, severity, radiation pattern, and precipitating factors. Pain has an insidious onset, gradually worsens, and is commonly located in the groin (83%), but is also reported in the lateral hip, anterior thigh, buttock, knee, lower back, lateral and posterior thigh [38, 39]. It is typically motion-related or position-related, notably with limitations in activities that involve squatting or assuming a crouched position [34, 40].

Physical Exam

In the majority of children, examination demonstrates reduced hip flexion and a positive provocative testing including the anterior impingement test (FADIR), FABER test, resisted straight leg raise, log roll test, and posterior impingement test [38, 41].

Diagnostic Studies

In FAI, imaging is used to evaluate the hip for abnormalities associated with impingement and to exclude arthritis, avascular necrosis, or other joint problems. Plain radiographs including an AP pelvic view and an axial cross-table view of the proximal femur are most useful. A Dunn/Rippstein view, preferably in 45° of hip flexion, can be obtained to reveal pathomorphologies of the ante-

rior femoral head–neck junction, and can serve as an alternative to the axial view. Labral tears, cartilage damage, and other pathologic signs of internal hip derangement can be confirmed or excluded via MRI or MR arthrography [34, 42].

Treatment(s)

Nonoperative management of FAI syndrome with physical therapy, pain management, and activity modification, in combination with intra-articular injections is effective in most adolescent patients [43, 44]. Surgical intervention via arthroscopic or open surgery can be considered in those who fail conservative treatment. The goal of surgery is to correct hip morphology and achieve impingement-free motion. Interventions can include reshaping the cam morphology, adjusting femoral torsion or neck angle, reorienting the acetabulum, or trimming the acetabular rim. If the labrum or articular cartilage is damaged, it can be resected, repaired, or reconstructed. Preexisting advanced osteoarthritis is the strongest predictor of poor outcome following FAI surgery [34]. There is no evidence that treating people with painless cam or pincer morphology will alter the risk of them developing FAI syndrome or osteoarthritis, so surgery is rarely indicated in these populations [38]. There is increasing popularity in regenerative medicine techniques as a treatment option, but more research is needed before recommendations can be made.

Stress Fractures

What Is the Pathology?

A stress fracture is described as bony microtrauma with accelerated bony remodeling in response to repetitive submaximal stress. The process of stress fractures occurs on a physiological and clinical continuum. Given this range of injury, it is helpful to distinguish earlier responses, termed *stress reactions*, from more advanced injuries, termed *stress fractures*, in which a distinct fracture line is evident on imaging. The ischiopubic ramus, oftentimes in the inferior pubic ramus adjacent to the symphysis, is the most common location of stress fractures in the pelvis. Femoral

neck stress fractures account for approximately 11% of stress fractures in young athletes. Etiology is likely multifactorial owing to the increased load after fatigue of supporting structures and contractile muscular forces acting across and on the bone. It is important to note that those with nutritional deficiencies and relative energy deficiency syndrome, characterized by changes in bone mineral density, low energy availability, and menstrual dysfunction, are at heightened risk for stress fractures. A thorough inquiry including history of menses, nutrition, exercise program, and fatigue is essential to screen for those at high risk [45].

Clinical Presentation

Typical history of a stress fracture includes localized pain that emerges after or toward the end of physical activity. Untreated stress reactions exhibit pain that occurs earlier during the physical activity and persists longer; with continued training, pain will linger throughout the training and carry on into daily ambulation [45]. Pelvic stress fractures can be mistaken for an adductor strain or osteitis pubis. Stress fractures mimic sacroiliitis in that they occur in the sacrum and usually present with unilateral pelvic pain localized to the sacroiliac joint area. Consider femoral stress fractures in any athlete, especially a distance runner who presents with hip, thigh, or groin pain. Symptoms are aggravated with weight bearing, and there is commonly reduced range of movement in the hip, particularly internal rotation [45].

Physical Exam

Direct palpation is a reliable physical exam technique, typically revealing local tenderness over the involved bone. Other useful, but not as reliable clinical tests include the hop test, fulcrum test, and Stork test to assess for fractures of the tibia, femur, and pars interarticularis, respectively [45].

Diagnostic Studies

Radiographic findings are typically seen after 2–8 weeks of symptoms. In the early stages of these injuries, the sensitivity of radiography may be as low as 10% and at follow-up, 30–70%. In the early phases of bone stress injuries, MRI may show signs of frac-

ture or callus that are not clearly depicted by plain radiographs. A focal region of periosteal bone formation is the most common sign in early stress fractures. The "gray cortex" sign, a cortical area of decreased density, may also be seen. Radionuclide scanning is a more sensitive but less specific method for imaging bony stress injuries [45].

Treatment(s)

Fractures are classified as high risk, medium risk, and low risk based on location. Medium-risk fractures are seen in the pelvis and high-risk fractures in the femoral neck. Medium- and low-risk fractures can be treated with a two-phase protocol. Phase 1 involves a multi-modal pain approach with local physiotherapy, non-steroidal anti-inflammatory medication, ice massage, and physical therapy modalities. Weight bearing is permitted as tolerated and the offending activity is discontinued. If the athlete is unable to ambulate pain-free, immobilization (i.e., a walking boot) should be temporarily utilized. A tailored activity program is designed to maintain strength and fitness but reduce impact loading to the skeleton. Phase 2 begins when the athlete is pain-free for 10–14 days and includes the gradual return to sport. The length of time depends on various factors, including the severity and chronicity of the condition and premorbid functional level of the athlete [45]. High-risk fractures of the femoral neck on the medial (compression) side can be treated conservatively while fractures on the lateral (tension) side require surgical referral and intervention due to high risk of delayed union, non-union, or displacement [34]. Surgical management of a stress fracture is also considered when there is failure of nonoperative management [45].

Return to Activities

In general, the level of pain guides the progression of conservative management from non-weight bearing to weight bearing and usually last 4–6 weeks [34]. An athlete can return to running 1 week following the resolution of focal bony tenderness, beginning at half their usual pace and distance, running only every other day for the first 2 weeks. A gradual increase in distance and frequency

is allowed over a 3–6-week period. Once they can run the distance required for training, the pace may be increased [45].

Coxa Saltans (Snapping Hip)

What Is the Pathology

The causes of coxa saltans can be classified as extra-articular (external or internal) and intra-articular. Extra-articular is more common than intra-articular, with external type being the most common. The extra-articular causes are further detailed in this section. The external type involves the iliotibial tract sliding over the greater trochanter. Thickening occurs in the posterior part of the iliotibial tract or the anterior border of the gluteus maximus enhancing the snapping. The internal type involves the musculotendinous iliopsoas snapping over the structures located behind it, most commonly the femoral head. Intra-articular lesions may be due to synovial chondromatosis, loose bodies, fracture fragments, and labral tears which may present a diagnostic challenge [46].

Clinical Presentation

In cases of coxa saltans of the internal and external types, the history is fairly diagnostic. Pain is generally the primary complaint, described as a snapping, painful sensation usually in the area of the greater trochanter or the anterior hip. With intra-articular lesions, the sensation is usually described as a clicking sensation instead of snapping [46].

Physical Exam

On exam, when suspicion for the internal type of coxa saltans is high, the examiner can frequently reproduce the snapping by flexing and extending the hip in the supine position. This can further be provoked by flexing and then abducting the hip, followed by extension and adduction. If the snapping results with these motions, the diagnosis can be corroborated by blocking the snapping through applying finger pressure over the iliopsoas tendon at the level of the femoral head. To assess for the external type of coxa saltans, the snapping can be reproduced by active hip flexion

in the side-lying position with the suspected side up. The snapping can be felt by the examiner with hand placed over the greater trochanter region [46].

Diagnostic Studies

Plain radiographs are an essential part of the workup to identify anatomic abnormalities; however, they should not be used in isolation, as there is nothing pathognomonic that will be identified on plain films. CT, MRI, dynamic ultrasound, and iliopsoas bursography can all be used to assist in the diagnosis [47]. Ultrasonography can be useful by directly and non-invasively visualizing the dynamic motion of the iliotibial band across the greater trochanter or assessing for intra-articular causes of snapping hip such as labral tears. MRI lacks this ability to evaluate the tendons dynamically [48].

Treatment(s)

Many children experience infrequent, benign snapping that does not require treatment. Intermittent external or internal snapping with initial onset within 6 months is best treated with rest and avoidance of triggering activities. If painful snapping is present with routine activities, nonoperative treatment may include rest and corticosteroid injection followed by a careful exercise program with stretching of the involved muscles. The vast majority of children with a symptomatic snapping hip improve with conservative therapy and regain normal use of the hip within 6–12 months under a controlled program. It is subsequently important to avoid repetitive snapping which may include alterations to the exercise program or sport participation [46].

External type snapping that does not improve with conservative therapy, is exceedingly rare, and may warrant surgery, which commonly involves excision of the greater trochanteric bursa with Z-plasty of the iliotibial band. Painful internal type snapping that is refractory to conservative therapy is also extremely rare. If surgery is required, good results are commonly obtained with lengthening of the posterolateral tendinous portion of the iliopsoas tendon [46].

Traumatic Hip Dislocation

What Is Pathology?

Traumatic hip dislocation in childhood is rare. In adults, they are most commonly posterior and associated with large-force trauma, such as motor vehicle accidents, but can rarely be seen in high-energy impact sports, like American football. In children, they may be seen with far less significant forces. Prompt recognition and reduction of traumatic hip dislocation is key [49, 50]. Posterior dislocation is the most common pattern of hip dislocation in the pediatric population. Owing to the skeletal immaturity in children, hip dislocation is more common than hip fracture. The most concerning complications of hip dislocation include sciatic nerve injury, posttraumatic osteoarthritis, and AVN (secondary to the dislocation causing a tear of the ligamentum teres), which occur in up to 10% of patients [34].

Clinical Presentation

Posterior dislocation usually presents with an alarmingly abnormal extremity position characterized by adduction, flexion, and internal rotation, often with shortening compared to the contralateral leg. This is commonly accompanied by an antalgic gait due to pain [51].

Physical Exam

On examination, ROM or resistance testing is not tolerated due to pain. A complete neurologic examination should be performed to evaluate for lumbosacral plexopathy, sciatic neuropathy, and femoral neuropathy [34].

Diagnostic Studies

Plain radiographs (AP pelvic and a 15° oblique lateral views) are necessary to confirm the diagnosis and assess for associated injuries. Plain films confirm the dislocation, reveal other concomitant injuries, and should be obtained before reduction is attempted and following reduction to confirm anatomic alignment [34, 51].

Treatment(s)

The goal of the treatment is to achieve an anatomic reduction with congruous hip joint surfaces by closed or open reduction. The timing between dislocation and reduction must be as short as possible to limit the risk of necrosis. There is no consensus on the duration of time required for immobilization after reduction, but 6 weeks in traction or in a hip spica cast, followed by immediate weight bearing, has been suggested by many authors [52]. There have been some case reports of patients doing well with non-weight bearing for 4 weeks while following posterior hip precautions (avoidance of hip flexion past 90°, hip adduction past neutral, and internal rotation of the hip) without the use of traction or hip spica, followed by progression of weight bearing and strengthening with physical therapy [49, 50]. If attempts at closed reduction are unsuccessful, surgery is indicated; it may also be required to repair displaced or comminuted fractures and remove intra-articular loose bodies [34]. The onset of gradual, progressive rehabilitation can occur a few days to a couple of weeks following reduction, depending on the patient's comfort level and whether a surgical intervention was performed [34].

Neoplastic and Infiltrative Hip Conditions

Osteoid Osteoma

What Is the Pathology?

Osteoid osteoma is the most common bone tumor of the hip, usually occurring in the femoral neck or shaft but can also occur in the acetabulum [53]. It is a benign, bone-forming tumor, typically seen in children between 4 and 12 years of age and more commonly affecting males.

Clinical Presentation

Presenting symptoms include deep, nocturnal pain in the hip, thigh, or knee that cannot be palpated and is relieved with NSAIDs [10].

Physical Exam

Physical exam often reveals an antalgic gait, decreased range of motion, and positive hip impingement signs [53].

Diagnostic Studies

Plain radiographs demonstrate a characteristic 1–1.5 cm nidus with surrounding sclerosis and cortical thickening. There also may be a valgus deformity of the femoral neck due to growth stimulation. Technetium-99m bone scans and MRI can also be used to visualize the tumor [53].

Treatment(s)

Historically, radiofrequency ablation or open resection procedures were utilized; however, there is growing evidence that osteoid osteomas resolve spontaneously. Symptomatically, these tumors can be treated with aspirin or NSAIDs, to which they are very responsive [53].

Malignancy

Hip pain and associated decreased range of motion and point tenderness have also been reported in several case studies and case series as a presenting sign of other malignancies including neuroblastoma and Ewing sarcoma, so malignancy must remain in the differential diagnosis when assessing hip pain [54, 55].

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Sports Trauma

7

Lyndsey N. Booker, Sara J. Lim,
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Introduction

Epidemiology of Hip Injuries in Sport

Hip injuries are a common complaint in athletes, with estimates of prevalence ranging from 6% to 14% of all sports injuries [1]. Athletes who play hockey, football, soccer, or rugby are at a higher risk due to sudden directional changes, rapid acceleration, and kicking [2]. Several risk factors have been identified, with prior hip/groin injury being the most significant [3]. Additionally, older age and weak hip musculature are important risk factors for hip injuries [3].

Anatomic Structures

The hip joint is a ball and socket joint comprising the acetabulum and femoral head. These structures are supported by the soft tissue components of the hip—the labrum and the joint capsule. The

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labrum is a fibrocartilaginous structure connected to the acetabular rim which stabilizes the joint by providing negative intra-articular pressure [4]. The capsule is composed of three ligaments—the iliofemoral ligament anteriorly, the ischiofemoral ligament posteriorly, and the pubofemoral ligament inferiorly [4].

There are 18 muscles that should be considered in the setting of acute hip pathology, and they are best grouped by action. The gluteal group's main action is hip abduction, and it includes the gluteus maximus, gluteus medius, gluteus minimus, and tensor fasciae latae. Gluteus medius is especially important in hip stabilization, and weakness or injury can result in a Trendelenburg gait. The hip adductor group consists of adductor longus, adductor brevis, adductor magnus, pectineus, and gracilis. The primary hip flexors are iliacus and psoas major; however, rectus femoris is also a weak hip flexor. The hip external rotators include obturator externus, obturator internus, piriformis, superior gemellus, inferior gemellus, and quadratus femoris [4].

Finally, it is important to consider the neurovascular supply of the hip. The femoral arteries supply most of the blood flow to the lower extremities, and the medial and lateral circumflex branches supply the femoral head within the hip. Another important supply to the femoral head is the foveal artery in the ligamentum teres, which is commonly disrupted in hip dislocations. The lumbosacral plexus provides sensory and motor innervation to the lower extremity including the hip [4] (Fig. 7.1).

Physical Examination of the Hip

Inspection includes observation of gait, lower limb and femur alignment, pelvic symmetry, and evidence of ecchymosis or swelling. Several structures should be palpated including the adductor muscles/tendons/entheses, iliopsoas muscle belly at the anterior hip joint, superficial hip abductors, greater trochanters, tendons of gluteus medius and minimus, and the bony pelvis including the pubic symphysis and ischial tuberosities. Active ROM should be observed including hip flexion, extension, abduction, adduction, and internal and external rotation both with the

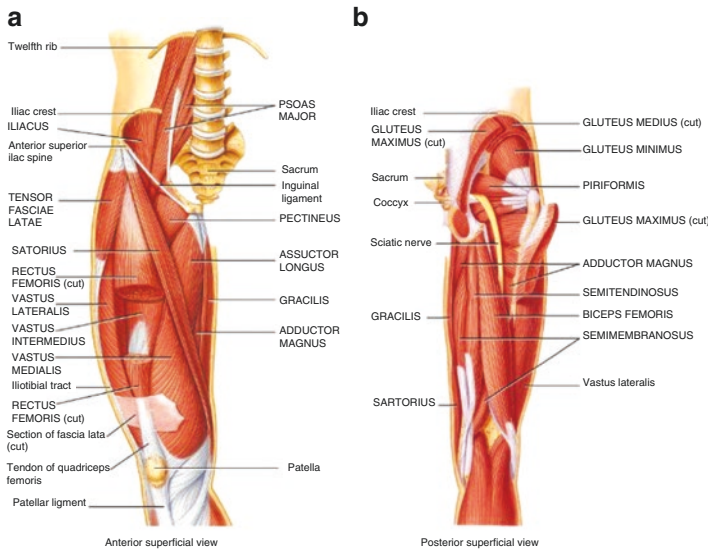

Fig. 7.1 Anatomy of the muscles surrounding the hip and pelvis with anterior (**a**) and posterior (**b**) views. (Source: Reproduced with permission from BoneandSpine.com)

hip in a neutral position and with 90° of flexion. Neurologic testing should include manual muscle testing, deep tendon reflexes, and sensory testing. Special tests include flexion/abduction/external rotation (FABER or Patrick's test), flexion/adduction/internal rotation (FADIR), log roll, Stinchfield's test, Ober's test and psoas muscle stretch/impingement (Thomas test). It is also helpful to test functional movements such as a single leg squat, step up/down, and the hop test [4].

Soft Tissue Contusions (Hip Pointer)

What Is the Pathology?

A "hip pointer" is a soft tissue contusion involving the iliac crest. This injury is typically sustained after direct trauma to the hip.

Clinical Presentation

Athletes will present with lateral hip pain and ecchymosis after sustaining a blow to an unpadded part of the iliac crest. While normally associated with contact sports (e.g., after a tackle in football or being checked into the boards in hockey), hip pointers have also been reported after falls in sports such as soccer or skiing [5].

Physical Exam

Inspection and palpation of the hip of an athlete will reveal ecchymosis and tenderness over the iliac crest. Additionally, they may be unable to bear weight on the affected lower extremity. If able to bear weight, they may have an antalgic gait [5]. Active range of motion will also be decreased on the affected side. The athlete may demonstrate pain with trunk flexion and rotation [6]. It is important to distinguish a hip pointer from other causes such as muscle tears, avulsion fractures, or acute compartment syndrome. A muscle tear can be distinguished from a hip pointer as there would likely be a decrease in strength on manual muscle testing [7]. An avulsion fracture may be differentiated by mechanism of injury. Instead of a direct blow, one may expect to see this injury present with sudden changes in rotational forces or if an eccentric load is greater than the strength of the insertion of the tendon or myotendinous junction [8]. Compartment syndrome should be suspected if pain is progressive, pain is out of proportion to exam, or there is tenseness noted on palpation [9].

Diagnostic Studies

Hip pointers are generally diagnosed on physical exam, but plain films may be helpful to rule out avulsion or apophyseal fractures [10]. In skeletally immature athletes, plain films may be especially helpful if pain does not improve in a few days or is wors-

ened by active or passive stretching of the muscle, as it may be a sign of an avulsion fracture [11]. MRI has been suggested as a more helpful imaging modality as it can reveal edema and subperiosteal hematoma [12].

Treatment

The first goal for the management of hip pointers is to minimize hematoma formation. This can be accomplished by icing and applying a compression wrap for 20 min every hour [13]. Conservative management includes pain control with acetaminophen and activity modification. There is a risk of worsening bleeding with NSAIDs, so these should be avoided if possible [6]. Physical therapy can be started once bleeding and swelling have been controlled and should focus on hip abductors and range of motion [5]. Injection of a local anesthetic into the point of maximal tenderness in the periosteum has been suggested as an intervention to decrease return to play time; however, it remains controversial [14]. There is a lack of data on the subject, but some studies have indicated that when used for hip pointers, the benefits of anesthetic injections outweigh the risks and likely do not have long-term safety issues [15, 16].

Return to Activities

Most athletes with hip pointers can return to sport in 1–3 weeks. However, severe cases may take up to 8 weeks [6].

Muscle Strain

What Is the Pathology?

A muscle strain is a traumatic injury to the muscle or tendon from an indirect force [17]. These injuries are graded on a scale of one

to three from order of mild to severe injuries. Grade one strains affect a few of the muscle or tendon fibers, and they may become painful with muscle contraction [17]. Grade two strains involve more fibers in the muscle-tendon unit, but the unit remains intact [17]. Grade three strains are complete ruptures of the muscle-tendon unit, which can be located at the tendon insertion site, myotendinous junction, or within the muscle or tendon [17]. The muscles most affected in the hip and thigh region include the proximal hamstrings, adductor muscle complex (adductor magnus, adductor brevis, adductor longus, gracilis, and pectineus), and rectus femoris [18].

Clinical Presentation

Acute muscular strains present with sudden onset of pain and weakness in the affected region. Proximal hamstring injuries occur near their origin at the ischial tuberosity, and athletes may report numbness and tingling after proximal hamstring injury if there is compression or irritation of the sciatic nerve [19]. Adductor muscle complex injuries occur either at the origin on the pubis or distally along the medial thigh within the muscle belly. Rectus femoris injuries occur at the origin on the anterior inferior iliac spine (AIIS) or within the muscle in the anterior thigh. Athletes in sports with high levels of kicking, sprinting, and cutting are at increased risk of sustaining an acute muscle strain [20, 21].

Physical Exam

Athletes will have pain in the strained muscle group on examination. Passive stretch and resisted activation of the affected muscle may exacerbate pain and demonstrate weakness [20]. Weight bearing on the injured leg can be difficult after an acute strain. If an athlete has a grade three strain, there may be ecchymosis and a visible bulge due to hematoma formation and retraction of the muscle [19].

Diagnostic Studies

Muscle strains are diagnosed clinically by history and physical examination. If ultrasound is available, injured tendons may appear to be thickened and heterogenous with edema surrounding the tendon [21]. Ultrasound of an acute muscle strain may demonstrate distortion of the normal muscle appearance with a focal area of heterogenous tissue [21]. High-grade strains can present as a soft tissue mass on ultrasound due to the retracted muscle fibers [21]. Evaluation of proximal hamstring avulsions can be difficult with ultrasound due to hematoma formation and muscle retraction [21].

MRI is a useful imaging modality for muscle injuries as it can better identify grade 3 muscle strain and avulsion injuries [19, 21].

Treatment

The initial management for low-grade muscle strains includes rest, ice, compression, and elevation (RICE). After the acute injury, the athlete should start a physical therapy program to progress stretching and active strengthening exercises [19]. Askling et al. found that a hamstring rehabilitation protocol that included lengthening-specific exercises was more effective than a conventional exercise program at returning Swedish elite jumpers and sprinters to their sport. The focus was on eccentric movements to increase flexibility, stabilize the trunk/pelvis, and increase strength [22].

Surgical intervention is indicated for proximal hamstring avulsions with greater than 3 cm retraction, and optimal timing of this is within 6 weeks of injury [19]. Rectus femoris avulsions should be managed surgically when there is loss of hip flexion strength [19]. Avulsions of the adductor tendons should also be repaired surgically.

Return to Activities

Lower grade muscle strains often have favorable outcomes after conservative management. Grade three muscle strains that require surgical intervention may require 3–7 months to return to sport depending on the injury location [19, 20].

Core Muscle Injury/Athletic Pubalgia

What Is the Pathology?

Athletic pubalgia (AP), also known as "core muscle injury" or "sports hernia," is defined as an injury to the muscular or fascial attachments to the anterior pubis without evidence of a true hernia [23]. This condition develops from an imbalance between the stronger hip adductors and weaker lower abdominals, which causes a shearing force across the hemipelvis and results in muscular overload with tearing of the transversalis fascia and/or overlying musculature [23].

Clinical Presentation

Athletes with AP will classically present with insidious onset unilateral lower abdominal, deep anterior groin, or proximal adductor pain. An acute presentation is rare but may occur with trunk hyperextension and hip hyperabduction that causes a partial or complete tear of the distal rectus abdominis/adductor aponeurosis. Pain will classically recur when the athlete tries to resume sports after a period of rest. Pain will also be worsened by sudden acceleration, twisting, turning, cutting, kicking, sit-ups, coughing, or sneezing. The pain may also radiate to the hip adductors, perineum, rectus abdominis muscles, inguinal ligament, or testicular area [23].

Physical Exam

Physical exam will be notable for tenderness at or above the pubic tubercle near the insertion point of the rectus abdominis or origin of the hip adductors without a palpable hernia. Special tests including FADIR (flexion, adduction, internal rotation) and FABER (flexion, abduction, external rotation) should be performed to rule out intra- and extra-articular hip pathologies that

may be present in addition to AP. Reproducible pain with a resisted sit up or single or bilateral resisted leg adduction is supportive of the diagnosis of AP [24].

Diagnostic Studies

Plain films of the hip and pelvis may be obtained to evaluate for intra- or extra-articular hip pathology such as arthritis, femoroacetabular impingement (FAI), or osteitis pubis. MRI is important when evaluating for AP as it can reveal rectus abdominis tears, which is pathognomonic for athletic pubalgia. The tear will appear as a cleft sign with hyperintensity on T2-weighted images at the rectus abdominis/adductor aponeurosis [24]. Dynamic US can demonstrate posterior abdominal wall deficiency when it is displaced anterior rather than becoming taut when the athlete strains; however, it is very subtle and operator dependent [23]. Diagnostic anesthetic injections into the hip joint may be helpful as relief of pain with the injection suggests an intra-articular pathology, while persistent pain in the lower abdomen and/or proximal adductor region is consistent with AP or adductor pathology [25].

Treatment

First-line treatment for athletic pubalgia is conservative management. Ellsworth et al. recommend a 4–6-week rest period followed by a four-phase rehabilitation program. Phase one (weeks 1–2) focuses on pain and edema control with stretching and hip and lumbar spine mobilization to increase ROM. Phase two (weeks 2–4) focuses on strengthening and adds cardiovascular warm-up with a bike or elliptical. Phase three (weeks 4–6) adds functional activities such as running. Phase four (weeks 6–8) incorporates sport/activity-specific activities [26]. Steroid injections at the rectus abdominis insertion site, conjoined tendon, or adductor tendon have been suggested, but there is not much data on short- or long-term efficacy [23]. If an athlete fails conserva-

tive management, surgical repair can be considered. There are several different techniques used which are variations of standard hernia repairs, including open, mini-open, and laparoscopic repairs. If FAI is found to be contributing to the symptoms, femoroacetabular surgery may be considered in a staged or concurrent approach [23].

Return to Activities

Athletes can return to sport after completing the phased rehabilitation program pain-free, generally 10–12 weeks after diagnosis of AP. For those that undergo surgical repair, a similar 6–8-week phased rehabilitation program has been described with athletes returning to play 8–12 weeks after surgery [26].

Hip Dislocations and Fractures

What Is the Pathology?

Dislocations and fractures of the hip are uncommon in sporting activities as they are associated with severe trauma. A hip dislocation occurs when the femoral head is displaced outside of the acetabulum, which significantly restricts the typical motion of the joint. Dislocations are characterized as anterior or posterior. Posterior dislocations account for up to 90% of the hip dislocations in sports [27, 28]. Hip fractures include fractures of the femoral head, femoral neck, intertrochanteric femur, femoral shaft, and the acetabulum (Fig. 7.2).

Clinical Presentation

High energy sports such as football, rugby, gymnastics, skiing, cycling, and basketball have a higher risk for traumatic injuries including hip dislocation and fracture.

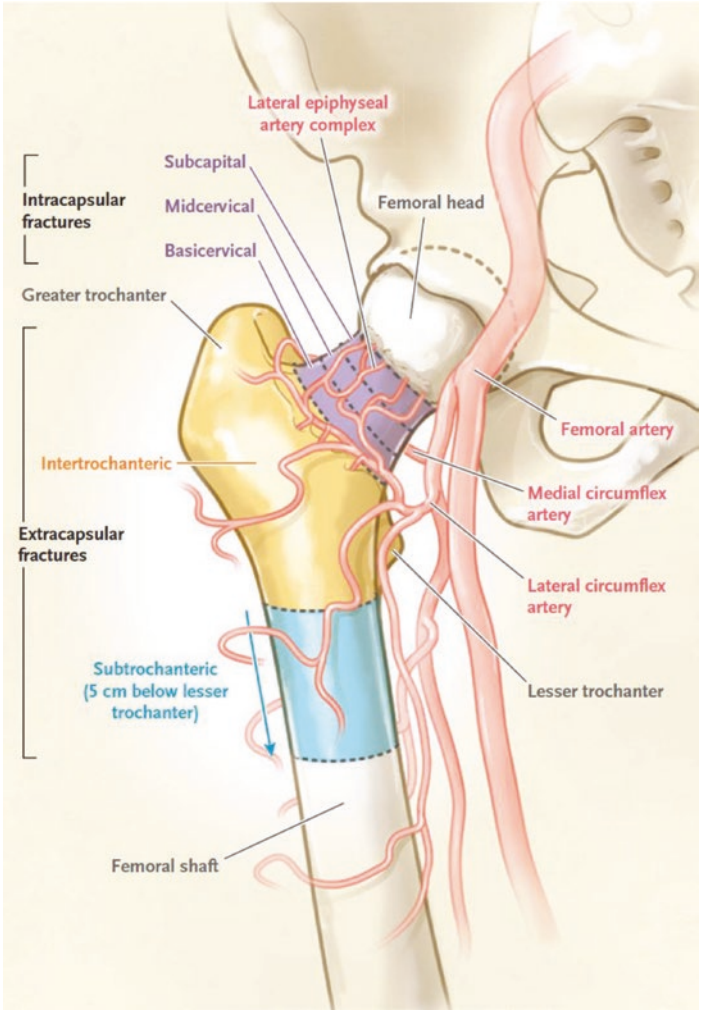

Fig. 7.2 Hip fractures are classified by their anatomic location being intracapsular or extracapsular. The common fracture sites include femoral head, femoral neck, intertrochanteric, and subtrochanteric. (Source: [29], Copyright © 2017 Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society)

Posterior dislocations occur when there is an axial force to the femur with the knee flexed, and the hip flexed, adducted, and internally rotated. Anterior dislocations involve an axial force on the flexed knee with the hip abducted and externally rotated.

Athletes who have sustained a hip fracture or dislocation typically have severe pain, and they are unable to bear weight through the affected lower extremity [30].

Physical Exam

For any athlete that is suspected of having a hip fracture or dislocation, efforts should be made to minimize movement of the lower extremity on initial evaluation. A thorough neurovascular examination should be completed. As noted previously, the femoral artery supplies most of the blood flow to the lower extremity. It can be compromised in certain fractures and dislocations which is a surgical emergency [27]. The sciatic nerve, coursing posteriorly, is the most common nerve injured in hip dislocations and femur fractures [27, 28]. Palpation of the injured area may reveal significant pain and spasm in the gluteal musculature. Checking leg lengths can be helpful to confirm the diagnosis of dislocation, as well as fracture [27]. Athletes with posterior dislocations typically present with their leg in a position with the knee flexed, and the hip flexed, adducted, and internally rotated. With anterior dislocations, the athlete's hip will be positioned in an abducted and externally rotated position. The femoral head can be palpated near the obturator foramen for posterior dislocations or near the anterior superior iliac spine with anterior dislocations [28].

Diagnostic Studies

Radiographs are the first-line imaging study to assess hip fractures or dislocations. Anteroposterior views of the pelvis and affected hip should be obtained. Additional views may include the lateral hip (cross table view), and anteroposterior and lateral

views of the ipsilateral knee [28]. The Judet view, an oblique view at 45°, is important for visualizing the acetabulum [27, 30]. For dislocations, radiographs should also be performed after reduction to confirm the appropriate positioning.

Computed tomography (CT) scans of the pelvis and hip can be used to evaluate femoral head abnormalities, osteochondral lesions, and acetabular fractures after hip reduction [28]. CT angiogram should be ordered if there is concern for vascular compromise with an asymmetric pulse [30].

MRI is used to evaluate occult femoral neck and intertrochanteric fractures [28]. If MRI is contraindicated or unavailable, thin-cut CT images can be obtained.

Treatment

In the setting of hip dislocation, hip fracture should be ruled out on radiographs before closed reduction is attempted. When the dislocation occurs during a sporting event, the athlete's initial evaluation takes place on the sideline or training room. If there is X-ray available on site, an experienced sports medicine physician may attempt to reduce a closed posterior hip dislocation. Otherwise, the emergency department is the most appropriate setting to manage the dislocation. Anterior dislocations should not be reduced on the sidelines due to the level of difficulty with reduction [27]. In general, closed reductions should occur within 6–12 h after injury to reduce the risk of avascular necrosis [27]. The key to closed reductions is ensuring adequate analgesia, conscious sedation, and muscle relaxation [27, 28]. Open reduction in the operating room is reserved for dislocations with femur fracture, hip joint instability, or if unable to reduce using a closed technique.

There are many hip reduction techniques that have been described although the Allis, Bigelow, and Stimson techniques are most widely used [28, 30]. The Allis technique is used most and requires an assistant to stabilize the pelvis as the treating provider pulls the leg with in-line traction. The hip is then flexed to 90°

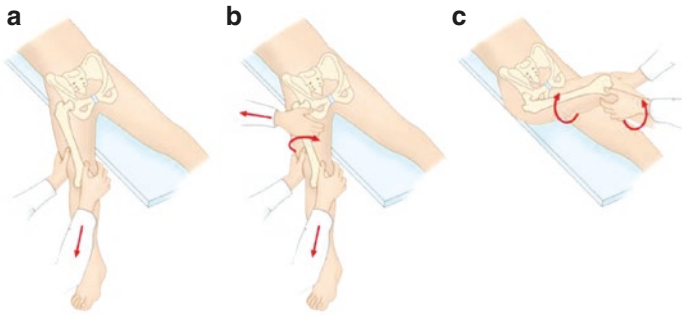

Fig. 7.3 Demonstration of the Allis technique for reducing a dislocation hip. The leg is pulled with in-line traction (**a**) while an assistant stabilizes the pelvis (**b**), and the knee is flexed to 90° with internal and external rotation until reduced (**c**). (Source: Reprinted from with permission from Musculoskeletal Emergencies, 1st Edition, Checo F, Shekhman M, Goldstein A, Erwteman AS, Chapter 13: Hip and Thigh, p 186–201, Copyright © 2012 with permission from Elsevier)

with alternating internal and external rotation until the hip is reduced (Fig. 7.3). The Bigelow technique requires the assistant to apply pressure to the anterior superior iliac spine (ASIS) as the provider flexes the hip to 90°, internally rotates and adducts the hip until it is reduced [30]. The Stimson technique requires the patient to lie in the prone position with their legs hanging off the table [30]. The hip and knee are flexed to 90° and a downward force is applied to the calf [30].

After a simple reduction is completed, the athlete is made non-weight bearing or foot flat touchdown weight bearing with use of crutches [27, 28]. Sometimes a knee immobilizer will be utilized to decrease hip flexion [28]. The athletes should also be counseled to avoid activities that increase hip internal and external rotation.

Surgical intervention is the typical management for hip fractures, and an orthopedic surgery consultation is necessary. Depending on the location of the fracture, traction may be used in the short term to help with pain relief and minimize motion through the fractured region [30]. Surgical techniques vary based on fracture location.

Return to Activities

Returning to sport after hip dislocation or fracture can be difficult. Rehabilitation is imperative to progress back to sport-specific activity. If surgical intervention is required, there is typically a period of protected weight bearing which can be prolonged depending on the severity of injury. Athletes should be counseled that recovery from a hip dislocation, with or without fracture may affect their future athletic career [30]. Return to sport after an isolated hip fracture without dislocation could take up to 4–6 months although some athletes may take up to a year or longer to completely recover motor strength [30]. Athletes should also be aware of complications following hip dislocation and fracture which include heterotopic ossification, avascular necrosis of the femoral head, osteoarthritis, and sciatic nerve injury [27–30].

Labral Tears

What Is the Pathology?

The labrum is a fibrocartilaginous ring attached to the rim of the acetabulum that forms a seal around the femoral head. It increases joint stability through the fluid seal, which produces a negative intra-articular pressure. Most labral tears occur in the presence of osseous pathologies; however, abnormalities may not always be seen in athletes [31]. For example, running-related labral tears are thought to result from repetitive hip hyperextension and external rotation causing joint instability and stress at the chondrolabral junction [32].

Clinical Presentation

Athletes with labral tears may present with a variety of symptoms. A retrospective study by Burnett et al. found that labral tears should be suspected in young, active patients with insidious onset

sharp and/or dull groin pain associated with activity with or without a history of trauma. However, patients also presented with locking, pain at night, pain in the buttocks, and pain with walking or pivoting [33]. Athletes may also describe catching, clicking, and joint instability [4].

Physical Exam

The clinical examination for labral tears can be difficult. Typically, athletes will have pain with FADIR (also called the anterior hip impingement test) in anterior labral tears and pain with the posterior impingement test in posterior labral tears. Passive range of motion may reproduce pain symptoms [4].

Diagnostic Studies

Anteroposterior (AP) pelvis and lateral hip plain films are often the first imaging studies ordered, but they are normal in the setting of an isolated labral tear. Radiographs may demonstrate acetabular dysplasia, with a lateral center-edge angle less than 20°. Dysplasia increases anterosuperior labral stress and can lead to tearing and degeneration of the labrum [34]. Additionally, radiographs are used to evaluate for femoroacetabular impingement, which is a risk factor for labral tears [35]. CT may not reliably detect labral tears and standard MRI can underestimate labral pathology. MR arthrography is more accurate but only has a specificity of 70%. The current gold standard for diagnosis is direct visualization of the labrum via arthroscopy [4, 34].

Treatment

Conservative management is recommended first; however, there are not well-defined parameters for this. Athletes should be counseled to limit activities, decrease weight on the affected side, and trial NSAIDs. However, pain will often return with resumption of

activities as the labrum is limited in its ability to heal [36]. It has been suggested that rehabilitation for labral tears should include a focus on reducing anteriorly directed forces on the hip through increased recruitment of muscles involved in hip motion, correct movement patterns in hip extension and gait, as well as providing education on avoiding pivoting motions [34]. If pain is persistent, interventions such as intra-articular steroid injections and platelet-rich plasma have been suggested although data on the benefits of these interventions is limited [37, 38]. If the athlete remains symptomatic despite conservative management, surgery may be indicated. Labral debridement is the most common procedure; however, studies have shown that labral repair has improved outcomes [39].

Return to Activities

Following hip arthroscopy, athletes will follow a graduated rehabilitation protocol initially focusing on pain control, ROM, and edema management before progressing to full passive and active ROM, strengthening, balance training, and sport-specific activity 3–4 months post-op if the athlete has regained full strength and coordination [40].

Apophyseal Injuries

What Is the Pathology?

An apophysis is an area of bone that has developed from a secondary ossification center. These apophyses can be seen radiographically in children and adolescents who are not skeletally mature. Avulsion fractures of the apophyses within the hip can occur from an acute injury or repetitive stress [41, 42]. The relatively weak growth plate can sustain injury after a forceful muscle contraction. The apophyses in the hip include the ischial tuberosity, iliac crest, anterior superior iliac spine (ASIS), anterior inferior iliac spine (AIIS), and greater and lesser trochanters and pubic sym-

Table 7.1 Common sites of apophysitis in the hip and pelvis with the associated muscle groups affected by this injury

Site of avulsion	Common muscles affected
Anterior inferior iliac spine	Rectus femoris
Ischial tuberosity	Hamstrings
Anterior superior iliac spine	Sartorius, tensor fascia lata
Greater trochanter	Gluteus medius and minimus
Lesser trochanter	Iliopsoas
Pubic symphysis	Rectus abdominis, hip adductors
Iliac crest	Abdominal muscles

Source: Adapted from Clin Sports Med, Volume 40, Schroeder PB, Nicholes MA, Schmitz MR, Hip injuries in the adolescent athlete, p 385–398, Copyright 2021, with permission from Elsevier

physis [18, 41, 42] (Table 7.1). The two most common areas for avulsion injury are the ischial tuberosity and AIIS [18, 41, 42]. These injuries are rare in skeletally mature athletes.

Clinical Presentation

Typical presentation includes acute pain and swelling in the affected area after a traumatic non-contact injury [18, 41, 42]. Some athletes may also have difficulty with weight bearing and functional weakness [18, 42]. Sports that require more rapid acceleration and deceleration movements, such as soccer, gymnastics, track, and field are at a higher risk for injury [42].

Physical Exam

Most athletes will have swelling, ecchymosis, and tenderness to palpation in the region of the injury. There will also be pain with activation of the affected muscle. Passive range of motion may be restricted due to pain, especially when the muscle attached to the avulsed fragment is stretched [41, 42].

Diagnostic Studies

Radiographs are the most helpful imaging study to confirm the diagnosis and to assess displacement of the avulsed fragment [18, 41, 42]. Radiographs also help to rule out additional pathology such as hip dislocations or acute fractures of the femur and pelvis. Rarely, a computed tomography (CT) may be necessary to assess the degree of avulsion [18].

Treatment

Conservative management with rest, NSAIDs, ice and protected weight bearing is the typical treatment if the avulsed fragment is displaced <2 cm [41–44]. Crutches can be used for protected weight bearing until the symptoms resolve [42]. Due to the risk of nonunion and ongoing pain, surgical intervention is recommended if the avulsed fragment is displaced >2 cm [41–44]. If an athlete is treated conservatively and continues to have functional impairments, late surgical intervention is indicated [41]. Physical therapy should start when symptoms resolve. The goal of physical therapy is to improve range of motion, incorporate isometric strengthening exercises, and gradually return to sport-specific activity.

Return to Activities

Overall, athletes can return to sport after they are able to demonstrate pre-injury strength and range of motion [18]. Athletes who were treated conservatively may return to sport as soon as 10–12 weeks after injury [43].

Greater Trochanter Pain Syndrome

What Is the Pathology?

Most athletes who have pain in the greater trochanter region can attribute this to chronic tendinopathy of the gluteus medius and/or minimus tendons [45]. Less frequently, an athlete may have acute bursitis in the setting of direct trauma. Bursae are fluid-filled sacs that serve to decrease friction between tissues in the body. The trochanteric bursae are susceptible to traumatic injury as they are located close to the skin with minimal subcutaneous tissue surrounding them [45].

Clinical Presentation

The athlete will present with pain along the lateral hip. Pain is worsened by lateral hip pressure, and it can radiate down the lateral leg or posterolateral thigh [46]. In chronic presentations, patients may describe a deep, achy pain that is exacerbated by lying on the affected side, ambulating, rising from a chair or climbing stairs [46].

Physical Exam

On exam, there is tenderness to palpation along the greater trochanteric region of the lateral hip. Ecchymosis at the lateral hip may be present after acute trauma [28]. Strength is not often affected in true acute bursitis, but strength testing may be limited by pain [45]. If true weakness is present, hip abductor tendinopathy or tear is more likely than acute bursitis [45]. Resisted hip abduction in a side lying position can reproduce symptoms, but passive hip range of motion should not be restricted. Ober test can be used to evaluate for tightness of the iliotibial (IT) band. As the IT band courses over the greater trochanter, tightness in this structure can lead to inflammation near the trochanteric bursa from repetitive catching on the lateral hip. Ober test is assessed by having the athlete lie on the unaffected side while the examiner pas-

sively extends and abducts the hip with the knee flexed to 90° [47]. If the thigh does not adduct when released, this is positive for IT band tightness.

Diagnostic Studies

Anteroposterior and lateral radiographs of the hip can be obtained in the setting of acute trauma to rule out fracture [28]. Ultrasound can be useful in the acute setting to evaluate for fluid within the subgluteus maximus, subgluteus medius, or subgluteus minimus bursae to diagnose a true bursitis. Assessing the integrity of the gluteal tendons can also be done with ultrasound. MRI imaging is usually not required for diagnosis, but it can be ordered if an athlete has not improved with conservative management to look for tendinopathy or tendon tears [45].

Treatment

Pain in the greater trochanteric region is typically managed by treating the underlying pathology. Rest, activity modification, and padding of the greater trochanter are conservative measures that can be used initially. For acute inflammation, ice and a short course of NSAIDs can be utilized. Physical therapy can help address core muscle and hip abductor strengthening in addition to a stretching program [28]. If pain persists after conservative management, a corticosteroid injection can help in more severe bursitis cases. Athletes should be counseled that the goal of the injection is to help with pain in the acute to subacute setting, but the injection should complement an ongoing rehabilitation program.

Return to Activities

Greater trochanteric pain is often self-limited with conservative treatment. An athlete can return to sport once the inflammation has resolved, and they are pain free with sport-specific activity.

Morel-Lavallée Lesion

What Is the Pathology?

A Morel-Lavallée lesion is a closed degloving injury involving the subcutaneous fat and the underlying superficial fascia that results after shear stress [48]. The potential space between these two layers fills with serosanguinous fluid and necrotic fat, and it is unable to drain internally due to the intact fascial layers [49]. A fibrous capsule with deposition of fibrin, hemosiderin, and granulation tissue may form if there is an ongoing systemic inflammatory response after the acute injury [49]. This is a less common traumatic injury that mostly affects the trochanteric region [48, 49]. These lesions can often be mistaken for an abscess, pseudolipoma, fat necrosis, or soft tissue sarcoma [48].

Clinical Presentation

Morel-Lavallée lesions typically occur in sports with high impact and increased sliding mechanisms such as football and baseball. Outside of sport, Morel-Lavallée lesions can be seen in people involved in motor vehicle collisions [49]. The time course for development of these lesions is variable. Some athletes present with symptoms after the initial injury although many times concomitant injuries such as fracture may delay the diagnosis. Athletes may present with edema, ecchymosis, pain, and abnormal skin contour in the affected area [48, 49].

Physical Exam

Most consistently, the physical exam will be notable for a large edematous area where the lesion is present. This mass may fluctuate on palpation. The skin can appear asymmetrically contoured compared to the unaffected side with or without ecchymosis [49]. Due to the disruption of cutaneous nerves with the shearing mech-

anism, athletes will also have hypoesthesia. In chronic lesions, the skin may appear dry, cracked, or have altered pigmentation [48].

Diagnostic Studies

In acute trauma settings, a radiograph is typically the first option for initial evaluation to assess the appearance of a soft tissue mass and to rule out acute fractures. In the emergency department, CT imaging is also utilized with trauma.

Ultrasound imaging is a helpful tool to evaluate acute injuries. It is also helpful for management depending on the severity of the injury. On ultrasound imaging of acute Morel-Lavallée lesions, the fluid collection will appear irregular and heterogenous [21, 48, 49]. In contrast, chronic injuries will show smoother margins with homogenous fluid [21, 48, 49].

MRI imaging is the most useful study to evaluate the chronicity of the lesion. Acute injuries demonstrate hypointense fluid collections on T1-weighted sequences with hyperintense fluid collections on T2-weighted sequences. Subacute injuries have hyperintense fluid collections on both sequences, while chronic injuries have a hypointense appearance on both sequences in addition to fibrous septations or calcifications within the fluid [48, 49].

Treatment

Management of Morel-Lavallée lesions varies. Conservative management for acute lesions involves compressive bandaging with or without fluid aspiration using a large bore needle [48, 49].

As these lesions are at risk for bacterial colonization, injuries that involve open fractures, closed fractures with an overlying lesion, deep infection, or skin necrosis typically require surgical intervention [49]. Fractures, either open or closed, may require delayed internal fixation after the initial surgical treatment. Surgical options for Morel-Lavallée lesions include either open

debridement or a minimally invasive technique using small incisions at the proximal and distal aspects of the lesion for drainage. Sclerotherapy has been described as an adjunct treatment for chronic lesions by promoting scar formation and aiding the closure of the dead space [49]. Agents that have been used for sclerotherapy include doxycycline, bleomycin, talc, alcohol, and tetracycline [49, 50].

Infection and recurrence of the lesion are two of the most common complications. Antibiotic treatment perioperatively and postoperatively are used to decrease the rate of infection after surgical intervention [49].

Return to Activities

There are no specific return-to-play guidelines for Morel-Lavallée lesions related to sport. In general, a timely diagnosis is important for these lesions as some athletes may present with delayed symptoms if a fluid collection is not apparent on initial injury. Conservative management without complications will allow athletes a faster recovery [48, 50]. Before returning to sport, the athlete should be pain free and have resolution of the Morel-Lavallée lesion.

Myositis Ossificans of the Hip

What Is the Pathology?

Myositis ossificans (MO) is a benign, ossifying soft tissue mass found in skeletal muscle [51]. The pathophysiology of MO is not well defined, but inappropriate differentiation of fibroblast cells into osteogenic cells is thought to mediate the process [52]. The most widely accepted hypothesis suggests that acute traumatic injury to skeletal muscle results in an inflammatory cascade which causes mesenchymal cells to differentiate and undergo endochondral bone formation. The inflammatory cascade is also thought to cause muscle damage, which further exacerbates formation of heterotopic bone [53].

Clinical Presentation

MO can have a variable presentation; however, most commonly an athlete may report persistent muscle pain and joint stiffness after blunt trauma or repetitive minor trauma to the soft tissue. Their symptoms will persist longer than what is expected for a muscle strain or contusion [54]. Contact sports appear to be more commonly associated with MO, especially American football [55]. However, repetitive minor trauma can also result in MO. For example, "rider's bone" describes MO found in adductor muscles in horseback riders. "Shooter's bone" describes MO in the deltoid found in marksmen. Additionally, MO has also been described in the soleus in ballet dancers [53].

Physical Exam

The most common initial sign of MO is decreased range of motion in an adjacent joint [56]. Additionally, edema may be noted. If the lesion causes mechanical irritation to a bursa or tendon, pain may be present [54]. If the lesion compresses neurovascular structures, the patient may have paresthesias, lymphedema, weakness, or even venous thromboembolism [54]. Symptoms may eventually resolve as the lesion matures [57].

Diagnostic Studies

The utility of bloodwork in diagnosing myositis ossificans has been studied but ultimately no lab has been identified that can accurately diagnose MO. Trending alkaline phosphatase may be helpful to ensure levels appropriately respond. Alkaline phosphatase will generally rise after the 3-week mark, peak at week 10, and return to baseline by week 18 [58]. As it generally correlates with maturity, it may be somewhat helpful in staging MO. However, it is important to note that other pathologies such as osteosarcoma can also cause an increase in alkaline phosphatase [53].

Radiographs are helpful to rule out other acute causes of focal pain such as fractures. In MO, radiographs are often negative until 2–6 weeks after the injury [53]. A soft tissue mass or periosteal reaction may be first observed before soft tissue calcifications appear around the 3–4-week mark. The calcifications may initially appear flocculent and amorphous before becoming more peripherally oriented and coarse appearing. Around the 6–8-week mark the calcifications mature to form a densely calcified peripheral rim with lucent center. After 6 months or more, the center may become lightly calcified. Mature lesions will sometimes adhere to the adjacent bone, but there is often a radiolucent cleft between the lesion and the bone [54].

Point-of-care ultrasound is gaining popularity as a first-line imaging modality. Some studies suggest that US should be the preferred method to screen for MO as changes can be seen as early as 3–5 weeks after injury [59]. When MO is visualized with US, a zonal appearance has been described: an outer hypoechoic zone surrounding the lesion, a middle hyperechoic zone representing the calcifying rim, and a central hypoechoic zone representing the central fibroblastic stroma [60].

CT may be helpful in diagnosing MO before calcifications become radiographically apparent. MO will initially appear as a soft tissue mass or edema. CT may reveal mineralization around the lesion up to 4–6 weeks earlier than MRI [53]. The center of the lesion will generally be more isodense than the surrounding muscle.

MRI is the gold standard but is often not the first choice for imaging due to cost. The appearance of MO on MRI will differ by stage, so MRI should be interpreted in conjunction with other imaging modalities. Early MO will demonstrate an isointense or slightly hyperintense signal on T1-weighted imaging. The lesion will appear hyperintense on T2-weighted images. For intermediate MO, the peripheral and central zones of the lesion will be isointense to hypointense compared to surrounding muscle. MRI obtained during this stage may show the zonal pattern that is diagnostic of MO. Additionally, surrounding edema can be seen on MRI obtained within 8 weeks of injury but may persist for several months. Mature MO has an MRI pattern of mature lamellar

bone—the mass will be well-defined and heterogeneous and will be isointense when compared to fat on T1- and T2-weighted imaging. MO is fully mature once surrounding edema is not present on MRI [61].

Treatment

Myositis ossificans is generally self-limited, so conservative management is an appropriate first step [62]. As MO is associated with hematoma formation, RICE (rest, ice, compression, and elevation) is recommended for 3–7 days. Some recommend the use of crutches to further limit hematoma expansion if MO is found in the lower extremities [63]. Gentle range-of-motion exercises may begin as early as 48–72 h post-injury [64]. Once the athlete can perform range-of-motion exercises pain-free, rehabilitation may be progressed to isometric training, followed by isotonic exercises, and finally isokinetic and dynamic exercises [54]. Given the efficacy of NSAIDs in the treatment of heterotopic ossification in spinal cord injury, it has been suggested that indomethacin can be used as prophylaxis for MO [65]. Extracorporeal shock wave therapy has also been suggested as an effective noninvasive treatment option; however, the data is limited [66, 67]. If an athlete remains symptomatic despite conservative measures, surgical excision may be considered. Surgery was historically delayed at least 6 months as it was thought that resection of a mature lesion would reduce the risk of recurrence [54]. However, earlier resection of lesions has been shown to have minimal risk of recurrence [68]. The decision to pursue surgery should be carefully discussed in a multidisciplinary setting.

Return to Activities

There are no specific guidelines for return-to-play in athletes with myositis ossificans. Symptoms usually resolve 6–8 weeks after initial injury [53]. A small ($n = 19$) retrospective case series found 90% of athletes returned to light physical activity 3 months after

diagnosis, 90% were back to pre-injury level of performance at 6 months, and all were back to pre-injury level of performance within a year [59].

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Fractures

8

Clayton Walker

Pathology Title: Hip Fractures

What Is the Pathology, How, Why?

Fractures of the proximal femur are colloquially known as hip fractures and are extremely common with 260,000–300,000 occurring in the United States annually [1]. Traumatic hip fractures are associated with bone fragility and commonly seen in elderly patients (mean age of 80 years old) with osteoporotic bone who sustained a low-energy trauma. The mechanism of injury can vary but is usually caused by falling on to the lateral aspect of the hip, around the greater trochanter, from a standing height [2]. Osteoporosis is more common in women due the effects of menopause, thus 77% of all traumatic hip fractures occur in women [3, 4]. In men, primary osteoporosis is less common so clinicians should be aware of secondary causes of osteoporosis in this population, which include hypogonadism, alcohol use, and corticosteroid use [4]. Because these fractures tend to occur in a frail population, they are associated with significant morbidity and mortality. One-year all-cause mortality ranges from 12% to 37%,

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with 9% being directly attributed to the hip fracture [5]. Function also declines significantly after injury with 10–20% of all survivors of hip fracture requiring a change in living arrangement due to immobility or inability to perform activities of daily living [5].

Although traumatic hip fractures are most commonly seen in patients over the age of 65, 3% occur in younger patients (<50 years old), usually after a high-energy trauma, like a motor vehicle accident or a fall from height [6]. The mean age for this type of injury is 32 years old with 77% occurring in male patients [6]. For a younger patient to fracture their hip in a trauma, the femur must be axial loaded while the hip is in the abducted position. If the hip is adducted and flexed at the time of impact, dislocation is more likely [2].

Traumatic hip fractures are classified based on what part of the femur is fractured. They are divided into two categories, intracapsular and extracapsular, with extracapsular fractures being further categorized as intertrochanteric or subtrochanteric [7]. Intracapsular fractures make up around 60% of all hip fractures and occur within the hip joint. They exclude fractures of the femoral head so are limited to fractures of the femoral neck [7, 8]. The medial femoral circumflex artery courses around the inferomedial hip capsule and gives off retinacular branches that are tightly adherent to the femoral neck making them susceptible to disruption if the fracture is displaced [9]. These branches provide blood supply to the femoral head so if they are disrupted, patients can develop avascular necrosis [7]. Intertrochanteric fractures make up about 36% of hip fractures and are those that occur distal to the femoral neck but proximal to the lesser trochanter [7, 8]. Fractures that occur in this area are subject to traction forces due to the greater trochanter being the attachment site of major hip adductors and the lesser trochanter being the attachment site of major hip flexors. Because of this, these types of fractures have a higher risk of malunion [7]. Subtrochanteric fractures make up roughly 4% of hip fractures and occur distal to the lesser trochanter but within the first 5 cm of femoral shaft [7, 8]. Abductors and external rotators of the hip attach at the proximal subtrochanteric area and adductors attach distally so bone fragments can be displaced by these muscular forces [7]. Additionally, this area has a weaker

blood supply than the intracapsular and intertrochanteric regions, which prolongs the healing process [7].

There are many different grading systems used to describe traumatic hip fractures, but the two most important things to determine are where the fracture is located (intracapsular, intertrochanteric, or subtrochanteric) and whether or not it is displaced. These two factors guide treatment and are reflected in grading systems like the Garden Classification, Pauwels' Classification, and AO/OTA Classification systems [10–12].

Atraumatic fracture is a term used to describe a fracture that is caused by a very low-energy mechanism that would usually be incapable of causing such an injury. Stress fractures, which are a type of atraumatic fracture, can occur at the femoral neck and can be classified as a fatigue fracture or an insufficiency fracture [13].

Fatigue fractures occur in normal bone that is subjected to repetitive stress causing microdamage that exceeds the bone's ability to heal and remodel [13]. Over time, this continued activity will lead to the bone breaking down and a stress fracture to form. These are commonly seen in younger people who abruptly increase their physical activity such as military recruits or runners. Although fatigue fractures are common, the femur is rarely implicated and only accounts for 7.2% of all cases of stress fracture in athletes [14]. When the femur is implicated, fractures will usually occur at the femoral neck and can be seen bilaterally in 9% of cases [15]. Because of the rarity of femoral stress fractures, consideration should be given to the patient's nutritional status as stress fractures can be seen more commonly in patients with relative energy deficiency in sport [16].

In contrast to fatigue fractures, insufficiency fractures result from normal stress being applied to abnormal bone. This can be seen in the context of many disease states, such as advanced osteoporosis, paget's disease, or in the case of pathologic fractures, tumor disrupting the bone's integrity [13].

Fatigue fractures of the femoral neck are classified by size and location. Fractures are considered "complete" if the fatigue line, which appears as a linear band of low signal intensity on MRI, spans greater than 50% the width of the femoral neck and "incomplete" if it spans less than 50% the width of the femoral neck [17].

Location is classified according to the Devas classification system as "tension-type" or "compression-type" [18]. Tension-type fractures involve the superior-lateral aspect of the femoral neck and are at a high risk for progressing to a complete fracture due to tension forces being applied across the fracture line. Compression-type fractures involve the inferior-medial femoral neck and are more amenable to conservative therapy due to compressive forces keeping the fracture well approximated. Complete fractures are at a high risk for sudden displacement which can compromise the blood supply to the femoral head and cause avascular necrosis. Avascular necrosis causes significant morbidity in the young, active patient population and is associated with delays in surgical intervention, so early diagnosis and surgical fixation is imperative for good outcomes [19].

Clinical Presentation

Patients who present with a traumatic hip fracture are often over the age of 65, have risk factors for osteoporosis, and present to the emergency department after low-energy trauma, like a fall from standing height. They will be unable to bear weight on the affected limb and will have severe pain that localizes to the groin. If the patient is younger with healthy bones, they will present with similar symptoms but after a high-energy trauma-like motor vehicle accident or a fall from height [2].

Stress fractures present differently. Patients will often present to an outpatient clinic as opposed the emergency department. They will complain of dull, vague pain that localizes to their buttocks, knee, thigh, groin, or back. The pain will have a gradual onset without a specific inciting event, like a trauma, and have progressively worsened over time. Most patients will be able to ambulate normally, with only 20% presenting with a limp or antalgic gait [20]. Pain will be exacerbated with weight-bearing activities such as running, jumping, or ascending stairs. Fatigue fractures are more commonly seen in younger patients who abruptly increase the intensity of their training, like runners or military recruits, so inquiring about activity level and recent

changes is important. Insufficiency fractures will present with similarly vague symptoms but are more likely to occur in older patients with underlying disease. In the older population especially, they may also have other pathology that mask the symptoms of a hip fracture such as osteoarthritis and chronic lower back pain.

Physical Exam

With traumatic hip fractures, the affected limb may be abducted, externally rotated, and shortened if the fracture is displaced. Pain will be severe, sharp, localize to the groin, and present at rest. Patients will be unable to bear weight on the affected limb. Pain is exacerbated by active and passive range of motion of the hip. Neurovascular exam is usually normal but should be done to assess for any vascular or neurologic compromise in the affected limb [2].

With stress fractures, the patient will be able to bear weight, but may present with a limp or antalgic gait [20]. Pain may be reproduced by extremes of hip range of motion, palpation of the inguinal area overlying the hip joint, and by inducing a load across the fracture [20, 21]. To induce a load across the fracture, the "hop test" can be used in clinics. The "hop test" requires the patient to jump up and down on the affected limb and has been shown to reproduce symptoms in 70% of patients [22]. The "fulcrum test" can also be used, which requires the examiner to create a fulcrum under the femur while the patient is seated and then apply a downward force to the distal end of the femur, but this is more sensitive for femoral shaft stress fractures [23].

Diagnostic Studies

Plain radiographs of the hip is usually sufficient to make the diagnosis of traumatic hip fracture. It is recommended to order an anterior-posterior view of the pelvis and a lateral view of the proximal femur to assess for displacement. Stress fractures, however,

are frequently occult on initial radiographs, thus advanced imaging is often needed [24]. MRIs are considered the gold standard to assess for stress fractures and are considered nearly 100% sensitive and specific [25]. On T2-weighted imaging, edema within the bone will appear as a hyperintense region and the fatigue line will appear as a linear band of low signal intensity that runs perpendicular to the line of force that crosses the femoral neck [25].

Treatment

Surgery is the mainstay treatment for traumatic hip fracture. Surgical fixation is the preferred treatment for extracapsular fractures and nondisplaced intracapsular fracturs, especially in younger patients, as it preserves the natural hip joint. If an intracapsular fracture is displaced, hip arthroplasty is preferred treatment (either total or partial hip arthroplasty). This is because the vascular supply to the femoral head is commonly compromised with displaced fractures, which can lead to avascular necrosis and nonunion (incidence of 10% and 30%, respectively, in patients treated with internal fixation) [26]. Both total and partial hip arthroplasties replace the head and neck of the femur thus eliminating the risk of avascular necrosis and nonunion. Arthroplasty has been shown to have significantly lower reoperation rates compared to surgical fixation (11% vs 40%) and is the gold standard of treatment for displaced intracapsular fractures in the elderly [26]. For patients younger than 50 years old with displaced intracapsular fractures, preservation of the natural hip joint is paramount so surgical fixation is recommended [6]. Definitive, non-operative treatment of hip fractures is very rarely indicated (around 2% of cases) [27]. This is reserved for patients who are extremely high-risk surgical candidates, patients who were non-ambulatory prior to the fracture and not in pain, and those who present late with partially healed fractures not affecting function [2]. Current guidelines recommend surgery within 24–48 h of admission, but there is emerging evidence that surgery done within 24 h is associated lower 30-day mortality (5.9% vs 6.5%)

and lower rates of medical complications (pulmonary embolism, myocardial infarction, pneumonia, etc.) [28, 29].

For stress fractures, management depends on the type. Any patient presenting with a complete fracture, meaning the fatigue line is greater than 50% the width of the femoral neck, or tension-type fracture should be made non-weight bearing and immediately referred for surgical fixation to avoid the possibility of sudden displacement of the fracture. For incomplete compression-type stress fractures, meaning the fatigue line is less than 50% the width of the femoral neck, a trial of conservative management is appropriate. The patient should be made non-weight bearing until they can ambulate without pain, then advance their weight-bearing activity as pain allows. Worsening pain or a plateau in progress would be an indication for reimaging. If the fracture has progressed, then referral for surgery is indicated [17].

Return to Activities

For traumatic hip fractures in the elderly, early mobilization and immediate full weight bearing has been shown to decreased morbidity and mortality and is thus the standard of care [29–32]. Movement restrictions are often recommended for patients whose surgery required disruption of the joint capsule, as seen with total or partial hip arthroplasty. This is done to reduce the chances of dislocating the prosthetic joint although evidence for their use is limited and evolving [33, 34]. The specifics of these hip precautions vary based on the surgical approach. With the posterior approach, it is recommended that patients avoid hip flexion past 90°, internal rotation, and adduction [35]. With the anterior approach, it is recommended that patients avoid hip extension and external rotation [35]. Patients will be placed on these precautions for at least 6 weeks with guarded progression thereafter [35]. Physical therapy should begin immediately and focus on improving gait and strength in the affected limb.

With stress fractures, if surgery is indicated, patients should remain non-weight bearing for 6 weeks postoperatively.

Afterwards, they can progress weight-bearing status as pain allows until pain-free full weight bearing is achieved. At 12 weeks, if the patient has achieved pain-free full weight bearing, physical therapy and a gentle running program can begin. Patient should be able to endure full activity without pain before returning to sport, which can take anywhere from 3 to 12 months to achieve. If the patient was managed non-operatively, they will follow a similar progression of activity. They should remain non-weight bearing until they are completely pain free. They can then progress their weight-bearing status as pain allows until they reach full activity. Once they are pain free with full activity, they can return to sport. This is usually achieved around 3 months but can take up to 7 months [36].

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9 Rheumatology and Infectious Diseases and Hip Pain

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Osteoarthritis

Hip osteoarthritis (OA) is the most common rheumatologic hip disease, affecting the quality of life, to varying degrees, of millions of people around the world [27, 30]. An aging population and increasing rates of obesity, has made OA one of the leading causes of musculoskeletal pain and disability in the United States [27, 29]. Defined as the failure of the structure and function of the hip synovial joint, the etiology of OA is multifaceted and includes mechanical, structural, environmental, and even genetic factors [27]. Although the hip joint requires a certain amount of load in order to maintain its structure and function, excessive load on the joint can lead to degradation over time [31]. Repetitive activity and loading of the joint, not offset by reciprocated time for tissue repair, may lead to an imbalance of bone resorption to deposition and eventual joint degeneration [31]. It is important to note that low levels of estrogen and vitamin D may cause a synergistic decline in cell turnover rate and bone deposition [27]. On a cellular level, inflammatory recruitment of polymorphonuclear leukocytes to the joint space leads to an effusion [31]. Pain is

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produced with neuronal sensitization of substance P nerve fibers as well as capsular distension caused by vasodilation and venous congestion from the inflammatory process [27].

Clinical Presentation

In general, pain and impaired function are the most common presenting symptoms or complaints of patients with hip OA. While catchy, the cliche, "true hip pain is groin pain" is also correct when it comes to clinical presentation of hip pain [1, 2, 25]. Most patients will describe their hip pain as pain that radiates into the groin, triggered by certain movements, particularly some combination of internal rotation, flexion, and adduction of the hip [1, 25]. Other patients, however, will complain of pain that radiates like a band around the hip and will present cupping the sides of their hip with their thumb and index finger in a "C sign" fashion. The most prevalent risk factor for hip OA is advanced age, usually those older than 45 years of age [27, 30]. Other risk factors that can lead to OA are female gender, obesity, occupations requiring repetitive stress on the hip, as well as pre-existing bone deformities, joint injuries, and prior surgeries [29, 30].

Physical Exam

As soon as the patient walks into the office, your physical exam can begin with a quick inspection of the individual's gait pattern. Notable for hip OA are Trendelenburg gait and antalgia with decrease in stance phase on the affected side [1, 25]. Palpation of bony landmarks as well as soft tissue areas such as the anterior hip or lateral hip may yield information on possible bursitis or tendinitis and range of motion (ROM) testing, starting with the log roll, can assess for pain within the hip, with or without radiation to the groin [1]. With every exam, comparison to the contralateral side is vital and can provide a baseline for decreased hip functionality. Other important ROM examinations involve hip flexion with

straight leg, deep flexion with knee bent, measuring popliteal angle to assess hamstring tightness, resisted straight leg raise to assess for pain within or around the hip, and assisted straight leg raise to assess for resolution of pain in the hip [1]. Strength and sensation examination, including reflexes, are important tests to confirm the patient is neurologically intact and rule out non-hip OA neurological issues, such as radiculopathy [2]. Special tests are reserved towards the end of the physical exam and as a general rule, it is the combination of these special exams, the history, and the rest of a thorough physical exam that will zero in the diagnosis. Recommended special examinations include Patrick's test (Flexion, Abduction, and External Rotation, FABER), Flexion, Adduction, and Internal Rotation (FADIR), scouring of the hip, resisted SLR to assess for hip flexor etiology of pain, and Ober's test to evaluate for tightness of the Iliotibial band [2]. The examination should be curtailed and guided by the history obtained, as to not elicit further pain unnecessarily, while done in a methodical way to ensure capturing of all key points in the exam [1]. A structured examination will facilitate an organized physical exam and should follow the "IPROMiSSSE" method of Inspection, Palpation, Range of Motion, (i), Strength, Sensation and Special Examinations (Table 9.1).

Treatments

The one curative treatment for hip OA is a hip replacement or total hip arthroplasty. Other, more conservative forms of surgical treatment, include osteotomy and hip resurfacing [28]. Due to its invasiveness, surgical treatment is reserved for patients with the most severe form of hip OA and undertaken when non-operative measures have been exhausted or if the patient's quality of life is no longer acceptable to them [1, 27]. Fortunately, conservative treatments for OA have expanded in their efficacy in recent years. Reducing pain while improving function and mobility is the goal of hip OA treatment and evidence suggests that this may be achieved in patients who incorporate moderate weight loss and

Table 9.1 Imaging of the hip. Adapted from [24, 25]

Diagnostic studies
Hip radiographs AP pelvis radiograph with lateral and false-profile views • AP pelvis: to assess for degree of joint space narrowing, presence of osteophytes, subchondral sclerosis, subchondral cysts, pelvic obliquity – Recommend weight-bearing radiographs for better assessment of joint spaces [3] • Cross table lateral or Lateral views: to assess for cam morphology, FH sphericity • False-profile view: to assess for acetabular morphology, and to calculate anterior CEA • Generally, findings of OA are joint space narrowing, osteophyte formation, and sclerosis of the subchondral bone plate with subchondral cyst formation as [3] • The Kellegren-Lawrence score is the most reliable radiologic classification system used [3]. It is graded from – 0—no radiographic findings of OA – 1—doubtful narrowing of joint space and possible osteophytic lipping – 2—definite osteophytes with possible narrowed joint space – 3—definite osteophytes with moderate joint space narrowing, some sclerosis – 4—definite osteophytes with severe joint space narrowing, subchondral sclerosis, and definite deformity of bone contour
Ultrasound of the hip Dynamic ultrasound of the hip joint is useful in evaluating joint effusion and synovitis with the ability to assess for increased vascularization. Ultrasound is also able to assess the presence of osteophytes and is very helpful for imaging guidance of intra-articular injections [3].
MRI of the hip Typically order without contrast; request proton density sequences • Wide field of view: including both hips to assess symmetry. • Allows for assessment and evaluation of a variety of tissue abnormalities not only of the articular cartilage and/or the acetabular labrum but also of the bone marrow, ligaments, and synovium [3]. • Sagittal view: to assess for cartilage, labral, psoas, and hamstring pathology. • Coronal view: to assess the labrum, capsule, gluteus medius and minimus, adductors, pubic symphysis. • Axial view: to assess the hip flexors, gluteal muscles, adductor muscles, and pubic symphysis for core muscle injuries [3].

Table 9.1 (continued)

Diagnostic studies
CT scans with three-dimensional reconstruction Computed tomography is useful in further evaluation of cam morphology, acetabular dysplasia, femoral and acetabular version as well as providing information on the three-dimensional surgical planning [3].

strengthening hip-stabilizing muscle groups through exercise [1, 28]. The main caveat of this course of treatment is patient buy-in and long-term compliance.

For hip OA exacerbations, pharmacologic treatment is first-line and among them is acetaminophen [28]. With an excellent side effect and benign safety profile, acetaminophen works effectively through its inhibition of the central nervous system on spinal nitric oxide mechanism and substance P receptors [28]. Similar in efficacy, NSAIDs are second-line treatments used for moderate to severe flare up of hip OA with a mechanism of action directed at inhibition of cyclooxygenase and leukotrienes [27]. Because of this mechanism, deleterious side effects such as gastric bleeding, nephrotoxicity, and cardiovascular risks need to be considered for those with long-term use [1, 27] Due to these adverse effects, there has been an uptick in use of topical NSAIDs and analgesics such as voltaren cream and lidocaine patches among others as adjuvant treatment.

For patients having difficulty sustaining their progress with exercise and weight loss and those unresponsive to pharmacologic treatment, intra-articular injections can be considered both for pain reduction and improved function [27]. It is often also considered for patients plateauing in their exercise or physical therapy program due to pain and those who want to maximize their conservative treatments and avoid surgery. When it comes to injections, corticosteroid is the first-line for hip OA and followed by more controversial and less supported evidence for the hip with visco supplementation injections [28]. It is recommended that intra-articular injections be done under imaging guidance with either ultrasound or fluoroscopy to maximize efficacy and

prevent adverse complications [1]. Other options such as platelet-rich plasma (PRP), prolotherapy and injectable NSAIDs can be considered, however, these medications are often cost prohibitive, physician-dependent or equipment-dependent. PRP, in particular, is still awaiting FDA approval and with mix evidence for its benefits [1, 27].

Return to Activities

Evidence suggests that patients, including those with advanced hip OA will respond to conservative management [1]. Pain-guided movement and exercise, without excessive loading that can lead to degradation of the joint, is essential to not only preserving the synovial hip joint but maintaining overall health and preventing deconditioning in such patients [2]. Exercises focused on strengthening the hip girdle stabilizers, core musculature as well as the hamstrings and quadriceps optimize the kinetic chain and play a vital role in a successful non-surgical return to activity [1, 2]. Patients must be periodically reminded that return to activity may not necessarily be a linear route, requiring re-assessments along their journey back to functionality.

Sacroiliitis

Sacroiliitis refers to inflammation at the sacroiliac joint secondary to chronic inflammatory arthritis and may be present in a wide range of conditions, most commonly Ankylosing Spondylitis (AS) or Reiter's syndrome [4]. Other disease processes that may lead to sacroiliitis include enteropathic arthritis (Crohn's disease or ulcerative colitis), rheumatoid arthritis, psoriatic arthritis, gout, osteoarthritis, and Behcet's disease [4]. Sacroilitis can also develop due to a traumatic event or infection.

In females, there may be increased incidence from pregnancy due to the hormonal changes and imbalances that affect the integrity of the SI joint [5]. It is characterized by the absence of Rheumatoid Factor (RF), association with human leukocyte antigen (HLA)-B27, a tendency for familial aggregation, and inflammation around the entheses (the site of tendon or ligament insertion into bone), uveitis, urethritis, and psoriatic skin lesion. Fibrosis and ossification within the SI joint may result from chronic inflammation of the sacroiliac (SI) joints [6].

Clinical Presentation

Sacroiliitis may present as generalized low back pain and morning stiffness, weakness of the hip musculature on the affected side, pain with prolonged standing and transitional movements, as well as difficulty ascending or descending stairs [4]. The condition presents in adolescence to middle-aged individuals and is seen as a predominantly male disease [5]. It is commonly associated with ankylosing spondylitis, a seronegative spondyloarthropathy characterized by chronic inflammation, back pain and stiffness of the spine, including the sacroiliac joint [4].

Physical Exam

As with general OA, it is recommended to follow the "IPROMiSSSE" method of Inspection, Palpation, Range of Motion, (i), Strength, Sensation, and Special Examination to ensure a thorough examination. Inspection of a patient with suspected sacroiliitis begins with observation and assessment of their gait and presence of antalgia, monitoring for Trendelenburg gait, a sign of weakness of the surrounding musculature around the SI joint [1]. By asking the patient to point with one finger to the area of their pain may yield positive for the Fortin-finger test with the

patient pointing to the PSIS or sacral sulcus of the affected SI joint. Palpation of the sacral sulcus near the PSIS and reproduction of patient's pain may be indicative of SI joint etiology. Range of motion testing with flexion, extension, abduction as well as internal and external rotation of the hip will reproduce the patient's pain originating from the posterior hip in the area of the affected SI joint [1, 25]. Neurologic examinations including strength and sensation are expected to be normal, equal, symmetric and intact throughout although, they may present with pain limited strength if acutely flared. For special examinations, the three most common provocation tests are the pelvic rock test, FABER (Flexion, ABduction, and External Rotation), and Gaenslen's test [4] (Table 9.2).

Table 9.2 Hip Diagnostics [24, 25]

Diagnostic studies
Hip radiographs Radiography has poor sensitivity and specificity especially in early disease, but it remains the firstline of imaging. A specific view is helpful called SI joint anteroposterior (AP) oblique view, which is a part of radiographic sacroiliac series. It is indicated for the assessment of the left and right SI joint for comparison contralaterally in the evaluation of sacroiliitis and ankylosing spondylitis [6]. It is also used to determine any dislocation or subluxation of the joint. Note that degree of obliquity can vary widely between patients. Radiographic findings include sclerosis of the endplates of the SI joint particularly on the iliac side, irregular joint end plates and widening of joint spaces (https://radiopaedia.org/articles/sacroiliac-joint-ap-oblique-view-1?lang=us).
Ultrasound Ultrasonography is helpful for image-guidance intervention such as an SI joint corticosteroid injection. It is not often used for diagnostic purposes although technically can be considered as it is excellent in dynamic imaging [6].

Table 9.2 (continued)

Diagnostic studies
MRI Better imaging modality in identifying early disease processes and inflammatory changes of joints especially when radiography is negative and high clinical suspicion for sacroiliitis [6]. MRI findings of sacroiliitis are synovitis and capsulitis characterized by thickening and contrast enhancement of the synovium and joint capsule, marrow edema in the bones adjacent to affected joints seen in T2-weighted images or STIR sequence (may be earliest finding), subchondral sclerosis that parallel the joint margins which presents as low signal intensity on all sequences, marginal foci of articular bone loss or erosions found more prominent at the anteroinferior and iliac side of the SI joint [6]. There may also be intra-articular high T1 signal that fills up excavated bone erosions, periarticular fat deposition (fat metaplasia) and associated ankylosis [6].
CT scan Computed tomography presents higher sensitivity and specificity, accuracy with better delineation of anatomy but due to higher exposure to radiation, it is recommended against the use of CT for evaluation of sacroiliitis [6].
Lab studies Laboratory studies are indicated especially when suspicion for associated AS, Reiter's or other rheumatologic arthritides. Studies should include WBC usually within normal but can be elevated with associated infection, elevated ESR/CRP, blood cultures may be positive in 50% and positive for the presence of HLA-B27 with negative rheumatoid factor [6].

Treatments

Sacroiliitis treatment is conservative management, depending on the underlying cause, and rarely requires surgical options. Overuse injury or trauma can be addressed with rest, activity modification, NSAIDs and if recalcitrant to more conservative measures, may benefit from corticosteroid injection [1]. In pregnancy, a serola belt or SI joint belt can make a significant difference in pain

tolerance and function, especially towards the later stages of the third trimester [7]. While pregnancy-associated sacroiliitis typically resolves after childbirth, it is important to assess the full hip as a rare condition of transient osteoporosis can be a serious complication that should not be missed [7]. For sacroiliitis of infectious etiology, intravenous antibiotics may be required until symptoms resolve and CRP returns to baseline, after which patients can be transitioned into oral antibiotics. Surgery, on rare occasions, may be needed should antibiotics fail and especially if a large abscess is present [5]. In sacroiliitis associated with spondyloarthropathy, NSAIDs may be firstline but may likely require the use of disease-modifying antirheumatic drugs (DMARDs) such as TNF inhibitors [5]. When treating pharmacologically, it is best to manage patients alongside rheumatology. Physical therapy directed towards the maintenance of good mobility of the hip girdles as well as spine and peripheral joints is of vital importance to preserve function and significantly improve pain [1]. Modalities such as cold and heat therapy, hydrotherapy, spa therapy, diathermy, and electric therapy are all indicated as adjuvant therapy to help improve pain tolerance [1].

Return to Activities

With the help of an astute physical therapist, a gradual return to activity program can be established, especially in patients who are affected by sacroiliitis associated with spondyloarthropathy [4]. Initial return can begin with pain-guided exercises and movements. As patient tolerance for pain and activity increases, a strengthening program of the hip girdle stabilizers, core musculature as well as the hamstrings and quadriceps muscles to optimize the kinetic chain can be established to return to full and near-baseline activity [1]. To increase capacity and endurance as well as reduce cardiovascular morbidity and mobility, aerobic training with brisk walking, cycling with upright bars, swimming, or other

aquatic therapy can be incorporated into any training regiment [1].

Osteonecrosis of the Hip

Osteonecrosis, or avascular necrosis, refers to decreased vascular supply to the bone, resulting in osteocytes or mesenchymal cell death [8]. Typically affecting patients in their late 30s or early 40s, gender predominance varies depending on the underlying cause of osteonecrosis. Osteonecrosis of the hip joint leads to subsequent collapse and flattening of the femoral head. While most cases are idiopathic, there are often both traumatic and non-traumatic causal relationships of osteonecrosis [8].

Traumatic causes may result from direct injury to blood supply as a result of fractures, dislocation subluxations, or radiation injury [8]. Caisson disease (also known as "the bends" or decompression sickness) causes formation of nitrogen within the arterioles, which can lead to osteonecrosis years after initial exposure. Non-traumatic risk factors are multifactorial in nature and are believed to result from conditions such as chronic alcohol use due to decreased osteogenesis, subsequent induction of adipogenesis, and lipid deposition in bone resulting in osteocyte death [9]. Sickle cell anemia (due to vascular occlusion of blood supply due to sickling), and other hemoglobinopathies, as well as conditions such as lupus or malignancies requiring chronic, high-dose courses of corticosteroids treatment may result in intravascular thrombotic occlusion, extravascular fat deposition, or any combination of these resulting in decreased blood supply [10]. Other chronic conditions that can predispose to osteonecrosis include HIV, pancreatitis, chronic renal failure, and smoking, among others. Osteonecrosis may be a complication of progression from transient osteoporosis of the hip, a benign, self-limited condition of unknown etiology which in women often occurs in the third trimester of pregnancy [7].

Clinical Presentation

Patients may complain of general hip irritability [11], anterior hip pain, which may refer to the groin, or less commonly the thigh and gluteal areas [8]. Pain may worsen with transition from sit to stand, walking up steps or inclined surfaces, as well any movement that loads the hip joint. While many cases will be gradual in onset, acute pain complaints can occur in the case of acute infarction events, which highlights the importance of identifying any and all predisposing comorbidities.

Physical Exam

Although physical exam findings may be nonspecific, it is important to perform a full hip examination, assessing for range of motion, inspecting the patient's gait pattern for antalgia, as well as a positive Trendelenburg sign. Tenderness to palpation in the affected groin may be present, and patients may complain of pain with the log roll, as well as reduced or painful ROM at the hip.

Diagnostic Studies

Plain A/P and lateral radiographs of the affected hip joint should be obtained, and if positive for osteonecrosis should be followed by plain films of the bilateral hip joints as half of osteonecrosis cases are bilateral in nature [12]. The pathognomonic crescent sign or area of subchondral lucency on plain X-ray suggests intra-articular fracture or subchondral collapse [13]. If osteonecrosis is not detected on plain radiographs, but is clinically suspected, a non-contrast MRI should be ordered, due to its high sensitivity and specificity for osteonecrosis, and greater ability to assess the size and extent of the lesion [26].

Findings can be staged according to the Ficat and Arlet classification system of the femoral head (Table 9.3).

Table 9.3 Ficat and Arlet classification system of the femoral head. Adapted from [26]

Classification	Clinical	Radiographs	MRI
Stage 0	No symptoms, preclinical	Normal	Normal
Stage 1	Possible groin pain	Normal or mild osteopenia	Possible edema
Stage 2	Groin pain and stiffness; pain with activity	Osteopenia and/or subchondral cysts; diffuse porosis; precollapse of joint space	Outlines area of involvement of the femoral head
Stage 3	Groin pain, stiffness, radiation of pain; pain with activity	Crescent sign and/or subchondral collapse (flattening) of joint with secondary degenerative changes; loss of sphericity of femoral head	Same as radiographs
Stage 4	Groin pain and limp; pain at rest	End-stage disease with collapse; extensive destruction of joint; reduced joint space	Same as radiograph

MRI, magnetic resonance imaging

Treatments

Treatment of osteonecrosis requires consideration of various factors, including but not limited to, presence of symptoms, femoral head collapse, extent of disease, and quality of life issues such as patient's preference, lifestyle, age, and comorbid conditions [8]. Therapies can be divided into operative and non-operative management and is generally decided based on the size of the lesion.

Non-operative Management

Initial management options focus on physical therapy, as well as resting and offloading the joint through the use of assistive

walking devices such as canes, crutches, and walkers. For pain management, oral medications such as NSAIDs, acetaminophen, and short courses of opioids may be helpful in managing pain. Evidence has been mixed regarding the efficacy and reliability of pharmacologic agents such bisphosphonates, statins, anticoagulants, and vasodilators to treat osteonecrosis [14]. Consequently, they are not generally recommended for routine treatment.

Operative Management

Core Decompression

Generally performed in the early stages of osteonecrosis for small lesions, this procedure involves drilling holes into the femoral head, relieving pressure, and ultimately pain, while stimulating a healing response through bone and vascular neogenesis [8]. Bone grafts, stem cells, and concentrated bone marrow injected into the necrosed bone have been hypothesized to slow disease progression and stimulate new bone growth [15].

Bone Grafting

Bone grafting can be combined with core decompression and involves transplanting of healthy bone to the area of necrotic or dead bone from one part of the patient's body (autograft) to another, or from a cadaver (allograft) [14]. Bone grafting can be categorized as vascularized or non-vascularized. Vascularized grafting involves taking a segment of the fibula and its blood supply and transplanting it to the area of a hole created in the femoral neck and head, improving perfusion of the bone. Non-vascularized grafting surgery is performed via three main techniques: (1) phemister, (2) trapdoor, and (3) lightbulb. The Phemister technique involves removal of a small section of bone, followed by debridement and then insertion of a cortical strut graft for cortical support. The trapdoor technique involves removal of a chondral window from the femoral head, allowing access to the necrotic bone region which is then removed with curettage or high-speed burr, and subsequently filled with bone graft. Finally, the lightbulb

technique involves removal of a cortical window from the femoral neck, debridement of the head from necrotic tissue, followed by packing with bone graft [16].

Platelet-rich plasma therapy has also been described in the literature as used to augment core decompression and bone grafting through increasing levels of the cytokines such as platelet-derived growth factor, and endothelial growth factor, among others to induce angiogenesis and osteogenesis [17].

Osteotomy

Osteotomy involves making cuts in the bone to remove necrotic bone from areas subject to weight-bearing forces. A potential consequence of this procedure is the difficulty in converting from osteotomy to total hip arthroplasty if it is ultimately needed.

Total Hip Arthroplasty (THA)

For patients with major femoral head collapse, THA replaces the necrotic bone of the femoral head with a prosthesis. In most cases, this approach is successful in relieving pain and restoring function. Younger patients may have to undergo a revision at some point in their lifetime, given the longevity of prosthetic hip replacements, lasting on average from 15 to 25 years [8].

Return to Activities

Return to activity is dependent on the patient's current symptoms and function in the context of non-operative or post-operative management. In most cases, returning to competitive sports is rarely a possibility.

Most patients undergoing THA should be able to resume regular, non-impact activities such as walking, swimming, and golf, for example, once comfort allows. High impact activities such as running, and heavy lifting, should be avoided, as well as activities that put the joint through extreme ranges of motion.

Septic Arthritis of the Hip

Septic arthritis refers to an infection of a joint, often due to a bacteria or other microorganisms. Although septic arthritis of the hip is less common compared to that of the knee, high clinical suspicion, and early diagnosis is essential given the destructive nature of septic arthritis on the joint [18], and it's potential for subsequent osteomyelitis, extra-articular abscess formation, pathologic dislocation, and sepsis if left untreated.

Septic arthritis is usually monomicrobial, often caused by Staphylococcus aureus, and MRSA, in adults [19], but can also be caused by other Gram-positive and Gram-negative organisms depending on the mechanism of infection. Most commonly, septic arthritis is a result of hematogenous spread, either through bacteremia or direct inoculation by trauma [18]. Bacterial seeding leads to an inflammatory response and release of cytokines and reactive oxygen species, and subsequent destruction of the joint [19]. Risk factors for septic arthritis include advanced age, preexisting joint disease, recent history of joint surgery or injection, immunocompromised state, intravenous drug use, skin or soft tissue infection, and indwelling catheters.

Clinical Presentation

Patients are often febrile, but septic arthritis should not be ruled out in afebrile patients [19] as this may be indicative of inability to mount an appropriate response to the infection, especially if immunocompromised. Clinical presentation includes joint pain, decreased range of motion or inability to bear weight.

The gold standard for diagnosis remains synovial fluid analysis and culture and should ideally be collected before the initiation of antibiotic treatment for improved diagnostic yield [19]. This can be performed via arthrocentesis using anatomic landmarks, as well as radiographic and ultrasound-guided imaging. Laboratory findings may commonly demonstrate nonspecific biomarkers including elevated white blood counts, sedimentation rates, and

C-reactive protein. Blood cultures should also be obtained to determine the extent of disease and to guide antibiotic treatment [19].

Physical Exam

Patients will commonly present with severe and/or acute onset hip pain, referred pain to the groin, as well as inability to bear weight. Unlike the knee joint, where erythema, warmth, and redness may be found on the physical exam, these findings may be absent or less visible in septic arthritis of the hip joint. Physical exam may reveal pain with passive range of motion, and relief of symptoms with hip flexion and external rotation as this allows the hip capsule to accommodate a greater amount of intra-articular volume [18].

Diagnostic Studies

Although plain radiographs are usually the initial image obtained for its ability to evaluate for arthritic changes, fractures, osteonecrosis, or other bony lesions, this diagnostic study may appear normal early in the disease course [20]. For this reason, additional imaging may be required. Ultrasound of the joint can help evaluate for effusion and better joint visualization during arthrocentesis. MRI is more sensitive for changes associated with fractures, early damage to the cartilage, effusions, osteochondral lesions, osteomyelitis, and extra-articular abscesses [18].

Treatments

Septic arthritis is a medical emergency, and patients with a high-likelihood for diagnosis should be referred to the emergency department for evaluation and work up. Patients admitted for further assessment receive follow-up of labs and cultures, intravenous antibiotics, as well as repeated drainage of the joint, if with

recurrent effusions [21]. Drainage can be done via needle aspiration, arthroscopy, or arthrotomy (open surgical drainage), with arthroscopy recommended for drainage of the hip for improved irrigation. In circumstances where there is a concern for a residual foreign body due to trauma, drainage is inadequate via aspiration or arthroscopy, or effusion persists after 7 days despite serial aspiration, open surgical drainage is recommended [21].

Antibiotic therapy should cover the most likely organism and should be tailored in consultation with infectious disease specialists appropriately as culture and susceptibility results return [22]. Duration of antibiotic therapy generally ranges from 2 to 6 weeks [19] although shorter or longer courses of intravenous and oral antibiotics may be warranted based on the organism isolated, and whether concomitant conditions such as bacteremia, endocarditis, or osteomyelitis are present.

In cases of destruction to the hip joint, and active bacterial infections, a two-stage total hip arthroplasty (THA) may be undertaken given the risk of performing a primary THA in the setting of active septic arthritis due to the risk of periprosthetic joint infection [22]. The first stage involves resection arthroplasty and often implantation of an antibiotic-loaded cement spacer or cement beads followed by the second stage at which time the patient undergoes definitive THA. Single stage or primary THA may be considered for treatment in patients with resolved or quiescent infection of the hip joint, with comorbid conditions, or who prefer to undergo less surgeries [23].

Return to Activities

Return to activity and functional status in the setting of septic arthritis of the hip is largely dependent on the patient's pre-existing comorbid conditions, duration of condition prior to initiation of therapy, and virulence of the infecting organism.

Most patients undergoing THA should be able to resume regular, non-impact activities such as walking, swimming, and golf,

for example, once comfort allows. High impact activities such as running, and heavy lifting, should be avoided, as well as activities that put the joint through extreme ranges of motion to reduce the risk of dislocation.

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Hip Pain Case Studies

10

Nathaniel Milburn and David Tietze

Chronic Hip Pain in a 7-Year-Old

History

A 7-year-old male presents to his pediatrician's office complaining of hip pain that has been present and escalating for the last 4 months. The parents initially attributed the hip pain to his high level of activity in sports but decided to bring him to be evaluated after they noticed him limping. He is normally very active, but with the ongoing hip pain has not practiced for over a week. The rest has not improved his pain at all. They have tried Tylenol and Motrin at home with minimal relief. They cannot recall a specific injury in the preceding 6 months.

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Further Patient Background

Review of systems
No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls.
Past medical Hx/surgeries
Concussion 2 years prior
Medications
Multivitamin
Protein powder
Creatine
Family history
None
Social history
Patient lives at home with his parents as an only child. He plays ice hockey, football, baseball, and runs track.

Initial physical exam findings
BMI: 17.8
Height: 4′3″
Gen: Alert and oriented to person, place, and time
Left hip
Inspection: No obvious deformity
Palpation
- No tenderness to palpation
- Points deep in groin to where pain is worst

Movement: Abduction limited due to pain (5°)
Strength: 5/5
Sensation: Normal
Special exam maneuvers
+ FADIR
+ FABER

Differential Dx (Table 10.1)

Table 10.1 Commonly considered differential diagnoses

Differential	Key findings
Slipped Capital Femoral Epiphysis (SCFE)	Typical patient is African American, obese, and occurs in both boys (age 13) and girls (age 11). Pain originates from hip joint with limited internal rotation and flexion (FADIR). Is a Salter-Harris type I fracture of the femoral head.
Femoral Acetabular Impingement (FAI)	Occurs in young healthy active patients due to formation of abnormal bone in a CAM and/or pincer deformity. Patients will display hip joint pain and limited ROM/pain with FADIR testing.
Legg-Calve-Perthes (LCP)	Osteonecrosis of the femoral head, typically in boys 4–8 years old. Physical exam with joint pain and limited abduction.
Developmental Dysplasia of the Hip (DDH)	Disorder screened for at birth but frequently missed, caused by inadequate coverage of the femoral head by the acetabulum. Patients may or may not have pain in the hip joint and often display hyper-mobility of the joint.
Stress Fracture	Relatively rare in children but may occur in athletes involved in endurance sports. May display a positive "hop test," need to be screened for relative energy deficiency [1].
Juvenile Idiopathic Arthritis (JIA)	May present with isolated joint pain. Patient may also have other joint involvement or subtle skin changes.
Transient Synovitis	Painful hip with limited range of motion. Has been described as a post-viral syndrome, but etiology is still unclear. Lifetime incidence approaches 3% typical age range of 3–8. May lead to LGP in 3.4% [2]
Leukemia (acute lymphoblastic) and other malignancies (osteoid osteoma, Ewing sarcoma, lymphoma, neuroblastoma)	Malignancy commonly presents with musculoskeletal pain, in ALL that rate is approximately 20% [3]. A high index of suspicion for various cancers should be on any musculoskeletal pain differential, especially if atypical or migratory.

Diagnostic Discussion

Hip pain in the pediatric age range is often overlooked and under treated in the general population. Any child with chronic hip pain, severe acute hip pain, or hip pain that continues to worsen with physical activity should be evaluated by a medical professional.

Our patient is having insidious hip pain that continues despite relative rest and has progressed to the point of limping. The physical exam for the hip often does not help distinguish between different diagnoses, as intra-articular processes all share similar findings. Interestingly, many patients that believe hip pain originates near the greater trochanter, while clinically, true intra-articular hip pain is typically noted within the groin. The positive FADIR testing is consistent with many of the diagnoses in the table, and further testing will be required prior to final diagnosis.

DDH is most frequent in girls, babies born breech, babies swaddled tight and may have an inheritable disposition [4]. Newborns are screened for this condition with the Barlow-Ortolani test although sensitivity (26%) is lacking for this purpose [5]. Pain often does not occur until later in life due to hypermobility and development of arthritis.

SCFE and LCP demonstrate an overlapping presentation; however, their typical patient differs. LCP tends to occur in younger patients, is less strongly associated with obesity, and its presentation at a later age may signify a more debilitating course. There is also some association with passive smoke exposure. On the other hand, SCFE patients tend to be older than 10, obese, and African American.

FAI is more commonly seen in the adult population starting around the age of 20. On exam it may demonstrate a block that limits range of motion, or the exam may be limited due to pain. There may be a link between FAI and being Caucasian, certain sports, and genetic disposition. However, more practitioners agree that it is brought on by repetitive movements that stress the bone in some way. Furthermore, given how it most frequently occurs later in life it would be the lowest on our differential prior to further work-up.

Stress fractures of the hip in the pediatric age group are relatively rare but increase in incidence with age. Although the pain may improve with relative rest and limiting weight bearing, the short period of rest this patient underwent would likely not be long enough to see improvement.

JIA typically does not affect solely the hip, preferring the knees and ankle. It is also a diagnosis of exclusion and would require working up the other various etiologies of hip/joint pain. Labs may be normal although ANA may be positive.

Patients with transient synovitis of the hip often present within 1 week of symptoms starting. It may be unilateral or bilateral, and most often do not have a fever. Laboratory analysis should be undergone although clinically it is difficult to distinguish from septic arthritis. Treatment involves the use of NSAIDS to help reduce the recovery time [6].

The various malignancies in the table are a representation of the high index of suspicion orthopedic providers must have for these conditions. ALL in particular may demonstrate bone pain, with children often presenting with a limp [7]. Red flag signs of malignancy include pain being worse at night, weight loss, and fevers.

Workup: Imaging

This X-ray of the patient demonstrates osteonecrosis and obliteration of the left femoral head (Fig. 10.1). One can see a normal appearing right femoral head with an open growth plate. This imaging is diagnostic for LCP disease. However, it should be noted that X-ray may be normal early in the course of disease, and thus normal images cannot rule out this diagnosis.

MRI of the left hip demonstrating collapsed and sclerotic epiphysis. There is also a cystic lesion in the metaphysis (Fig. 10.2).

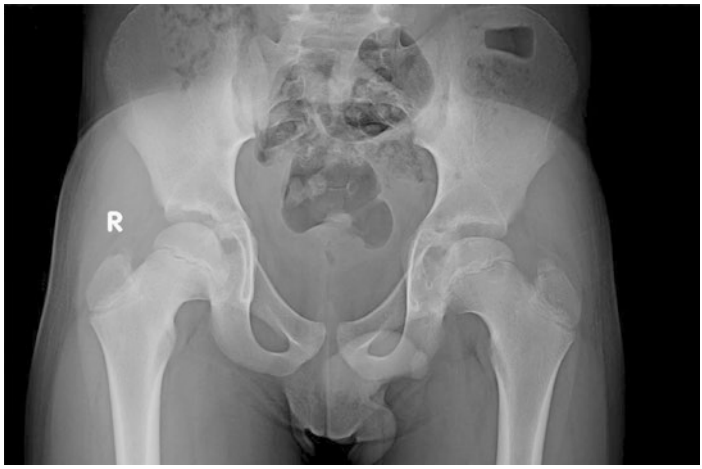

Fig. 10.1 One view AP X-ray of pelvis. (Case courtesy of Dr. Paresh K. Desai, Radiopaedia.org. From the case rID: 19979)

Management

In general, LCP may be treated conservatively with rest and use of crutches to reduce forces through the femoral head. In addition, NSAID for pain control, PT for ROM exercises, and bracing to limit abduction should be discussed.

Should the child be older than 8 at the time of diagnosis, there may be less plasticity of the acetabulum and thus referral to a surgeon for consideration of an osteotomy is appropriate.

Key Take-Aways

1. Hip pain in the pediatric patient should be assessed and worked up appropriately as there are a variety of conditions that can have significant long-term deleterious effects.
2. Pediatric hip pain that is insidious and frequently will have the child continuing to walk on the affected leg include: SCFE, Legg-Calve-Perthes, and DDH.
3. A more acute hip pain in the pediatric patient should alert the provider to a different diagnosis, such as: septic arthritis, malignancy, rheumatologist, and transient synovitis.

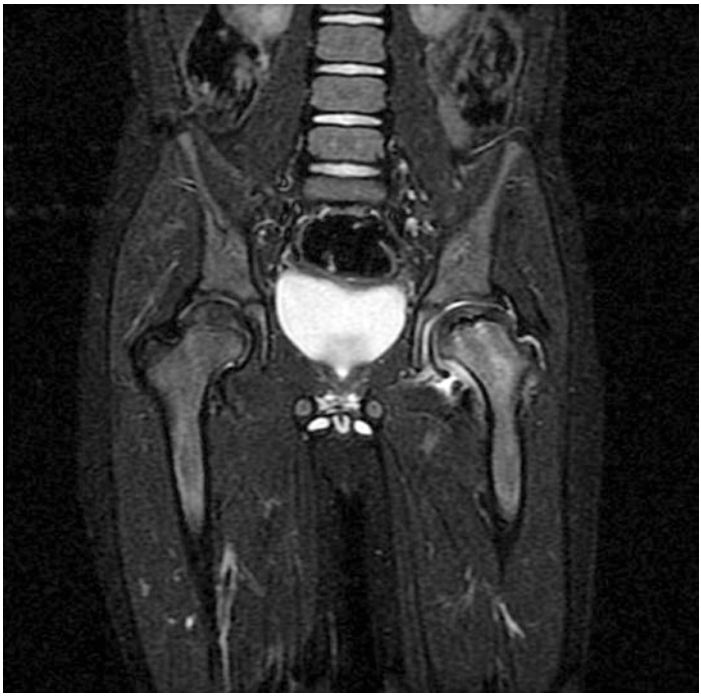

Fig. 10.2 Coronal MRI slice of same pelvis. (Case courtesy of Dr. Ali Abougazia, Radiopaedia.org. From the case rID: 22659)

Traumatic Hip Pain

History

The patient is a 20-year-old male collegiate football quarterback seen on the field after a play is blown dead. The preceding play he was tackled from behind landing on his abdomen, and he has not yet moved from the prone position with clear discomfort. After being approached by the medical staff he informs them that his pain level is 10/10 and he cannot move his left leg.

Further Background

The patient has suffered from multiple orthopedic issues in the past including a torn ACL in his right knee, ATFL sprain in bilateral ankles, a herniated L4 disc, and two prior concussions. There is no notable history of hip pain noted in the preseason physical forms.

Review of systems	**Physical exam findings**
Nothing of note other than the acute injury	*BMI* of 24 kg/m^2
Past medical Hx/surgeries	*Gen*: In acute distress, obvious extreme pain
ACL repair 2 year prior	**Hip**
Discectomy 3 years prior	*Inspection*: Unable to assess due to pads
Medications	*Range of motion*: Left leg internally rotated and shorter compared to right
Pre-game toradol shot for knee pain	*Strength*: Unable to active left leg, possibly due to pain
Family history	*Sensation*: Grossly intact to light tough
None	*Reflexes*: 1+ bilateral patellar and Achilles
Social history	*Tone*: Normal
No ETOH	**Gait**: Unable to walk
	Specialized exam maneuvers
	Unable to test FADIR/FABER on patient due to pain

Differential Dx (Table 10.2)

Workup: Imaging

Right Hip XR

XR hip demonstrating a posterior dislocation (direction is termed which way the femoral head dislocates compared to the acetabulum) (Fig. 10.3).

Table 10.2 Commonly considered differential diagnoses

Differential diagnoses	Key findings
Herniated disc	May occur acutely from injury with variable pain and sensory changes dependent on level of injury. Often displays a positive straight leg test and may cause acute weakness.
Pelvic ring fracture	Painful injury that may occur due to trauma or insufficiency in an older population. If suspected, a physical exam should include a pelvic compression to determine if there is an unstable fracture that "closes."
Dislocated hip	A high force injury that can cause severe pain. Often the patient will also suffer fractures of the femoral head and/or acetabulum during the event.
Femur fracture	A femoral shaft fracture may occur due to high forces and may be a limb threatening emergency. Due to compartment syndrome, may get a variety of symptoms with increased thigh pressures.

Discussion

A posterior dislocated hip is the cause of this patient's symptoms. Often in patients with severe orthopedic pain the physical exam can be unreliable and difficult to perform. Thus, a wide differential should be considered including any limb and/or life-threatening diagnoses. After the most likely diagnosis is determined, appropriate imaging should be obtained.

While a femoral shaft fracture could cause a similar clinical picture, it would not typically cause the internal rotation of the lower extremity [8]. In addition, pain would be felt near the fracture site, most often further down the shaft. A pelvic fracture in similar fashion should be considered, but also would be unlikely to produce the rotation of the lower extremity.

While a herniated disc could cause the extreme pain and weakness, it typically demonstrates a myotomal/dermatomal distribution. Again, it would also be unlikely to cause the internal rotation of the hip found on physical exam.

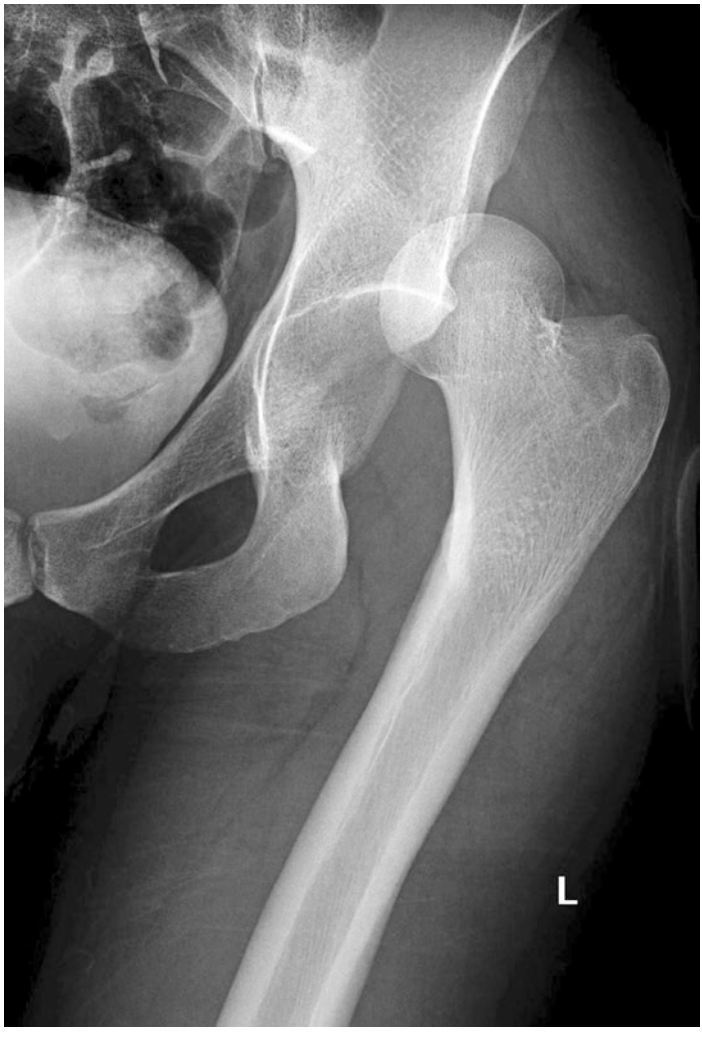

Fig. 10.3 One view X-ray left hip. (Case courtesy of Dr. Hani Makky Al Salam, Radiopaedia.org, rID: 10397)

Management

The most important aspect of hip dislocation treatment is timely reduction by a qualified medical professional which will help reduce the risk of post-reduction AVN [9]. X-rays are typically taken pre-reduction to confirm the diagnosis, and then adequate sedation/pain control should be administered prior to attempting a closed reduction. Post reduction CT scans may be considered to evaluate for associated fractures (especially of the posterior acetabular rim) and loose bodies. Should a closed reduction fail, an open reduction will be required (Fig. 10.4).

In the event there are no fractures or loose bodies, conservative management after reduction should include protected weight bearing for up to 6 weeks and progressive physical therapy. Both fractures and loose bodies will most likely require surgical intervention. Long term, patients may suffer from AVN, post-traumatic osteoarthritis, sciatic nerve injury, and heterotopic ossification [11].

Key Take-Aways

1. Hip dislocations are a high energy injury that require a high level of suspicion and prompt management.
2. Post reduction CT scans should be taken to assess for fractures and loose bodies.
3. Even after successful reduction, patients may encounter long term setbacks including AVN, HO, and osteoarthritis of the hip.

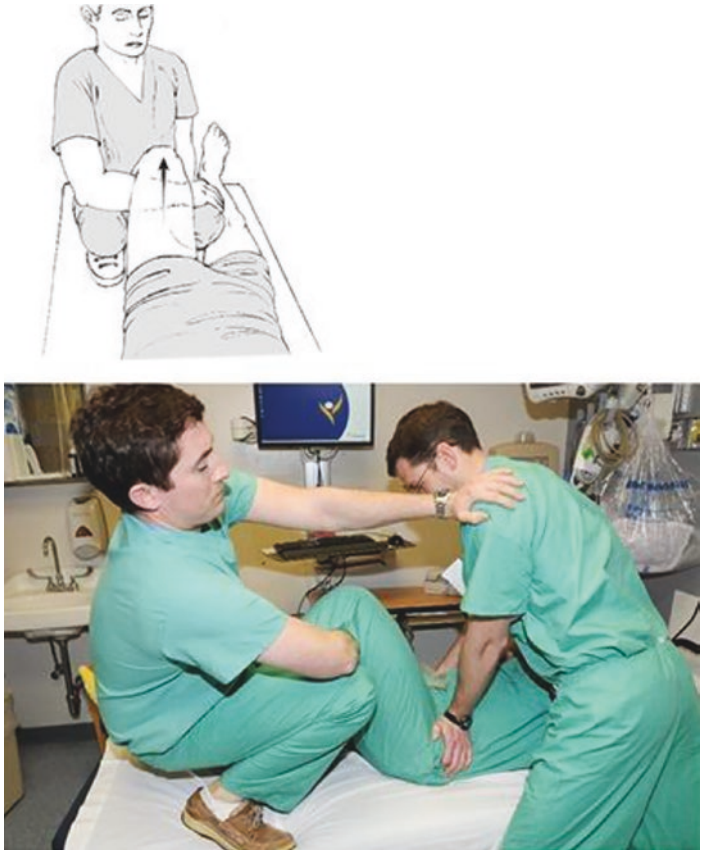

Fig. 10.4 The "Waddell technique" for reducing a hip [10]. (Case courtesy of Dr. Hani Makky Al Salam, Radiopaedia.org, rID: 10397)

Anterior Hip Pain in an Adult Male

History

The patient is a 48-year-old male without a significant past medical history who presented to the clinic with left sided hip pain. The pain is localized to the left anterior hip, is characterized as a

deep, sharp pain graded 3/10 in severity and began over 20 years ago with no acute injury. Mechanical locking, catching and stiffness are also noted. He denies pain with ambulation. The pain is aggravated by sitting for long periods of time, twisting the wrong way, and running. He has tried activity modification and Meloxicam with mild temporary alleviation of symptoms.

Review of systems No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls. **Past medical Hx/surgeries** None **Medications** Meloxicam as needed **Family history** None **Social history** Running, lifting weights	**Physical exam findings** *BMI* of 23.4 kg/m^2 *Gen*: Not in acute distress, well developed **Hip** *Inspection*: no bony abnormalities, no muscular atrophy *Palpation*: Non tender to palpation *Range of motion* – 100° of flexion – 70° of external rotation – 15° of internal rotation. *Strength*: 5/5 in flexion, extension, adduction and abduction *Sensation*: Grossly intact to light touch *Reflexes*: 1+ bilateral patellar and Achilles *Specialized testing*: Reported pain reproduced with flexion, adduction and internal rotation **Gait**: Slight limp **Specialized exam maneuvers** – Pain reproduced with flexion, adduction and internal rotation – No pain with flexion, abduction and external rotation – Negative on log roll – Negative for heel strike

Differential Dx (Table 10.3)

Workup: Imaging

Left Hip X-Ray

Diagnostic Discussion

In this patient with anterior hip pain, the differential can be narrowed through obtaining a thorough history and exam maneuvers that help pinpoint internal vs. external hip pathology. The absence of notable trauma on history makes a significant ligament or tendon tear unlikely. The patient's age, lack of trauma history and decreased motion on internal rotation, suggest a degenerative condition. However, since he has had symptoms for over 20 years, a

Table 10.3 Commonly considered differential diagnoses

Differential diagnoses	Key findings
Acetabular labral tear	Typically caused by repetitive impact to the hip. Flexion, adduction and internal rotation can reproduce symptoms.
Femoroacetabular Impingement	Structural anterior hip pain that is associated with CAM and Pincer lesions. Pain reproduced with hip flexion, adduction and internal rotation.
Hip flexor strain	Pain is reproducible upon resisted hip flexion.
Athletic pubalgia	Occurs acutely or chronically. Pain is often reproduced upon a resisted sit-up or hip adduction. Palpation of the pubic symphysis can also illicit symptoms.
Stress fracture	Often caused by repetitive stress on the hip, associated with swelling and pain, positive log roll and/or heel strike testing.
Osteoarthritis	Depending on the stage of osteoarthritis, pain can be intermittent & sharp with associated stiffness, with or without chronic aching pain. Worsened by joint use and relieved by rest. Joint deformity and swelling can also be present. Age and previous joint trauma history are important to consider with this diagnosis.

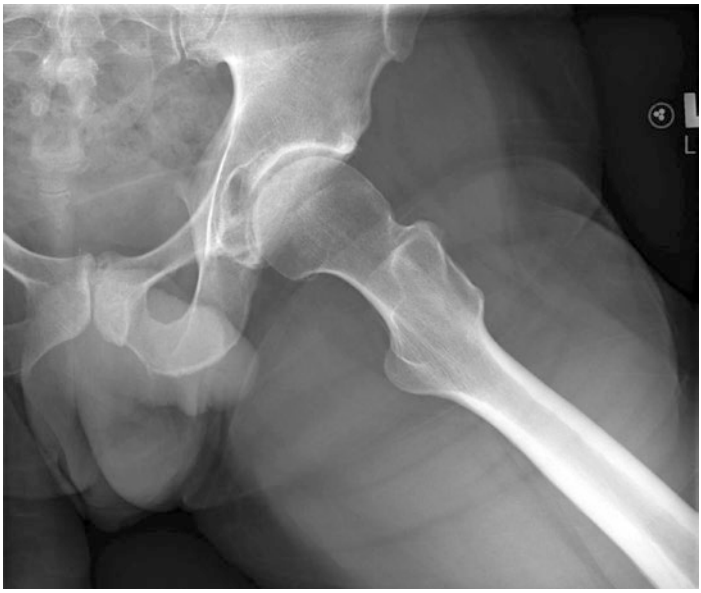

Fig. 10.5 Cam morphology with mild obvious femoral neck anterolateral convexity

congenital etiology for motion loss should be considered as well. Full strength without reproduction of symptoms upon flexion, make a flexor strain less likely as well. An acetabular labral tear could be considered due to mechanical symptoms noted in the patient's history.

Femoroacetabular impingement should be considered in patients with a history of hip pain and decreased range of motion (Fig. 10.5). Although commonly referred anteriorly, the pain has been described laterally and posterior as well. X-ray radiography can help discern structural causes of intra-articular pathology such as an overt fracture, arthritis, or Cam and Pincer lesions in FAI. CT can also be used to identify structural causes. MRI arthrogram can be used to evaluate the acetabular labrum, other soft tissues, and occult fractures. This patient has Femoroacetabular impingement.

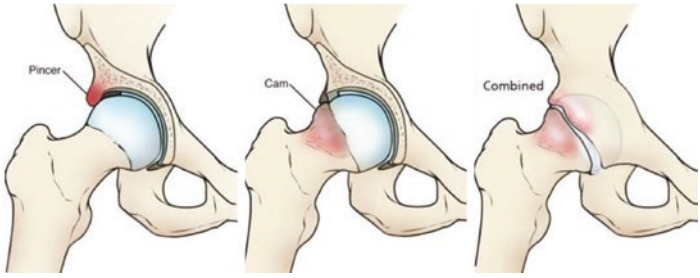

Fig. 10.6 Cam, Pincer and Combined lesions in the hip [12]

Femoroacetabular impingement with associated labral tear is the most likely cause of this patient's pain and dysfunction. Pain on FADIR test and reduced motion point towards an intra-articular pathology (Fig. 10.6).

It is important to distinguish FAI from osteoarthritis for purposes of treatment. Whereas osteoarthritis includes a deterioration of articular cartilage, FAI is an overgrowth of bone on the femoral neck, acetabulum, or both. It is, however, possible to have both FAI and osteoarthritis in the same hip joint as FAI is a risk factor for the development of osteoarthritis [13].

Management

Conservative, nonoperative management of Femoroacetabular impingement is preferred in patients who have minimal symptoms or lack mechanical symptoms. A regimen for this patient may include the following, with gradual resolution of symptoms.

- Nonsteroidal anti-inflammatory drugs
- Physical therapy

If there is no improvement in symptoms after undergoing an appropriate course of conservative therapy, or symptoms progress, it is reasonable to refer to Orthopaedic surgery for operative management.

Intra-articular steroid injections should be used sparingly in this condition. Due to evidence suggesting corticosteroids have a detrimental effect on articular cartilage, it may be prudent to avoid use in younger, non-arthritic patients, opting instead for earlier surgical intervention [14].

Operative management should be considered with failure of conservative therapy, mechanical symptoms, or patients with minimal arthritic symptoms.

Operative treatment for these fractures includes either an arthroscopic osteotomy, open osteotomy, or both in some cases. Athletes can expect to return to play about 7 months after surgical intervention.

Key Take-Aways

1. Diagnosing Femoroacetabular impingement includes a thorough history, relevant clinical exam and imaging to support the diagnosis.
2. Physical exam maneuvers such as a positive impingement sign are useful in diagnosing a Femoroacetabular impingement. Confirmatory testing with X-rays, CT or MRI can help show a Cam and/or Pincer lesion.
3. Conversative management is preferred in patients with minimal, non-mechanical symptoms. Operative management should be decided based on a case-by-case basis and may be preferred in younger patients for hip preservation.

Anterior Hip Pain in a Young Female

History

The patient is a 17-year-old female without significant past medical history who presents to the clinic with left sided hip pain. The pain is localized to the left anterior hip, is characterized as a progressive sharp pain graded 6/10 in severity and began 3 weeks ago with no acute injury. She denies having pain with ambulation. About 6 weeks previously she increased her workouts to include a boot camp that focused on plyometrics. The pain is aggravated

by sitting for long periods of time and getting up in the morning. She has tried naproxen, ice, heat and massage with mild temporary alleviation of symptoms.

Further Background

In addition to the boot camp, the patient also runs 25+ miles a week and does not engage in resistance training. Menstrual cycles have been regular and consistent. Diet is restricted by a self-diagnosed gluten-allergy.

Review of systems No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls. **Past medical Hx/surgeries** None **Medications** Naproxen as needed **Family history** None **Social history** Works out 6 days a week	**Physical exam findings** *BMI* of 19.5 kg/m^2 *Gen*: Not in acute distress, well developed **Hip** *Inspection*: No bony abnormalities, no muscular atrophy *Palpation*: Non tender to palpation *Range of motion*: – 135° of flexion – 90° of external rotation – 30° of internal rotation *Strength*: 5/5 in flexion, extension, adduction and abduction *Sensation*: Grossly intact to light touch *Reflexes*: 1+ bilateral patellar and Achilles *Specialized testing*: Reported pain reproduced with flexion, adduction and internal rotation **Gait**: Normal **Specialized exam maneuvers** – Pain reproduced with flexion, adduction and internal rotation – No pain with flexion, abduction and external rotation – Negative on log roll – Positive for heel strike

Differential Dx (Table 10.4)

Workup: Imaging

Table 10.4 Commonly considered differential diagnoses

Differential diagnoses	Key findings
Acetabular labral tear	Typically caused by repetitive impact to the hip. Flexion, adduction and internal rotation can reproduce symptoms.
Femoroacetabular Impingement	Structural anterior hip pain that is associated with CAM and Pincer lesions. Pain reproduced with hip flexion, adduction and internal rotation.
Hip flexor strain	Pain is reproducible upon resisted hip flexion.
Athletic pubalgia	Occurs acutely or chronically. Pain is often reproduced upon a resisted sit-up or hip adduction. Palpation of the pubic symphysis can also illicit symptoms.
Stress fracture	Often caused by repetitive stress on the hip, associated with swelling and pain, positive log roll and/or heel strike testing.
Osteoarthritis	Depending on the stage of osteoarthritis, pain can be intermittent & sharp with associated stiffness, with or without chronic aching pain. Worsened by joint use and relieved by rest. Joint deformity and swelling can also be present. Age and previous joint trauma history are important to consider with this diagnosis.

Left Hip X-Ray (Fig. 10.7)

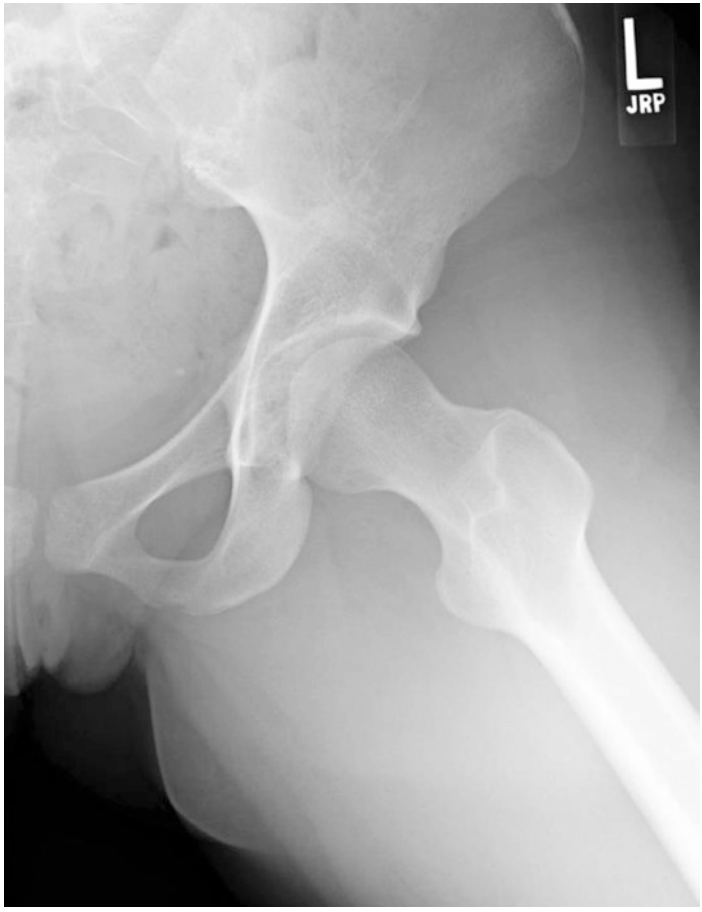

Fig. 10.7 Cranial acetabular retroversion. Joint space is maintained

Left Hip MRI Arthrogram (Fig. 10.8)

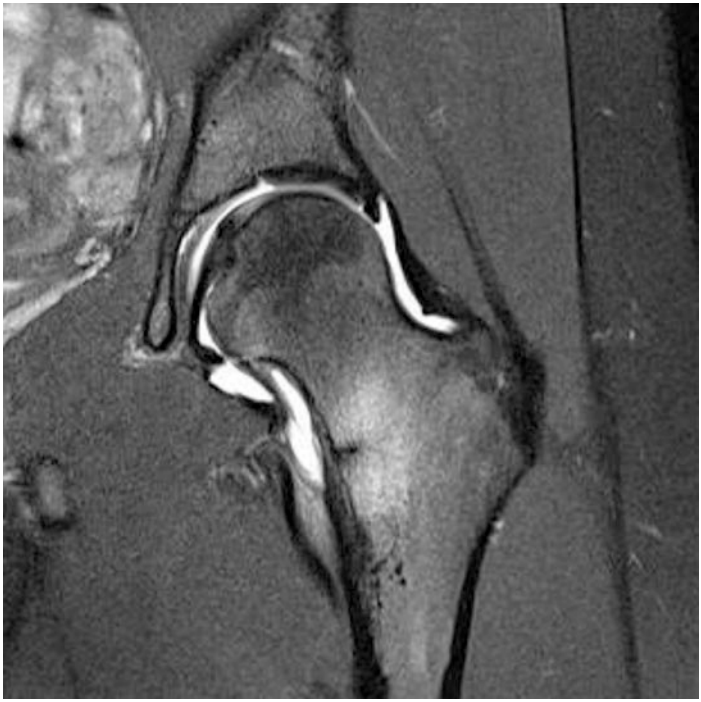

Fig. 10.8 Nondisplaced stress fracture pattern at medial trochar, involving only one quarter of the width of proximal femur with moderate surrounding bone marrow edema suggest ongoing mechanical stress and/or reparative inflammation

Diagnostic Discussion

In this patient with anterior hip pain, the differential can be narrowed through obtaining a thorough history and exam maneuvers that help pinpoint specific structural damage. The absence of notable trauma in history makes a significant ligament or tendon tear unlikely. The patient's age, lack of trauma history and full/symmetric range of motion limits the likelihood of a degenerative condition. Full strength without reproduction of symptoms upon flexion, make a flexor strain less likely as well. An acetabular labral tear could be considered due to the presence of reproducible pain upon intra-articular stress.

Femoral neck stress fractures should be considered in patients with a history revealing repetitive impact and/or increase in activity level. Compounding factors would include low BMI, dietary restrictions, and other signs of relative energy deficiency in sport (RED-S). X-ray radiography can help discern structural causes of intra-articular pathology (overt fracture or arthritis). A three-phase bone scan can be used for suspected stress fractures by utilizing a radioactive tracer to see an area of high bone turnover. It is highly specific, so a negative scan rules out the possibility of a stress fracture [15]. MRI Arthrogram can be used to evaluate the acetabular labrum, other soft tissues, and occult fractures. This patient has a femoral stress fracture.

Procedures and lab tests
Serum calcium, vitamin D_2, TSH, PTH, ferritin, CBC, metabolic panel, and appropriate sex hormones can be used to see if there is a secondary cause for the fracture to have developed. A DEXA scan can be used to determine bone density in appropriate patients.

A stress fracture of the femur is the most likely cause of this patient's pain and dysfunction. Pain on heel strike and FADIR tests (Fig. 10.9) point towards an intra-articular pathology that likely is secondary to repetitive impact on the hip. With her dietary restriction, there is concern for a nutritional deficiency that may be reducing the body's energy reserve.

Fig. 10.9 FADIR testing [16]

While stress fractures often occur in activities that involve repetitive impact, older patients may obtain insufficiency fractures with little to no trauma. A history of osteoporosis or osteopenia can often contribute to this diagnosis. Confirmatory testing can be done through MRI, especially for small defects or if the patient is still early in the course. Radiograph and ultrasound can be used as initial imaging modalities but are limited by their inability to fully visualize occult pathology.

Management

Conservative, nonoperative management of femoral neck stress fractures is preferred in patients who have a compression (inferior) sided fracture or a fracture line that is less than 50% of the femoral neck width. A regimen for this patient may include the following, with gradual resolution of symptoms over 6–8 weeks.

- Offloading the hip with crutches until fully weight bearing without pain
- Acetaminophen for pain relief
- Physical therapy
  - Goals are to recondition after a period of immobilization

If there is no improvement in symptoms after undergoing an appropriate course of conservative therapy, or symptoms progress to an overt fracture, it is reasonable to refer to Orthopaedic surgery for operative management.

Intra-articular steroid injections should not be administered in patients with a stress fracture.

Operative management should be considered with failure of conservative therapy, displaced fractures, compression sided fractures with a fracture through more than 50% of the femoral neck width, or tension sided (superior) fractures.

Operative treatment for these fractures includes open reduction and internal fixation with three 6.5 or 7.0 mm cannulated screws through the femoral neck [17].

Areas of Research

- Most patients will improve with a conservative approach, but for elite and professional athletes' biologic options such as PRP and BMAC may offer a minimally invasive option that can expedite healing and allow for earlier return to sports participation [18]

Key Take-Aways

1. Diagnosing a stress fracture includes the history of rapidly increasing activity, gradually increasing pain with activity and a high clinical suspicion.
2. Physical exam maneuvers such as the log roll, heel strike and FADIR are useful in diagnosing stress fractures. Confirmatory testing with MRI or three phase bone scan is indicated if patients meet criteria with normal X-rays.
3. Conversative management is preferred in patients with compression (inferior) sided fractures that do not involve over 50% of the femoral neck width. Initial immobilization followed by a progressive, gradual therapy program is preferred in most patients. Operative management (ORIF) should be decided based on the specific fracture pathology or failure of conservative measures.

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