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.

References

1. Cifu DX. Braddom's physical medicine and rehabilitation. Philadelphia: Elsevier Health Sciences; 2020.
2. Frontera WR, DeLisa JA, Gans BM, Robinson LR. DeLisa's physical medicine and rehabilitation: principles and practice. Philadelphia: Lippincott Williams & Wilkins; 2019.
3. Feger J. Osteoarthritis of the hip: radiology reference article. Radiopaedia Blog RSS. 10 Dec 2021. Retrieved 10 Jan 2022 from https://radiopaedia.org/articles/osteoarthritis-of-the-hip?lang=us.
4. Slobodin G, Hussein H, Rosner I, Eshed I. Sacroiliitis–early diagnosis is key. J Inflamm Res. 2018;11:339.
5. Krueger C, Garfin S. Sacroiliitis. Orthobullets. 2020. Retrieved 10 Jan 2022 from https://www.orthobullets.com/spine/2040/sacroiliitis.
6. Bernard SA, Kransdorf MJ, Beaman FD, Adler RS, Amini B, Appel M, et al. ACR Appropriateness Criteria® chronic back pain suspected sacroiliitis-spondyloarthropathy. J Am Coll Radiol. 2017;14(5):S62–70.
7. Asadipooya K, Graves L, Greene LW. Transient osteoporosis of the hip: review of the literature. Osteoporos Int. 2017;28(6):1805–16.
8. Lespasio MJ, Sodhi N, Mont MA. Osteonecrosis of the hip: a primer. Perm J. 2019;23 https://doi.org/10.7812/TPP/18-100.
9. Wang Y, Li Y, Mao K, Li J, Cui Q, Wang GJ. Alcohol-induced adipogenesis in bone and marrow: a possible mechanism for osteonecrosis. Clin Orthop Relat Res. 2003;410:213–24.
10. Beckmann R, Shaheen H, Kweider N, Ghassemi A, Fragoulis A, Hermanns-Sachweh B, et al. Enoxaparin prevents steroid-related avascular necrosis of the femoral head. Sci World J. 2014;2014:347813.
11. Madden CC, Netter FH. Netter's sports medicine. Philadelphia: Saunders/Elsevier; 2010.
12. Laporte DM, Mont MA, Mohan V, Jones LC, Hungerford DS. Multifocal osteonecrosis. J Rheumatol. 1998;25(10):1968–74.
13. Zhang QY, Li ZR, Gao FQ, Sun W. Pericollapse stage of osteonecrosis of the femoral head: a last chance for joint preservation. Chin Med J. 2018;131(21):2589–98.
14. Cohen-Rosenblum A, Cui Q. Osteonecrosis of the femoral head. Orthop Clin N Am. 2019;50(2):139–49.

15. Jager M, Jelinek EM, Wess KM, Scharfstadt A, Jacobson M, Kevy SV, Krauspe R. Bone marrow concentrate: a novel strategy for bone defect treatment. Curr Stem Cell Res Ther. 2009;4(1):34–43.
16. Sultan AA, Khlopas A, Surace P, Samuel LT, Faour M, Sodhi N, et al. The use of non-vascularized bone grafts to treat osteonecrosis of the femoral head: indications, techniques, and outcomes. Int Orthop. 2019;43(6):1315–20.
17. Han J, Gao F, Li Y, Ma J, Sun W, Shi L, et al. The use of platelet-rich plasma for the treatment of osteonecrosis of the femoral head: a systematic review. BioMed Res Int. 2020;2020:2642439.
18. Lum ZC, Shieh AK, Meehan JP. Native adult hip with bacterial septic arthritis. JBJS Rev. 2018;6(10):e2.
19. Mathews CJ, Weston VC, Jones A, Field M, Coakley G. Bacterial septic arthritis in adults. Lancet. 2010;375(9717):846–55.
20. Hassan AS, Rao A, Manadan AM, Block JA. Peripheral bacterial septic arthritis: review of diagnosis and management. JCR J Clin Rheumatol. 2017;23(8):435–42.
21. Nair R, Schweizer ML, Singh N. Septic arthritis and prosthetic joint infections in older adults. Infect Dis Clin. 2017;31(4):715–29.
22. Hipfl C, Karczewski D, Oronowicz J, Pumberger M, Perka C, Hardt S. Total hip arthroplasty for destructive septic arthritis of the hip using a two-stage protocol without spacer placement. Arch Orthop Trauma Surg. 2021:1–10. https://doi.org/10.1007/s00402-021-03981-2.
23. Papanna MC, Chebbout R, Buckley S, Stockley I, Hamer A. Infection and failure rates following total hip arthroplasty for septic arthritis: a case-controlled study. Hip Int. 2018;28(1):63–7.
24. Gonzalez-Fernandez M, Schaaf S, editors. Handbook of physical medicine and rehabilitation. New York: Springer Publishing Company; 2021.
25. Jacobson J, Girish G, Jiang Y, Sabb B. Radiographic evaluation of arthritis: degenerative joint disease and variations. Radiology. 2008;248(3):737–47.
26. Lespasio MJ, Sodhi N, Mont MA. Osteonecrosis of the hip: a primer. The Permanente Journal. 2019;23. p. 18–100. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6380478/.
27. Lespasio MJ, Sultan AA, Piuzzi NS, Khlopas A, Husni ME, Muschler G F, Mont MA. Hip osteoarthritis: a primer. The Permanente Journal. 2018;22
28. Watts E, S Incavo et al. “Hip Osteoarthritis.” Orthobullets; 2022. https://www.orthobullets.com/recon/5005/hiposteoarthritis. updated 3/8/2022.

29. Felson DT, Nevitt MC, Yang M, Clancy M, Niu J, Torner JC, Zhang Y. A new approach yields high rates of radiographic progression in knee osteoarthritis. The Journal of rheumatology. 2008;35(10):2047–54.
30. Cooper C, Inskip H, Croft P, Campbell L, Smith G, Mclearn M, Coggon D. Individual risk factors for hip osteoarthritis: obesity, hip injury and physical activity. American journal of epidemiology. 1998;147(6):516–522.
31. Aresti N, Kassam J, Nicholas N, Achan P. Hip osteoarthritis. BMJ. 2016:354.