Acute Disorders of the Joints and Bursae

Radiographic Clues to Diagnosis

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The majority of joint disorders are nonemergent, but the possibility of patients presenting to the ED with a true emergency, such as septic arthritis, cannot be overlooked. The etiologies of joint pain are varied, and it is often challenging to differentiate between chronic and acute disease. However, radiographs can help point to diagnosis and treatment and elucidate whether there is a need for further imaging or referral. Herein, warning signs are reviewed and a radiographic guide is provided to aid in the diagnosis of disorders of the joints and bursae.

Joint pain is an exceedingly common complaint, causing more than 1.7 million people to seek care in EDs throughout the country every year.[1] While arthralgias can affect individuals in all age ranges, their prevalence increases exponentially with age. According to CDC figures, half of people 65 years and older have been diagnosed with some form of arthritis.[2] While the majority of cases are nonemergent, a small percentage of patients with a joint complaint may have a true medical emergency that warrants immediate intervention. Adding to the complexity are the many differing etiologies of arthralgias. Often, the challenge for the emergency physician is to differentiate between exacerbations of chronic disease and an acute, potentially debilitating or even life-threatening disease process, while providing care for many patients in a busy ED.

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This article highlights warning signs related to disorders of the joints and bursae that may help the provider recognize the need for referral, specific treatment, or further diagnostic tests. Key features of the most common and/or serious arthropathies are summarized in the Table.

OSTEOARTHRITIS

Osteoarthritis, or degenerative joint disease, is one of the most common causes of joint pain in the general population. Severe osteoarthritis has the potential to cause significant functional disability, especially in patients older than 65. According to one study, nearly 20% of people in this age-group have osteoarthritis of the knee.[3] Prevalence increases dramatically with age.[4]

Symptoms generally consist of progressive pain in one or more joints, crepitus, joint tenderness, and decreased range of motion and commonly manifest in

TABLE. Characteristics and Exam Findings of Various Arthropathies

	Osteoarthritis	Inflammatory Arthritis	Septic Arthritis
History	Chronic; insidious onset; common in the elderly	Chronic with flares	Acute onset; fever; non–weight-bearing
Physical exam	Polyarticular	Polyarticular; PIP/MCP joints	Monoarticular; erythema; warmth
Synovial fluid:			
Appearance	Transparent straw-yellow	Cloudy or transparent yellow	Purulent/bloody
WBC count (cells/μL)	<2,000 (<25% PMN cells)	200–50,000 (>50% PMN cells)	>50,000 (>50% PMN cells)
Culture	Negative	Negative	>50% positive

PIP = proximal interphalangeal; MCP = metacarpophalangeal; WBC = white blood cell; PMN = polymorphonuclear.
Information extracted from Brent[10] and Burton.[11]

patients older than 60 years.[4] In addition to articulate cartilage deterioration and osteophyte formation seen on plain radiographs of patients with osteoarthritis, MRI reveals changes to subchondral bone, synovium, and meniscus. Bone marrow lesions seen on MRI in subchondral bone predict worsening osteoarthritis. In advanced disease states, MRI often reveals subchondral cyst-like lesions and flattening of the articular surface, or subchondral bone attrition, which is not related to trauma. Researchers postulate that the pain of this disease is likely secondary to synovitis that occurs as cartilage slowly deteriorates. As the synovium thickens and produces effusions within the joint spaces, the result is pain, which is exacerbated by range of motion.[5]

Not surprisingly, MRI is neither possible nor practical for the evaluation of joint pain in many EDs. However, there is little evidence to support the decision to obtain plain radiographs of a joint in a patient with established osteoarthritis. Of course, it is not unusual for patients to present to the ED with symptoms consistent with osteoarthritis but no previous diagnosis. Such cases may represent an initial presentation of osteoarthritis or of another arthropathy with a symptomatic profile similar to that of osteoarthritis. For these patients or for any patient with joint pain that raises suspicion for alternate etiologies, radiographs may be helpful.

Radiographic findings of osteoarthritis include joint space narrowing and osteophyte formation (Figure 1). There is a strong correlation between radiographic evidence of osteoarthritis and complaints of stiffness, aching, crepitus, and pain in the affected joint.[6] Radiographs are warranted only if the patient complains of the above symptoms for more than 4 weeks without improvement after conservative care, ie, rest and NSAID use. Additionally, radiographs may provide useful information if some other underlying pathology is suspected.[4]

Approximately one-third of patients with radiographic evidence of osteoarthritis will have a joint effusion.[7] Moderate to large effusions are associated with knee pain and are common in advanced osteoarthritis. These effusions are caused by damage to ligaments, menisci, and cartilage. Occasionally, loose bodies may form. Characteristic of severe osteoarthritis, loose bodies may consist of either chondral or meniscal fragments or detached osteophytes. As they migrate within the joint space, loose bodies can cause inflammation in a joint that is already damaged. Since physical exam is not usually diagnostic, patients with a suspiciously painful joint in which more serious etiologies have been excluded should be referred to an orthopedist for arthroscopy or MRI.[5]

Treatment of osteoarthritis consists of weight loss

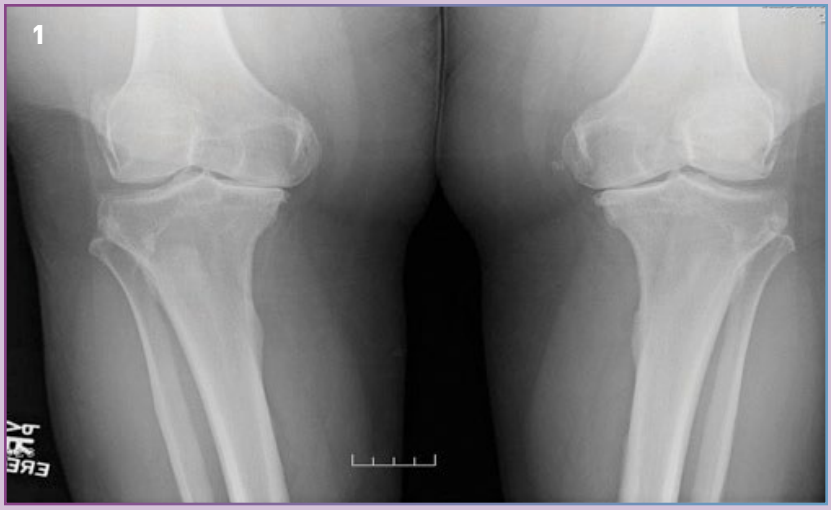

Figure 1. Osteoarthritis of the knees with degenerative changes, including joint space narrowing and osteophytes.

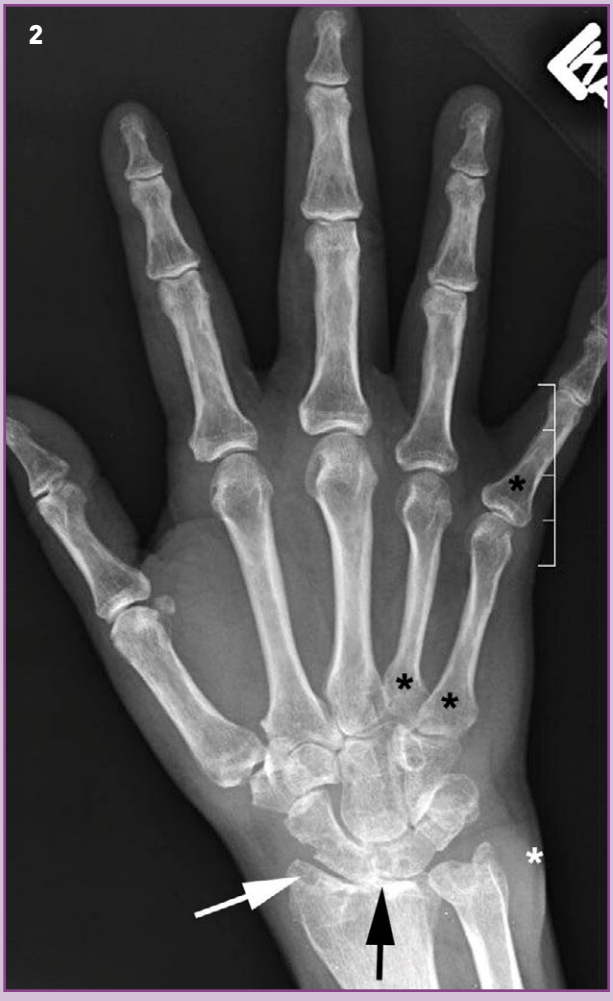

Figure 2. PA view of the hand in a patient with RA. Patients who present to the ED with a previously undiagnosed inflammatory arthropathy typically have early disease, rather than the much more radiographically apparent advanced disease. One of the early findings of an inflammatory arthropathy is periarticular osteopenia (black asterisks), which occurs as a result of inflammation-induced hyperemia. Focal soft tissue swelling may be seen surrounding involved joints or at sites of other commonly involved soft tissue structures, such as the extensor carpi ulnaris tendon (white asterisk) in the hand. As the disease progresses and cartilage is destroyed, periarticular erosions (white arrow) and subsequent joint space narrowing (black arrow) are seen. If these early inflammatory changes are absent or equivocal, MRI with contrast may be utilized to directly evaluate for evidence of synovial inflammation.

Images courtesy of Keith D. Hentel, MD

(for overweight patients), modified exercise programs, and low-impact aerobics, including range-of-motion exercises and strengthening of the quadriceps muscles. Exacerbations of chronic osteoarthritis should be treated with acetaminophen, NSAIDs (topical or oral), and COX-2 inhibitors. Although rarely performed in the ED, intra-articular injections of corticosteroids can offer short-term relief. Referral for arthroscopy, on the other hand, has been shown to be effective only for treatment of intra-articular loose bodies, and is not diagnostic or otherwise therapeutic.[8]

Another common finding in patients with advanced degenerative joint disease is a fluid collection in the popliteal space, commonly known as a Baker cyst. This is a misnomer, as the fluctuance palpated is actually synovial fluid that has been extruded from the posteromedial joint capsule and is not a discrete collection. The synovial fluid collects between the semimembranous and medial gastrocnemius bursae when there is increased pressure in the joint from meniscal tears or large effusions. Baker cysts are clinically significant because there is a small risk of rupture or hemorrhage into the popliteal space. Signs of rupture include calf pain, which may be confused with DVT. After DVT has been ruled out, patients with Baker cyst rupture should be treated with rest, elevation, and intra-articular triamcinolone. This can be administered directly into the joint space of the anterior knee, since it communicates with the Baker cyst.[9]

INFLAMMATORY ARTHRITIS

Rheumatoid arthritis (RA) is the most common form of inflammatory arthritis, affecting one in every 100 adults in the United States.[10] Undiagnosed patients who have this condition may initially present to the ED. RA should be suspected in patients with joint pain and morning stiffness lasting longer than 15 minutes, fatigue, tenderness, swelling, and painful range of motion.[10] It is two to three times more common in women than men, and RA typically has an earlier age at onset than does osteoarthritis, with which RA is often confused. This disorder is characteristically progressive, polyarticular, and symmetric in its presentation.[11]

Due to the progressive nature of inflammatory arthritis, patients who do not receive specialized intervention are at risk for joint destruction and subsequent disability. This is especially true in women, in whom the progression of disease is often accelerated compared to that in men. Identifying patients with RA is important also because of the myriad comorbidities with which it is associated. Increased risk of cardiovascular disease and a predisposition to infection are concerns of particular relevance for the emergency physician.[10]

Since treatment delays such as misdiagnosing new-onset RA as osteoarthritis can lead to substantial disability in as little as 4 months, patients should be referred to a rheumatologist promptly if they meet the following criteria: swelling in three or more joints, prolonged morning stiffness, or involvement of the proximal interphalangeal, metacarpophalangeal, or metatarsophalangeal joints.[10] The distal interphalangeal joint is usually spared. Radiographic findings in inflammatory/rheumatoid arthritis are depicted in Figures 2, 3, and 4.

In general, acute flares in patients with previously diagnosed RA should be treated symptomatically. NSAIDs and corticosteroids have long been considered the primary treatment. Disease-modifying antirheumatic drugs (DMARDs), such as methotrexate and tumor necrosis factor α (TNF-α) inhibitors, have previously been reserved for advanced disease. However, they are now being employed by rheumatologists not only to drive early RA into remission but also as maintenance therapies. Early, intensive therapy has increasingly been shown to slow progression of joint damage. This kind of treatment requires close follow-up with a specialist. Therefore, the main goals of the emergency physician are to rule out septic arthritis, treat acute flares symptomatically, and make rheumatology referrals in a timely manner.[12]

ANKYLOSING SPONDYLITIS

Ankylosing spondylitis is a less common form of inflammatory arthritis, affecting approximately 0.1% of the population.[10] Aside from considerable peripheral arthritis, hallmarks of this debilitating disease include back pain and decreased spinal mobility. Patients with ankylosing spondylitis are also predisposed to uveitis and inflammatory bowel disease; in addition, the incidence of cardiovascular disease in these patients is even

Figure 3. AP view of the knee. In large joints, an inflammatory arthritis affects the entire synovium, resulting in narrowing of all joint space compartments, as observed in this radiograph showing diffuse narrowing of both the medial (white arrow) and lateral (black arrow) tibiofemoral joint spaces. This differs from the asymmetric pattern of joint space narrowing seen in osteoarthritis, which occurs due to "wear and tear" within the joint.

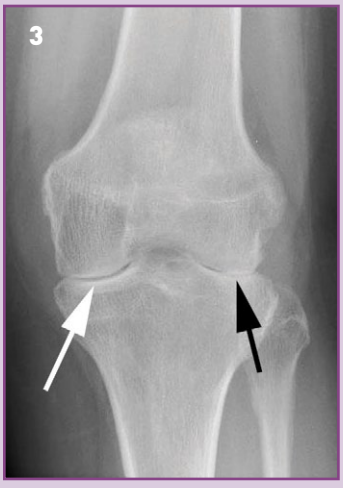

Figure 4. PA view of the hand. In advanced inflammatory arthritis, the synovial erosions result in osseous destruction, demonstrated in this radiograph by the absence of the distal ulna (black arrow), diffuse loss of joint spaces as seen in the carpal joints (red asterisk), and subluxations and dislocations as seen in the metacarpophalangeal joints (white arrows). The ulnar deviation of the subluxed phalanges as seen in this case is typical for rheumatoid arthritis. In cases of advanced inflammatory arthritis, advanced diagnostic imaging such as CT and MRI should be utilized to evaluate for specific complications such as pathologic fracture or infection.

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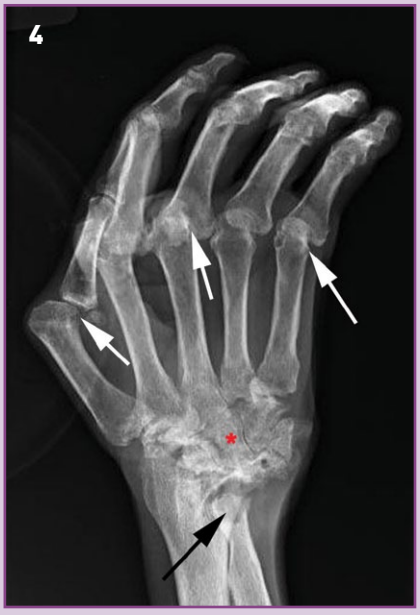

higher than that in patients with RA.[10]

Signs and symptoms of ankylosing spondylitis include chronic lower back pain that lasts more than 3 months and morning stiffness that lasts longer than 30 minutes but improves with exercise. The disease occurs in persons younger than 45 years. These patients also usually test positive for the HLA-B27 serotype. Diagnosis is made by evidence of sacroiliitis on radiographs (Figure 5).[10] In advanced disease, plain radiography may also reveal the characteristic "bamboo spine" that is caused by the fusing of vertebral bodies.[11]

REACTIVE ARTHRITIS

Formerly known as Reiter syndrome, reactive arthritis is an inflammatory arthritis that occurs in response to an infectious etiology. Symptoms often follow gastrointestinal illnesses caused by *Salmonella, Shigella,* and *Campylobacter* species but may also be seen with sexually transmitted infections such as chlamydia. If chlamydia is detected, treatment with doxycycline or erythromycin is warranted. Sexual partners should be treated as well.[13] Unfortunately, research does not demonstrate a symptomatic benefit greater than that achieved with placebo in the treatment of *Salmonella,*

Shigella, Campylobacter, and *Yersinia* infections in patients who have reactive arthritis.[14] In general, patients should be treated symptomatically and referred to a rheumatologist if symptoms persist for more than 6 months. In such cases, patients occasionally benefit from treatment with DMARDs or sulfasalazine.[13]

CRYSTAL ARTHROPATHIES

Gout is the most common inflammatory arthritis in men older than 40 years. It is caused by the precipitation of uric acid crystals within the joint spaces of individuals with hyperuricemia (>7 mg/dL). The classic initial presentation of the disease consists of intermittent severe attacks of pain in a single joint, specifically the first metatarsophalangeal joint. As the disease progresses, attacks may become more frequent but are often less severe. Although gout is usually monoarticular, it can be polyarticular in women. It also has a later age at onset in women, typically after age 60.[15]

Acute gouty arthritis usually presents with rapid onset of pain in a single joint, progressing to severe pain in a matter of hours. The affected joint will be swollen, erythematous, and exquisitely tender to even light palpation. Symptoms usually resolve with treatment, or in a matter of days if left untreated. Identifying precipitating events may help patients avoid future attacks. Common triggers include trauma, alcohol, contrast dye, salicylates, and diuretics. Because rapid changes in the body's uric acid level have been known to trigger gout flares, allopurinol is not started during an acute attack.[15] Radiographic findings in gout are seen in Figure 6.

Diagnosis of new-onset gout is made by arthrocentesis and examination of synovial fluid. Needle-shaped, negatively birefringent crystals visualized under polarizing light microscope are diagnostic, and the synovial fluid will reveal an inflammatory picture with an increased white blood cell (WBC) count with a predominance of polymorphonuclear cells. Treatment consists of NSAIDs, specifically indomethacin. Additionally, colchicine may be given every hour until pain relief is achieved or until the patient experiences side effects such as abdominal cramping, nausea, vomiting, or diarrhea. There is conflicting evidence for the use of corticosteroids,[16] which have historically been reserved for patients who cannot tolerate indomethacin and colchicine. Treatment is most effective within the first 48 hours of an attack. Patients should continue treatment for the next 3 days following resolution of symptoms.[15,17]

Although most cases of gout present to the ED in the second, or acute, stage, the initial stage is actually asymptomatic hyperuricemia. Most cases of hyperuricemia are due to decreased excretion of uric acid by the kidneys instead of increased production. There is a positive correlation between level of total body urate level and incidence of gout. After many years of acute and intermittent gout attacks, patients who are not adequately treated will often progress to the final stage of chronic tophaceous gout. This stage is marked by persistent pain and destruction in previously symptomatic joints, attributed to bony deposits visible on radiographs.[15]

Maintenance therapy for gout is aimed at lowering the total body uric acid level to 5 mg/dL or less. Ideally, patients should be referred to their primary care provider or a rheumatologist for monitoring and long-term treatment with allopurinol. For patients with frequent flares, daily maintenance with NSAIDs or colchicine may be required.[15]

SEPTIC ARTHRITIS

Bacterial invasion of a joint in the form of septic arthritis is a true medical emergency. The prevalence of septic arthritis is 2 to 10 cases per 100,000 persons.[12] It typically is the result of hematogenous seeding but can also be caused by contiguous spread or direct inoculation of a joint.[11] Destruction of cartilage and underlying bone occurs as inflammatory cytokines cause the release of proteolytic enzymes within the joint. If septic arthritis is not recognized and treated in a timely fashion, irreversible impairment of the joint can occur in as little as 10 days.[18] Rampant bacterial invasion within a joint can progress to fulminant sepsis and even death in a matter of days to weeks if left untreated. Mortality has been reported to be as high as 15% in these patients, with many survivors experiencing permanent disability due to the resulting joint injury.[19]

Septic arthritis should be suspected in patients who present with rapidly progressive pain, swelling, and

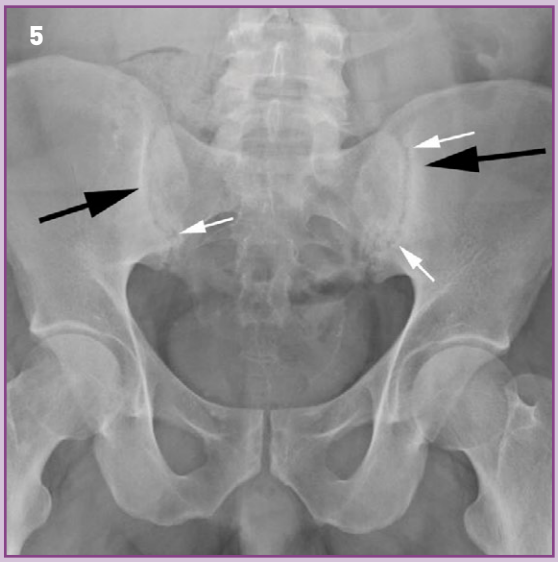

Figure 5. AP view of the pelvis showing increased sclerosis surrounding the sacroiliac joints bilaterally (black arrows). The osseous changes in any process involving the sacroiliac joints appear more prominent on the iliac side of the joint because the iliac cartilage is thinner than the cartilage of the sacrum. Note that in sacroiliitis due to inflammatory conditions, the margins of the joint are irregular, with small erosions (white arrows) caused by the inflamed synovium, as observed in this case. These erosions help to distinguish sacroiliitis from other processes that may cause sclerosis around the joint, such as a degenerative arthritis. Sacroiliitis may be a result of several inflammatory conditions, but the symmetric involvement of the bilateral sacroiliac joints is typical of ankylosing spondylitis. The same pattern may also be associated with inflammatory bowel disease. If findings are equivocal on radiographs, either CT or MRI may be utilized to further evaluate the sacroiliac joints. When available, MRI is preferred because it does not use ionizing radiation.

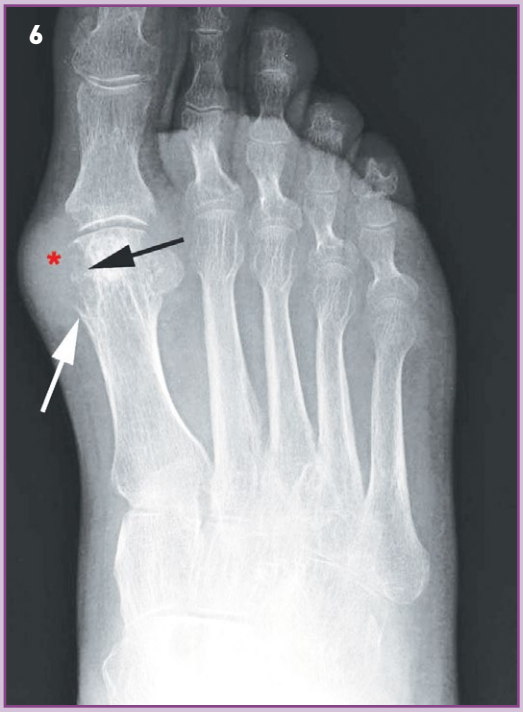

Figure 6. AP view of the foot in a patient with gout. Radiographs are typically normal in early gout, and several years of active disease may be required for the disease to become radiographically apparent. Typical findings include focal soft tissue nodules (red asterisk), representing a soft tissue tophus, and periarticular erosions (black arrow) with characteristic overhanging edges of reparative bone (white arrow). These overhanging edges, which are caused by the bone's healing response around the tophi, may occur in gout, as opposed to an inflammatory arthritis, due to the long time frame in which the erosions develop. Advanced imaging does not typically play a role in the diagnosis of gout.

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erythema of a single joint. Acute synovitis of the knee is most common, but other large- and medium-sized joints may also be affected. Occasionally, septic arthritis is polyarticular, but usually with presentations in an additive and ipsilateral fashion, such as involvement of the right knee followed by the right hip.[20] In nongonococcal septic arthritis, the most common organism implicated is *Staphylococcus aureus*, followed by *Streptococcus pneumoniae* and gram-negative bacilli.[21] The incidence of methicillin-resistant *S aureus* is increasing, with one study attributing approximately 50% of culture-positive cases in adults to MRSA.[22]

It is worth noting that septic arthritis does occur in children, in whom its presentation is similar to that in the adult population: with fever, arthralgia, and joint swelling. Pediatric mortality from septic arthritis has been reported at 7.3% across all pediatric age-groups; death usually results from sepsis. Caksen et al found that almost half of septic arthritis cases in a pediatric population had coexisting osteomyelitis.[23] The most commonly implicated organism is *S aureus*. Although *Haemophilus influenzae* was previously implicated as one of the top pathogens in pediatric septic arthritis, its prevalence has decreased with immunization. Now the bacterial etiology is similar to that observed in the adult population, with the exception of neonates and infants, in whom disease due to *Candida* and group B streptococcus must be considered.[23] Figures 7 and 8 demonstrate radiographic findings in septic arthritis.

Diagnosis of a septic joint is particularly challenging in the setting of RA. Affected patients have impaired host immunity as well as decreased phagocyte function, and often have atypical presentations. They are also more likely to be taking immunosuppressive agents such as glucocorticoids, DMARDs, and anti-TNF-α agents.[18] Half of patients with RA who have a septic joint are afebrile at initial presentation.[24] The disease is more frequently polyarticular in these patients as well, and onset is more likely to be reported as insidious.[24] Since septic arthritis can be difficult to distinguish from a typical RA flare, the decision to proceed with arthrocentesis must be carefully considered in this population. Septic arthritis should be suspected in patients with RA if the flare seems more severe than usual for a given patient, the patient is unable to bear weight on the affected joint, there is a significant decrease in range of motion, or there are any systemic symptoms associated with the joint pain. While patients experiencing an exacerbation of RA may have significant pain and swelling, overlying erythema of the joint is rare and should be investigated. If any of the above is discovered upon exam, it is advisable to analyze the synovial fluid directly in order to rule out septic arthritis. Culture of synovial fluid is especially critical, as patients with RA are more likely to have an opportunistic or a rare pathogenic infection of the joint. Studies show that elderly patients, those colonized with *S aureus*, diabetic patients, and patients with cellulitis or decubitus ulcers overlying a joint are also at increased risk for nongonococcal septic arthritis.[18,24]

Retrospective studies have found that certain chronic diseases such as reactive arthritis, ankylosing spondylitis, RA, and other connective tissue diseases causing long-term damage to articular surfaces put patients at increased risk for acquiring septic arthritis.[25] Other predisposing factors include prosthetic joints, recent arthrocentesis or instrumentation, and history of IV drug use.[25] A recent study from Hong Kong indicates particular vigilance for septic arthritis is warranted in patients with end-stage renal disease who undergo hemodialysis. These patients are at increased risk not only as a result of frequent instrumentation and vascular access, but also due to decreased humoral immunity.[26]

The diagnosis of septic arthritis cannot be made based on history and physical exam alone. Systemic leukocytosis, increased erythrocyte sedimentation rate, and high C-reactive protein level may all be present in septic arthritis; however, research has shown that these findings are neither sensitive nor specific enough to make the diagnosis conclusively.[18] Therefore, analysis of the synovial fluid is essential if a septic joint is suspected in an ED patient.[21] Removal of the effusion by arthrocentesis is not only diagnostic but also therapeutic. (See "Arthrocentesis," page 14.)

After arthrocentesis is performed, broad-spectrum antibiotics should be started empirically if suspicion for septic arthritis is high. Otherwise, antibiotic therapy may be guided by Gram stain results. Since gonococcal arthritis is the most common cause of septic arthritis in young adults, these patients may be safely treated with

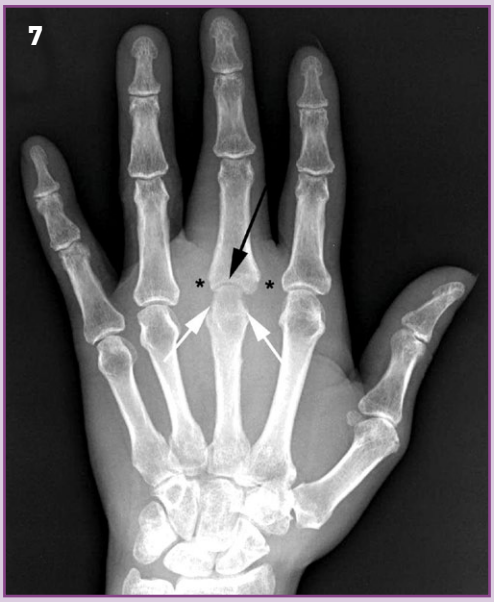
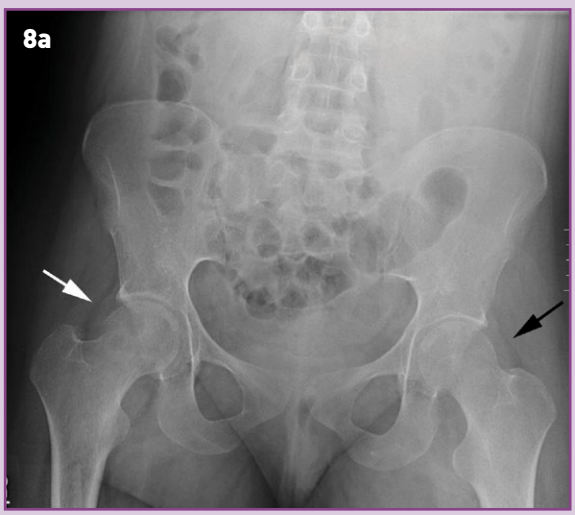


Figure 7. PA view of the hand in a patient with septic arthritis of the third metacarpophalangeal joint. Radiographic findings that may be seen in septic arthritis are similar to those found in inflammatory arthropathy, but they are typically confined to a single joint. These findings include focal soft tissue swelling (black asterisks), articular erosions (white arrows), and joint space narrowing (black arrow). However, the erosions and joint space narrowing are radiographically present only after septic arthritis has caused advanced cartilage loss. Therefore, to preserve joint function, it is important to diagnose septic arthritis prior to the development of radiographic findings. MRI with contrast is the preferred test to evaluate for soft tissue and articular infection.



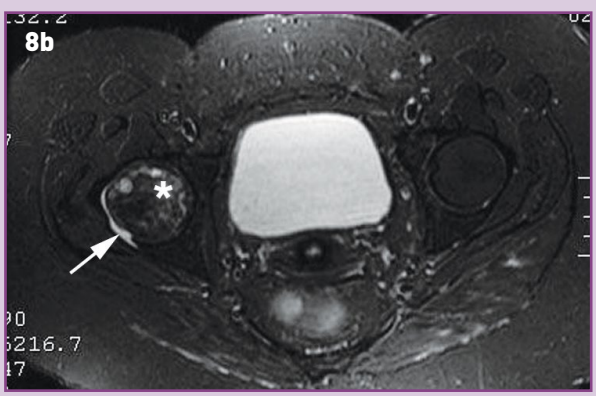


Figure 8. **(8a)** AP view of the pelvis in a patient with septic arthritis of the right hip. In some large joints, evidence of soft tissue inflammation and joint effusions can be very difficult to recognize, and therefore, early septic arthritis often remains radiographically occult. One may look for secondary signs of inflammation and effusion, such as bulging of the periarticular fat plane (white arrow), suggesting the presence of a hip joint effusion. Note the normal fat plane (black arrow) on the contralateral side.
(8b) An axial T_2-weighted fat-suppressed (edema-sensitive) image from an MRI examination further supports the diagnosis of septic arthritis by showing areas of bright signal in the bone (white asterisk), representing marrow edema, and bright signal in the joint space (white arrow), representing effusion.

Images courtesy of Keith D. Hentel, MD

penicillin alone once the organism is positively identified.[11] Patients with community-acquired disease who have gram-positive organisms on Gram stain of synovial fluid may be treated with IV cefazolin.[18] Patients who were recently hospitalized or otherwise at risk for nosocomial infection should be treated with vancomycin. If synovial fluid analysis reveals gram-negative organisms, ceftriaxone is an appropriate choice. It is appropriate to double cover for *Pseudomonas* with ceftazidime and gentamicin in IV drug users and in patients who have a negative Gram stain result with a suspicious clinical presentation. Admission for prolonged parenteral antibiotics and repeat therapeutic arthrocentesis is almost always indicated. Patients usually require at least 2 weeks of IV antibiotics followed by oral antibiotics after discharge.[18]

In addition to initiation of broad-spectrum antibiotics, septic arthritis requires emergent orthopedic consultation.[21] Referral for arthroscopy and possible washout of the joint should be considered if the patient has a complex or loculated joint effusion that cannot be properly aspirated or if there is coexisting osteomyelitis. The joint may be partially immobilized for the patient's comfort, with early range-of-motion exercises followed by physical therapy as inflammation resolves. In the setting of joint prosthesis, an orthopedic surgeon must be consulted prior to any attempt at arthrocentesis. However, antibiotic treatment should not be delayed in anticipation of the consultation. In some cases, removal of hardware may be necessary, since the surfaces of prosthetic joints are predisposed to bacterial colonization. For patients undergoing long-term immunosuppressive therapy, temporarily stopping or decreasing these medications may be required.[18]

ARTHROCENTESIS

The diagnostic study of choice for all joint effusions is examination of the synovial fluid. Thus, arthrocentesis can be a powerful diagnostic tool that may frequently alter patient course and treatment, especially in the case of septic arthritis or the crystal arthropathies.[27]

In patients with suspected joint effusions that are difficult to fully appreciate on physical exam, bedside ultrasonography may prove valuable. It can help determine the size, location, and extent of an intra-articular fluid collection. Recent research suggests that the use of ultrasonography may facilitate joint aspiration, especially in smaller or more technically challenging joints. Comparison of ultrasound-guided arthrocentesis with standard landmark technique has yielded many promising results. In one study, not only did patients rate pain as lower when ultrasound guidance was used in the procedure, but physicians also rated the procedure as easier to perform, and they completed it faster.[28] Additional research is needed to confirm the growing body of evidence supporting the use of ultrasound in arthrocentesis.

Some investigators have also suggested that the appearance of a joint effusion on ultrasound can provide information about its etiology. Since the crystalline material characteristic of gout is more reflective of sound waves than pure synovial fluid, it can be identified within the intra-articular space.[27]

Arthrocentesis should be performed using aseptic technique. Before arthrocentesis is performed, the overlying skin should be examined thoroughly for any lesions or cellulitis, the presence of which is a contraindication to the procedure. Introducing a needle intra-articularly through such lesions increases the risk for septic arthritis. Anticoagulation with warfarin, on the other hand, is not an absolute contraindication to arthrocentesis. Risks for patients taking these agents are low, as long as the INR is not supratherapeutic.[27] Prior to arthrocentesis, the patient should be screened for any history of recent trauma to the area and for other risk factors for hemarthrosis, such as hemophilia. Other relative contraindications include prosthetic joints, as these are more prone to septic arthritis following the procedure. If available, orthopedic consultation is warranted for suspected septic prosthetic joints if arthrocentesis is being considered in the ED.[11]

Synovial Fluid Analysis

Once synovial fluid has been obtained, it should be sent for Gram stain and culture, WBC count with differential, and wet prep for crystal identification. If gonococcal or anaerobic organisms are suspected, specific culture mediums may be required.[11] Glucose and protein measurement may also aid in the diagnosis of septic arthritis. Prior to receiving lab results, how-

ever, the physician can often obtain useful information merely by observing the appearance of the aspirated fluid. Normal synovial fluid is transparent and usually colorless, while synovial fluid obtained from a joint with a noninflammatory arthritis typically has a yellow hue but maintains its transparent nature. Inflammatory arthropathies, on the other hand, produce cloudy yellow fluid that can be difficult to differentiate from the synovial fluid in septic arthritis, which may be frankly purulent.[11]

Normal synovial fluid may have a WBC count of up to 200/µL but will have a low percentage of polymorphonuclear cells, usually less than 25%. Synovial fluid in noninflammatory arthritis such as osteoarthritis may have up to 2,000 WBCs/µL but has a relatively normal differential. In gout, pseudogout, RA, and the spondyloarthropathies, the WBC count increases to 200 to 50,000/µL. In these inflammatory arthropathies, polymorphonuclear cells may account for over 50% of the WBCs. WBC counts exceeding 50,000/µL suggest gonococcal or nongonococcal septic arthritis. Gram staining of synovial fluid will reveal organisms in 70% of patients with septic arthritis. This may be useful in guiding antibiotic therapy.[11]

CONCLUSION

Joint pain has a wide range of etiologies and may vary greatly in its presentation throughout all age ranges. The emergency physician must remain especially vigilant for septic arthritis, maintaining a high index of suspicion for this emergent condition whenever a patient presents with a swollen, painful joint. In elucidating the causes of most nontraumatic joint pain, taking a thorough history and performing a careful physical exam, followed by examination of the synovial fluid when indicated, are key. Patients with suspicious findings on physical exam or laboratory results should be treated aggressively and referred early to a specialist. **EM**

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