# Native Joint Bacterial Septic Arthritis in the Adult

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**Abstract:** Septic arthritis (SA) is an uncommon but potentially life-threatening condition; it occurs when microorganisms invade the joint space by direct inoculation or by hematogenous spread. Up to a third of patients with septic arthritis suffer long-term disability. Challenges in the management of septic arthritis include selection of appropriate antimicrobials and selection of an appropriate joint fluid drainage method [1]. In this review, I will focus on the epidemiology, mechanism, pathogenesis, clinical signs, diagnosis, and treatment of native joint bacterial septic arthritis in the adult.

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## Introduction

An estimated 20,000 cases of septic arthritis occur in the United States each year. Septic arthritis occurs most commonly in the elderly and in children less than 3 years of age. The average hospital stay is about 7 days, with an estimated annual cost of approximately USD 750 million [1]. The introduction of microorganisms into the joint space can result from procedures such as joint surgery, joint aspiration, or intra-articular injection. Almost any microorganisms can cause septic arthritis. Bacteria are the most common, but viruses, mycobacteria, and fungi have also been implicated. Risk factors for septic arthritis include prosthetic joints, preexisting arthritis, and immunodeficiency. The most common organism identified in septic arthritis in adults is *Staphylococcus aureus*, responsible for 40–50% of cases [2]. The next most common causative organisms are groups A and B streptococci, which are especially common among the elderly and those with chronic diseases such as diabetes or cirrhosis. Gram-negative organisms, though less common, are also frequently seen. These include *Neisseria gonorrhoeae*, *Neisseria meningitides*, and Gram-negative enteric organisms. *Pseudomonas* should be suspected in patients who use IV drugs. In approximately 20 to 50% of

cases of septic arthritis, no causative organisms are identified. This is most often due to administration of antibiotics prior to arthrocentesis but may also suggest infection with obligate intracellular or other fastidious organisms.

## Pathophysiology

The synovium is highly vascularized and lacks a protective basement membrane; thus, the most common route of bacterial entry is via hematogenous spread. Joints with preexisting damage, such as in rheumatoid arthritis or osteoarthritis, are especially prone to septic arthritis [2]. Less commonly, joint infection occurs as a result of direct inoculation after trauma or surgery. Rarely, septic arthritis is caused by intra-articular injections or arthrocentesis. However, outbreaks associated with injection safety breaches have been reported. Examples of such breaches include using the same syringe to deliver medication to more than one patient, using single-dose or single-use medications for more than one patient, and failing to use an aseptic technique when preparing and administering injections [3].

Upon introduction to the joint space, microorganisms trigger an acute inflammatory reaction that may progress to involve the surrounding bone. Destruction of the articular surface may follow due to the release of inflammatory cytokines and proteases.

### Signs and Symptoms

Symptoms of SA include acute onset of a decreased range of joint motion, localized pain, tenderness, and swelling of the affected joint, often associated with fever. The absence of fever does not rule out a diagnosis of SA, especially in elderly patients, with only 30 to 60% of cases having fever upon presentation.

### Laboratory Findings

Unfortunately, there is no single clinical sign or laboratory test (excluding cultures) that can conclusively differentiate between SA and non-SA [4]. Blood cultures are positive in about 10% of patients with negative synovial fluid (SF) cultures. Many patients have an elevated WBC count, but by itself, this is not an independent predictor of SA. The serum erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are often elevated in SA. Nevertheless, these can also be elevated in noninfectious inflammatory conditions. Thus, despite the good sensitivity for an ESR of over 30 mm/h, the associated specificity is only about 30% for SA in adults. Similarly, CRP values of over 100 mg/liter have a reported sensitivity of 80% for SA, but specificities range from 30 to 70% [4].

In more than 50% of adult cases, the knee is the site of the infection, followed by the shoulders, wrists, hips, and ankles. The hip is the most commonly affected joint in children. Less commonly affected joints, such as the sacroiliac (SI) joint, sternoclavicular joint, and symphysis pubis, can have unique presentations. Septic arthritis of the SI joint can present with buttocks pain, gait changes, and fever. Infection of the symphysis pubis can occur with pelvic malignancies and in female patients who have undergone urinary incontinence surgeries, or in IV drug users, and can present with fever, suprapubic pain, and an antalgic gait [5,6]. Sternoclavicular and acromioclavicular joint infections are notable in that they often present only with pain and usually require surgical resection with a thoracic surgeon [7,8]. Most patients with septic arthritis of the symphysis pubis complain of pubic pain. It occurs most commonly in women undergoing urinary incontinence surgery but may also be seen in athletes or as the result of trauma or IV drug use. *Staphylococcus aureus* is the usual cause, but *Pseudomonas aeruginosa* is commonly seen in intravenous drug users [5].

Septic arthritis is usually monoarticular but is oligoarticular in approximately 10 to 20% of cases. Multiple joint involvement is typically seen in patients with overwhelming sepsis, multiple systemic comorbid conditions, or RA [9].

The most important procedure for the timely and accurate diagnosis of septic joints is arthrocentesis. Culture of synovial fluid remains the gold standard for the diagnosis of SA. Ideally, joint aspiration should be performed prior to the initiation of antimicrobials. It is important to remember that the presence of crystals in SF does not exclude infection.

Gram staining can provide immediate information and is often positive in bacterial arthritis, with a sensitivity between 30 and 50%. Synovial fluid cultures are very sensitive in nongonococcal bacterial arthritis and are positive in the majority of cases. Their sensitivity can be compromised in patients who have received antibiotic treatment prior to obtaining cultures and can also be falsely negative in infections caused by fastidious and obligate intracellular organisms.

Other clues to the diagnosis of SA include a low synovial fluid glucose level and elevated protein or lactic acid levels; however, those laboratory values are nonspecific and can be found in all forms of inflammatory arthritis. An elevated synovial WBC count greater than 50,000/μL is very suggestive of SA, but such elevations are also seen with other inflammatory arthritides, and lower synovial WBC counts do not preclude SA [10,11].

There is growing interest in the use of metagenomic sequencing to identify the etiology of infection, especially in culture-negative cases. However, there is little information yet available to determine its utility in cases of SA [12].

## Imaging

The first imaging study performed of a potentially infected joint should be a radiograph, and it should be compared with a baseline image if available. Characteristic findings include joint space widening, periarticular fat pad displacement, and, occasionally, erosive changes. Advanced imaging methods, such as CT and MRI, can be especially useful for examining the SI and hip joints where inflammatory changes may be difficult to identify on a plain radiograph.

## Differential Diagnosis

The main differential diagnoses for bacterial arthritis are other causes of acute arthritis and include acute crystal arthritis (gout and pseudogout), reactive arthritis, RA, and Lyme disease. Crystal arthritis is usually the first diagnosis to consider because it can present in a very similar fashion and can coexist with bacterial arthritis. The diagnosis is usually made by visualizing monosodium urate or calcium pyrophosphate crystals in gout and pseudogout. It is critical to remember that neither the presence of crystals in the synovial fluid nor the absence of a positive synovial fluid Gram stain can definitively exclude bacterial arthritis.

Patients with reactive arthritis may report a recent gastrointestinal or genitourinary tract infection and may report other symptoms such as skin lesions or ocular inflammation. Patients with RA typically present with chronic polyarthritis that is symmetrical and not associated with fever or leukocytosis; however, they can still present with an acute monoarthritis with elevated synovial fluid WBC counts. It should be noted that if an RA patient with good disease control acutely presents with an inflamed joint, then septic arthritis should be considered.

## Treatment

The cornerstones of treatment include adequate drainage and antimicrobials. The initial choice of antibiotics is generally based on epidemiological factors, such as age, risk factors, and clinical presentation, in addition to Gram stain results. No randomized controlled trials to date have compared different antibiotic regimens for the treatment of bacterial arthritis, nor is there strong evidence to guide the duration of treatment [13].

Vancomycin is generally the initial antibiotic used in the United States. If the patient is elderly, immunocompromised, or an IV drug abuser, vancomycin plus a third-generation cephalosporin such

as ceftriaxone should be administered. The initial antibiotic regimen should be adjusted based on culture, and susceptibility results. If the clinical picture is suggestive of disseminated gonococcal or meningococcal infections, IV ceftriaxone provides adequate coverage. If the patient has an indwelling venous catheter or is an IV drug user, then *P. aeruginosa* should be suspected, and an anti-pseudomonal beta-lactam should be administered. The use of intra-articular antibiotics is not advised because it adds no therapeutic benefit and can potentially damage cartilage and the synovium.

Intravenous antibiotics are typically administered for about 14 days followed by a similar-duration course of oral antibiotics, if oral options are available. There is growing evidence that oral antibiotics may be used earlier, once the patient responds and there is an oral option available. In one study, oral antibiotic therapy was noninferior to intravenous antibiotic therapy when used during the first 6 weeks for complex orthopedic infection, as assessed by treatment failure at 1 year [14].

Septic arthritis, as with any other form of a closed infection, needs drainage in conjunction with antibiotics. However, there have not been randomized controlled trials in adults comparing drainage methods, namely, daily needle aspiration, arthroscopic drainage, or open surgical drainage via arthrotomy. The choice has generally depended on procedural availability. One study found that patients hospitalized in the orthopedic unit were more likely to be treated with surgical intervention compared with patients hospitalized in the medical unit, with no statistically significant difference in treatment failure rates between the two groups [13,15].

Daily needle arthrocentesis has many advantages. It can be performed safely at the bedside, it can be performed in patients who are poor surgical candidates, and it allows for daily analysis of the synovial fluid. The main disadvantages are the inability to debride infected tissue and incomplete drainage of loculated effusions. Additionally, some joints such as the hips and shoulders are not easily accessible or adequately drained by arthrocentesis. The benefits of surgical drainage include the ability to debride necrotic tissue, obtain biopsies, and evaluate for a potential synovectomy. These benefits may be more pronounced in infections of the knees, wrist, and shoulder [13].

## Summary

Septic arthritis in adults continues to be an important infection that can be difficult to diagnose but requires prompt treatment from a multidisciplinary team. There should be a low threshold to obtain synovial fluid from a patient with a monoarticular arthritis as examination of the synovial fluid is critical in differentiating infectious from non-infectious causes. Treatment paradigms are changing and often include oral antibiotic therapy. Additional information on the optimal duration of therapy is sorely needed.

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