Septic arthritis in adults

Authors: Don L Goldenberg, MD, Daniel J Sexton, MD
Section Editor: Denis Spelman, MBBS, FRACP, FRCPA, MPH
Deputy Editor: Keri K Hall, MD, MS

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INTRODUCTION

Septic arthritis is synonymous with an infection in a joint. Septic arthritis is usually caused by bacteria but can also be caused by other microorganisms. Septic arthritis due to bacterial infection is often a destructive form of acute arthritis [1].

The epidemiology, microbiology, clinical manifestations, diagnosis, differential diagnosis, and treatment of septic arthritis of native joints due to typical bacteria are reviewed here. An overview of monoarthritis in adults is presented separately. (See "Monoarthritis in adults: Etiology and evaluation".)

Issues related to prosthetic joint infection are discussed separately. (See "Prosthetic joint infection: Epidemiology, microbiology, clinical manifestations, and diagnosis" and "Prosthetic joint infection: Treatment".)

Issues related to septic arthritis in children are discussed separately. (See "Bacterial arthritis: Clinical features and diagnosis in infants and children" and "Bacterial arthritis: Treatment and outcome in infants and children".)

Issues related to gonococcal arthritis, lyme arthritis, and viral causes of arthritis are discussed separately. (See "Disseminated gonococcal infection" and "Musculoskeletal manifestations of Lyme disease" and "Viruses that cause arthritis".)

EPIDEMIOLOGY

In the United States in 2012, septic arthritis was responsible for 16,000 emergency department visits [2].

Predisposing factors include [3-8]:

- Advance age
- Pre-existing joint disease
- Recent joint surgery or injection
- Skin or soft tissue infection
- Injection drug use (IDU)
- Indwelling catheters
- Immunosuppression (including diabetes)

Most commonly, septic arthritis arises via hematogenous seeding. Bacteremia is more likely to localize in a joint with pre-existing arthritis (such as rheumatoid arthritis, osteoarthritis, gout, pseudogout, Charcot arthropathy), particularly if associated with synovitis [9-11]. Patients with rheumatoid arthritis, may have additional predisposing factors, such as intra-articular steroid injections and use of immunosuppressive medications [12-15].

In one series including more than 500 adult cases of native joint septic arthritis in New Zealand between 2009 and 2014, large joint septic arthritis (involving the knee, shoulder, hip, ankle, wrist, or elbow) occurred more frequently than small joint septic arthritis (involving the finger, toe, acromioclavicular, or sternoclavicular joints; incidence 13 versus 8 per 100,000 person-years) [16].

IDU has become an increasingly important risk factor for septic arthritis in the United States in the context of the opioid epidemic; consequently, septic arthritis has been observed more frequently in younger patients with no other comorbidities [17]. In one study in Boston, IDU was a factor in 20 percent of septic arthritis cases between 2009 and 2018 (compared with 10 percent between 1990 and 2008) [18]. Most of these infections were due to *Staphylococcus aureus*; one-third were methicillin resistant. There was frequent involvement of the sacroiliac, acromioclavicular, and facet joints.

MECHANISM OF INFECTION

Septic arthritis develops as a result of hematogenous seeding, direct inoculation of bacteria into the joint, or contiguous spread from an adjacent soft tissue or bone infection [10,11,19].

Most commonly, septic arthritis arises via hematogenous seeding of the synovial membrane (which has no limiting basement membrane, thus allowing organisms to enter the joint space). Septic arthritis due to hematogenous seeding is most commonly caused by organisms with propensity to adhere to the synovial tissues (such as *S. aureus*) [20]. Hematogenous seeding most commonly involves large joints [16].

Bacterial arthritis can also occur via direct inoculation of bacteria into the joint; mechanisms include bite wounds, trauma, arthroscopy or other surgery, and intra-articular injection [21,22]. Septic arthritis following an intra-articular injection is uncommon; rarely it occurs in clusters due to unsafe injection practices [21,23].

Rarely, septic arthritis develops via extension of infection into the joint space from adjacent tissues. In the setting of long-bone osteomyelitis, infection within the metaphysis can break through the bone cortex, leading to discharge of pus into the joint. This occurs in joints in which the metaphysis is within the capsular reflection (eg, knees, hip, shoulder, and elbows). (See "Nonvertebral osteomyelitis in adults: Clinical manifestations and diagnosis", section on 'Hematogenous osteomyelitis'.)

Other examples include complications of subclavian vein and femoral catheterization (resulting in septic arthritis of the sternoclavicular and hip joints, respectively), and ruptured diverticulitis, resulting in dissection of infection via the retroperitoneal space into the posterior thigh and hip joint [24-26].

MICROBIOLOGY

Numerous pathogens are capable of causing septic arthritis (table 1). The microbiology of septic arthritis depends in part on the mechanism of infection and relevant epidemiologic exposures. (See 'Epidemiology' above.)

Septic arthritis is usually monomicrobial. *S. aureus* (including methicillin-resistant *S. aureus*) is the most common cause of septic arthritis in adults [4,11,27]. Other gram-positive organisms such as streptococci are also important potential causes of septic arthritis [28]. *Streptococcus pneumoniae* is responsible for a small percentage of cases of septic arthritis in adults [29].

Septic arthritis due to gram-negative bacilli generally occurs in older adults, in patients with underlying immunosuppression, or intravenous drug users; it may also occur as a complication of trauma. Patients with underlying immunosuppression and intravenous drug users are at risk for infection due to *Pseudomonas* [30].

Polymicrobial septic arthritis is uncommon; it may occur in the setting of penetrating trauma involving the joint space or via hematogenous seeding in patients with polymicrobial bacteremia. Small joint septic arthritis is more likely to be polymicrobial and caused by streptococci, *Eikenella*, or anaerobic bacteria [16].

CLINICAL MANIFESTATIONS

Signs and symptoms — Patients with septic arthritis usually present acutely with a single swollen and painful joint (ie, monoarticular arthritis) [31]. Joint pain, swelling, warmth, and restricted movement occur in 80 percent of patients with septic arthritis [3]. Most patients with septic arthritis are febrile; however, older patients with septic arthritis may be afebrile [3].

The knee is involved in more than 50 percent of cases; wrists, ankles, and hips are also affected commonly [10]. Infection of the symphysis pubis is uncommon [32]. Involvement of axial joints (such as the sternoclavicular, acromioclavicular, or sternomanubrial joint) may occur in people who inject drugs (PWID) [18,33]. (See "Pelvic osteomyelitis and other infections of the bony pelvis in adults".)

Oligoarticular or polyarticular infection (usually two or three joints) occurs in approximately 20 percent of patients with septic arthritis. Polyarticular septic arthritis is most likely to occur in patients with sepsis and underlying rheumatoid arthritis or other systemic connective tissue disease [34].

Septic arthritis may be a presenting manifestation of infective endocarditis; this occurs most commonly among PWID [35]. (See "Clinical manifestations and evaluation of adults with suspected left-sided native valve endocarditis".)

In septic arthritis that occurs as a result of hematogenous seeding, blood cultures are positive in approximately 50 percent of cases [11]. Laboratory findings such as elevated white blood cell count, erythrocyte sedimentation rate, and C-reactive protein are common but nonspecific [36].

Physical examination — The physical examination should include thorough evaluation of all joints for erythema, swelling, warmth, and tenderness [3]. Infected joints are characteristically painful and usually demonstrate an effusion, both of which are associated with limited active and passive range of motion. External findings such as swelling, erythema, and warmth may be less prominent in the setting of septic arthritis involving the hip, shoulder or spine joints. In addition, physical exam findings may be less prominent in patients who are older adults and/or immunocompromised.

DIAGNOSIS

General approach — The diagnosis of septic arthritis should be suspected in patients with acute onset of at least one swollen, painful joint, with or without relevant risk factors (including bacteremia, pre-existing joint disease, and immunosuppression).

The diagnosis of septic arthritis is made based on synovial fluid analysis and culture (algorithm 1 and table 2). Septic arthritis may be definitively established in the setting of positive synovial fluid Gram stain and/or culture [3]. In patients with purulent synovial fluid (leukocyte count >20,000 cells/microL, mostly neutrophils) but negative synovial fluid cultures, a presumptive diagnosis of septic arthritis may be made. (See 'Interpreting synovial fluid test results' below and 'Differential diagnosis' below.)

In addition, blood cultures (two sets) and, when indicated, radiographs, ultrasound, or imaging studies of the involved joint should be obtained.

In the setting of septic arthritis due to organisms that commonly cause endocarditis (such as *S. aureus*, streptococci, or enterococci) with no clear predisposing cause, evaluation for endocarditis should be pursued. (See "Clinical manifestations and evaluation of adults with suspected left-sided native valve endocarditis".)

Obtaining clinical specimens — Collection of synovial fluid and blood cultures should be performed prior to administration of antibiotics. If synovial fluid cannot be obtained with closed needle aspiration, the joint should be aspirated under radiographic guidance. Certain joints (such as the hip or sacroiliac joint) may require surgical arthrotomy, which may be accompanied by irrigation and drainage. The only definitive way to diagnose a septic joint is via synovial fluid culture.

Synovial fluid should be sent for Gram stain, bacterial culture, white blood cell count with differential, and assessment for crystals (monosodium urate and calcium pyrophosphate crystal deposition crystals) with a polarizing microscope.

Synovial fluid may be sent for culture in a sterile tube (ideally with an anticoagulant such as ethylenediaminetetraacetic acid [EDTA] to guard against clotting) and/or in blood culture bottles (aerobic and anaerobic) [37-40]. If blood culture bottles are used, synovial fluid also should also be sent in a sterile container to allow Gram stain microscopy. Use of blood culture bottles may increase the likelihood of recovering nonpathogenic skin contaminants; in such cases, culture results should be interpreted in the context of the Gram stain result. The use of blood culture bottles has no significant advantage when common causes of septic arthritis such as *S. aureus* is

suspected [41] but may have advantages in detecting unusual organisms such as *Kingella* and *Brucella* [42]. *Kingella kingae* occurs more commonly in children than adults. (See "Bacterial arthritis: Clinical features and diagnosis in infants and children".)

Nucleic acid amplification tests such as polymerase chain reaction and other advanced diagnostic methods such as MALDI-TOF mass spectometry may be useful when routine cultures are negative and the suspicion for infection remains high; in infections due to *N. gonorrhoeae* cases (see "Disseminated gonococcal infection"); or cases in which prior treatment with antibiotics is suspected as having led to a spuriously negative routine culture. When positive, such test results need to be interpreted carefully and correlated with Gram stains and clinical and epidemiologic findings, because extremely small amounts of contaminating bacterial material can lead to a false-positive result [43].

Measurement of procalcitonin levels (in the serum and/or synovial fluid) has been proposed as tools for diagnosis of septic arthritis, particularly when synovial fluid is difficult to obtain or in patients with coexisting inflammatory arthritis.

The sensitivity of serum procalcitonin level is relatively low (pooled sensitivity in 10 studies 0.54 [95% CI 0.41-0.66]), as is the negative likelihood ratio (0.49 [95% CI 0.38-0.62]); thus, such a test cannot be used to rule out septic arthritis. The specificity of serum procalcitonin is higher (pooled sensitivity in one meta-analysis 0.95 [95% CI 0.87-0.96]); thus a positive test may be useful occasionally in deciding if a difficult-to-access joint such as the hip or sacroiliac joint should be aspirated [44,45]. There are limited data to suggest that synovial fluid procalcitonin levels may be more predictive of septic arthritis than blood levels [46]; however, such testing is not needed if a culture is obtained at the time of joint aspiration.

Other synovial fluid assays (such as synovial fluid lactate, glucose, C-reactive protein, polymerase chain reaction, or immunoassays) are not useful for diagnosis of septic arthritis [47-49].

Synovial biopsy is rarely necessary, but may be indicated if there is evidence of concurrent contiguous osteomyelitis, in rare cases in which joint aspiration fails to provide a satisfactory sample for diagnostic testing, or when infection or coinfection with *M. tuberculosis* or other slow growing pathogens is a possibility.

Interpreting synovial fluid test results — In the setting of septic arthritis, synovial fluid analysis typically demonstrates the following (table 2 and algorithm 1) [11]:

- Leukocyte count of 50,000 to 150,000 cells/microL (mostly neutrophils) [37]. The likelihood of septic arthritis increases as the synovial fluid leukocyte count increases [4,16]. High

synovial fluid white blood cell counts can also occur in other conditions, so it is important to interpret the results of synovial fluid testing in the overall clinical context. (See 'Differential diagnosis' below.)

- Gram stain is positive in some cases; the sensitivity is 30 to 50 percent [3]. False-positive results may reflect precipitated crystal violet and mucin in the synovial fluid, which can mimic the appearance of gram-positive cocci. False-negative results may occur if crystals are present or if clotting occurs [38,50].

- Synovial fluid culture is positive in the more than 60 percent of patients with nongonococcal bacterial arthritis [51]. Negative synovial fluid cultures may occur in the setting of recent antimicrobial therapy or infection with a fastidious organism.

  In one retrospective study including 383 patients with suspected septic arthritis (of whom 82 patients received antibiotics prior to the initial synovial fluid analysis), a leukocyte count between 16,000 and 33,000 cells/microL with >90 percent neutrophils was strongly suggestive of septic arthritis [52].

Radiographic imaging — Radiographs of the involved joint should be obtained for evaluation of concurrent bone and joint disease; in addition, baseline radiography is often useful to guide subsequent management decisions. For joints that are difficult to examine (such as the hip or sacroiliac joint), computed tomography or magnetic resonance imaging are useful for detection of effusion. Nuclear imaging is not warranted for suspected septic arthritis. There are insufficient data to support a role for fluorodeoxyglucose-positron emission tomography scans in diagnosis of septic arthritis [53]. (See "Imaging techniques for evaluation of the painful joint".)

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of septic arthritis includes infectious as well as noninfectious illnesses (table 3 and table 4) [54]. (See "Monoarthritis in adults: Etiology and evaluation".)

Other causes of infection include septic bursitis and alternate infectious causes of arthritis:

- Septic bursitis – Septic bursitis refers to inflammation of the bursa due to infection; the mechanisms for development of septic bursitis are the same as the mechanisms for development of septic arthritis. The diagnosis of septic bursitis is confirmed by culture of fluid from the affected bursa. (See "Septic bursitis".)

- Gonococcal arthritis – Gonococcal arthritis typically presents acutely in sexually active individuals with fever, chills, skin lesions, polyarthralgias, and tenosynovitis, evolving into a persistent monoarthritis or oligoarthritis. The diagnosis of disseminated gonococcal infection is made by identification of *Neisseria gonorrhoeae* (either through molecular testing or culture) via nucleic acid amplification testing or culture of a specimen of blood, synovial fluid or tissue, skin lesion, or other nonmucosal site. Culture requires processing on chocolate agar plates, Thayer-Martin medium, or other selective gonococcal medium; the organism cannot be cultured on routine culture media. (See "Disseminated gonococcal infection".)

- Lyme disease – Lyme disease should be suspected in patients with an acute monoarthritis in the setting of epidemiologic exposure in an endemic area; erythema migrans rash, fever, and migratory arthralgias may occur weeks or months prior. The diagnosis is established via serologic testing. (See "Musculoskeletal manifestations of Lyme disease", section on 'Diagnosis of Lyme arthritis'.)

- Tuberculous arthritis – Tuberculous arthritis should be suspected in patients with indolent presentation of persistent culture-negative oligoarthritis or monoarthritis, in the setting of relevant epidemiologic exposure. The sensitivity of synovial fluid Ziehl-Neelsen stain for detection of acid-fast bacilli is low; the diagnosis is established via synovial membrane histopathology and culture. (See "Bone and joint tuberculosis", section on 'Arthritis'.)

- Viral causes of arthritis – Viral causes of arthritis typically present with polyarthritis; they include chikungunya, dengue fever, Zika virus, parvovirus, Ross River virus, Barmah Forest virus, and rubella. A number of other viruses including enterovirus, adenovirus, and alphaviruses may also cause arthritis. (See "Viruses that cause arthritis".)

- Fungal arthritis – Fungal arthritis should be suspected in patients with indolent presentation of persistent culture-negative oligoarthritis or monoarthritis, in the setting of relevant epidemiologic exposure, penetrating trauma, or immunosuppression. Fungal causes of arthritis include sporotrichosis, coccidioidomycosis, candidiasis, and others [55]. The diagnosis is established via fungal stain and culture of synovial fluid or via synovial membrane histopathology and culture.

Noninfectious causes of arthritis include trauma and inflammatory arthritis:

- Acute traumatic arthritis – Acute traumatic arthritis usually causes bloody synovial fluid and is generally associated with a history of significant trauma to the joint. (See "Overview of hemarthrosis", section on 'Traumatic'.)

- Crystal-induced arthritis (gout or pseudogout) – Manifestations of crystal-induced arthritis include monoarthritis and leukocytosis. Clues suggestive of gout include involvement of the first metatarsophalangeal joint, prior self-limited attacks of arthritis, and presence of tophi. The diagnosis of crystal-induced arthritis can be established by synovial fluid analysis demonstrating monosodium urate crystals of gout or calcium pyrophosphate dihydrate crystals of pseudogout.

  Concurrent crystal-induced and septic arthritis can occur [56]. In patients with concurrent gout and septic arthritis, the synovial fluid Gram stain may be negative; thus, cultures should be performed if concurrent infection is suspected [50]. The synovial fluid leukocyte count is often above 50,000/mm^3 in gout and calcium pyrophosphate crystal deposition disease; therefore, this finding cannot be used to distinguish from septic arthritis [57]. (See "Clinical manifestations and diagnosis of gout" and "Clinical manifestations and diagnosis of calcium pyrophosphate crystal deposition (CPPD) disease".)

- Reactive arthritis or spondyloarthritis – Chronic inflammatory joint disease can present with a new swollen joint, simulating septic arthritis; this is especially common in the seronegative spondyloarthropathies such as reactive arthritis. Most patients with reactive arthritis have recent genitourinary or gastrointestinal signs or symptoms, conjunctivitis, or skin or mucus membrane lesions. Occasionally, patients with ankylosing spondylitis present with acute-onset hip arthritis that mimics septic arthritis. (See "Reactive arthritis" and "Diagnosis and differential diagnosis of axial spondyloarthritis (ankylosing spondylitis and nonradiographic axial spondyloarthritis) in adults".)

- Avascular necrosis – Avascular necrosis refers to damage of the bone vasculature leading to mechanical bone failure; it can occur in the context of a number of conditions. It usually occurs in the anterolateral femoral head, although it may also affect the femoral condyles, humeral heads, proximal tibia, vertebrae, and small bones of the hand and foot. It typically presents with localized pain. The diagnosis is established via radiographic imaging. (See "Clinical manifestations and diagnosis of osteonecrosis (avascular necrosis of bone)".)

- Rheumatoid arthritis – Rheumatoid arthritis (RA) is typically a symmetric, chronic polyarthritis; however, acute or subacute exacerbation of one or a few joints can occur. The diagnosis may be difficult to establish because the clinical findings may be atypical; many patients with RA and superimposed septic arthritis present indolently (rather than acutely), often with little fever or leukocytosis. Conversely, RA itself may present with a "pseudoseptic arthritis" picture, including an explosive acute synovitis with a marked synovial fluid leukocytosis. The diagnosis of RA is established via clinical criteria

summarized separately. (See "Diagnosis and differential diagnosis of rheumatoid arthritis".)

TREATMENT

Management of acute bacterial arthritis consists of joint drainage and antibiotic therapy (algorithm 2 and algorithm 3) [58].

Joint drainage — In general, patients with septic arthritis warrant joint drainage, since this condition represents a closed abscess. Approaches to joint drainage for management of septic arthritis in adults include needle aspiration, arthroscopic drainage, or arthrotomy (open surgical drainage). The choice of approach depends on clinical factors including the joint affected and the duration of infection.

For septic arthritis of the knee, elbow, ankle, or wrist, the joint may be drained via needle aspiration or arthroscopy. For septic arthritis of the hip, shoulder, or difficult-to-access joint (such as the sternoclavicular joint), the joint should be drained by arthroscopy. In any joint, arthroscopy may facilitate more thorough irrigation [59-62]. In one retrospective series including 72 cases of septic knee arthritis and 25 cases of septic hip arthritis, aspiration and surgical drainage were equally effective; aspiration was associated with a shorter hospital stay (21 versus 33 days) [63].

Surgical drainage is warranted in the following circumstances [10,11,64-66]:

- Adequate drainage cannot be achieved by needle aspiration or arthroscopy
- Suspicion for penetrating trauma with a residual foreign body
- Joint effusion persists after seven days of serial aspiration

Patients with severe infection may require repeated aspirations or arthroscopic irrigations, and in some cases, synovectomy [8]. Serial synovial fluid analyses should be performed; as infection is treated, these findings should demonstrate sterilization of the fluid and decreasing total white blood cell count. Adequacy of drainage may also be assessed clinically (based on improvement in fever curve, white blood cell count, joint swelling, and pain).

Antibiotic therapy

Initial approach — The initial choice of empiric antimicrobial therapy should cover the most likely pathogens. The approach below is supported by case series [4]; there are no randomized trials.

- If the initial Gram stain of synovial fluid demonstrates gram-positive cocci, empiric treatment with vancomycin (table 5) should be administered (algorithm 2) [67].

  - Patients with septic arthritis due to methicillin-susceptible *S. aureus* should be treated with a beta-lactam agent such as cefazolin (2 g intravenously [IV] every eight hours), nafcillin or oxacillin (2 g IV every four hours), or flucloxacillin (2 g IV every six hours). Patients who are allergic to penicillin can be treated with vancomycin.

  - Patients with septic arthritis due to methicillin-resistant *S. aureus* should be treated with vancomycin; if this is not feasible due to allergy or drug intolerance, reasonable alternative agents include daptomycin (6 mg/kg/day IV), linezolid (600 mg orally or IV twice daily), or clindamycin (600 mg orally or IV three times daily) [67].

- If the initial Gram stain of synovial fluid demonstrates gram-negative bacilli, treatment should be guided by risk for *Pseudomonas* infection (algorithm 2):

  - Patients with risk for *Pseudomonas* infection (eg, immunosuppressed patients and people who inject drugs [PWID]) warrant empiric coverage for *Pseudomonas* infection.

    - For patients who have sepsis or septic shock, have neutropenia and bacteremia, have severe burns, or are in a setting where the incidence of resistance to the chosen antibiotic class is high (eg, >10 to 15 percent), we administer empiric therapy with a combination of two antipseudomonal agents from different antibiotic classes (eg, a beta-lactam with an aminoglycoside or a fluoroquinolone) (table 6). (See "Principles of antimicrobial therapy of Pseudomonas aeruginosa infections", section on 'Indications for combination therapy'.)

    - For patients without any of these additional risk factors for mortality or resistant organisms, we administer empiric treatment with a single antipseudomonal agent (eg, ceftazidime 2 g IV every eight hours or cefepime 2 g IV every 8 to 12 hours).

  - Patients with no risk factors for *Pseudomonas* infection warrant treatment with a third-generation cephalosporin (eg, ceftriaxone 2 g IV once daily or cefotaxime 2 g IV every eight hours).

  Empiric antimicrobial therapy should be tailored to antimicrobial susceptibility data when available.

- If the initial Gram stain of synovial fluid is negative but synovial fluid cell count is consistent with septic arthritis, the approach depends on individual clinical circumstances (algorithm 3) [30]:

- For immunocompetent patients without confounding factors (such as trauma), we suggest treatment with vancomycin.

- For patients with traumatic bacterial arthritis, we suggest treatment with vancomycin plus a third-generation cephalosporin (ceftriaxone or cefotaxime).

- For immunocompromised patients and PWID, we suggest treatment with vancomycin plus a cephalosporin with activity against *Pseudomonas* (ceftazidime or cefepime).

For patients with septic arthritis associated with an animal bite, the approach to antibiotic selection is discussed separately. (See "Animal bites (dogs, cats, and other animals): Evaluation and management", section on 'Spectrum of therapy'.)

The initial antibiotic regimen should be tailored to culture and susceptibility results when available. As an example, vancomycin should be discontinued in patients with staphylococcal or streptococcal infections that are susceptible to beta-lactam therapy.

In some circumstances, parenteral therapy may be switched to oral therapy following debridement and finalization of microbiology data [68]. Considerations include pathogen susceptibility to an oral antimicrobial agent with good bioavailability and substantial barrier to use of outpatient parenteral therapy. We do not use oral therapy for treatment of septic arthritis in the context of *S. aureus* bacteremia, poor compliance, or gastrointestinal conditions that could interfere with absorption [69].

There is no role for intra-articular antibiotics, since systemic antibiotic therapy produces adequate drug levels in joint fluid. Furthermore, direct instillation of antibiotics into a joint may induce an inflammatory response and carries a risk of iatrogenic complications such as secondary infection [11].

Duration — The optimal duration of antimicrobial therapy for treatment of septic arthritis is uncertain. The approach below is supported by case series [4].

For patients with septic arthritis due to *S. aureus* in the setting of concomitant bacteremia (but no evidence of endocarditis), we administer parenteral therapy for four weeks.

For patients with septic arthritis due to *S. aureus* in the absence of concomitant bacteremia or signs of endocarditis, we administer parenteral antibiotics for at least 14 days, followed by oral therapy for an additional 7 to 14 days. The choice of oral antibiotic regimen for completion of therapy depends on the pathogen:

- For septic arthritis due to methicillin-sensitive *S. aureus*, suitable choices include dicloxacillin (500 mg orally every six hours), flucloxacillin (500 mg orally every six hours), or cephalexin (500 mg orally every six hours). Patients who are allergic to penicillin can be treated with clindamycin (600 mg orally every eight hours).

- For septic arthritis due to methicillin-resistant *S. aureus*, suitable choices include clindamycin, trimethoprim-sulfamethoxazole, doxycycline (or minocycline), linezolid (or tedizolid), and rifampin in combination with either ciprofloxacin or fusidic acid (table 7).

For patients with septic arthritis due to organisms that are susceptible to oral agents with high bioavailability (such as a fluoroquinolone), we favor treatment with a short course (four to seven days) of parenteral therapy, followed by 14 to 21 days of oral therapy. Compliance and response to therapy should be monitored carefully in such cases.

For patients with septic arthritis due to difficult-to-treat pathogens (such as *P. aeruginosa* or *Enterobacter* spp), longer courses of outpatient parenteral antibiotic therapy (eg, three to four weeks) may be necessary, especially if the response to therapy is slow or the patient is immunosuppressed.

For patients with septic arthritis and contiguous osteomyelitis, a long (four to six week) course of antibiotics may be indicated. (See "Nonvertebral osteomyelitis in adults: Treatment".)

For patients with septic arthritis in the setting of endocarditis, the duration of therapy is guided by the duration required for treatment of endocarditis. (See "Antimicrobial therapy of left-sided native valve endocarditis" and "Antimicrobial therapy of prosthetic valve endocarditis".)

One randomized trial suggested two weeks of antibiotic therapy may be noninferior to four weeks of therapy after surgical drainage; however, of the 154 patients enrolled, most had septic arthritis of the wrist or hand (64 percent); only 1 hip infection and 14 knee infections were included [70]. Furthermore, there no cases of infection due to methicillin-resistant *S. aureus*, and more than two-thirds of patients had pathogens other than *S. aureus*.

OUTCOME

In the United States, these were 13,700 hospital admissions for septic arthritis in 2012 [2]. Average length of stay was seven days and only 40 percent of patients were discharged home. Discharge to a rehabilitation or nursing facility, longer hospital stay, and worse outcome correlated with age >50 years, Medicaid and self-pay as primary payer, teaching hospital status,

heart failure, and diabetes. There was a 3 percent mortality rate during the primary hospitalization.

Unfortunately, there are few studies on the long-term joint outcome in patients with septic arthritis. In most, the functional outcome has depended on host factors (such as pre-existing joint disease), the virulence of the infecting organism, and duration of infection prior to initiation of therapy.

In one study including 121 adults with septic arthritis, a poor joint outcome (as defined by the need for amputation, arthrodesis, prosthetic surgery, or severe functional deterioration) occurred in one-third of the patients; adverse prognostic factors included older age and pre-existing joint disease [9].

The pathogen may also influence the outcome of treatment. In studies of septic arthritis due to *S. aureus,* poor joint outcomes have been observed in up to half of patients following completion of therapy [11,71]. In a three-year period, 93 patients with *S. aureus* arthritis were identified and 40 percent were methicillin-resistant *S. aureus* [72]. More than 90 percent of the cases were community acquired and 44 percent of the patients had diabetes mellitus. The in-hospital mortality rate was 5.4 percent. In contrast, a study of patients with pneumococcal septic arthritis noted a return to baseline joint function (or only mild limitation of joint motion) following therapy in 95 percent of cases [29].

Mortality due to septic arthritis depends on comorbid conditions such as advanced age, renal or cardiac disease, and immunosuppression. The mortality rates in most series range from 10 to 15 percent [9]. Polyarticular septic arthritis, particularly when it is due to *S. aureus* or occurs in the presence of rheumatoid arthritis, has an extremely poor prognosis, with mortality rates as high as 50 percent [34]. Mortality due to septic pneumococcal arthritis was reported as 19 percent in one series [29].

In a series of 55 patients treated with arthroscopic lavage and antibiotics for septic arthritis of the knee, medical comorbidities, increased age, and multiple medication use had worse outcomes [73].

The outcome of septic arthritis in people who inject drugs (PWID) is worse than in most other patient groups. In a nation-wide series of septic knees, the proportion of patients with injection drug user-related septic arthritis increased from 5 percent in 2000 to 11 percent in 2013 [74]. PWID-related cases were more likely to require repeat surgical procedures, longer hospital stays, and had higher mortality rates.

In one series including more than 500 adult cases of native joint septic arthritis in New Zealand between 2009 and 2014, large joint septic arthritis was associated with a higher rate of treatment failure than small joint septic arthritis (23 versus 12 percent) [16]. Adverse outcomes included death (5 percent), relapse (5 percent), reinfection (6 percent), and amputation (3 percent).

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Septic arthritis in adults".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Septic arthritis (The Basics)")
- Beyond the Basics topics (see "Patient education: Arthritis (Beyond the Basics)" and "Patient education: Joint infection (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Pathogenesis** – Septic arthritis refers to infection in a joint. Most commonly, septic arthritis arises via hematogenous seeding. It may also develop as a result of direct inoculation of bacteria into the joint. Rarely, septic arthritis develops via extension of

infection into the joint space from adjacent tissues. (See 'Introduction' above and 'Mechanism of infection' above.)

- **Microbiology** – Septic arthritis is usually monomicrobial. *Staphylococcus aureus* (including methicillin-resistant *S. aureus*) is the most common cause of septic arthritis in adults (table 1). Other gram-positive organisms such as streptococci are also important potential causes. Septic arthritis due to gram-negative bacilli generally occurs in older adults, patients with underlying immunosuppression, or intravenous drug users; it may also occur as a complication of trauma. Rarely, nonbacterial organisms, such as fungi, can cause septic arthritis. (See 'Microbiology' above.)

- **Clinical manifestations** – Patients with septic arthritis usually present acutely with a single swollen and painful joint (ie, monoarticular arthritis); oligoarticular or polyarticular infection occurs in approximately 20 percent of patients. Joint pain, swelling, warmth, and restricted movement occur in most cases. Patients with septic arthritis are usually febrile; older patients may be afebrile. The knee is involved in more than half of cases; wrists, ankles, and hips are also affected commonly. (See 'Clinical manifestations' above.)

- **Approach to diagnosis** – The diagnosis of septic arthritis should be suspected in patients with acute onset of at least one swollen, painful joint. Prior to administration of antibiotics, synovial fluid should be sent for Gram stain, bacterial culture, white blood cell count with differential, and assessment for crystals. In addition, blood cultures (two sets) and radiographs of the involved joint should be obtained. (See 'Diagnosis' above.)

- **Interpreting diagnostic tests** – The diagnosis of septic arthritis is established based on synovial fluid analysis and culture (algorithm 1 and table 2). A definitive diagnosis is made if synovial fluid Gram stain and/or culture is positive. If synovial fluid Gram stain and culture are negative, a presumptive diagnosis can be made based on purulence of the synovial fluid (leukocyte count >20,000 cells/microL, mostly neutrophils). (See 'Diagnosis' above.)

- **Management** – Treatment of acute bacterial arthritis consists of joint drainage or debridement coupled with antibiotic therapy. (See 'Treatment' above.)

  - **Joint drainage or debridement** – Approaches to joint drainage for management of septic arthritis include needle aspiration, arthroscopy, or arthrotomy (open surgical drainage). (See 'Joint drainage' above.)

  - **Empiric antibiotic selection** – The choice of empiric antimicrobial therapy should cover the most likely pathogens (see 'Initial approach' above):

- If the initial Gram stain of the synovial fluid shows gram-positive cocci, treatment consists of vancomycin (algorithm 2).

- If the initial Gram stain of the synovial fluid shows gram-negative bacilli, treatment should be guided by risk for Pseudomonal infection (algorithm 2). Patients with risk for *Pseudomonas* infection (eg, immunosuppressed patients and people who inject drugs [PWID]) warrant empiric coverage for *Pseudomonas* infection.

  For patients who have sepsis or septic shock, have neutropenia and bacteremia, have severe burns, or are in a setting where the incidence of resistance to the chosen antibiotic class is high (eg, >10 to 15 percent), we suggest treatment a combination of two antipseudomonal agents from different antibiotic classes (eg, a beta-lactam with an aminoglycoside or a fluoroquinolone) (table 6) (**Grade 2C**). (See "Principles of antimicrobial therapy of Pseudomonas aeruginosa infections".)

  For patients without any of these additional risk factors for mortality or resistant organisms, we suggest treatment with a single antipseudomonal agent (table 6) (**Grade 2B**). (See "Principles of antimicrobial therapy of Pseudomonas aeruginosa infections".)

  For patients with no risk factors for *Pseudomonas* infection, treatment consists of a third-generation cephalosporin.

- If the initial Gram stain of synovial fluid is negative but synovial fluid cell count is consistent with septic arthritis, the approach depends on individual clinical circumstances (algorithm 3).

  For immunocompetent patients without confounding factors (such as trauma), we suggest treatment with vancomycin (**Grade 2C**).

  For patients with traumatic bacterial arthritis, we suggest treatment with vancomycin plus a third-generation cephalosporin (**Grade 2C**).

  For immunocompromised patients and PWID, we suggest treatment with vancomycin plus a cephalosporin with activity agains*t Pseudomonas* (ceftazidime or cefepime) (**Grade 2C**).

- **Targeted antibiotic therapy** – Once microbiology and susceptibility data are available, antibiotic therapy should be narrowed.

- **Duration of therapy** – The duration of therapy is tailored to individual clinical circumstances as described above. (See 'Antibiotic therapy' above.)

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Topic 7666 Version 51.0

GRAPHICS

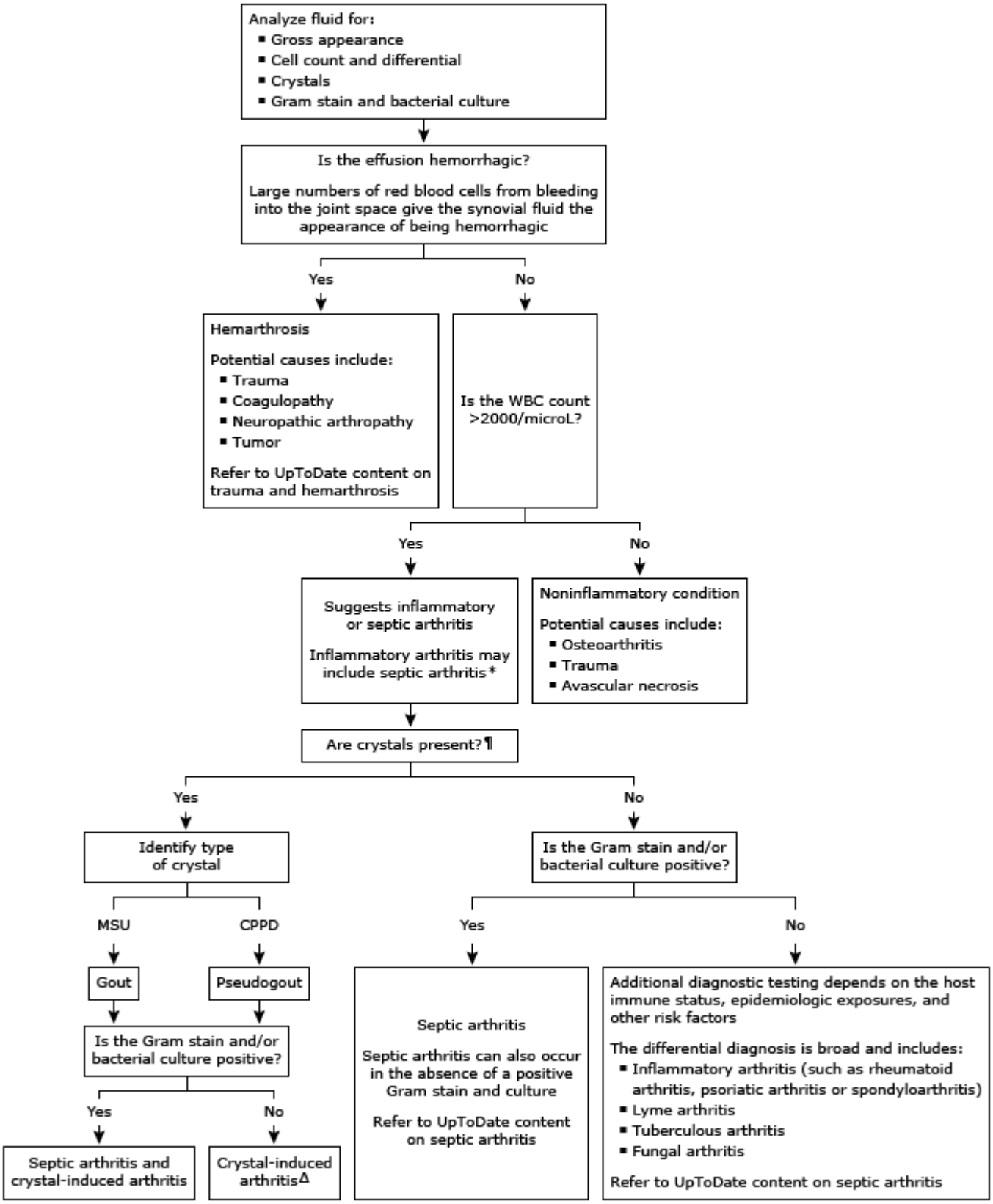
Causes of infectious arthritis

Organism	Clinical clues
Staphylococcus aureus	Healthy adults, skin breakdown, previously damaged joint (eg, rheumatoid arthritis), prosthetic joint
Streptococcal species	Healthy adults, splenic dysfunction
Neisseria gonorrhoeae	Healthy adults (particularly young, sexually active), associated tenosynovitis, vesicular pustules, late complement deficiency, negative synovial fluid culture and Gram stain
Aerobic gram-negative bacteria	Immunocompromised hosts, gastrointestinal infection
Anaerobic gram-negative bacteria	Immunocompromised hosts, gastrointestinal infection
Brucellosis	Zoonosis
Mycobacterial species	Immunocompromised hosts, travel to or residence in an endemic area
Fungal species (*Candida* species, sporotrichosis, *Cryptococcus*, blastomycosis, coccidioidomycosis)	Immunocompromised hosts
Spirochete (*Borellia burgdorferi*)	Exposure to ticks, antecedent rash, knee joint involvement
Mycoplasma hominis	Immunocompromised hosts with prior urinary tract manipulation

Refer to separate UpToDate topic for discussion of viral causes of arthritis.

Graphic 57688 Version 10.0

Guide to interpretation of synovial fluid analysis



WBC: white blood cell; MSU: monosodium urate; CPPD: calcium pyrophosphate crystal deposition.

* Septic arthritis is typically associated with synovial fluid white blood cell counts >20,000 cells/microL, but lower counts may be observed, especially for arthritis due to disseminated gonococcal infection. With most bacterial organisms, particularly *Staphylococcus aureus*, the synovial fluid white blood cell count is typically >50,000 cells/microL (and often >100,000 cells/microL).

¶ Crystal-induced arthritis may still be considered despite the absence of identified crystals; false-negative results occur, especially with CPPD.

Δ If treatment of crystal-induced arthritis does not result in clinical improvement, consider other inflammatory or infectious arthridites.

Graphic 111452 Version 2.0

Classification of joint fluid based on synovial fluid characteristics

	Noninflammatory (such as osteoarthritis)	Inflammatory (such as rheumatoid arthritis)	Septic	Hemorrhagic
WBC count (cells/microL)	<2000	2000 to 20,000	>20,000*	Up to 1 WBC for every 1000 RBCs
Percent neutrophils	<25%	50 to 75%	>75%	<50%¶
Crystal examination by polarized microscopy	Negative	May demonstrate uric acid or CPPD crystals	Negative	Bloody
Stain, culture for microorganisms	Negative	Negative$^{\Delta}$	May be positive (depending on organism, prior antibiotic exposure)	Negative

WBC: white blood cell; RBC: red blood cell; CPPD: calcium pyrophosphate dihydrate deposition.

* With most bacterial organisms, particularly *Staphylococcus aureus*, the synovial fluid WBC count is typically >50,000 cells/microL (and often >100,000 cells/microL). However, lower counts (in the inflammatory range) may be observed in the setting of septic arthritis, especially for disseminated gonococcal infection or if antibiotics were administered prior to joint fluid sampling.

¶ Hemorrhagic synovial fluid usually has <50% neutrophils; however, in some cases a higher proportion may be observed, reflecting the peripheral neutrophil count.

Δ Inflammatory findings are typically observed in the setting of rheumatologic conditions and crystal-induced arthritis; however, concomitant infection also warrants consideration, and synovial fluid culture should be obtained routinely during synovial fluid analysis. In the setting of noninfectious inflammatory arthritis, synovial fluid leukocyte counts may be >20,000 cells/microL (often termed "pseudoseptic"). In general, the higher the synovial fluid leukocyte count, the greater the concern for septic arthritis.

Graphic 76506 Version 12.0

Differential diagnosis of acute monoarthritis

Infection
Bacterial
Fungal
Mycobacterial
Viral
Spirochete
Crystal induced
Monosodium urate
Calcium pyrophosphate dihydrate
Hydroxyapatite
Calcium oxalate
Lipid
Hemarthrosis
Trauma
Anticoagulation
Clotting disorders
Fracture
Pigmented villonodular synovitis

Tumor
Tenosynovial giant cell tumor (formerly pigmented villonodular synovitis)
Chondrosarcoma
Osteoid osteoma
Metastatic disease
Systemic rheumatic disease
Rheumatoid arthritis
Spondyloarthritis
Systemic lupus erythematosus
Sarcoidosis
Osteoarthritis
Erosive variant
Intraarticular derangement
Meniscal tear
Osteonecrosis
Fracture
Other
Plant thorn synovitis

Graphic 62597 Version 5.0

Major causes of inflammatory polyarthritis

Infectious arthritis

- Bacterial
  - Lyme disease
  - Bacterial endocarditis
- Viral
- Other infections

Postinfectious (reactive) arthritis

- Rheumatic fever
- Reactive arthritis
- Enteric infection

Other seronegative spondyloarthritides

- Ankylosing spondylitis
- Psoriatic arthritis
- Inflammatory bowel disease

Rheumatoid arthritis

Inflammatory osteoarthritis

Crystal-induced arthritis

Juvenile idiopathic arthritis

Systemic rheumatic illnesses

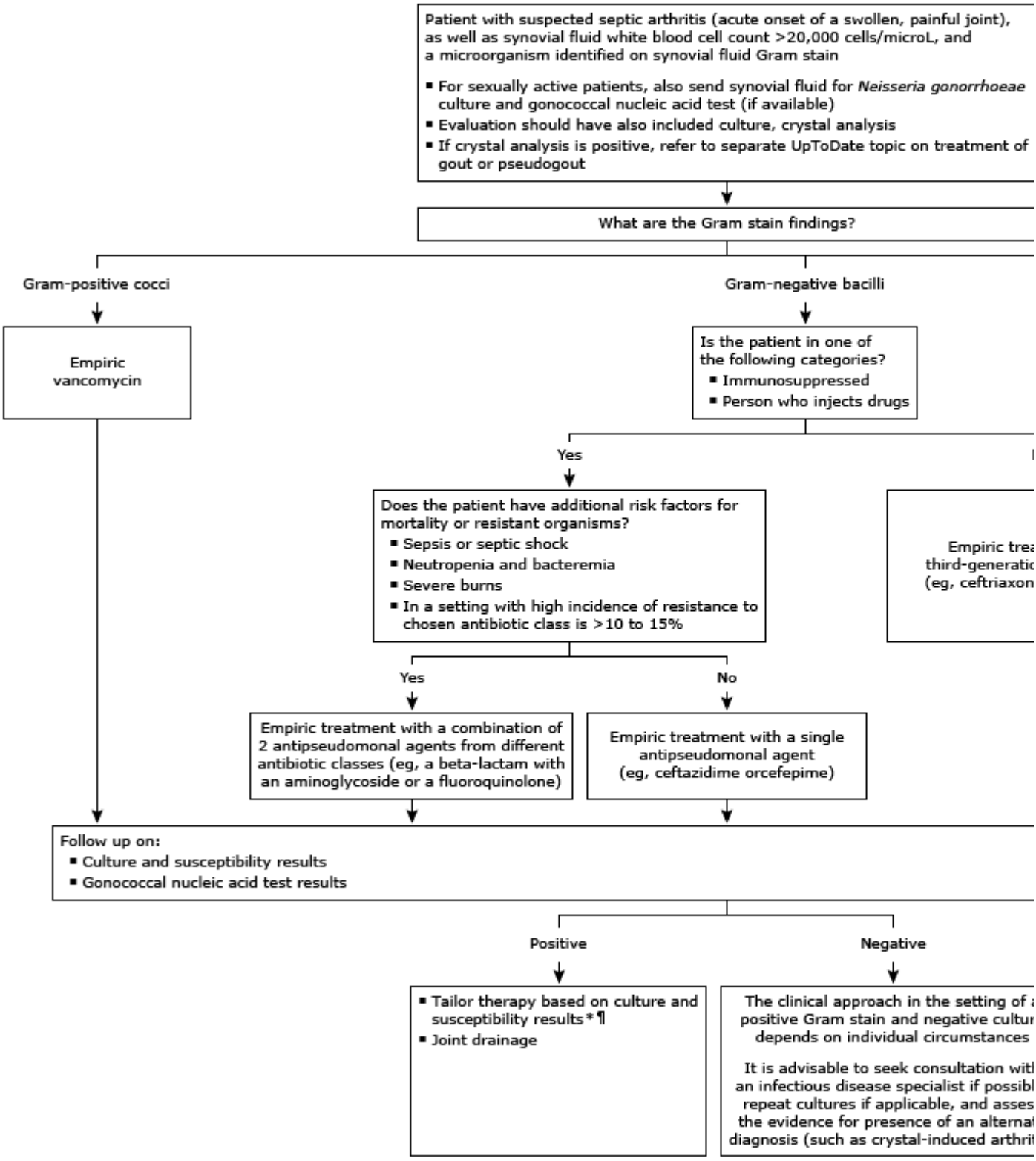
- Systemic lupus erythematosus
- Systemic vasculitis
- Systemic sclerosis
- Polymyositis/dermatomyositis
- Adult-onset Still's disease
- Behçet syndrome
- Relapsing polychondritis
- Autoinflammatory disorders

Other systemic illnesses

- Sarcoidosis
- Palindromic rheumatism
- Familial Mediterranean fever
- Malignancy
- Hyperlipoproteinemias

Graphic 74266 Version 8.0

Approach to patients with suspected septic arthritis and positive synovial fluid



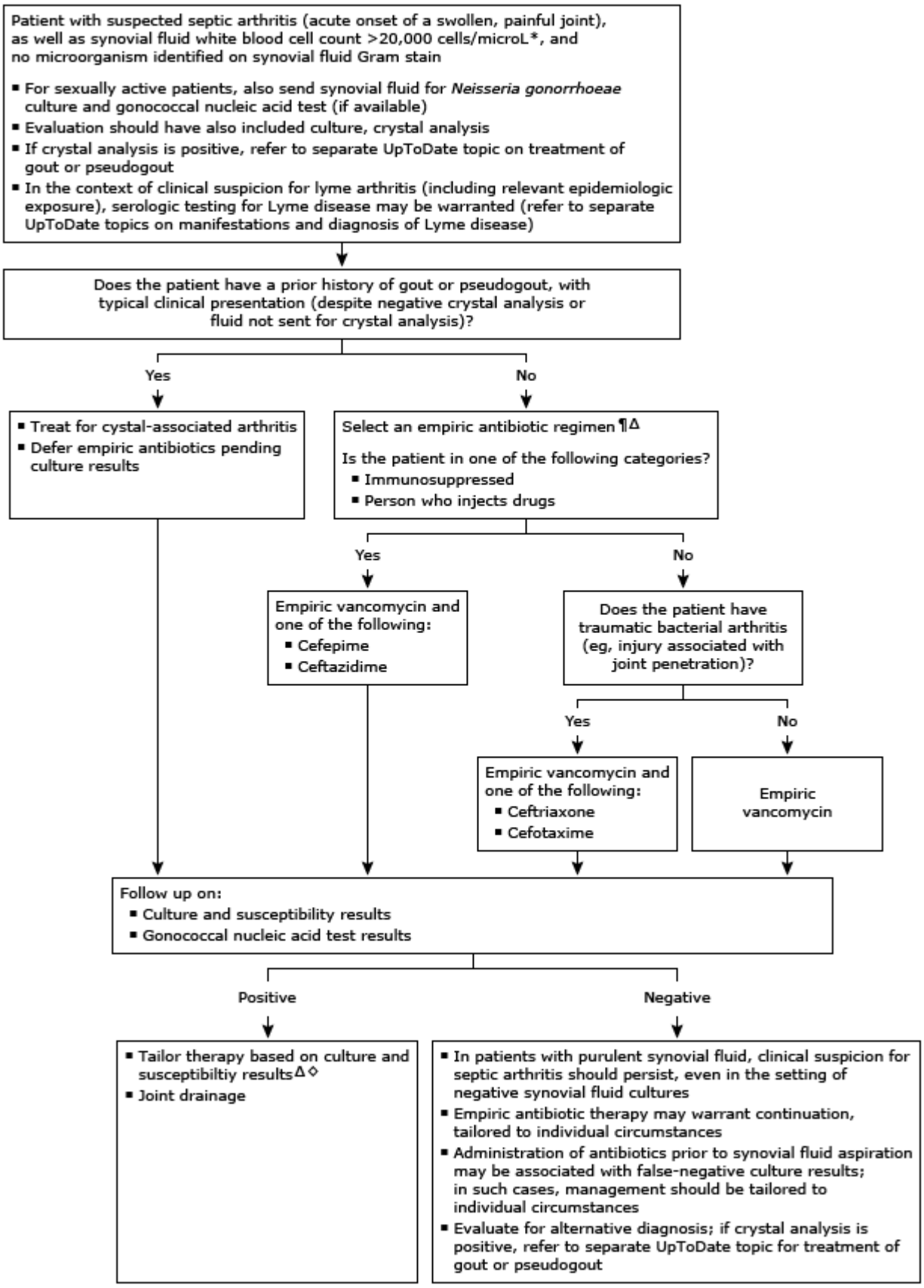
This algorithm summarizes an approach to evaluation and management of patients with suspected septic a stain. Issues related to septic arthritis associated with animal bites are discussed separately (refer to UpToD related to septic bursitis (refer to UpToDate topic on septic bursitis).

* Refer to UpToDate topic on disseminated gonococcal infection for details on treatment regimens.

¶ The duration of therapy for septic arthritis is tailored to individual clinical circumstances (refer to UpToDat discussion).

Graphic 134805 Version 1.0

Approach to patients with suspected septic arthritis and negative synovial fluid Gram stain



This algorithm summarizes an approach to evaluation and management of patients with suspected septic arthritis and negative synovial fluid Gram stain. Issues related to septic arthritis associated with animal bites are discussed separately (refer to UpToDate topic on animal bites), as are issues related to septic bursitis (refer to UpToDate topic on septic bursitis).

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* With most bacterial organisms, particularly *Staphylococcus aureus*, the synovial fluid white blood cell count is typically >50,000 cells/microL (and often >100,000 cells/microL). However, lower counts may be observed in the setting of septic arthritis, especially for disseminated gonococcal infection or if antibiotics were administered prior to joint fluid sampling.

¶ If there is high suspicion for disseminated gonococcal infection (eg, tenosynovitis or dermatitis, evidence of urogenital, rectal or pharyngeal gonococcal infection), including empiric treatment for gonococcal infection is reasonable.

Δ Refer to separate UpToDate topic on disseminated gonococcal infection for details on treatment regimens.

◇ The duration of therapy for septic arthritis is tailored to individual clinical circumstances (refer to UpToDate topic on septic arthritis for further discussion).

Graphic 134806 Version 1.0

Approach to vancomycin dosing for adults with normal kidney function*

Loading dose (for patients with known or suspected severe *Staphylococcus aureus* infection)¶	Load 20 to 35 mg/kg (based on actual body weight, rounded to the nearest 250 mg increment; not to exceed 3000 mg). Within this range, we use a higher dose for critically ill patients; we use a lower dose for patients who are obese and/or are receiving vancomycin via continuous infusion.
Initial maintenance dose and interval	Typically 15 to 20 mg/kg every 8 to 12 hours for most patients (based on actual body weight, rounded to the nearest 250 mg increment). In general, the approach to establishing the vancomycin dose/interval is guided by a nomogram.Δ
Subsequent dose and interval adjustments	Based on AUC-guided (preferred for severe infection)[1] or trough-guided serum concentration monitoring.◇

AUC: area under the 24-hour time-concentration curve.

* Refer to the UpToDate topic on vancomycin dosing for management of patients with abnormal kidney function.

¶ For patients with known or suspected severe *S. aureus* infection, we suggest administration of a loading dose to reduce the likelihood of suboptimal initial vancomycin exposure. Severe *S. aureus* infections include (but are not limited to) bacteremia, endocarditis, osteomyelitis, prosthetic joint infection, pneumonia warranting hospitalization, infection involving the central nervous system, or infection causing critical illness.

Δ If possible, the nomogram should be developed and validated at the institution where it is used, to best reflect the regional patient population. Refer to UpToDate topic on vancomycin dosing for sample nomogram.

◇ Refer to the UpToDate topic on vancomycin dosing for discussion of AUC-guided and trough-guided vancomycin dosing. For patients with nonsevere infection who receive vancomycin for <3 days (in the setting of stable kidney function and absence of other risk factors for altered vancomycin kinetics), vancomycin concentration monitoring is often omitted; the value of such monitoring prior to achieving steady state (usually around treatment day 2 to 3) is uncertain.

Reference:

1. *Rybak MJ, Le J, Lodise TP, et al. Therapeutic Monitoring of Vancomycin for Serious Methicillin-Resistant Staphylococcus Aureus Infections: A Revised Consensus Guideline and Review by the American Society of Health-System Pharmacists, the Infectious Diseases Society of America, the Pediatric Infectious Diseases Society, and the Society of Infectious Diseases Pharmacists. Am J Health Syst Pharm 2020; 77:835.*

Graphic 128911 Version 4.0

Antibiotics used for the treatment of *Pseudomonas aeruginosa* infections in adults

Class	Agent	Dose
Penicillin-beta-lactamase combinations*	Piperacillin-tazobactam	4.5 g IV every 6 hours
	Ticarcillin-clavulanate (not available in the United States or Canada)	3.1 g IV every 4 hours
Cephalosporins	Ceftazidime	2 g IV every 8 hours
	Cefepime	2 g IV every 8 or 12 hours¶
	Cefoperazone	2 g IV every 12 hours
	Cefiderocol$^{\Delta}$	2 g IV every 8 hours
Monobactams	Aztreonam	2 g IV every 8 hours
Fluoroquinolones$^{\diamond}$	Ciprofloxacin	400 mg IV every 8 to 12 hours or 750 mg orally every 12 hours
	Levofloxacin	750 mg IV or orally once daily
Carbapenems§	Meropenem	1 g IV every 8 hours
	Doripenem	500 mg IV every 8 hours
	Imipenem	500 mg IV every 6 hours¥
Advanced beta-lactamase inhibitor combinations$^{\Delta}$	Ceftazidime-avibactam	2.5 g IV every 8 hours
	Ceftolozane-tazobactam	1.5 to 3 g IV every 8 hours‡
	Imipenem-cilastatin-relebactam	1.25 g IV every 6 hours
Aminoglycosides†	Tobramycin	Dosing of aminoglycosides is discussed in detail in a dedicated topic
	Gentamicin	
	Amikacin	
	Plazomicin	
Polymyxins**	Colistin	Dosing of polymyxins is discussed in detail in a dedicated topic
	Polymyxin B	

Doses refer to intravenous administration. Doses listed are for patients with normal renal function; dose adjustments may be warranted for renal impairment.

IV: intravenous; MIC: minimum inhibitory concentration.

* Ticarcillin (3 g every 4 hours) and piperacillin (4 g every 4 hours) each have antipseudomonal activity but are not available in the United States as single agents without the beta-lactamase inhibitor.

¶ We aim to use the higher dose, particularly for severe infections or neutropenic patients, but dosing should take into account the condition treated, the MIC of the isolate, the potential for toxicity, and other patient specific factors.

Δ The novel cephalosporin cefiderocol and combination agents that include a cephalosporin or carbapenem plus a beta-lactamase inhibitor are generally reserved for infections resistant to other agents. The addition of vaborbactam to meropenem does not enhance the clinical activity of meropenem against carbapenem-resistant *P. aeruginosa*.

◇ Fluoroquinolones are the only class of antibiotics with antipseudomonal activity that have an oral formulation.

§ Carbapenems given as a single therapy have the propensity to induce resistance during treatment.

¥ Among the carbapenems, we favor meropenem or doripenem over imipenem, which has a higher propensity to induce resistance during treatment. For severe infection (eg, septic shock), imipenem can be given up to a dose of 1 g every 8 hours.

‡ Pulmonary infections are treated with the 3 g every 8 hour dose.

† Aminoglycosides are generally used in combination with a beta-lactam and should NOT be used as a single agent for infections other than those of the lower urinary tract. When using an aminoglycoside as part of therapy for an infection with a high risk of *P. aeruginosa*, we favor tobramycin over gentamicin as it has greater intrinsic antipseudomonal activity; however, it may not be widely available.

** Polymyxins are generally reserved for the treatment of serious infections caused by *P. aeruginosa* isolates resistant to other agents. In such cases, they are often administered in combination with other antimicrobial agents and with a loading dose preceding the standing dose. Refer to other UpToDate content for more details on polymyxin dosing.

Graphic 91115 Version 14.0

Oral antimicrobial agents for completing treatment of septic arthritis due to methicillin-resistant *Staphylococcus aureus* (MRSA) in adults*

Treatment	Adult dose
Clindamycin	600 mg orally three times daily
Trimethoprim-sulfamethoxazole (cotrimoxazole)	2 DS tablets orally twice daily (alternatively: 4 mg/kg per dose [trimethoprim component] orally twice daily [maximum 2 DS tablets twice daily])
Doxycycline	100 mg orally twice daily
Minocycline	200 mg orally once, then 100 mg orally twice daily
Linezolid¶	600 mg orally twice daily
Rifampin$^{\Delta}$	300 to 450 mg orally twice daily◇
PLUS	
One of the following agents:	
Ciprofloxacin	500 to 750 mg twice daily
Fusidic acid (where available)§	500 mg orally 3 times daily

The doses above are intended for patients with normal renal function; dosing may require adjustment in patients with renal insufficiency.

DS: double strength (ie, 160 mg trimethoprim with 800 mg sulfamethoxazole per tablet).

* Following joint drainage, the typical duration of antibiotic therapy for treatment of septic arthritis is three to four weeks; we typically administer parenteral antibiotics for at least 14 days followed by oral therapy for an additional 14 days. If linezolid is used as initial therapy, it may be administered orally for the entire duration given its high oral bioavailability.

¶ The mechanism of action for tedizolid is comparable with that of linezolid; however, thus far there are no data/experience regarding use of tedizolid for treatment of septic arthritis.

Δ Patients who cannot take rifampin because of drug resistance, allergy, toxicity, intolerance, or drug-drug interactions should remain on intravenous antistaphylococcal therapy for 4 to 6 weeks (before transitioning to antibiotic suppression with an oral regimen, if warranted).

◇ We favor administration of rifampin 450 orally twice daily; the dose may be reduced to 300 mg orally twice daily in the setting of nausea.

§ Not available in the United States. Fusidic acid should not be used alone; it must be combined with a second active agent to reduce the likelihood of selection for drug resistance. When rifampin is combined with fusidic acid, fusidic levels may be reduced.

Data from:

1. *Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis 2014; 59:e10.*
2. *Liu C, Bayer A, Cosgrove SE, et al. Clinical Practice Guidelines by the Infectious Diseases Society of America for the Treatment of Methicillin-Resistant Staphylococcus Aureus Infections in Adults and Children. Clin Infect Dis 2011; 52:e18.*

Graphic 114503 Version 2.0

Contributor Disclosures

Don L Goldenberg, MD No relevant financial relationship(s) with ineligible companies to disclose. **Daniel J Sexton, MD** Equity Ownership/Stock Options: Magnolia Medical Technologies [Medical diagnostics]. Consultant/Advisory Boards: Magnolia Medical Technologies [Medical diagnostics]. All of the relevant financial relationships listed have been mitigated. **Denis Spelman, MBBS, FRACP, FRCPA, MPH** No relevant financial relationship(s) with ineligible companies to disclose. **Keri K Hall, MD, MS** No relevant financial relationship(s) with ineligible companies to disclose.

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