INTRODUCTION

Many mechanisms provoke acute joint symptoms: degradation and degeneration of articular cartilage (osteoarthritis), deposition of immune complexes or immune system–related phenomena (rheumatoid arthritis, rheumatic fever and possibly, a component of gonococcal arthritis), crystal-induced inflammation (gout and pseudogout), seronegative spondyloarthopathies (ankylosing spondylitis [see chapter 282, "Systemic Rheumatic Diseases"] and reactive arthritis [postinfectious with HLA-B27 susceptibility]), and bacterial invasion (gonococcal and nongonococcal septic arthritis, including Lyme arthritis) or viral invasion (viral arthritis). These processes impact joint capsules and surfaces, resulting in a cascade of reactive and inflammatory events. Septic arthritis is invasion of a joint by an infectious agent with organism proliferation and associated inflammation; bacterial arthritis is a subset of septic arthritis. Under ideal conditions, the infectious agent is recoverable from the joint fluid in septic arthritis, but in clinical practice, this is often not the case. This chapter reviews the common causes and treatments of acute nontraumatic joint pain. Joint injuries are discussed in section 22, "Injuries to Bones and Joints," and disorders due to repetitive use syndromes are discussed in section 23, "Musculoskeletal Disorders," by anatomic site.

CLINICAL APPROACH TO ACUTE JOINT PAIN

Septic arthritis is the most important consideration in the evaluation of a swollen, warm, and painful joint. Urgent treatment may prevent both joint destruction and mortality (11% with treatment).1,2 The diagnosis of septic arthritis is clinical and is supported by diagnostic tests.1,2 No single diagnostic parameter is sufficiently sensitive to screen patients for septic arthritis including synovial WBC counts.3

CLINICAL FEATURES AND RISK FACTORS

Risk factors (Table 284-1),3,4 the number of joints involved (Table 284-2), and the migratory pattern (Table 284-3), if one exists, aid in the differential diagnosis. Approximately 85% of patients with nongonococcal septic arthritis present with a single joint infected; Staphylococcus aureus and Streptococcus pneumoniae are more likely to infect two or more joints simultaneously.5,6,7,8 Septic arthritis involving more than one joint can occur in rheumatoid arthritis (50%), immunocompromise, gout, diabetes, and/or renal disease; the mortality rate is significantly higher in patients with polyarticular septic arthritis (11% vs 30%).5,7 Recent joint surgery and cellulitis overlying a prosthetic hip or knee are the only findings on history or physical examination that significantly alter (both increase) the probability of nongonococcal septic arthritis.3
# Risk Factors for Nongonococcal and Gonococcal Septic Arthritis

<table>
<thead>
<tr>
<th>Nongonococcal</th>
<th>Gonococcal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injection drug use*</td>
<td>HIV infection*</td>
</tr>
<tr>
<td>Diabetes mellitus*</td>
<td>Injection drug use*</td>
</tr>
<tr>
<td>Rheumatoid arthritis*</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>Prosthetic joint, knee, * or hip*</td>
<td>Menses</td>
</tr>
<tr>
<td>Immunosuppression, HIV*</td>
<td>Systemic lupus erythematosus</td>
</tr>
<tr>
<td>Age: &gt;80 y old*</td>
<td>Complement deficiency</td>
</tr>
<tr>
<td>Skin ulceration and/or infection*</td>
<td></td>
</tr>
<tr>
<td>Hemophilia</td>
<td></td>
</tr>
<tr>
<td>Hypogammaglobulinemia</td>
<td></td>
</tr>
<tr>
<td>Malignancy</td>
<td></td>
</tr>
<tr>
<td>Hemodialysis</td>
<td></td>
</tr>
<tr>
<td>Liver disease</td>
<td></td>
</tr>
<tr>
<td>Alcoholism</td>
<td></td>
</tr>
<tr>
<td>Steroid therapy</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviation:** HIV = human immunodeficiency virus.

*Risk factors supported by epidemiologic study.*
### TABLE 284-2

**Differential Diagnosis of Arthritis by Number of Affected Joints**

<table>
<thead>
<tr>
<th>Number of Joints</th>
<th>Differential Considerations for Typical Presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = Monoarthritis</td>
<td>85% of nongonococcal septic arthritis*&lt;br&gt;Crystal-induced (gout, pseudogout)&lt;br&gt;Gonococcal septic arthritis&lt;br&gt;Trauma-induced arthritis&lt;br&gt;Osteoarthritis (acute)&lt;br&gt;Lyme disease&lt;br&gt;Avascular necrosis&lt;br&gt;Tumor</td>
</tr>
<tr>
<td>2–3 = Oligoarthritis†</td>
<td>15% of nongonococcal septic arthritis, more common with <em>Staphylococcus aureus</em> and <em>Streptococcus pneumoniae</em>&lt;br&gt;Lyme disease&lt;br&gt;Reactive arthritis (Reiter’s syndrome)&lt;br&gt;Gonococcal arthritis&lt;br&gt;Rheumatic fever</td>
</tr>
<tr>
<td>&gt;3 = Polyarthritis†</td>
<td>Rheumatoid arthritis&lt;br&gt;Systemic lupus erythematosus&lt;br&gt;Viral arthritis&lt;br&gt;Osteoarthritis (chronic)&lt;br&gt;Serum sickness&lt;br&gt;Serum sickness–like reactions</td>
</tr>
</tbody>
</table>

* Involvement of more than one joint does not rule out septic arthritis.

†The distinction between oligoarthritis and polyarthritis varies in the literature with a cut point of either three or four joints.

### TABLE 284-3

**Common Joint Disorders with a Migratory Distribution Pattern**

- Gonococcal arthritis
- Acute rheumatic fever
- Lyme disease
- Viral arthritis
- Systemic lupus erythematosus
SYNOVIAL FLUID ANALYSIS

When septic arthritis is suspected, aspirate joint fluid, and obtain analysis and culture of the aspirate to direct treatment.\textsuperscript{1,2} Table 284-4 provides diagnostic guidance based on synovial fluid results in the context of different patient characteristics.\textsuperscript{1–12}
**TABLE 284-4**  
Septic Arthritis: Joint Aspiration Results in Different Patient Groups

<table>
<thead>
<tr>
<th>Key Factor</th>
<th>Patient Status</th>
<th>Joint Aspiration</th>
<th>Diagnostic Considerations/Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive Gram stain</td>
<td>Acute joint pain and swelling</td>
<td>Gram stain positive for bacteria</td>
<td>Initiate empiric IV antibiotics, admit to the hospital, monitor culture and patient course (positive Gram stain is found in &lt;50% of patients with septic arthritis).</td>
</tr>
<tr>
<td>Classic synovial WBC count</td>
<td>Acute joint pain and swelling</td>
<td>&gt;50,000 WBC/mm(^3) or &gt;90% PMNs</td>
<td>Synovial fluid with &gt;50,000 WBC/mm(^3) is 56% sensitive and 90% specific for septic arthritis. Initiate empiric IV antibiotics and hospital admission.</td>
</tr>
<tr>
<td>Increased sensitivity of lower synovial WBC counts</td>
<td>Acute joint pain and swelling in a patient with risk factors for septic arthritis or systemic signs of infection</td>
<td>&gt;25,000 WBC/mm(^3) or &gt;90% PMNs</td>
<td>Synovial fluid with &gt;25,000 WBC/mm(^3) is 73% sensitive and 77% specific for septic arthritis. Consider empiric IV antibiotics and admission to the hospital for monitoring of patient course and cultures.</td>
</tr>
<tr>
<td>Acute gout with coexisting septic arthritis</td>
<td>Acute joint pain and swelling; patient with acute gout or history of gout with systemic signs of infection</td>
<td>Crystals, &gt;2000 WBC/mm(^3), or &gt;90% PMNs</td>
<td>Crystal-induced arthritis may coexist with septic arthritis; cell counts are &lt;6000 WBC/mm(^3) in 10% of infected joints; more than one joint is involved in 10%–45%. Look for infected tophi. Consider empiric IV antibiotics and admission to the hospital for monitoring of patient course and cultures.</td>
</tr>
<tr>
<td>Prosthetic joint</td>
<td>Acute pain and swelling in patient with prosthetic joint</td>
<td>&gt;10,000 WBC/mm(^3), &gt;90% PMNs</td>
<td>Consult operating orthopedic surgeon before aspiration if possible. AAOS definition for acute periprosthetic infection is 3 of the following: (1) CRP elevated above 100 milligrams/L and ESR elevated above local norm, (2) synovial WBC &gt;10,000/mm(^3), (3) synovial PMNs &gt;90%, (4) positive culture, and (5) positive histologic analysis of periprosthetic tissue.</td>
</tr>
<tr>
<td>Key Factor</td>
<td>Patient Status</td>
<td>Joint Aspiration</td>
<td>Diagnostic Considerations/Management</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>-----------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Immunocompromise</td>
<td>Joint swelling in an immunocompromised patient or systemic signs of infection in a patient with immunocompromise</td>
<td>&gt;200 WBC/mm³, &gt;25% PMNs</td>
<td>Immunocompromised patients sustain septic arthritis with diminished immune response; cell counts and percent PMNs are frequently lower than in immunocompetent patients with similar infections. Consider empiric IV antibiotics and admission to the hospital for monitoring of patient course and cultures.</td>
</tr>
<tr>
<td>Gonococcal arthritis</td>
<td>Monoarticular or polyarticular joint pain in a patient with history of unprotected sex (primarily in young patients)</td>
<td>10,000–80,000 WBC/mm³</td>
<td>Positive culture in &lt;50% of infected joints; collect urogenital cultures plus pharynx and rectum cultures as determined by history. Consider empiric IV antibiotics and admission to the hospital for monitoring of patient course and cultures.</td>
</tr>
<tr>
<td>Rheumatoid arthritis with coexisting septic arthritis</td>
<td>Joint pain and/or swelling in a patient with rheumatoid arthritis</td>
<td>2000–120,000 WBC/mm³</td>
<td>Severe pain and limited range of motion may be absent in patients on immunosuppression. Look for infected rheumatoid nodules or ulcerated foot calluses; source in 76% of cases. Consider empiric IV antibiotics and admission to the hospital for monitoring of patient course and cultures.</td>
</tr>
<tr>
<td>Lyme disease</td>
<td>Acute joint pain in a patient living in a Lyme disease–endemic area or with a history of rash or tick bite</td>
<td>200–300,000 WBC/mm³</td>
<td>Consider empiric antibiotics and close follow-up to monitor culture and patient course; admit if clinical picture is indistinguishable from septic arthritis. Arthralgia appears months after initial symptoms. Joint effusion (moderate to large) may be out of proportion to the patient’s pain (mild to moderate). Knee is the most common affected joint.</td>
</tr>
<tr>
<td>Post trauma</td>
<td>Joint trauma several days prior, initial swelling, now increasing pain</td>
<td>0–2000 WBC/mm³, &lt;25% PMNs, 0–500 RBC/mm³</td>
<td>Posttraumatic effusions may become infected in patients with skin infections or bacteremia. Aspiration of the joint reduces pain for approximately 1 week, but has no effect on long-term disability.</td>
</tr>
</tbody>
</table>
### Key Factor

<table>
<thead>
<tr>
<th>Patient Status</th>
<th>Joint Aspiration</th>
<th>Diagnostic Considerations/Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient with acute joint pain and suspected swelling, with or without other symptoms or signs to suggest septic arthritis</td>
<td>&quot;Dry tap&quot;</td>
<td>Major causes of dry tap are mistaken physical diagnosis of effusion; blockage of the needle by plica, fat, or debris; or synovial fluid with high viscosity or true lipoma arborescens (benign replacement of subsynovial tissue by fat cells). Use US to determine true effusion and direct needle to largest collection of fluid.</td>
</tr>
<tr>
<td>Patient with sufficient joint pain and swelling to warrant arthrocentesis, no comorbidities, absent signs and symptoms of sepsis</td>
<td>&lt;200 WBC/mm³, &lt;25% PMNs</td>
<td>Normal WBC cell counts and differential percentages make the diagnosis of septic arthritis unlikely in a patient without comorbidities or objective signs of infection. A mechanism should be in place for timely follow-up of culture results if they turn positive.</td>
</tr>
</tbody>
</table>

**Abbreviations:** AAOS = American Academy of Orthopedic Surgeons; CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; PMNs = polymorphonuclear leukocytes.

*All patients who received joint aspiration for suspected septic arthritis should have cultures of synovial fluid, blood, and any nonjoint source clinically suspected of infection (e.g., skin, urine).*

Analyze joint fluid for Gram stain, leukocyte count with differential, and a wet preparation for crystals. Glucose, protein, and lactate dehydrogenase levels do not direct treatment decisions. Synovial lactate levels may prove an aide in identifying septic arthritis if future studies confirm preliminary reports. Culture for gonococci and anaerobes, in addition to typical gram-positive and -negative organisms.

### SERUM LABORATORY STUDIES

Serum erythrocyte sedimentation rate and C-reactive protein levels are commonly elevated in several acute inflammatory and reactive arthritides (gonococcal and nongonococcal septic arthritis, crystal-induced, spondyloarthropathies, and rheumatoid and Lyme arthritis) but are not helpful for establishing a specific diagnosis in adults. However, erythrocyte sedimentation rate and C-reactive protein are recommended as an aid to monitor response to therapy by the British Society of Rheumatology Guidelines and the American Academy of Orthopedic Surgeons guidelines include erythrocyte sedimentation rate and C-reactive protein as part of their minor criteria for the diagnosis of acute periprosthetic joint infection (Table 284-4). The American Academy of Orthopedic Surgeons also recommends that an elevation of either erythrocyte sedimentation rate or C-reactive protein be used as criterion to aspirate a painful prosthetic joint with increased warmth. The sensitivity of the serum WBC count in adults for the diagnosis of nongonococcal bacterial septic arthritis is approximately 60%. Blood cultures should be obtained before antibiotic therapy for presumptive or possible septic arthritis. However, the sensitivity for identifying the causative
organism in adults and children with nongonococcal bacterial septic arthritis is 23% to 36%. Elevated procalcitonin levels provide 90% specificity to help rule in the diagnosis of septic arthritis but are only 67% sensitive in screening for the diagnosis. Laboratory studies can aid in the diagnosis at follow-up. Possible studies include Lyme titer, rheumatoid factor, antinuclear antibodies, antineutrophil cytoplasmic antibodies, HLA-B27 tissue typing, lupus anticoagulant, and repeat synovial fluid analysis.

**IMAGING**

Bedside US is useful to identify joint effusion and aids successful joint aspiration. Obtain radiographs of an inflamed joint if trauma, tumor, avascular necrosis, and osteomyelitis are diagnostic considerations. Radioisotope scanning is not usually required for ED diagnosis but can be useful to detect osteomyelitis, occult fracture, avascular necrosis, or tumor. MRI is not recommended for routing assessment of septic arthritis but may be helpful for difficult diagnostic cases. MRI is more sensitive to identify joint effusion than specific for the diagnosis of septic arthritis.

**ARTHROCENTESIS**

Prepare the site to avoid bacterial contamination. The skin overlying the affected joint should be free of cellulitis or impetigo to avoid contamination of the joint space during arthrocentesis. Orthopedics should be consulted before aspiration of a prosthetic joint for direction in diagnostic workup and interpretation of results. Other relative contraindications to joint aspiration are coagulopathy, including hemarthrosis in hemophiliac patients before factor replacement.

Cleanse a large area overlying and adjacent to the affected joint with povidone-iodine solution. After air drying, clean the skin with an alcohol wipe to remove the povidone-iodine solution from the skin surface. Removal of the overlying povidone-iodine prevents the introduction of the povidone-iodine antiseptic into the joint, which can result in chemical irritation or sterilization of the aspiration sample. Next place sterile drapes over the site and maintain sterile technique throughout the procedure.

Anesthetize the skin and soft tissues overlying the joint with a 25- to 30-gauge needle. Avoid intra-articular injection of anesthetic because the anesthetic can inhibit bacterial growth and may result in a spuriously negative culture in an early septic joint.

Use a large-bore needle (18 or 19 gauge) for aspiration of fluid from large joints. Use smaller-bore needles for small joints (no smaller than 22 gauge). Choose a syringe large enough to accommodate the anticipated volume of fluid within the joint space. Remove as much synovial fluid as possible to obtain a good diagnostic sample and to relieve pain from joint capsule distention. Promptly send aspirated fluid to the laboratory for culture, Gram stain, leukocyte count with differential, and crystal analysis. US should be used in the event of a dry tap (see Table 284-4).

**SHOULDER JOINT ASPIRATION**

US can facilitate shoulder aspiration. The anterior or posterior approach can be used.

**Anterior Approach**
Have the patient sit upright, facing you, and externally rotate the humerus. Insert the needle just lateral to the coracoid process, between the coracoid process and the humeral head (Figure 284-1A). Direct the needle posteriorly. If it is difficult to locate the coracoid process, the posterior approach to the glenohumeral joint may be easier.

**Posterior Approach**

Sit the patient upright with the back facing you. Palpate the spine of the scapula to its lateral limit: the acromion. Identify the posterolateral corner of the acromion. Use a 1.5-inch needle. The point for needle insertion is 1 cm inferior and 1 cm medial to the posterolateral corner of the acromion (Figures 284-1B and 284-2). Direct the needle anterior and medial toward the presumed position of the coracoid process. The glenohumeral joint is located at a depth of approximately 1.0 to 1.5 inches.

**FIGURE 284-1.**

Shoulder arthrocentesis. A. anterior approach. B. Shoulder Arthrocentesis posterior approach.

---

**A**

www.accessmedicine.com
Copyright © McGraw-Hill Education. All rights reserved.

**B**

www.accessmedicine.com
Copyright © McGraw-Hill Education. All rights reserved.

**FIGURE 284-2.**

Shoulder arthrocentesis, posterior approach.
ELBOW JOINT ASPIRATION

Use a lateral or posterior approach to the elbow joint. Do not use a medial approach to avoid neurovascular structures. Place the elbow in 90-degree flexion, resting on a table, with the hand prone to widen the joint space. Locate the radial head, lateral epicondyle of the distal humerus, and the lateral aspect of the olecranon tip. These three landmarks form the anconeus triangle. The center of this triangle is the site for needle entry into the skin. Using the tip of the gloved index finger of the nondominant hand, palpate a sulcus just proximal to the radial head. The sulcus is the needle entry point. Direct the needle medial and perpendicular to the radius toward the distal end of the antecubital fossa (Figure 284-3).

FIGURE 284-3.
Arthrocentesis of the elbow.
WRIST JOINT ASPIRATION

Landmarks for wrist arthrocentesis are palpable with the wrist in a neutral position. The landmarks are the radial tubercle of the distal radius, the anatomic snuffbox, the extensor pollicis longus tendon, and the common extensor tendon of the index finger (Figure 284-4). Insert the needle perpendicular to the skin, ulnar to the radial tubercle and the anatomic snuffbox, between the extensor pollicis longus (just ulnar to the extensor pollicis longus) and the common extensor tendons.

FIGURE 284-4.
Arthrocentesis of the wrist. [Images used with permission of Sandra Werner.]
HIP JOINT ASPIRATION

Hip arthrocentesis may be performed by an anterior or medial approach. If local practice dictates open surgical assessment and drainage, an orthopedic consultant will often perform this procedure. US-guided arthrocentesis by an emergency physician or radiologist is also acceptable if local practices and training are in place to support this approach (Figure 284-5). Controversy exists regarding the utility of US or MRI as a screening test before open surgical evaluation. Immediate consultation with an orthopedic surgeon is therefore desirable when a diagnosis of septic hip arthritis is considered.

FIGURE 284-5.

KNEE JOINT ASPIRATION

The knee joint can be entered either medial or lateral to the patella. With the patient supine, fully extend the knee and make sure the quadriceps muscle is relaxed. Identify the midpoint of the patella. The insertion point of the needle is located approximately 1 cm inferior to the patellar edge, either lateral (Figure 284-6) or medial (Figure 284-7) to the middle of the patella. Direct the needle posterior to the patella and horizontally toward the joint space. Compression or "milking" applied to both sides (proximal and distal) of the joint space by an assistant who is using sterile technique may facilitate aspiration of small amounts of fluid. In patients with obese or large knees, it may be necessary to use a needle longer than 1.5 inches to enter the joint space. Figure 284-8 demonstrates the use of US to visualize a knee joint effusion.

FIGURE 284-6.
A. Landmarks for knee arthrocentesis. D = distal femur; P = patella; T = tibia. B. Arthrocentesis of the knee, lateral approach.
Arthrocentesis of the knee, medial approach.

Bedside US of the knee in long axis (A) and short axis (B) demonstrating joint effusion.
ANKLE JOINT ASPIRATION

Ankle arthrocentesis may be performed at either the tibiotalar joint (medial approach) (Figure 284-9) or the subtalar joint (lateral approach) (Figure 284-10). The medial approach is generally preferred.

FIGURE 284-9.
Arthrocentesis of the ankle, medial approach.

www.accessmedicine.com
Copyright © McGraw-Hill Education. All rights reserved.
FIGURE 284-10.
Arthrocentesis of the ankle, lateral approach.
Medial (Tibiotalar)

Approach Have the patient supine with the foot initially perpendicular to the leg. This position facilitates the location of a sulcus lateral to the medial malleolus and medial to the tibialis anterior and extensor hallucis longus tendons (Figures 284-9 and 284-11A and B). Then plantar flex the foot with the needle entering the skin overlying the sulcus. Angle the needle slightly cephalad as it passes between the medial malleolus and the tibialis anterior tendon.

FIGURE 284-11.
A. US-guided approach to medial ankle arthrocentesis. Arrow points to the ankle effusion. B. Arrow points to needle in the tibiotalar joint, medial approach with transducer in the sagittal plane. [Images used with permission of Sandra Werner.]

Lateral (Subtalar)

www.accessmedicine.com
Copyright © McGraw-Hill Education. All rights reserved.

www.accessmedicine.com
Copyright © McGraw-Hill Education. All rights reserved.
Approach Keep the patient's foot perpendicular to the leg. Enter the subtalar joint just below the tip of the lateral malleolus (Figure 284-10). Direct the needle medially toward the joint space.

SEPTIC ARTHRITIS

An acute hot, swollen, and tender joint (or joints) with restriction of movement is bacterial nongonococcal septic arthritis until proven otherwise. Clinical features, risk factors, and treatment differ for bacterial, nongonococcal septic arthritis and gonococcal arthritis (Tables 284-1, Tables 284-1, 284-2, 284-3, 284-4). For management of septic arthritis in infants and children, see chapter 140, "Musculoskeletal Disorders in Children." In young adults, sexual activity increases the prevalence of gonococcal arthritis and reactive arthritis (formerly known as Reiter's syndrome) associated with chlamydial urethritis.

BACTERIAL NONGONOCOCCAL SEPTIC ARTHRITIS

Although no clinical pattern is diagnostic of bacterial nongonococcal septic arthritis, certain general observations are helpful. Joint pain (85%), a history of joint swelling (78%), and fever (57%) are the only findings that occur in >50% of patients with bacterial nongonococcal septic arthritis. Sweats (27%) and rigors (19%) are less common findings.

The involved joint can become exquisitely painful over a few hours. The patient may splint the affected joint to relieve pain with movement. Joint effusion may be small or large. Resistance to passive and active movement and limitation of full joint movement are notable findings but are common with gout without infection and may be absent in immunosuppressed patients. Although joint aspiration and analysis are essential to the diagnosis, the sensitivity of any one finding for the diagnosis of nongonococcal septic arthritis is only moderate. For example, the sensitivity of joint fluid WBC to make the diagnosis of nongonococcal septic arthritis using the commonly quoted cutoff of 50,000 cells/mm³ is only 56%. The sensitivity of erythrocyte sedimentation rate for nongonococcal septic arthritis using a cutoff of 30 mm/h ranges between 76% and 96%, but this test is nonspecific.

If a septic arthritis diagnosis cannot be reliably excluded after clinical evaluation, including arthrocentesis, admit the patient for parenteral antibiotics and pain control until synovial fluid culture results are available. Antibiotic coverage is directed at staphylococcal and streptococcal species including methicillin-resistant S. aureus. Vancomycin plus a third-generation cephalosporin is the preferred therapy (Table 284-5).
**TABLE 284-5**

**Commonly Encountered Organisms in Septic Arthritis in Adolescents and Adults**

<table>
<thead>
<tr>
<th>Patient/Condition</th>
<th>Expected Organisms</th>
<th>Antibiotic Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young healthy adults, or patients with risk factors for <em>Neisseria gonorrhoeae</em></td>
<td><em>Staphylococcus, N. gonorrhoeae, Streptococcus</em>, gram-negative bacteria</td>
<td><strong>Vancomycin</strong>, 15 milligrams/kg IV load, if Gram stain reveals gram-positive organisms in clusters. Ceftriaxone, 1 gram IV, or imipenem, 500 milligrams IV, should be used/added if either gram-negative organisms are present or no organisms are present on Gram stain and <em>N. gonorrhoeae</em> is suspected (also culture urethra, cervix, or anal canal as indicated).</td>
</tr>
<tr>
<td>Adults with comorbid disease (rheumatoid arthritis, human immunodeficiency virus, cancer) or injection drug users</td>
<td><em>Staphylococcus</em>, gram-negative bacilli</td>
<td><strong>Vancomycin</strong>, 15 milligrams/kg IV load, plus <em>cefepime</em>, 2 grams IV, or imipenem, 500 milligrams IV. <em>Meropenem</em> 1 gram IV may be used as an alternative agent.</td>
</tr>
<tr>
<td>Sickle cell patients</td>
<td><em>Salmonella</em> (increasingly <em>Staphylococcus</em>)</td>
<td><strong>Vancomycin</strong>, 15 milligrams/kg IV load, plus <em>ciprofloxacin</em>, 400 milligrams IV. Imipenem, 500 milligrams IV, may be used as an alternative agent.</td>
</tr>
</tbody>
</table>

*Recommendations differ from the 2006 British Society of Rheumatology treatment guidelines due to the rising incidence of methicillin-resistant *Staphylococcus aureus* septic arthritis.

Consult orthopedic surgery for possible joint irrigation in the operating room if the joint aspiration is positive for infection. Repeat closed-needle aspiration, arthroscopic irrigation, or less commonly, open surgical drainage may be required, depending on a number of factors, including consultant preference, patient age, affected joint, comorbid illnesses, and likelihood of septic source. Consultation with infectious disease may be required to determine ideal antibiotic choice in select patients. GONOCOCCAL SEPTIC ARTHRITIS

Gonococcal arthritis is the most common cause of septic arthritis in young sexually active adults. Joint infection will typically have a prodromal phase in which migratory arthritis and tenosynovitis predominate before pain and swelling settle on one or more septic joints. Vesiculopustular lesions, especially on the fingers, may be found (see Figure 149-33 in chapter titled "Sexually Transmitted Infections").

Synovial fluid cultures are often negative in gonococcal arthritis, with only 25% to 50% of cases yielding positive identification of the organism. Cultures of the posterior pharynx, urethra, cervix, and rectum (as directed by history of sexual contact) before antibiotic treatment increase the culture yield. Cases of gonococcal arthritis suspected clinically should be treated despite negative initial results while waiting for all culture results to return.
Treatment for gonococcal arthritis follows the same general principles as treatment for nongonococcal septic arthritis (see Table 284-5 for details). However, gonococcal arthritis does not yield joint destruction with the frequency of nongonococcal arthritis, and therefore, surgical intervention is rarely needed. Daily joint aspiration is routinely done until there is clinical improvement. Neisseria gonorrhoeae, in the setting of arthritis, remains sensitive to third-generation cephalosporin therapy (see chapter 149).

CRYSTAL-INDUCED SYNOVITIS (GOUT AND PSEUDOGOUT)

Crystal-induced synovitis is primarily an illness of middle-aged and elderly adults. Uric acid (gout) and calcium pyrophosphate (calcium pyrophosphate deposition, or pseudogout) are the two most common crystalline agents, with gout representing the most common form of inflammatory joint disease in men >40 years old. The classic description of gout is monoarthritis involving the great toe or knee joint in a man >40 years old. Gout is less common in women during their reproductive years but may occur in association with periods of increased insulin resistance such as gestational diabetes.

CLINICAL FEATURES

Joint pain develops over hours. An acute gout or pseudogout attack often follows trauma, surgery, a significant illness, or change in medication. Gout results from precipitation of urine acid crystals in the joint, and pseudogout results from calcium pyrophosphate crystals. Crystalline involvement of joints has a predilection for the foot and knee. Although the first metatarsophalangeal joint is a classic focus for acute gout, no joint is the exclusive site of involvement for either crystal.

DIAGNOSIS

The diagnosis of a crystal-induced synovitis is by joint aspiration and identification of crystals through a polarizing microscope. Uric acid crystals (gout) appear needle shaped and blue when the source of light is perpendicular to the crystal (negative birefringence). Calcium pyrophosphate (pseudogout) is yellow in this alignment (positive birefringence), with a rhomboid shape. Crystals are located within phagocytes from aspirates of synovial fluid, or within inflamed tissues adjacent to the affected joint.

Serum uric acid levels are not generally useful for diagnosis, as up to 30% of patients will have normal uric acid levels during an acute gout attack. There is no elevation of serum uric acid, calcium, or phosphate in pseudogout. The joint aspirate WBC can be elevated with gout and pseudogout. However, the presence of crystals, the absence of bacteria on Gram stain or culture, and frequently, the dramatic response to nonsteroidal anti-inflammatory drugs (NSAIDs) clarify the diagnosis. When the diagnosis of a septic joint cannot be excluded, hospital admission until cultures and/or clinical response clarify the diagnosis is the safest course of action.

TREATMENT

When the diagnosis of gout or pseudogout is established, treatment is an NSAID for 5 to 7 days. First-line treatment is indomethacin (or naproxen). Do not give NSAIDs to patients with renal insufficiency. For patients with normal renal function, the initial dose of indomethacin is 50 milligrams. Therapy is continued three times a day for 3 to 7 days as needed. Substantial pain relief typically occurs within 2 hours of NSAID administration. Colchicine is an
alternative agent to treat acute gout and pseudogout in patients with normal renal and hepatic function. Oral colchicine is typically administered at a dose of 0.6 milligram/h until intolerable side effects (vomiting or diarrhea) or efficacy ensues. IV administration of colchicine can be associated with serious side effects, with risks such as bone marrow suppression, neuropathy, myopathy, and death. For patients with renal insufficiency, narcotic analgesics are needed for pain relief because NSAIDs and colchicine are generally avoided. Prednisone is a first-line treatment option provided the patient is not diabetic.

Discharge is typical unless pain is not controlled or if septic arthritis is a consideration. Once acute symptoms have resolved, long-term control may be achieved with reduction or elimination of gout-inducing agents (diuretics, aspirin, or cyclosporine) and treatment with prophylactic drugs, such as allopurinol or probenecid. There is no effective prophylaxis for pseudogout.

**VIRAL ARTHRITIS**

The most common causes of viral arthritis in the United States are parvovirus B19, rubella, and hepatitis B (Table 284-6).
### Table 284-6

**Common Causes of Viral Arthritis**

<table>
<thead>
<tr>
<th>Virus</th>
<th>Prevalence of Arthritis</th>
<th>Findings</th>
<th>Duration</th>
<th>Additional Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parvovirus B19</td>
<td>Children 10%</td>
<td>Polyarticular</td>
<td>2–8 wk or chronic</td>
<td>Causes erythema infectiosum in children, rarely causes aplastic crisis</td>
</tr>
<tr>
<td></td>
<td>Adults 50%–70%</td>
<td>Symmetric</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rubella</td>
<td>Adults 50%</td>
<td>Polyarticular</td>
<td>5–7 d</td>
<td>Relapse</td>
</tr>
<tr>
<td>Epstein-Barr virus</td>
<td>1%–5%</td>
<td>Poly- or monoarticular</td>
<td>1–12 wk</td>
<td>Autoantibodies</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>10%–25%</td>
<td>Migratory</td>
<td>1–3 wk</td>
<td>Vasculitis</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>10%</td>
<td>Polyarticular</td>
<td>Chronic</td>
<td>Vasculitis</td>
</tr>
<tr>
<td>HIV</td>
<td>10%–50%</td>
<td>Mono- or oligoarticular</td>
<td>Chronic</td>
<td>Viral load &gt;10,000 copies of HIV RNA, CD4 count &lt;350 cells</td>
</tr>
<tr>
<td>Alphaviruses</td>
<td>&gt;50%</td>
<td>Oligoarticular</td>
<td>1–4 wk</td>
<td>More common in Asia, Africa; fever, myalgias</td>
</tr>
</tbody>
</table>

*Abbreviation: HIV = human immunodeficiency virus.*

*Alphaviruses include: Sindbis virus, Mayaro virus, Ross River virus, Semliki Forest virus, O’nyong-nyong virus, Chikungunya virus, and Barmah Forest virus.*

**PARVOVIRUS ARTHRITIS**

Parvovirus B19 causes *erythema infectiosum* in children. In adults, this rash occurs in less than half of patients. Joint involvement is more common in adults, with morning stiffness, swelling, erythema, and a presentation similar to acute presentations of rheumatoid arthritis.\(^{28,29}\)

**HEPATITIS VIRUS ARTHRITIS**

The most commonly involved joint with hepatitis B is the knee. Signs and symptoms are fever and lymphadenopathy, followed by joint pain, and then jaundice. Immune complexes in the synovium are responsible for synovial inflammatory changes. Hepatitis C causes a polyarticular arthritis, which may become chronic.\(^{29}\)

**RUBEELLA ARTHRITIS**
The arthritis of rubella is uncommon in children and adult males, but occurs in 50% of adult females with acute rubella, appearing soon after the emergence of the classic rash. The arthritis is polyarticular, most frequently involving the wrist, hand, knee, ankle, and elbow.

**ALPHAVIRUS AND HUMAN IMMUNODEFICIENCY VIRUS ARTHRITIS**

Alphaviruses are common causes of arthritis in Africa, Asia, Australia, the West Pacific, and South America. Human immunodeficiency virus may cause an associated arthralgia/arthritis, a reactive arthritis, or psoriatic arthritis. In human immunodeficiency virus, CD4 counts below 350 cells/mm$^3$ increase the likelihood of bacterial arthritis. Counts below 200 cells/mm$^3$ are associated with an opportunistic cause of arthritis.Overall management is discussed in chapter 154, "Human Immunodeficiency Virus Infection."

**LYME DISEASE**

The arthritic manifestations of Lyme disease occur weeks, months, or years after primary, stage I infection. Symptoms include monoarticular or oligoarticular asymmetric joint involvement. Large joints are most often affected, particularly the knee. A migratory pattern of oligoarthritis may be noted in addition to brief attacks of bursitis and tendonitis.

The diagnosis of Lyme arthritis is initially suspected in patients residing in, or with a recent visit to, an endemic area. A history of tick bite or erythema chronicum migrans rash (see Figure 160-1 in chapter titled "Zoonotic Infections") is helpful but often absent. Arthrocentesis yields an inflammatory synovial fluid, usually with negative cultures. For detailed discussion of the diagnosis and treatment of Lyme disease, see chapter 160. Given the difficulty of making a definitive diagnosis in many patients, treatment of suspected Lyme arthritis is often initiated on the grounds of high clinical suspicion. Treatment is administered for 4 weeks, with a number of antibiotics recognized as effective, including ceftriaxone, 1 gram IV twice daily, switching to PO after clinical improvement; doxycycline, 100 milligrams PO twice daily; amoxicillin, 500 milligrams three times daily; or cefuroxime, 500 milligrams twice daily.30

**HEMARTHROSIS**

**TRAUMATIC HEMARTHROSIS**

Traumatic hemarthrosis has a high association with ligamentous injury or an intra-articular fracture. Effusions following trauma may range from small minor effusions to large painful fluid collections that impede range of motion. Aspiration of very large traumatic effusions will provide pain relief for approximately 1 week and increase range of motion but has no effect on long-term outcome.12 Treatment of traumatic hemarthrosis consists of immobilization, ice, and elevation of the affected joint. In the absence of a fracture or significantly unstable joint requiring immediate orthopedic evaluation, follow-up is needed for possible ligamentous and articular injuries.

**SPONTANEOUS HEMARTHROSIS**

Spontaneous hemarthrosis usually indicates underlying systemic illness and should prompt consideration for primary or secondary coagulopathies. Hemophiliacs should receive specific clotting factor replacement for hemarthrosis (see chapter 235, "Hemophilias and von Willebrand’s Disease"). Joint aspiration for acute hemarthrosis in hemophilia is controversial but recommended by some for a large hemarthrosis that can be aspirated during the first 12 hours of
symptoms. Joint aspiration should only be performed after factor replacement. Follow-up and/or consultation should be provided with hematology and orthopedics.

RHEUMATOID ARTHRITIS

Rheumatoid arthritis is typically a progressive disease, with polyarticular involvement of symmetric joints and sparing of the distal interphalangeal joints. Women are affected more commonly than men. Patients describe stiffness of the joints occurring after prolonged periods of inactivity (morning stiffness). A "boggy," slightly edematous synovium may be palpated. Arthrocentesis of synovial fluid is typically noted for an inflammatory profile. For further discussion of systemic clinical features of rheumatoid arthritis, see chapter 282.

Salicylates or other NSAIDs are the cornerstone of treatment for an acute exacerbation. Corticosteroids may be used for brief periods, with long-term therapy using agents such as methotrexate, leflunomide, sulfasalazine, and other nonbiologic and biologic disease-modifying antirheumatic drugs. Consider septic arthritis in patients with an acute episode of arthritis who are also receiving immunosuppressives.

OSTEOARTHRITIS

Osteoarthritis is distinguished from rheumatoid arthritis by a lack of constitutional symptoms and/or multisystem involvement. Destruction of joints in osteoarthritis may involve the distal interphalangeal joints, with less dramatic symmetric, polyarticular exacerbations. Although osteoarthritis is a chronic, polyarticular disease, patients may present with an acute monoarthritis exacerbation, typically of the knee. Effusions are small and difficult to aspirate. If fluid is aspirated, it is noninflammatory.

Radiographs demonstrate characteristic joint space narrowing due to destruction of articular cartilage. Treatment is joint rest and NSAIDs or acetaminophen in the setting of GI complications. Systemic corticosteroids are not indicated, although intra-articular corticosteroids may be administered by a primary care physician or orthopedist.

REACTIVE ARTHRITIS

Reactive arthritis (formerly known as Reiter’s syndrome) is a seronegative spondyloarthropathy characterized by an acute, asymmetric oligoarthritis occurring 2 to 6 weeks after an infectious illness. The classic triad of Reiter’s syndrome is arthritis, urethritis, and conjunctivitis. A history of all three components is not necessary for diagnosis. Chlamydia or Ureaplasma are common inciting infectious agents (postvenereal reactive arthritis). Enteric infections may precipitate a reactive arthritis (postdysentery reactive arthritis). Implicated agents of postdysentery reactive arthritis are Salmonella, Shigella, Yersinia, and Campylobacter, and possible agents include Escherichia coli and Clostridium difficile. Conjunctivitis occurs in one third of postvenereal and >50% of postdysentery forms of reactive arthritis.

Joint involvement in reactive arthritis typically involves the lower extremities, including the feet. Back and buttock pain may occur. A diffuse swelling of an entire digit (sausage digit) may be found as well but is not specific to reactive arthritis. Synovial fluid aspirates demonstrate an inflammatory profile. Treatment has traditionally been supportive, with emphasis on pain control with NSAID therapy. Antibiotics were previously thought to be of no benefit, but now long-term combination antibiotic therapy is being used for Chlamydia-induced reactive arthritis, using
rifampicin combined with either doxycycline or azithromycin.\textsuperscript{35} Arthroscopic synovectomy may also provide benefit.\textsuperscript{36} Suspected cases should be referred to rheumatology for confirmation of diagnosis and management.

\textbf{BURSITIS}

\textbf{NONSEPTIC BURSITIS}

Bursitis is an inflammatory process involving one of the >150 bursae in the body, but most commonly the bursa overlying the elbow or the knee (see also \textit{chapter 281}, "Hip and Knee Pain").\textsuperscript{37} Bursitis can be caused by repetitive trauma or can be associated with gout, pseudogout, or rheumatoid arthritis. Repetitive activities that can precipitate bursitis are identified by the typical names given: "carpet layer's or housemaid's knee" (prepatellar bursitis) or "student's elbow" (olecranon bursitis). The affected bursa is easily palpated but is not tender and not erythematous. Bursal enlargement is usually chronic or progressive but not acute. If bursitis is acute, consider septic bursitis (see "\textit{Septic Bursitis}" below). In nonseptic bursitis, there is no limitation of, or pain upon, joint movement. The skin over the bursa may be thickened and calloused, indicating chronic repetitive trauma or pressure. Treatment is NSAIDs and elimination of activities that produce symptoms. Aspiration and drainage of bursal fluid is controversial (if infection is not suspected), because bursal fluid often reaccumulates after aspiration.

\textbf{SEPTIC BURSITIS}

Unlike septic arthritis, septic bursitis is more likely secondary to bacterial spread from a skin lesion or local cellulitis to an injured or inflamed bursa. Therefore, cultures more closely reflect skin flora.\textsuperscript{37} Septic bursitis is characterized by acute pain, tenderness, erythema of the affected bursa, and overlying warmth when compared with the unaffected side.\textsuperscript{37,38} The most common sites for septic bursitis are the prepatellar bursa (50\% to 53\%) and the olecranon bursa (40\% to 45\%).\textsuperscript{38,39,40} Fever occurs in <50\% of patients with septic bursitis.\textsuperscript{38} Pain can occasionally be mild (10\%) but is usually moderate or severe.\textsuperscript{39} Associated cellulitis of the surrounding skin may be evident.

Most authors recommend the aspiration of bursal fluid if septic bursitis is considered.\textsuperscript{37,39} Bursal aspiration can be diagnostic and therapeutic. Bursal fluid demonstrates characteristic findings in infection (\textit{Table 284-7}).\textsuperscript{37} Culture is the definitive test for presence or absence of infection. Diagnosis is presumed by one of the following criteria based on bursal fluid results: positive Gram stain, >3000 WBC/mm\textsuperscript{3}, >50\% polymorphonuclear cells, glucose <31 milligrams/dL, or bursal to serum glucose ratio of <50\%.\textsuperscript{37}
**TABLE 284-7**

**Characteristics of Bursal Fluid in Patients with Septic and Nonseptic Olecranon and Prepatellar Bursitis**

<table>
<thead>
<tr>
<th></th>
<th>Septic</th>
<th>Traumatic and Idiopathic</th>
<th>Crystal Induced</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Appearance</strong></td>
<td>Purulent or serosanguineous</td>
<td>Straw colored, serosanguineous, or bloody</td>
<td>Straw colored to bloody</td>
</tr>
<tr>
<td><strong>Leukocytes/mm³</strong></td>
<td>Range, 350–392,000; mean, 54,330 ± 34,197; &gt;3000 is considered diagnostic</td>
<td>Range, 0–11,700; mean, 2475 ± 1988</td>
<td>Range, 1000–6000; mean, 2900</td>
</tr>
<tr>
<td><strong>Differential count</strong></td>
<td>&gt;50% polymorphonuclear cells is considered diagnostic</td>
<td>Predominantly mononuclear</td>
<td>Highly variable</td>
</tr>
<tr>
<td><strong>Ratio of bursal fluid to serum glucose</strong></td>
<td>&lt;50% in 90% of cases (diagnostic)</td>
<td>&gt;50%, 70%–80% in 98% of cases</td>
<td>Unknown</td>
</tr>
<tr>
<td><strong>Gram stain</strong></td>
<td>Positive in 70% (diagnostic)</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td><strong>Crystals present</strong></td>
<td>No *</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Culture results</strong></td>
<td>Positive (diagnostic)</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

*The presence of crystals does not rule out infection.

*S. aureus* accounts for the majority of infections, but *Staphylococcus epidermidis* and *Streptococcus* species are also encountered. Septic bursitis generally responds well to oral antibiotics, with emphasis on coverage of *Staphylococcus* and *Streptococcus* species. With the high prevalence of methicillin-resistant *S. aureus*, adjust antibiotic choice according to local sensitivities. Conditions that require hospital admission for incision and debridement and IV antibiotics include sepsis, extensive purulent bursitis, extensive surrounding cellulitis, suspected joint involvement, immunocompromise, or failure to respond to a course of oral antibiotics. See specific treatment recommendations below.

**OLECRANON BURSITIS**

The olecranon bursa overlies the olecranon process on the extensor surface of the elbow. The bursa is tense and edematous. Pain elicited with range of motion at the elbow is minor until the motion tightens and compresses the distended overlying bursa. Gouty tophi on the extensor surface of the elbow may be palpable or visible if the cause of bursitis is crystal-induced bursitis. If bursal fluid is aspirated, uric acid crystals are evident on microscopy.

To aspirate the olecranon bursa, prepare the bursal skin and use antiseptic technique. The patient’s arm can be extended to allow for maximal bursal distention. Use a lateral approach to the affected bursa. Remove as much fluid as
possible, and send the aspirate to the laboratory for analysis for WBC, Gram stain, crystals, glucose, and culture.

Treatment depends on patient condition; if septic, the patient should be treated with vancomycin, 15 milligrams/kg, plus piperacillin/tazobactam, 4.5 grams IV, or meropenem, 500–1000 milligrams IV. Most patients can be treated as outpatients with a 14-day course of oral antibiotics.\textsuperscript{37,38,39} Common antibiotics chosen include clindamycin, 300 milligrams three times per day for 10 days, or dicloxacillin, 500 milligrams four times per day.\textsuperscript{39} Trimethoprim-sulfamethoxazole is an alternative.\textsuperscript{38} Steroids are not indicated in the ED because infection cannot be definitively excluded by negative culture results. Admission is indicated for clinical toxicity, extensive surrounding cellulitis, failure of outpatient treatment, or immunocompromise. Some patients benefit from surgical excision of the bursa sac.\textsuperscript{40}

**PREPATELLAR BURSITIS**

Bursitis may affect any of the four bursae surrounding the extensor aspect of the knee (see Figure 281-6). A history of overuse or repetitive trauma to the prepatellar area is typical.\textsuperscript{37} The noninfected or aseptic bursa is enlarged and taut but nontender and not warm. There is full range of motion of the knee. If septic patellar bursitis is a consideration (Figure 284-12), aspirate the prepatellar bursa to obtain fluid for analysis. Prepare the skin overlying the bursa and use aseptic technique. Use either a lateral or medial approach. Fluid analysis and treatment are the same as for septic olecranon bursitis (see "Olecranon Bursitis" above).

**FIGURE 284-12.**


---

**REFERENCES**


**USEFUL WEB RESOURCES**
