# Osteomyelitis

## may be classified based on the duration of illness

### Chronic osteomyelitis

• **The hallmark of chronic osteomyelitis** is the **presence of dead bone (sequestrum)**. Other common features of chronic osteomyelitis include **involucrum** (reactive bony encasement of the sequestrum), **local bone loss**, and, if there is extension through cortical bone, **sinus tracts** .

**Sequestra** are usually present; they form as a result of bone ischemia and necrosis in the context of blood vessel compression due to elevated medullary pressure associated with bone marrow inflammation. Sequestra can be seen radiographically.

• The presence of a sinus tract is pathognomonic of chronic osteomyelitis

• Chronic osteomyelitis may manifest as pain, erythema, or swelling, sometimes in association with a draining sinus tract; fever is usually absent.
• Chronic osteomyelitis may also present with intermittent flares of pain and swelling.

### Acute osteomyelitis

• Acute osteomyelitis typically presents with **gradual onset of symptoms over several days**.

• evolves over several days to weeks and can progress to a chronic infection.

• Patients usually present with a **dull pain** at the involved site, with or without movement.
**Local findings** (tenderness, warmth, erythema, and swelling ) and **systemic symptoms** (fever, rigors) may also be present .

## may be classified based on the mechanism of infection

Nonhematogenous osteomyelitis can occur **as a result of contiguous spread of infection to bone from adjacent soft tissues and joints or via direct inoculation of infection into the bone** (as a result of trauma or surgery).

### Nonhematogenous osteomyelitis

• Among younger adults, nonhematogenous osteomyelitis occurs most commonly in the setting of **trauma and related surgery**. Among older adults, nonhematogenous osteomyelitis occurs most commonly as a result of **contiguous spread of infection to bone from adjacent soft tissues and joints**.

• **Risk factors for nonhematogenous osteomyelitis** include poorly healing soft tissue wounds (including decubitus ulcers), presence of orthopedic hardware, diabetes, peripheral vascular disease, and peripheral neuropathy.

• Nonhematogenous osteomyelitis may be **polymicrobial** or **monomicrobial**.

• **Staphylococcus aureus** (including methicillin-resistant S. aureus), **coagulase-negative staphylococci**, and **aerobic gram-negative bacilli** are the most common organisms.

### Hematogenous osteomyelitis

• Hematogenous osteomyelitis is caused by **microorganisms that seed the bone in the setting of bacteremia**

• Hematogenous osteomyelitis is the most common form of osteomyelitis in infants and children.
• In adults, **vertebral osteomyelitis** is the most common form of hematogenous osteomyelitis. Most cases occur in patients >50 years.

• Hematogenous osteomyelitis is usually **monomicrobial.**

• **S. aureus** is by far the most commonly isolated organism. **Aerobic gram-negative rods** are identified in up to 30 percent of cases.

• **Tuberculous osteomyelitis** usually occurs from reactivation of tuberculous bacilli lodged in bone during the mycobacteremia occurring at the time of the primary infection

# Osteomyelitis

## PATHOGENESIS

• Normal bone is highly resistant to infection.

Osteomyelitis develops when there is a large inoculation of organisms, presence of bone damage, and/or presence of hardware or other foreign material.

• The pathogenesis of osteomyelitis is **multifactorial** and **poorly understood**; important factors include **the virulence of the infecting organism(s)**, **the host immune status**, and **the bone vascularity**.
• Bacteria have a number of virulence determinants that may contribute to development of osteomyelitis.

• **Adherence** appears to play a central role in the early stages of S. aureus-induced osteomyeliti or arthritis.
S. aureus adheres to a number of components of bone matrix including fibrinogen, fibronectin, laminin, collagen, bone sialoglycoprotein, and clumping factor A.

• **S. aureus** can survive intracellularly in cultured osteoblasts.
Persistence of intracellular pathogens within **osteoblasts** may also be an important factor in the pathogenesis of osteomyelitis.
When digested by osteoblasts, S. aureus undergoes phenotypic alteration, which renders it more resistant to the action of antimicrobials.
This may explain in part the high relapse rate of osteomyelitis treated with antimicrobials for a short duration.

## Clinical approach and diagnosis

**• In general, the diagnosis of osteomyelitis is established via culture obtained from biopsy of the involved bone.**
**A diagnosis of osteomyelitis may be inferred in the following circumstances:**

• Clinical and radiographic findings typical of osteomyelitis and positive blood cultures with a likely pathogen (such as Staphylococcus aureus);
in such cases, bone biopsy is not required but may be useful, particularly if subsequent therapeutic debridement is needed.

• Bone histopathology consistent with osteomyelitis in the absence of positive culture data (particularly in the setting of recent antibiotic administration).

• Suggestive clinical and typical radiographic findings and persistently elevated inflammatory markers, in circumstances with no positive culture data and a biopsy is not feasible.

## Management

• Findings of osteomyelitis on radiographic imaging should prompt bone biopsy for culture and histology to confirm the diagnosis and to guide antimicrobial therapy, unless blood cultures are positive for a likely pathogen
(such as S. aureus, a gram-negative enteric rod, or Pseudomonas aeruginosa).

• Patients with negative Gram stain and culture results should be treated with an antimicrobial regimen with activity against the common causes of vertebral osteomyelitis, including staphylococci, streptococci, and
gram-negative bacilli.

## Complications

**<u>Complications of osteomyelitis include:</u>**

- Sinus tract formation
- Contiguous soft tissue infection
- Abscess
- Septic arthritis
- Systemic infection
- Bony deformity and Fracture
- Malignancy

# Septic arthritis

## Overview

• Septic arthritis is synonymous with an infection in a joint.

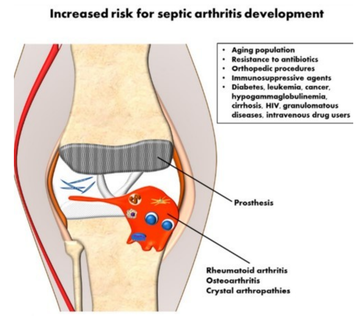
• Septic arthritis is usually caused by **bacteria** but can also be caused by **other microorganisms**.
→ Septic arthritis due to bacterial infection is often **a destructive form of acute arthritis**

## Etiology

• Septic arthritis is usually **monomicrobial** .
• **S. aureus** (including methicillin-resistant S. aureus) **is the most common cause of septic arthritis** in adults.
→ **Other gram-positive organisms such as streptococci** are also important potential causes of septic arthritis.

## Pathogenesis (Roots of infection)

• 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.
• **direct inoculation of bacteria into the joint .**
• **contiguous spread from an adjacent soft tissue .**
• **bone infection .**

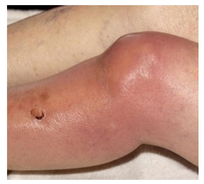



## clinical manifestations

• Patients with septic arthritis usually present acutely with a **single swollen** and **painful joint** (ie, monoarticular arthritis).
→ **Joint pain**, **swelling**, **warmth**, and **restricted movement** occur in 80% of patients with septic arthritis.

**Most patients** with septic arthritis are **febrile**; however, **older patients** with septic arthritis may be **afebrile**.

• The knee is involved in more than 50% of cases; wrists, ankles, and hips are also affected commonly



## Diagnosis

• The diagnosis of septic arthritis is made based on **synovial fluid analysis** and **culture**.
• In addition, blood cultures and, when indicated, radiographs, ultrasound, or imaging studies of the involved joint should be obtained.
→ 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**.

**Septic arthritis /** DIAGNOSIS

| 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 |
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| 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.

## Treatment

**Drainage therapy** — needle aspiration, arthroscopic drainage, or arthrotomy

**Antibiotic therapy**

• If the initial Gram stain of synovial fluid demonstrates **gram-positive cocci** → empiric treatment with **vancomycin**

• If the initial Gram stain of synovial fluid demonstrates **gram-negative bacilli** → treatment should be guided by risk for **Pseudomonas infection**

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 **preexisting joint disease .**

# Animal bites

## Epidemiology and Manifestations

- **90%** of animal bites → dog bites - occur most often in children .
- In children, dog bites usually involve the **head and neck**; in adolescents and adults, dog bites usually involve the **extremities** .
- Dog bites may be associated with a range of injuries, **from minor to major wounds**.

- **10%** of animal bites → cat bites - occur most often in adult women .
- Cat bites usually occur on the **extremities** and **tend to penetrate deeply, with higher risk of deep infection** (abscess, septic arthritis , osteomyelitis, tenosynovitis, bacteremia, or necrotizing soft tissue infection) **than dog bites** .

**Infections are much more common after cat bites (up to 50 percent of wounds) than dog bites .**

- Bite wound infection may be :

**- Superficial** (eg, **cellulitis**, with or without abscess) .
Clinical manifestations of superficial infection include **fever, erythema, swelling, and warmth, purulent drainage, and/or lymphangitis**.
An associated superficial abscess may present as a tender, erythematous, fluctuant nodule.

**- Deep** (**abscess**, **septic arthritis**, **osteomyelitis**, **tenosynovitis**, or **necrotizing soft tissue infection**).

## Etiology

**Bite wound culture yields five types of bacterial isolates. Mixed aerobic and anaerobic bacteria are observed in 60% of cases; skin flora are isolated in about 40% of cases**

### Bartonella henselae

- **B. henselae** is responsible for a disease acquired **after exposure to cats** (e.g., scratches, bites, contact with the contaminated feces of cat fleas): **cat-scratch disease**, 1–3 weeks after inoculation.

- **Symptoms** typically include **a non-painful bump** or **blister at the site of injury** and **painful** and **swollen lymph nodes .**

- B. henselae may be **transmitted via the bite of an infected cat;** other forms of **transmission** include **cat scratches, flea exposure, and contact with cat saliva via broken skin or mucosal surfaces.**

- The incubation period for B. henselae infection is 7 to 14 days.

### Anaerobes (Bacteroides, fusobacteria, Porphyromonas, Prevotella)

### Pasteurella

Pasteurella in 50% of dog bites and 75% of cat bites .

### The oral flora of the biting animal and human skin flora (such as staphylococci and streptococci ) .

### Capnocytophaga canimorsus

Capnocytophaga canimorsus can cause bacteremia and fatal sepsis after animal bites, especially in patients with asplenia, alcoholism, or underlying hepatic disease.

## Diagnosis

- The physical examination should ensure that the patient is **hemodynamically stable** and **should assess for injuries to adjacent structures**, especially for bites with deep puncture wounds on the head, neck, trunk, or close to joints.

- Bite wounds should be **evaluated carefully for foreign material** , and a **neurovascular assessment should be performed in areas distal to the wound**.