Musculoskeletal Infections in the Emergency Department

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KEYWORDS

• Osteomyelitis • Spondylodiscitis • Spinal epidural abscess
• Posttraumatic osteomyelitis • Septic arthritis • Periprosthetic joint infection
• Emergency department

KEY POINTS

- Patients with musculoskeletal infections can have heterogeneous presentations, as the signs and symptoms are often occult and nonspecific.
- *Staphylococcus aureus* is the most common microorganism associated with bone and joint infections.
- Definitive diagnosis requires sampling of affected tissue for Gram stain and microbiologic culture.
- The mainstay of treatment for bone and joint infections is antibiotic therapy, but surgical consultation for irrigation and/or debridement may be necessary in certain clinical situations.

INTRODUCTION

Bone and joint infections are a relatively uncommon cause of musculoskeletal complaints among patients seeking care in the emergency department (ED). Atypical and nonspecific presentations can be misleading, and definitive diagnosis of infection challenging, often requiring invasive and time-consuming procedures. This review outlines the clinical signs and symptoms that should lead emergency physicians to consider a musculoskeletal infection, the diagnostic workup, and key therapeutic interventions when the clinical suspicion for infection is high. The approach to osteomyelitis, spondylodiscitis, spinal epidural abscess, antibiotic prophylaxis for an open fracture, septic arthritis, and periprosthetic joint infection in the ED will serve as the primary FOCI.

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OSTEOMYELITIS

Osteomyelitis is an inflammatory reaction of the bone due to infection, most often bacterial in nature. Infection can involve the bone marrow, cortex, periosteum, or surrounding soft tissues, leading to destruction of any or all of these anatomic structures.[1] In a US population-based study conducted in Olmstead County, Minnesota, the overall incidence of osteomyelitis increased from 11.4 cases per 100,000 person-years in the period from 1969 to 1979 to 24.4 per 100,000 person-years in the period from 2000 to 2009.[2] Although rates remained stable among children and adults younger than 50 years, incidence nearly tripled among those 60 years or older, fueled by a significant rise in diabetes-related osteomyelitis over the past 4 decades.

Osteomyelitis develops from 1 of 3 mechanisms of pathogenesis: bacteremia leading to hematogenous seeding of bone, contiguous spread of infection from adjacent soft tissue to bone, or direct inoculation of microorganisms into bone. Hematogenous osteomyelitis results either from the introduction of microorganisms into the bloodstream (eg, via injection drug use or an infected central venous catheter) or an infection elsewhere that has now been complicated by bloodstream involvement (eg, endocarditis, urinary tract infection). Osteomyelitis due to contiguous spread occurs most frequently in the setting of skin breakdown (eg, diabetic foot ulcer, vascular ulcer, or pressure-related decubitus ulcer) and soft tissue infection extending to underlying bone. Infected joints, both native (septic arthritis) or prosthetic, and other infected orthopedic devices can likewise involve adjacent bone. Osteomyelitis from direct inoculation classically arises in the setting of an open fracture or surgery. Patients with osteomyelitis will often have one or more pathologic risk factors associated with these mechanisms (**Table 1**). *Acute* osteomyelitis progresses over days to weeks and is characterized by inflammation of viable bone. In contrast, *chronic* osteomyelitis evolves over weeks, months, or even years and is distinguished by progression to osteonecrosis with the formation of sequestrum, often in the setting of recurrent or refractory infection.

Several classification systems exist to categorize osteomyelitis. The Waldvogel classification system differentiates osteomyelitis by mechanism of pathogenesis, focusing on hematogenous seeding, contiguous spread, and vascular insufficiency.[3] The Cierney-Mader classification system organizes osteomyelitis by the extent of host anatomic involvement, physiologic status, and comorbid factors that may influence clearance of infection.[4]

Table 1
Risk factors for development of osteomyelitis

Mechanism of Pathogenesis	Risk Factor
Hematogenous seeding	Injection drug use Central venous catheter or other long-term vascular device Urinary tract infection Immunosuppression (including chronic corticosteroid use)
Contiguous spread from adjacent tissues	Extremes of age Diabetes mellitus Vascular insufficiency Abscess/cellulitis/infected ulcer
Direct inoculation	Prior orthopedic surgery or indwelling orthopedic hardware Trauma (open fracture) Human/animal bite