Osteomyelitis of the Foot and Ankle

Diagnosis, Epidemiology, and Treatment

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KEYWORDS

• Osteomyelitis • Diabetic foot • Imaging • Surgical treatment • Laboratory studies • Adjuvant therapies

KEY POINTS

- Osteomyelitis of the foot and ankle is a common, potentially devastating condition with diagnostic and treatment challenges.
- History and physical examination, laboratory studies, vascular studies, histologic and microbiologic analyses, and various imaging modalities contribute to the diagnosis and treatment.
- Treatment should take a multidisciplinary approach to optimize patient factors, ensure eradication of the infection, and restore function.
- Surgical treatment needs to consider the physiology of the infection and the patient, must be extensive, and may use multiple techniques to achieve successful outcomes.
- Adjuvant therapies and novel laboratory markers may enhance outcomes as they are further studied and used.

INTRODUCTION

Osteomyelitis of the foot and ankle can be extremely debilitating to patients and a management challenge to the orthopedic surgeon. In the preantibiotic era, acute staphylococcal osteomyelitis carried a mortality rate of 50%.[1] Osteomyelitis of the foot and ankle can arise from multiple etiologies, and one of the most frequently encountered clinical scenarios is in the context of diabetic foot infections. The incidence of diabetic

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foot infections is 36.5 per 1000 persons per year, with a lifetime incidence of patients with diabetes developing a diabetic foot ulcer of 25%.[2–5] Underlying osteomyelitis is present in 20% to 68% of diabetic foot ulcers.[6–9] The presence of osteomyelitis in diabetic foot infections has an amputation rate of up to 66%.[9,10] In-hospital mortality associated with osteomyelitis in one study was 1.6%.[11] The economic burden of osteomyelitis is severe, with a median length of stay of 7 hospital days, mean hospital charges $19,000, and the direct costs of amputation associated with osteomyelitis exceeding $34,000.[2,3,11] Understanding how to accurately diagnose and effectively treat osteomyelitis is critical for the foot and ankle surgeon.

CLASSIFICATION AND PATHOPHYSIOLOGY OF OSTEOMYELITIS

Several classifications of osteomyelitis and diabetic foot wounds exist. Classification of osteomyelitis popularized by Waldvogel focused on the duration and mechanism of infection.[12] The duration of osteomyelitis is classified as acute, subacute, or chronic. Acute osteomyelitis refers to inflammatory bone changes caused by pathogens with symptoms manifesting within 2 weeks of infection.[12,13] Histologic findings of acute osteomyelitis include microorganisms, neutrophil infiltration, and congested or thrombosed nutrient blood vessels.[10,14] Chronic osteomyelitis is defined by the presence of necrotic bone and the absence of osteocytes, and symptoms may not occur until 6 weeks of infection.[10,12–14] Mechanisms of infection in osteomyelitis include hematogenous or exogenous spread. Hematogenous osteomyelitis involves bacteremia and seeding of the bone with an organism from a remote source.[12] Hematogenous osteomyelitis is primarily seen in pediatric patients, patients with chronic indwelling catheters, and intravenous drug abusers.[15] It generally occurs in bones with rich blood supply, such as the metaphases of long bones in children and the vertebral bodies of adults.[12,16] Exogenous osteomyelitis occurs from direct inoculation of the bone caused by contiguous spread from adjacent tissue, open fractures, penetrating trauma, or iatrogenic postsurgical contamination.[12,15,16] In diabetic foot osteomyelitis, there typically is contiguous spread from adjacent soft tissue infection or ulcer.

The pathophysiology of osteomyelitis begins as the infection spreads through the periosteum or is seeded hematogenously and extends within the medullary canal. The increased intramedullary pressure secondary to inflammation leads to bone necrosis and the overlying periosteal reaction begins the formation of new bone, creating an involucrum.[1] Inflammatory factors and leukocytes further contribute to bone necrosis and destruction. Local vascular channels are compressed and obliterated by the inflammatory process, creating areas of necrosis and sequestra where antibiotic penetration is insufficient.[1] At the edge of the infarcted microvascular channels, there is relative hyperemia, which causes bone dissolution and localized osteoporosis secondary to increased osteoclastic activity.[1] Osteoclastic activity is further stimulated by inflammatory factors, such as interleukin (IL)-1 and tumor necrosis factor released by inflammatory cells in response to bacterial antigens, leading to further attempts at remodeling because of dissolution.[6]

The Cierny classification of chronic osteomyelitis uses the anatomic location and extent of infection and also considers the physiologic factors of the patient.[17] There are four anatomic types: medullary (I), superficial (II), localized (III), and diffuse (IV). Based on the comorbidities and clinical status of the patient, the physiologic class of the host is defined as normal (A host), compromised (B host), or prohibitive (C host). This classification is presented in **Table 1**.

Consideration of surrounding soft tissues or staging of diabetic foot wounds is also important in foot and ankle osteomyelitis. The Wagner classification of diabetic foot