

and skin and soft tissue damage may need to be treated during the same intervention.

PROGNOSIS

Primary sternal osteomyelitis poses a minimal mortality risk. In contrast, the in-hospital mortality rates from secondary sternal osteomyelitis are 15–30% after sternal surgery.

FOOT OSTEOMYELITIS

PATHOGENESIS

Osteomyelitis of the foot usually occurs in patients with diabetes, peripheral arterial insufficiency, or peripheral neuropathy and after foot surgery. These entities are often linked to each other, especially in diabetic patients with late complications. However, foot osteomyelitis is also seen in patients with isolated peripheral neuropathy and can manifest as implant-associated osteomyelitis in patients without comorbidity due to a deep wound infection after foot surgery (hallux valgus surgery, arthrodesis, total ankle arthroplasty). Foot osteomyelitis is acquired almost exclusively by the exogenous route. It is a complication of deep pressure ulcers and of impaired wound healing after surgery.

EPIDEMIOLOGY

The incidence of diabetic foot infection is 30–40 cases/1000 persons with diabetes per year. The condition starts with skin and soft tissue lesions and progresses to osteomyelitis, especially in patients with risk factors. About 60–80% of patients with diabetic foot infection have confirmed osteomyelitis. Diabetic foot osteomyelitis increases the risk of amputation. With adequate management of the early stage of diabetic foot infections, the rate of amputation can be lowered.

RISK FACTORS

Risk factors for diabetic foot infection are (1) peripheral motor, sensory, and autonomic neuropathy; (2) neuro-osteoarthropathic deformities (Charcot foot; Fig. 158-5); (3) arterial insufficiency; (4) uncontrolled hyperglycemia; (5) disabilities such as reduced vision; and (6) maladaptive behavior.

MICROBIOLOGY

The correlation between cultures from bone biopsy and those from wound swabs or even deep soft tissue punctures is poor. Consistent

results have been found in only 13–43% of cases in various studies. The correlation is better when *S. aureus* is isolated (40–50%) than when anaerobes (20–35%), gram-negative bacilli (20–30%), or coagulase-negative staphylococci (0–20%) are identified. When only bone biopsy samples are considered, the leading pathogens are *S. aureus* (30–40%), anaerobes (10–20%), and various gram-negative bacilli (30–40%). The precise distribution depends on whether the patient already has been treated with antibiotics. Anaerobes are especially prevalent in chronic wounds. Pretreatment typically selects for *P. aeruginosa* or enterococci.

DIAGNOSIS

In many cases, foot osteomyelitis can be diagnosed clinically, without imaging procedures. Most clinicians rely on the “probe-to-bone” test, which has a positive predictive value of ~90% in populations with a high pretest probability. Thus, in a patient with diabetes who is hospitalized for a chronic deep foot ulcer, the diagnosis of foot osteomyelitis is highly probable if bone can be directly touched with a metal instrument. In a patient with a lower pretest probability, MRI should be performed because of its high degree of sensitivity (80–100%) and specificity (80–90%). Plain radiography has a sensitivity of only 30–90% and a specificity of only 50–90%; it may be considered for follow-up of patients with confirmed diabetic foot osteomyelitis.

TREATMENT FOOT OSTEOMYELITIS

As mentioned above, correlation between cultures of bone and those of wound swabs or wound punctures is poor. Antibiotic treatment should be based on bone culture. If no bone biopsy is performed, empirical therapy chosen in light of the most common infecting agents and the type of clinical syndrome should be given. Wound debridement combined with a 4- to 6-week course of antibiotics has been shown to render amputation unnecessary in about two-thirds of patients. According to the 2012 Infectious Diseases Society of America Clinical Practice Guideline for the Diagnosis and Treatment of Diabetic Foot Infections, the following management strategies should be considered. If a foot ulcer is clinically infected, prompt empirical antimicrobial therapy may prevent progression to osteomyelitis. When the risk of methicillin-resistant *S. aureus* is considered high, an agent active against these strains (e.g., vancomycin) should be chosen. If the patient has not recently received antibiotics, the spectrum of the selected antibiotic must include gram-positive cocci (e.g., clindamycin, ampicillin-sulbactam). If the patient has received antibiotics within the past month, the spectrum of empirical antibiotics should include gram-negative bacilli (e.g., clindamycin plus a fluoroquinolone). If the patient has risk factors for *Pseudomonas* infection (previous colonization, residence in a warm climate, frequent exposure of the foot to water), an empirical anti-pseudomonal agent (e.g., piperacillin-tazobactam, cefepime) is indicated. If osteomyelitis is suspected either on clinical grounds (probe to bone) or on the basis of imaging procedures (MRI), bone biopsy should be performed. If not all infected bone is surgically removed, the patient should be treated for 4–6 weeks in line with the identified pathogen(s) and their susceptibility. Treatment should initially be given by the IV route. Whether therapy can later be administered by the oral route depends on the bioavailability of oral drugs that cover the infecting agents. If dead bone cannot be removed, long-term therapy (at least 3 months) should be considered. In such cases, cure of osteomyelitis is usually the exception, and repetitive suppressive treatment may be needed.



FIGURE 158-5 Neuropathic joint disease (Charcot foot) complicated by chronic foot osteomyelitis in a 78-year old woman with diabetes mellitus complicated by severe neuropathy.