Anaerobic Osteomyelitis in Children

Claudia M. Espinosa, MD, * Matthew M. Davis, MD, MAPP, † and Janet R. Gilsdorf, MD‡

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Anaerobic bacteria are a recognized cause of osteomyelitis in children, and more than 800 cases have been reported. Yet, the prevalence of anaerobic osteomyelitis is unknown, in part because of the technical difficulties in isolating and identifying many of these organisms. The most common anaerobic isolates reported in children with osteomyelitis are Bacteroides spp., Peptostreptococcus, Clostridium spp., Pseudomonas, and aerobic isolates.1–7,10 Foul odor of pus, once considered a characteristic of anaerobic infections, is not always present.1,3,4,8 Complications of anaerobic osteomyelitis in children are similar to those of aerobic osteomyelitis and include progression to chronic infection, deformity, and pathologic fracture.

Diagnosis
Isolation of anaerobic microorganisms from bone tissue provides the definitive diagnosis of anaerobic osteomyelitis. Use of anaerobic containers for collection and transport and fresh culture media, avoidance of contamination with normal flora, and rapid processing of the sample maximize recovery.1–4 Because anaerobic organisms exhibit variable sensitivity to oxygen tension, subculturing may be necessary for species identification.4,8

Identification and resistance testing of anaerobes are not routine in all laboratories due to cost and lack of standardization of the methods.13 Antibiotic discs and spot tests may yield presumptive identification, but definitive identification may require biochemical testing, low-molecular-weight fatty acid profiling, or 16S rRNA sequencing. Identification kits that require a shorter incubation period in an aerobic environment (preformed enzymes) or longer incubation in an anaerobic environment (inducible enzymes) have good sensitivity (78%–79%).13 Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry provides accurate and rapid identification for commonly isolated anaerobic bacteria such as Bacteroides.
Infection. Ultrasound has high sensitivity and specificity values support the diagnosis, as do radiographs showing periosteal new bone formation (imitating malignant bone tumors), but these changes occur after 2 weeks. Radionuclide scanning with technetium may be positive before standard radiographic changes appear and may localize an infection. Ultrasound has high sensitivity (82%–100%) and specificity (75%–96%) if symptoms are localized, as-assesses the extent of soft-tissue compromise, and helps to differentiate acute from chronic osteomyelitis. Evidence of bone necrosis or abscess formation on imaging studies suggests possible anaerobic infection.

Treatment

Treatment of anaerobic osteomyelitis includes management of pain, immobilization, adequate drainage of purulent or necrotic material (if present), and antibiotic therapy. Surgical debridement is required for patients with bony abscesses, necrotic bone, decubitus ulcers, poor response to antimicrobial therapy, or an infected foreign body, or orthopedic device. The appropriate length of antibiotic therapy remains controversial and depends on whether the infection is acute or chronic. Most experts recommend 4 to 6 weeks for acute osteomyelitis. Treatment of chronic osteomyelitis is more variable, ranging from 4 weeks to more than 6 months after debridement of devitalized tissue. Empiric therapy should be directed to major aerobic and anaerobic organisms, depending on the site of the infection and the local patterns of microbial resistance. Transition to oral therapy is acceptable after inflammatory signs subside.

Increasingly many anaerobic bacteria produce beta-lactamase, limiting the therapeutic value of penicillin. In such infections, beta-lactams with beta-lactamase inhibitors or antibiotics such as clindamycin, cefoxitin, a carbapenem, or metronidazole are indicated. Recently, resistance of anaerobes to amoxicillin/clavulanate and piperacillin/tazobactam (2%–11% of cases); cefoxitin (3%–17%); and clindamycin (10%–40%), depending on the organism and geographic area, has been reported. Quinolones, widely used in adults, have not been well studied in the pediatric population. Thus, they are not licensed for use in children, but have been shown in clinical practice to be safe. In general, clindamycin, vancomycin, and linezolid have excellent bone concentrations, target some anaerobic bacteria and can be used as initial empiric therapy. Once culture and susceptibility test results are available, antibiotic treatment should be tailored.

Conclusion

Bone infections in children caused by anaerobic bacteria have been diagnosed more frequently in recent years due to improvements in bacterial isolation techniques and increased awareness. Attention to the risk factors for anaerobic bacteria, use of standardized isolation methods, monitoring susceptibility patterns, and targeting the treatment to specific organisms when possible will improve the outcome of anaerobic bone infection in children.

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References