Published in final edited form as:

Pediatr Infect Dis J. 2011 May ; 30(5): 422–423. doi:10.1097/INF.0b013e318217ca0e.

Anaerobic Osteomyelitis in Children

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Keywords

anaerobes; osteomyelitis

Anaerobic bacteria are a recognized cause of osteomyelitis in children,^{1–7} and more than 800 cases have been reported.^{3,4} 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. group, actinomycetes, *Fusobacterium* spp., anaerobic Gram positive organisms, *Clostridium* spp., *Peptostreptococcus*, *Bacillus* spp., *Corynebacterium* spp., and, more recently, *Prevotella* spp., *Porphyromonas* spp., and *Propionibacterium* spp. (especially associated with orthopedic devices).^{1–5,7–10}

Pathogenesis

Although osteomyelitis caused by aerobic bacteria in children usually results from hematogenous spread during bacteremia, anaerobic osteomyelitis usually results from spread from a contiguous infection.^{1,2,8} Processes that disrupt normal blood flow to the bone, such as trauma, surgery, chronic or acute ischemia, malignant growth, or metabolic disorders, predispose to anaerobic bacterial replication.⁸ In children, conditions associated with anaerobic osteomyelitis include mastoiditis, otitis media, sinusitis, periodontal abscess, human bites, decubitus ulcers, ischemia (necrotic tissue), and, less frequently, trauma and diabetes mellitus.^{1,3–5} In general, causative agents of anaerobic osteomyelitis belong to mucous membrane or skin flora contiguous to the infected site. For example, skull and hand infections and those associated with human bites reflect oral mucosa pathogens and perianal infections reflect gastrointestinal flora.^{3,4}

Polymicrobial infections and coinfection with aerobic bacteria, common in anaerobic osteomyelitis, are associated with increased severity and with treatment failure.^{1,3,8,11} A possible explanation is that aerobic bacteria metabolize available oxygen, thus reducing the tissue oxidation potential to a level that allows anaerobic bacteria to grow.⁸

Clinical Manifestations and Complications

Anaerobic osteomyelitis may be asymptomatic.^{1,7,8,12} In a series of 26 pediatric patients, the duration of symptoms before diagnosis varied widely from 7 to 37 days. Systemic symptoms, however, including low grade fever and localized pain, were found in 69% and 85% of cases, respectively, at admission.³ Infections associated with prosthetic and other

orthopedic devices may occur up to 24 months after surgery, or even later.^{9,10} 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, sub-culturing may be necessary for species identification.^{3,4,8}

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* (identifies up to 97.5%), *Fusobacterium*, and *Prevotella*.¹³

When bone tissue is not available for anaerobic culture, blood culture, and anaerobic culture of infected fluid contiguous to bone may yield the infecting organism. Similar to aerobic osteomyelitis, leukocytosis, and elevated sedimentation rate and C-reactive protein values support the diagnosis, as do radiographs showing periosteal new bone formation (imitating malignant bone tumors), but these changes occur after 2 weeks.⁸ Radionuclide scanning with technetium may be positive before standard radio-graphic changes appear and may localize an infection.¹⁴ Ultrasound has high sensitivity in detecting subperiosteal and soft-tissue fluid collections. Computed tomography scanning is useful if bone sequestrum is present or if the anatomic site is difficult to assess with magnetic resonance imaging (ie, evaluation of the pelvis or scapula). On the other hand, magnetic resonance imaging has high sensitivity (82%–100%) and specificity (75%–96%) if symptoms are localized, assesses the extent of soft-tissue compromise, and helps to differentiate acute from chronic osteomyelitis.^{4,14} 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.¹⁻⁴ 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 months¹⁰ 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.⁴ 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.¹³ 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.¹⁵ In general, clindamycin, vancomycin, and linezolid have excellent bone concentrations, target some anaerobic bacteria and can be used as initial empiric therapy.⁴ 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.

Acknowledgments

Claudia Espinosa, MD, thanks the Michigan Institute for Clinical and Health Research for a Clinical and Translational Science Award.

Supported by Eunice Kennedy Shriver National Institute of Child Health and Human Development (T32 HD007534) (to C.M.E.).

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