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Current Recommendations in the Management of Osteomyelitis of the Hand and Wrist

Hitoshi Honda, MD[a] and **Jay R. McDonald, MD**[b,a]

[a]Division of Infectious Diseases, Washington University of School of Medicine, Saint Louis, MO

[b]St. Louis VA Medical Center, St. Louis, MO

Abstract

Management of osteomyelitis of the hand and wrist is a multidisciplinary and individualized process. Suboptimal management can result in poor functional outcomes. A rational approach to management includes careful consideration of the pathogenesis, microbiology, diagnostic options, and surgical and medical treatment of this disease.

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Osteomyelitis, a pyogenic bony infection, can occur in virtually any bone, including the bones of the hand and wrist. It can be caused by direct inoculation of pathogens, spread from adjacent tissue, or hematogenous spread.[1] In the hand and wrist, the majority of osteomyelitis is related to direct inoculation, including penetrating trauma and post-surgical infection.

Once a pathogen is introduced into tissue, it replicates and spreads along anatomical planes into areas of least resistance. While intact cortex of bone provides at least a mechanical barrier to pathogen penetration, traumatized bone is easily infected. Local inflammation leads to increased tissue pressure, lower pH and oxygen tension, leading to the formation of microthrombi within the intraosseous vessels and bony necrosis. A nidus of necrotic bone, called a sequestrum, provides a safe harbor for pathogens because of lack of vascularity and thus poor drug penetration. When an infected sequestrum is present, non-surgical cure is nearly impossible. The presence of implanted hardware affords additional protection to pathogens, as many bacteria surround themselves with a protective barrier called a biofilm on non-organic surfaces.

Diagnosis of osteomyelitis can be challenging. Inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein tend to be elevated during osteomyelitis and decrease with effective therapy, but they are useful only to monitor response to therapy. MRI, CT scan, and 3-phase bone scan are marginally better than plain radiographs for diagnosing osteomyelitis, but all have poor sensitivity and specificity. Bone biopsy with characteristic pathology and positive culture is the gold standard diagnosis. The presence of infected material immediately adjacent to bone should be treated as osteomyelitis.

Osteomyelitis occurring after penetrating trauma or contaminated open injury is most often polymicrobial. Likely organisms may differ depending on the milieu at the time of injury. Contamination by human mouth flora, including after a bite, often includes viridans

Corresponding author: Jay R. McDonald, MD, Washington University School of Medicine, Division of Infectious Diseases, 660 S. Euclid Ave., Campus Box 8051, Saint Louis, MO 63110, Phone: (314) 454-8354, Fax: (314) 454-5392, Email: jmcdonal@im.wustl.edu.
Coauthor: Hitoshi Honda, MD, Washington University School of Medicine, Division of Infectious Diseases, 660 S. Euclid Ave., Campus Box 8051, Saint Louis, MO 63110, Phone: (314) 454-8354, Fax: (314) 454-5392, Email: hhonda@im.wustl.edu

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streptococci, *Staphylococcus aureus*, gram positive anaerobes, and *Eikenella corrodens*. Contamination by water may introduce water-borne bacteria such as *Pseudomonas* or other gram negative species. Soil contamination may introduce gram negative organisms and occasionally low-virulence fungi and atypical mycobacteria. *Pasteurella multocida* must be a consideration after dog and cat bites.

Osteomyelitis which is hematogenous, as well as post-operative infections after clean surgery, are more likely to be caused by a single organism. Hematogenous osteomyelitis is most commonly caused by *S. aureus*, streptococci, and enteric gram negative bacilli such as *Escherichia coli*. Post-operative infections are typically caused by skin flora, including staphylococci and streptococci. Especially in the presence of implanted hardware, organisms of low pathogenicity can cause indolent infection. Coagulase negative staphylococci, *Propionibacterium acnes*, and Bacillus species are examples of organisms which must be taken seriously in the presence of hardware.

S. aureus is a virulent organism capable of causing significant tissue invasion and bone destruction, and merits its own discussion. The rapid emergence of community associated methicillin resistant-*S. aureus* (CA-MRSA) since the 1990's has changed antimicrobial management for osteomyelitis; CA-MRSA are not susceptible to beta-lactam antibiotics, and therefore vancomycin must generally be included in empiric therapy for osteomyelitis when CA-MRSA is a potential pathogen.[2] Though CA-MRSA isolates are often susceptible to clindamycin, trimethoprim-sulfamethoxazole, macrolides, and quinolones, the role of these antimicrobials in the treatment of CA-MRSA osteomyelitis remains unclear.

Microbiological diagnosis is imperative in order to identify causative organisms and their antimicrobial susceptibilities. Ideally, antibiotics are delayed until appropriate cultures are obtained, but this may not be prudent when signs of acute illness are present. Intraoperative cultures for both aerobic and anaerobic bacteria should be obtained, since superficial cultures usually do not correlate with the causative pathogens in osteomyelitis. In chronic infections and immunosuppressed hosts, cultures for fungi and mycobacteria should also be sent.

Management of osteomyelitis of the hand and wrist should consist of a combined surgical and medical approach to achieve the most favorable outcome. Non-surgical cure is occasionally achieved in acute osteomyelitis, but should not be routinely attempted. Infected hardware should be removed, at least temporarily, whenever possible.[3] Temporary external fixation prior to hardware re-implantation should be considered. When hardware removal is not possible, chronic suppressive therapy after initial antimicrobial treatment is often necessary.

In general, bactericidal, parenteral antimicrobials are preferred for treatment of osteomyelitis. Initial empiric therapy should include antimicrobials that are active against gram positive and gram negative bacteria. Many clinicians prefer vancomycin for gram positive coverage, with target troughs from 15 to 25 mcg/ml. Newer agents such as daptomycin and linezolid may be effective as well, though there is less experience with their use in osteomyelitis. In addition, empiric therapy should include gram negative coverage, such as a third- or fourth-generation cephalosporin, or a flouroquinolone. Additional or alternative antimicrobials may be appropriate for specific situations according to suspected microbiology of the infection.

Once causative pathogens are identified, empiric therapy should be changed to pathogen-specific therapy. Parenteral antimicrobials with less frequent dosing may be desirable for outpatient parenteral antimicrobial therapy. Switching to oral antimicrobials with excellent bioavailability is sometimes possible, but should be done cautiously and with consultative advice. Optimal duration of treatment has not been adequately studied. We commonly treat for 4 to 6 weeks, and sometimes longer based on severity and response to therapy.

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Broad conclusions about preferred antimicrobials in osteomyelitis are not possible based on existing data. There are some studies of penetration of different antibiotics into bone. In experimental models of *S. aureus* osteomyelitis, bone-to-tissue concentrations of first generation cephalosporins was 7% or less, and concentrations for vancomycin and quinolones were 12–15%.[4] Clindamycin concentrations in debrided bone have generally been above the minimum inhibitory concentration for isolated pathogens.[5] However, these studies have used various non-standardized methodologies, and the results have not been correlated with outcomes in clinical studies.

Osteomyelitis of the hand and wrist can result in significant morbidity and functional loss. Optimal management requires a multidisciplinary approach, involving surgeons and infectious diseases specialists to preserve function and maximize the likelihood of positive outcome.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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