CHILDREN'S ACUTE HEMATOGENOUS OSTEOMYLITIS: REVIEW ARTICLE

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ABSTRACT

The most vascularized areas of the developing skeleton are frequently affected by the common invasive infection known as acute hematogenous osteomyelitis (AHO). This infection mostly affects children and is frequently spread hematogenously. In pediatric patients, the most common cause of acute hematogenous osteomyelitis (AHO) is now Staphylococcus aureus. The patient's age is frequently linked to the particular organism that was discovered in cases of bacterial osteomyelitis. In order to effectively treat pediatric AHO, antibiotic therapy must be initiated early. Parenterally administered antimicrobial drugs, such as vancomycin and clindamycin, must be given for at least four to six weeks.

A review of the literature is conducted with regards to the management, symptoms, and etiology of AHO in children. We offer a paradigm for empirical therapy based on published recommendations and the body of relevant literature.

Keywords: *children, antibiotics, bone infection, pediatric infectious disorders, acute hematogenous osteomylitis*

INTRODUCTION

Osteomyelitis (OM) is an acute or chronic bone infection that typically affects preschool-aged children, with a male-to-female ratio of 2:1. The long bones and vertebrae are the skeletal components that sustain damage R[1–2]. The most frequent kind of bone infection in children, acute hematogenic osteomyelitis (AHOM), which is divided into three stages: acute (symptoms last less than two weeks), subacute (symptoms last two weeks to three months), and chronic (long-term infection that develops over months to years) R[3]. bone degeneration, with an approximate yearly incidence of 8 per 100,000 youngsters in wealthy income-producing nations. The majority of cases of the infection have bacterial aetiology R[1]. While the most common way for children to become infected with osteomyelitis is through hematogenous inoculation of the bone during a bacteremia episode, osteomyelitis can also arise from direct inoculation from a penetrating trauma or can spread from a contiguous site of infection R[4].

Clinical manifestations :

Acute hematogenous osteomyelitis primarily affects children, and it usually shows up at the metaphysis of long bones. Patients usually present one to three days after the onset of symptoms, which usually include warmth in the afflicted area, erythema, fever, pain, and edema. The specific timing of symptoms depends on the microbiological etiology, but patients usually experience them 6–8 days prior to presentation. Furthermore, patients frequently describe a history of mild blunt trauma to the affected area. Although AHO can affect any part of the skeleton, the long bones of the lower extremities are among the most usually affected, followed by the pelvis R[5]. In a single-center investigation, the most often affected bones were the tibia, fibula, pelvis, and femur, in that order R[6]. When the child's condition and level of discomfort allow, a musculoskeletal examination should be performed with the utmost care; this is important because 5–10% of kids may have multifocal AHO[6].

Pathogenesis of AHO:

Although the exact cause of AHO in children is unknown, it is widely thought to result from the peculiar structure of developing bones in conjunction with transitory bacteremia. According to the widely accepted mechanism of pathogenesis, which was first proposed by Hobo and Trueta R[7], trauma causes the production of a suppurative focus in the metaphysis. Infection spreads to the medullary cavity, through the cortex elavates the periosteum, leading to sub-periosteal abscess. Infection does not reach the joint cavity because the epiphyseal cartilage is highly resistant to the spread of infection.

The necrotic bone that results from inflammation, bacterial toxins, ischemia caused by inflammatory thrombosis, pressure from exudates, and periodontal elevation all cause the cortical blood vessels to stretch. Osteoclasts break down the necrotic bone and separate it into normal bone, which is called the sequestrum. The periosteum surrounding the sequestrum exposes a new bone called the involucurum, which has holes called cloaca at the sites of repture of subperiosteal abscess, which open on the skin due to multiple sinuses releasing pus R[8].

Microbiological etiology:

The particular microorganism(s) recovered from bacterial osteomyelitis patients are frequently linked to the patient's age or the clinical situation [Tables 1 and 2]. [Table 3] summarizes the relative frequency of recovery of common organisms in AHO R[5]. Staphylococcus aureus is the pathogen most frequently linked to AHO.

group A streptococcus (GAS) in 80% of culture-positive cases R[9]. Gram-negative enteric bacteria, group B streptococcus, and S aureus are common infections in newborns. Neisseria gonorrhoeae should be taken into account in newborns and teenagers who are sexually active R[9]. In addition to S aureus, Salmonella spp. are often responsible for osteoarticular infections in children with sickle cell anemia R[9]. Since the broad introduction of the immunization program in the 1990s, Haemophilus influenzae type b (Hib)-caused osteomyelitis has become extremely rare in high-income countries9. In the past, Hib was responsible for 10%–15% of instances of osteomyelitis in children under the age of three who were not immunized in low-income nations.

AHO brought on by CA-MRSA, or community-associated methicillin-resistant S aureus, has grown widespread in several nations.R[4-5] MRSA prevalence varies greatly by location. Thirty to forty percent of juvenile osteoarticular infections were linked to MRSA in one US investigation R[9].

TABLE 1: Organisms Commonly Isolated in Osteomyelitis Based on Patient Age

Infants:(<1 Year)	**1-Group B streptococci** **2-Staphylococcus aureus** **3-Escherichia coli**
Youngsters (ages 1 to 16)	**1- S.aureus** **2- Streptococcus pyogenes** **3- Haemophilus influenza**

TABLE 2: Organisms Isolated in Bacterial Osteomyelitis

Organism	Comments
Staphylococcus aureus	Organism most often isolated in all types of osteomyelitis
Coagulase-negative staphylococci	Foreign-body–associated infection or Propionibacterium species
Enterobacteriaceae species or Pseudomonas aeruginosa	Common in nosocomial infections
Streptococci or anaerobic bacteria	Associated with bites, fist injuries caused by contact with another person's mouth, diabetic foot lesions, decubitus ulcers
Salmonella species or Streptococcus pneumoniae	Sickle cell disease
Bartonella henselae	Human immunodeficiency virus infection
Pasteurella multocida or Eikenella corrodens	Human or animal bites
Aspergillus species, Mycobacterium	Immunocompromised patients avium-intracellulare or Candida albicans
Mycobacterium tuberculosis	Populations in which tuberculosis is prevalent
Brucella species, Coxiella burnetii	Population in which these pathogens (cause of chronic Q fever) or other are endemic fungi found in specific geographic areas

Note: Reproduced with permission from Waldvogel FA, Lew DP, Osteomyelitis. 1997 N Engl J Med;336:999–1007.

Management:

Children's AHO is managed by a multidisciplinary team that includes radiologists, orthopedic surgeons, pediatric infectious disease specialists, and pediatricians. The causal organism, infection site, related consequences, and clinical and laboratory response to treatment all influence the first choice, course, and length of antibiotic therapy[13].

Antimicrobial therapy, debridement with management of resultant dead space, and, if necessary, stabilization of bone comprise the treatment following the initial evaluation, staging, and establishment of microbial etiology and susceptibilities.[12] Early antibiotic therapy yields the best results in the majority of patients with osteomyelitis.

For an appropriate rate of cure, antimicrobials should be provided for a least of four weeks, ideally six weeks [Table 4].

The optimum course of treatment for acute hematogenous osteomyelitis is a four- to six-week course of adequate antibiotic medication. Antibiotics and continuous debridement are the usual treatments for persistent osteomyelitis.

The length of AHOM therapy, both intravenously and orally, is a topic of much discussion in the scientific literature. For simple AHOM, the average treatment duration is four weeks, with a range of three to six weeks R[14-22].

The majority of trials on oral therapy for AHOM use high-dose clindamycin, amoxicillin, or cephalosporin alone or in conjunction with rifampicin R[1-2]

Table 3: Recommended Dosage for Oral Antibiotics

Cephalexin	100 mg/kg daily in 4 divided doses (max daily dosage 4 g)
Amoxicillin-clavulanate	80 mg/kg daily in 3 divided doses (max daily dosage 2 g)
Amoxicillin	75–100 mg/kg daily in 3 divided doses (max daily dosage 3 g)
Clindamycin	30–40 mg/kg daily in 3–4 divided doses (max daily dosage 1.8 g)
TMP-SMX	8 mg/kg daily of TMP in 2 divided doses (max daily dosage 320 mg of TMP)
Rifampicin	10–20 mg/kg daily in 1–2 divided doses (max daily dosage 600 mg)

Table -4

Intravenous antibiotic dosage Antibiotic	Recommended dose
Amoxicillin/clavulanate	75–100 mg/kg daily of amoxicillin in 3–4 divided doses (max 1 g/dose)
Ampicillin/sulbactam	100–200 mg/kg daily of ampicillin in 4 divided doses (max 2 g/dose)
Cephazolin	150 mg/kg daily in 3–4 divided doses (max 2 g/dose)
Ceftazidime	150 mg/kg daily in 3 divided doses (max 2 g/dose)
Ceftriaxone	50–100 mg/kg daily (max 2 g)
Clindamycin	45 mg/kg daily in 3 divided doses (max 900 mg/dose)
Oxacillin	150–200 mg/kg daily in 4 divided doses (max 2 g/dose)
Gentamycin neonates ≥35 weeks of gestational age:	4 mg/kg daily during the first week of life, then 5 mg/kg daily > 1 month-10 years: 8 mg/kg the first day, then 6 mg/kg daily > 10 years: 7 mg/kg daily the first day, then 5 mg/kg daily
Linezolida < 12 years:	30 mg/kg daily in 3 divided doses (max 600 mg/dose) > 12 years: 600 mg twice a day Vancomycin 45 mg/kg daily in 3

Surgical dissection:

In patients with persistent osteomyelitis, surgical debridement can be technically challenging. R[22] The debridement's quality is the most important factor in essential component of effective management. Following debridement and bone excision, the dead space left by tissue removal must be completely filled up.

According to studies, in 90% of instances of AHO, effective antibiotic therapy alone—without surgical intervention—might be sufficient. Drainage of purulent collections may promote pain relief and facilitate a more rapid response to medical therapy. In general, indications for surgical intervention may include (but are not limited to) the presence of subperiosteal, intraosseous, or adjacent soft tissue abscesses or failure to improve with medical therapy alone. The indications for surgical management in AHO include persistent symptoms (fever, local inflammation) not responding to empiric antibiotic therapy, the presence of periosteal or other deep soft tissue abscess (more common with MRSA or strains expressing virulence genes like PVL].

Conclusion :

AHO is a dangerous bacterial infection that affects youngsters that is somewhat prevalent. Although a diverse range of microorganisms can cause this disease, S. The primary cause is aureus. For these patients, a multidisciplinary strategy that takes into account combined medical and surgical care should be taken into consideration. Vancomycin and clindamycin are well-established traditional treatments that show promising therapeutic results in pediatric patients.

Considering the paucity of pediatric data, daptomycin and linezolid could be regarded as second-line treatment alternatives for individuals who are not responding to or tolerating vancomycin and clindamycin.

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