Pediatric Hematogenous Osteomyelitis: New Trends in Presentation, Diagnosis, and Treatment

John P. Dormans, MD, and Denis S. Drummond, MD

Abstract

The character of acute hematogenous osteomyelitis (AHO) in North American children has changed significantly during the past several decades. Although the typical clinical picture of established acute osteomyelitis in children (illness, dehydration, and an acutely painful limb) is still seen, more subtle presentations appear more frequently. Children often present with subacute osteomyelitis. Less common variants include Brodie's abscess, subacute epiphyseal osteomyelitis, and chronic recurrent multifocal osteomyelitis. Some patients present with a bone lesion that may be confused with other disease entities, including neoplasms. Biopsy is often needed to clarify the diagnosis. With the trend toward more invasive procedures in the neonatal intensive care unit, neonatal osteomyelitis is also seen more frequently. Advances in imaging technology, particularly improvements in technetium bone scanning and the advent of magnetic resonance imaging, have contributed to more precise diagnosis and better management of AHO. With the increased concern about medical economics, the recent trend toward decreasing the duration of intravenous antibiotic treatment of these infections appears to be appropriate as long as certain criteria are met. Neither surgery nor antibiotics alone will be associated with successful treatment in all cases, and this fact may explain the rare but continued morbidity that is still seen in children with AHO.

J Am Acad Orthop Surg 1994;2:333-341

The clinical presentation of acute hematogenous osteomyelitis (AHO) in children has changed in several ways over the past few decades. Fulminating infection is seen less frequently than before. Instead, atypical forms of infection, including subacute osteomyelitis, are more common, and children often present less ill and with less destructive radiologic features than previously described. This may be due to a variety of reasons, including modification of the clinical course by antibiotics given before admission and possibly increased awareness and earlier presentation to a medical facility, which results in earlier diagnosis.

Historical Perspective

Before the advent of antibiotics, AHO had a mortality rate as high as 45%.[1,2] After the introduction of penicillin in 1944, the mortality rate improved to 1% or less, and the incidence of osteomyelitis decreased.[2] Most cases were caused by penicillin-sensitive *Staphylococcus* and *Streptococcus* organisms and, with widespread use of penicillin, were successfully treated without early surgery. With the development of antibiotic-resistant organisms and the frequent abandonment of surgical intervention, morbidity, mortality, and recurrence of infection increased in the 1950s and 1960s.[1] Today, with appropriate antibiotics and aggressive therapy, recurrence of osteomyelitis is unusual.

Definitions

Acute hematogenous osteomyelitis can be classified by patient age (neonatal, childhood, or adult), causative organism (pyogenic or granulomatous infection), onset (acute, subacute, or chronic), and route of infection (hematogenous or by means of direct inoculation). Chronic osteomyelitis is defined by most authors as osteomyelitis with symptoms that have been present for more than 1 month. This review will concentrate on childhood acute pyogenic hematogenous osteomyelitis, childhood subacute pyogenic hematogenous osteomyelitis, and neonatal acute pyogenic hematogenous osteomyelitis.

Dr. Dormans is Assistant Professor of Orthopaedic Surgery, University of Pennsylvania, Philadelphia. Dr. Drummond is Professor of Orthopaedic Surgery, University of Pennsylvania.

Reprint requests: Dr. Dormans, Children's Hospital of Philadelphia, Division of Orthopaedic Surgery, Second Floor, Wood Building, 34th and Civic Center Boulevard, Philadelphia, PA 19104.

Pediatric Acute Hematogenous Osteomyelitis

Pediatric AHO usually occurs in the first decade. No consistent peak incidence by age group is described in the literature. There is a male predominance in most series[1-4]; however, some series show an almost equal male-female ratio. The greater occurrence in boys described in most series may be related to the role of trauma in the development of osteomyelitis in children.

Acute hematogenous osteomyelitis may coexist with septic arthritis. This occurs particularly in patients less than 12 to 18 months of age due to the unique blood supply of the chondroepiphysis. It may also occur in joints with intra-articular metaphyses (proximal humerus, proximal femur, distal lateral tibia, and proximal radius).

Pathogenesis

While it is apparent that trauma and bacteremia contribute to the development of osteomyelitis in children, neither will cause bone infection on its own. The role of trauma in the pathogenesis of AHO was implicated by Whalen et al in 1988.[5] They showed that trauma increases the chance of development of osteomyelitis when there is concurrent bacteremia. In their rabbit-model study, trauma appeared to be associated with regional ischemia and a generalized lowered resistance to infection. Other factors may include illness, malnutrition, and immune system deficiency.

Infection begins in the metaphyseal venous sinusoid, where there is a change from high-flow arterioles to low-flow venous sinusoids. Evidence suggests that there is a poorly developed reticuloendothelial system there, with lack of local resistance due to the absence of tissue macrophages.[5] In 1921, Hobo[6] described vascular loops in this area, with arterioles that take sharp bends at the physis and empty in venous lakes. Hobo believed that, with turbulence in this area, bacteria lodged in these sharp bends; their accumulation, combined with a lack of phagocytic reaction in the area, led to infection. Subsequent electron microscopic studies have shown terminal capillary branches in this area.

With the development of infection, there is thrombosis of the medullary vessels and suppression of the mobilization of infection-fighting cells (Fig. 1, A). Subsequently, an exudate is formed, which, if untreated, exits the porous metaphyseal cortex and elevates the periosteum to form a subperiosteal abscess or septic arthritis if the metaphysis is intra-articular (Fig. 1, B). If the periosteum remains viable, an involucrum is produced (Fig. 1, C).

The most common sites for AHO are the growing ends of long bones: the distal femur, proximal tibia, proximal femur, distal humerus, and distal radius. The predilection for these sites may be due to sluggish circulation near the physis favoring deposition of bacteria, or it may be due to the lack of tissue-based macrophages.[7] The lower extremities are involved more often than the upper, which may be related to the higher likelihood of trauma in these areas.

Evaluation and Diagnosis

History and Physical Examination

The clinical picture is still the most important factor in making the diagnosis. A high index of suspicion is required. There is a history of recent or concurrent infection in one third to one half of patients. Unexplained bone pain and fever should suggest osteomyelitis until proved otherwise. Recent studies show that a significant percentage of children do not fit the usual stereotype of an ill-appearing child with high fevers and high white blood cell (WBC) counts.[8,9] In fact, 36% of the patients in the series of Scott et al[9] had admission temperatures of less than 37.5° C, and 41% had WBC counts less than 10,500 / mm^3. At our institution, subacute osteo-

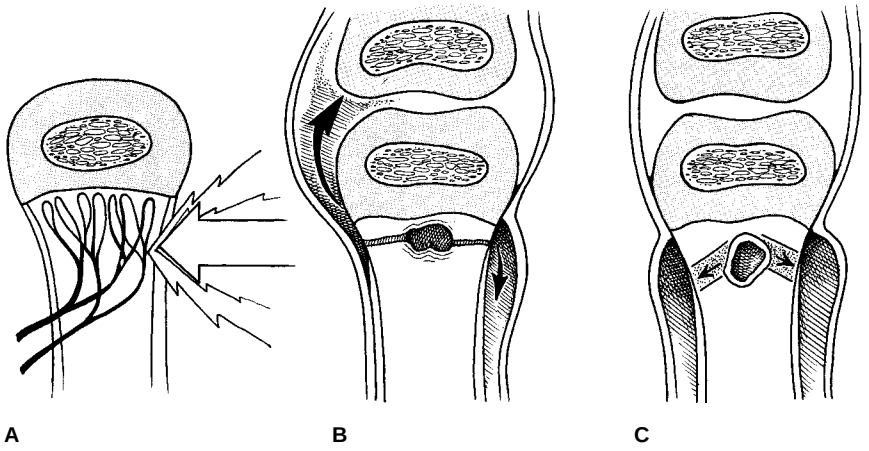

Fig. 1 **A**, The combination of bacteremia and trauma favors the development of infection in the metaphyseal venous sinusoids. **B**, The infection will eventually track through the porous metaphyseal cortical surface and elevate the surrounding periosteum. If the metaphysis is intra-articular, the infection will break into the joint and cause concurrent septic arthritis. C, The elevated periosteum lays down new bone initially (involucrum), and the dead medullary (or cortical) bone becomes a sequestrum.

myelitis presenting as a neoplasm is a not infrequent occurrence.[9]

On physical examination, swelling and refusal to move the limb are often noted. Some patients, however, are restless, as they are unable to relieve the pain of increased interosseous pressure by changing positions. There is usually tenderness with palpation and increased warmth, and occasionally there is an adjacent sympathetic joint effusion. There is no true joint irritability as there is in septic arthritis (unless there is concurrent septic arthritis). The history and physical examination are usually much more helpful in differentiating cellulitis from osteomyelitis. The characteristic erythema and swelling, which usually appear in 2 to 3 days with cellulitis, take 3 to 4 weeks to appear with untreated osteomyelitis.

Laboratory Studies

The WBC count is frequently but not invariably elevated; however, the erythrocyte sedimentation rate (ESR) is elevated in the majority of patients.[9] An elevated ESR is not a reliable sign in neonates or children with sickle cell anemia, nor is it a good way of following the resolution of infection during the first week, as it lags behind the improvement seen clinically.[4] The clinical value of C-reactive protein was recently evaluated by Unkila-Kallio et al[10] and found to be better in reflecting the effectiveness of therapy and predicting recovery from AHO than the ESR and the WBC count. Specifically, they found that the C-reactive protein concentration was elevated at the time of admission in 98% of cases, that the peak C-reactive protein level was reached on day 2 after admission, and that the level decreased very rapidly, normal values being reached within a week of admission.

Blood cultures were positive in approximately 40% to 50% of the cases in most series.[4] It is important to culture other body sites to increase the possibility of identifying the causative organism.

Radiography

Plain radiographs show a deep soft-tissue swelling and loss of tissue planes by 3 days, but no bone changes are seen until after a week or more.[4,8] A recent trend is for patients increasingly to appear with no changes on admission radiographs or even on radiographs obtained up to 2 weeks after the onset of symptoms, due to either suppression of infection with antibiotics or indolence of the infection.[9]

Radionuclide Studies

Technetium-99m diphosphonate bone scanning is useful in locating the area of involvement, especially in areas where localization may be difficult, such as the spine and pelvis. Bone scans are also useful in looking for multiple sites of involvement (usually in neonates) and in detecting osteomyelitis associated with septic arthritis. Increased uptake on a bone scan is the most common finding in AHO; in contrast, decreased uptake may indicate avascular necrosis.

Technetium bone scanning is not indicated in every case of osteomyelitis. It may not be accurate in very early cases, particularly earlier than 24 hours after the onset of infection,[7,11,12] because there may not yet be stimulation of bone turnover. Scans may be misleading, especially in the early stages of infection, in neonates, and in patients with sickle cell disease.[4,11,12] In addition, bone scans are nonspecific because increased uptake of the radionuclide may also indicate trauma, tumor, or infection. There is a 4% to 20% false-negative rate with technetium bone scanning.[9]

Canale et al[13] have shown that bone aspiration will not significantly affect the results of bone scanning if the bone scan is obtained within 48 hours after aspiration. Therefore, aspiration and treatment should not be delayed in order to obtain a bone scan.

Scans obtained with gallium-67 citrate and with indium-labeled leukocytes are more expensive and entail more radiation exposure. They also take longer to complete, which may delay the initiation of treatment. These studies are not often useful for the evaluation of AHO in children.

As single photon emission computed tomography (SPECT) becomes more widely used, this tool may also become useful in evaluating children with osteomyelitis.

Magnetic Resonance Imaging

Magnetic resonance (MR) imaging is a useful tool for evaluating patients with osteomyelitis. In one study comparing MR imaging and bone scanning in the evaluation of 35 patients with suspected acute osteomyelitis, both modalities had a sensitivity of 100%. However, MR imaging provided higher specificity and accuracy at a statistically significant level.[14] Magnetic resonance imaging offers the sensitivity of bone scanning but with better soft-tissue resolution, is more useful in differentiating cellulitis from osteomyelitis, and can be used to identify abscesses, sequestra, and sinus tracts. The MR imaging study is also more useful in differentiating abnormal bone marrow (i.e., intraosseous extent) than bone scanning, computed tomography, or plain radiography.[12] However, MR imaging lacks specificity in determining whether abnormal changes are due to osteomyelitis. It is especially useful in the axial skeleton.[14]

The classic MR findings of osteomyelitis are a decrease in the normally high signal intensity of marrow on T1-weighted images and normal or increased signal intensity on

T2-weighted images.[15] This is due to the replacement of marrow fat by inflammatory cells and edema, which are lower in signal intensity than fat on T1-weighted images and are higher in signal intensity than fat on T2-weighted images.

Magnetic resonance imaging may also be useful in differentiating acute from chronic osteomyelitis. Cohen et al[16] found that the best predictors of acute osteomyelitis were poorly defined soft-tissue planes, absence of cortical thickening, and a poor interface between normal and diseased marrow. In contrast, chronic osteomyelitis (defined in their study as continuation of clinical symptoms for more than 3 months, the presence of sclerosis and thickening of the bones on plain radiographs, and/or pathologic findings of chronic inflammation or sequestrum formation) was suggested by the presence of a well-defined soft-tissue abnormality, a thickened cortex, and a relatively good interface between normal and diseased marrow.

Magnetic resonance imaging studies of neonates should be done by a team familiar with this age group. Frequent monitoring, supervision, and adequate sedation to prevent movement are required.

Aspiration and Bacteriologic Study

Aspiration may be the most valuable clinical test. It serves two purposes: it usually allows the establishment of a bacteriologic diagnosis (even if there is no abscess), and it can be used to determine the presence or absence of an abscess that may require surgical drainage. It can also be done very quickly, within hours of first seeing the patient. Aspiration should be done if osteomyelitis is suspected on the basis of the clinical picture and workup. If a malignant tumor, such as a Ewing's sarcoma, is a clinical possibility, aspiration can be deferred to a formal open biopsy once staging studies have been done.

Aspiration is performed at the point of maximal tenderness and swelling using a trocar 16- or 18-gauge spinal needle. Aspiration is done subperiosteally first. If purulent material is obtained, the needle is withdrawn, and the fluid is transported to the laboratory. If no purulent or abnormal fluid is obtained, intraosseous aspiration can be done through the thin, porous metaphyseal bone. All material obtained should be sent for Gram stain and cultures. At our institution, in addition to the Gram stain, fluid is sent for aerobic, anaerobic, fungal, and tuberculosis cultures. Aspiration is positive in approximately 60% of patients, with the rate of retrieval of the causative organism ranging from 61% to 90%.[9] While awaiting the culture results, we begin antibiotic therapy based on the "best educated guess" of which organism is predominant (Table 1).

Staphylococcus aureus remains the most predominant organism, accounting for infection in 60% to 90% of patients.[4,7,8] There appears to have been no significant change in the predominance of *S aureus* since the preantibiotic era.[2] Streptococci, pneumococci, *Kingella kingae* organisms, and Gram-negative bacteria are occasionally the etiologic agents. Streptococci may be seen with infections secondary to the infected skin lesions associated with measles and chicken pox. Gram-negative organisms account for fewer than 5% of cases, with *Haemophilus influenzae* being the predominant organism in this group. *Salmonella* is seen in patients with sickle cell disease, but is less common than *S aureus*.[4,7]

Treatment

The principles of treatment are (1) identification of the organism, (2) selection of the correct antibiotic, (3) delivery of the antibiotic in sufficient concentrations and for sufficient duration, and (4) arrest of tissue destruction.[4]

Antibiotic Therapy

As stated previously, antibiotic therapy is begun as soon as all cul-

Table 1
Initial Antibiotic Therapy for Osteomyelitis

Patient Type	Probable Organism*	Initial Antibiotic
Neonates	Group B *Streptococcus*, *Staphylococcus aureus*, or Gram-negative rods (*Haemophilus influenzae*)	Cefotaxime, 100-120 mg/kg of body weight for 24 hr, or oxacillin and gentamicin, 5.0-7.5 mg/kg for 24 hr
Infants and children	*S aureus* (90% of cases)	Oxacillin, 150 mg/kg for 24 hr
If allergic to penicillin		Cefazolin, 100 mg/kg for 24 hr
If allergic to penicillin and cephalosporins		Clindamycin, 25-40 mg/kg for 24 hr, or vancomycin, 40 mg/kg for 24 hr
Patients with sickle cell disease	*S aureus* or *Salmonella*	Oxacillin and ampicillin or chloramphenicol or cefotaxime, 100-120 mg/kg for 24 hr

*Overall, 80% of cases are due to *Staphylococcus aureus*.

tures have been obtained; the selection of antibiotic is based on the "best educated guess" (Table 1) and is changed, if necessary, once the culture results are available. Coagulase-positive *Staphylococcus* organisms are responsible for the vast majority of cases of AHO in otherwise healthy children; therefore, an antibiotic that effectively combats this organism is used. If the patient is not allergic to penicillin, a beta-lactamase-resistant semisynthetic penicillin should be used. Since methicillin carries a significant risk of causing interstitial nephritis and nafcillin can be associated with skin sloughing if subcutaneous infiltration occurs, oxacillin is the antibiotic generally used at our institution in this situation. The recommended dosage of oxacillin is 150 mg/kg of body weight in divided doses given over 24 hours.

The duration and route of administration of the antibiotic have been the subject of considerable debate, with no resolution as yet. At one time, children with AHO were treated routinely for 6 weeks with intravenous antibiotics in the hospital setting. More recently, a regimen of 3 weeks of intravenous antibiotics followed by 3 weeks of oral antibiotics has been adopted. In reality, route of administration is less important than attainment of the appropriate serum concentration, and the combination of intravenous followed by oral antibiotic administration has become the standard care at most institutions.

The most important variable influencing the duration of antibiotic therapy is clinical response, but other factors include the age of the patient, the site of infection, the amount of destruction, and previous surgery. Once a clinical response to antibiotic treatment has been seen, a switch to oral antibiotics can be made.[7] Intravenous antibiotics are usually given for a minimum of 7 to 10 days at our institution.

As mentioned previously, in addition to the patient's temperature curve and clinical examination findings, the C-reactive protein level can be used to follow the response to antibiotics.[10] Several prerequisites must be met, however. Jackson and Nelson[8] have stated that contraindications to an early change to oral antibiotics include an inability to swallow or retain the medicine, lack of identification of the etiologic agent, inability of the laboratory to obtain serum bactericidal levels, an infection caused by an organism for which no effective oral antibiotic exists (e.g., *Pseudomonas*), and lack of clinical response to intravenous antibiotics. Peak serum levels of antibiotic may be helpful in demonstrating adequate drug absorption by the gastrointestinal tract. These are obtained by drawing a blood sample 1 hour after the oral administration of the drug. The bactericidal level of the drug in the blood should be 1:8 or greater.[7] Antibiotic therapy (combined intravenous and oral administration) should be continued for a total of 6 weeks.[7]

The usual oral antibiotic is dicloxacillin, given in a dosage of 50 mg/kg for 24 hours, or cephalexin, given in a dosage of 150 mg/kg for 24 hours. Antibiotic choices for patients who are allergic to penicillin are shown in Table 1.

Surgical Options

The major indication for surgical debridement is to evacuate a subperiosteal abscess or to remove dead or avascular bone. Neither antibiotics nor surgery alone will be successful in all cases. We believe that aspiration of pus is an indication for operative intervention. Periosteal destruction can occur if pus accumulates for any length of time. Surgery is also effective in removing sequestra in chronic infections. Surgical technique requires making a small skin incision, opening the periosteum, and drilling the cortex. Operative drainage of the subperiosteal abscess can preserve the periosteal blood supply. The need to drill the cortex in acute osteomyelitis is disputed by some authors.[1] In addition to routine contact cultures, primary bone cultures may increase the likelihood of obtaining the offending organism. Suction irrigation tubes may be used, but we prefer a closed suction drainage system for 24 to 48 hours. Insertion of a central line while the patient is under the same general anesthetic may be useful for long-term intravenous access.

Aggressive osseous debridement is the most important aspect of treatment for chronic osteomyelitis,[17] defined by most authors as osteomyelitis with symptoms that have been present for more than 1 to 3 months. Daoud and Saighi-Bouaouina[17] reviewed the data on 34 children with chronic hematogenous osteomyelitis and concluded that the status of the periosteum of the involved bone is of primary importance, both in predicting the subsequent evolution of the disease and in planning treatment. The status of the periosteum is best evaluated in these cases on the basis of the presence or absence of an involucrum, which forms as the result of subperiosteal new bone formation. The role of early sequestrectomy for patients with chronic osteomyelitis has been debated. Some have attributed poor outcomes to sequestrectomy performed too early and/or too extensively and have recommended delayed debridement and sequestrectomy to allow development of the involucrum and revascularization of the sequestrum. Others have recommended early operative sequestrectomy to permit rapid resolution of the infection and regeneration of bone. Daoud and Saighi-Bouaouina[17] found that if a sequestrum is present, early sequestrectomy is indicated to help control the infection without

preventing the formation of the involucrum.

Whether medical treatment only should be used for AHO continues to be controversial. Surgery may not be indicated if the diagnosis is made early and if there is no abscess. Cole et al[18] created a classification of osteomyelitis with treatment implications: their three types are early-acute (patient is older than 1 year of age with an acute febrile illness of less than 48 hours' duration), late-acute (patient with severe osteomyelitis and an abscess is older than 1 year of age and presents 5 or more days after the onset of symptoms), and neonatal/infantile (patient is less than 1 year of age). Use of intravenous antibiotics followed by oral antibiotics without surgery was successful in 92% of their early-acute cases. Recent studies have demonstrated a 90% response rate to medical management alone when the treatment was initiated within the first few days after the onset of symptoms.[18,19] If there is no early clinical response to medical management (within 36 hours), abscess formation becomes a possibility, and reevaluation is necessary. Once a clinical response has been achieved, a change to oral antibiotics is appropriate.

Neonatal Osteomyelitis

Neonatal osteomyelitis differs from AHO in children because (1) the blood supply to the chondroepiphysis is different, (2) causative organisms are different, and (3) the immune system is still developing in the neonate.

Before secondary ossification centers appear, the metaphyseal vessels penetrate directly into the chondroepiphysis (Fig. 2). Infection starting in the metaphysis can readily invade and destroy the chondroepiphysis and subsequently invade the joint. These transphyseal vessels persist until 12 to 18 months of age.[20] The physis serves as a mechanical barrier to infection in older children. Additionally, the osseous architecture of the neonate is more fragile and easily injured by the infectious process. Permanent growth arrest can occur. The long tubular bones are involved most commonly in neonatal osteomyelitis, but membranous bones, such as the maxilla, may also be involved. The proximal femur is most commonly involved, with destruction of the femoral head often occurring as a result. In up to 40% of neonates with AHO, multiple sites are involved.[7]

Group B streptococci are now the predominant organisms responsible for neonatal osteomyelitis.[21] Other causative organisms include *S aureus*, Gram-negative bacilli, and *Streptococcus pneumoniae*.[21] Invasive procedures, such as fetal monitoring, heel punctures, and placement of umbilical catheters, are related to the increased incidence of neonatal osteomyelitis. *Staphylococcus* organisms are most commonly associated with these manipulative procedures.

Neonates are often unable to mount a significant inflammatory response to infection. The infection tends to spread very rapidly and often involves multiple sites. Despite this, the temperature and WBC count may be normal. Physical examination usually discloses swelling and/or loss of movement, but may reveal fewer physical findings than would be seen in an older patient. Aspiration should be performed on any suspicious bone or joint. Technetium bone scans may be useful in detecting multiple sites of involvement; however, false-negative studies sometimes occur, and bone scans may not show all infected sites. Treatment requires early diagnosis, prompt surgical drainage if an abscess has developed, and administration of the appropriate antibiotics. While sequestra, draining sinuses, and chronic infection rarely develop,

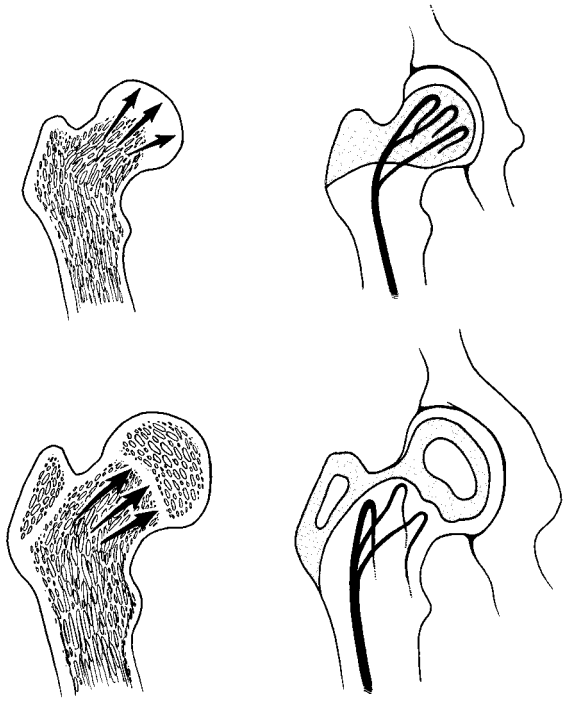

Fig. 2 Relation of blood supply to the proximal femur and spread of infection (arrows). **Top,** In the neonate, the metaphyseal vessels penetrate directly into the chondroepiphysis, allowing an infection in the metaphysis to readily invade and destroy the chondroepiphysis and subsequently invade the joint. **Bottom,** In the older child, the physis serves as a mechanical barrier to the spread of infection.

destruction of the chondroepiphysis, physis, and joint may occur if these structures are involved. With early diagnosis and treatment (in the absence of epiphyseal and joint involvement), the prognosis is usually quite good.

Table 2
Comparison of Acute and Subacute Hematogenous Osteomyelitis

	Subacute	Acute
Pain	Mild	Severe
Fever	Few patients	Majority of patients
Loss of function	Minimal	Marked
Prior antibiotic therapy	Often (30%-40% of patients)[15]	Occasionally
Elevated WBC count	Few	Majority of patients
Elevated ESR	Majority of patients	Majority of patients
Blood cultures	Few positive	50% positive[8]
Bone cultures	60% positive	85% positive
Initial radiographs	Frequently abnormal	Often normal
Site	Any location (may cross physis)	Usually metaphysis

Subacute Hematogenous Osteomyelitis

Subacute hematogenous osteomyelitis is most likely due to an altered host-pathogen relationship combined with increased host resistance, decreased virulence of the causative organism, and/or antibiotic modification. In a retrospective study of subacute hematogenous osteomyelitis in children, Roberts et al[22] found that antibiotics had been given to 40% of patients for infections other than osteomyelitis. As a result, the children appeared less ill and displayed less toxicity than children with acute osteomyelitis, frequently presenting with a limp but without the features of "typical osteomyelitis." Because children with subacute osteomyelitis appear less ill, they frequently present later in the course of their disease.

The features of acute and subacute hematogenous osteomyelitis are compared in Table 2. Most cases of subacute hematogenous osteomyelitis are due to *Staphylococcus* organisms, but recently *Streptococcus* organisms have been found.[22-24] Often, no organism is isolated. Subacute hematogenous osteomyelitis may be confused with neoplasms such as Ewing's sarcoma and osteoid osteoma. Pain is the most consistent symptom, and constitutional symptoms are usually mild. The ESR, however, is often elevated.

Radiographic bone lesions, which may be difficult to differentiate from lesions due to other diseases, are commonly seen. Gledhill and McIntyre[23] described four radiographic types. Type I is a solitary localized zone of radiolucency surrounded by a reactive zone suggestive of eosinophilic granuloma or Brodie's abscess. Type II is a metaphyseal lesion associated with loss of cortical bone. Type III is a diaphyseal lesion with excessive cortical reaction. Type IV is a lesion associated with onionskin layering of subperiosteal bone. This classification has been modified to include other variants of infections (Fig. 3).[22]

In patients older than 18 months of age, AHO rarely crosses the physis; however, subacute osteomyelitis frequently does cross the physis. There is rarely permanent damage to the growth plate in these patients.[7]

When the cortex is destroyed (Gledhill type II) or when there is extensive cortical reaction (Gledhill type IV), neoplasms such as eosinophilic granuloma, Ewing's sarcoma, and osteogenic sarcoma should be considered in the differential diagnosis. In as many as 50% of cases, subacute osteomyelitis is confused with tumor.[22] A biopsy is usually needed for definitive diagnosis. Antibiotics are given postoperatively and were used for 6 weeks in the series of Gledhill and McIntyre.[23] In cases in which extensive destruction is seen, postoperative cast immobilization should be considered to prevent fractures.

Brodie's Abscess

Since Brodie described localized metaphyseal tibial abscesses without any associated systemic illness in 1832, these lesions have been known as Brodie's abscesses. Brodie's abscess may be thought of as a form of subacute pyogenic osteomyelitis. Most Brodie's abscesses occur in the metaphysis (Gledhill type I lesions) and usually respond well to surgical debridement and postoperative antibiotics.

Primary Epiphyseal Osteomyelitis

Epiphyseal osteomyelitis may be either acute or subacute. Primary subacute epiphyseal osteomyelitis was described by King and Mayo[24] in 1969 and can be thought of as a Brodie's abscess of the epiphysis. Subacute osteomyelitis appears to act the same whether it occurs in the epiphysis or the metaphysis. Most

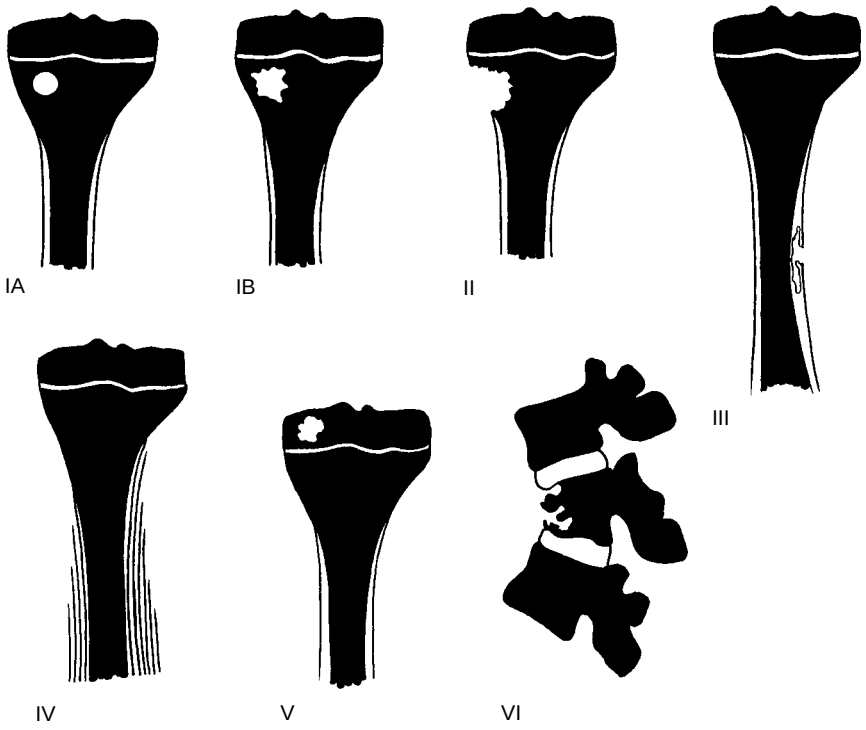

Fig. 3 Modified classification of subacute osteomyelitis.22 Type IA is characterized by a punched-out radiolucency suggestive of eosinophilic granuloma. Type IB is similar but has a sclerotic margin and represents a classic Brodie's abscess. Type II is a metaphyseal lesion associated with loss of cortical bone. Type III is a diaphyseal lesion with excessive cortical reaction. Type IV is a lesion associated with onionskin layering of subperiosteal bone. Type V is a concentric epiphyseal radiolucency. Type VI is an osteomyelitic lesion of a vertebral body.

authors have found no evidence of invasion of subacute epiphyseal osteomyelitis into the metaphysis.[22,24,25] While most cases of epiphyseal osteomyelitis are subacute, acute cases have been reported.

A biopsy is usually needed for diagnostic purposes and for differentiation of the lesions from neoplastic lesions, including osteoid osteoma. Most often, no causative organism is found, but when one is isolated, it is usually *S aureus*.[25] At surgery, granulation tissue is usually found instead of pus.[25] Surgical debridement followed by intravenous and oral administration of antibiotics is indicated. Some lesions will heal with antibiotics alone.[22]

Summary

Although typical AHO in children is common, more subtle presentations (e.g., subacute osteomyelitis, Brodie's abscess, subacute epiphyseal osteomyelitis, chronic recurrent multifocal osteomyelitis, neonatal osteomyelitis, and infections simulating neoplasm) appear more frequently. Wider recognition of these less common varieties, advances in imaging techniques (e.g., technetium bone scanning and MR imaging), and a better understanding of the pathophysiology of these infections as it relates to treatment have contributed to improved management of these conditions.

Acknowledgments: The authors wish to thank Lou Bell, MD, and Elizabeth Torg for their help in preparing this manuscript.

References

1. Gilmour WN: Acute haematogenous osteomyelitis. *J Bone Joint Surg Br* 1962;44:841-853.
2. McHenry MC, Alfidi RJ, Wilde AH, et al: Hematogenous osteomyelitis: A changing disease. *Cleve Clin Q* 1975; 42:125-153.
3. Morrey BF, Peterson HA: Hematogenous pyogenic osteomyelitis in children. *Orthop Clin North Am* 1975;6(4):935-951.
4. Morrissy RT: Bone and joint infections, in Morrissy RT (ed): *Lovell and Winter's Pediatric Orthopaedics*, 3rd ed. Philadelphia: Lippincott, 1990, vol 1, pp 539-561.
5. Whalen JL, Fitzgerald RH Jr, Morrissy RT: A histological study of acute hematogenous osteomyelitis following physeal injuries in rabbits. *J Bone Joint Surg Am* 1988;70:1383-1392.
6. Hobo T: Zur Pathogenese der akuten haematogenen Osteomyelitis, mit Berücksichtigung der Vitalfärbungslehre. *Acta Scholae Med Kioto* 1921;4: 1-29.
7. Green NE, Edwards K: Bone and joint infections in children. *Orthop Clin North Am* 1987;18(4):555-576.
8. Jackson MA, Nelson JD: Etiology and medical management of acute suppurative bone and joint infections in pediatric patients. *J Pediatr Orthop* 1982; 2:313-323.
9. Scott RJ, Christofersen MR, Robertson WW Jr, et al: Acute osteomyelitis in children: A review of 116 cases. *J Pediatr Orthop* 1990;10:649-652.
10. Unkila-Kallio L, Kallio MJT, Eskola J, et al: Serum C-reactive protein, erythrocyte sedimentation rate, and white blood cell count in acute hematogenous osteomyelitis of children. *Pediatrics* 1994;93:59-62.
11. Herndon WA, Alexieva BT, Schwindt ML, et al: Nuclear imaging for muscu-

loskeletal infections in children. *J Pediatr Orthop* 1985;5:343-347.
12. Wegener WA, Alavi A: Diagnostic imaging of musculoskeletal infection: Roentgenography; gallium, indium-labeled white blood cell, gammaglobulin, bone scintigraphy; and MRI. *Orthop Clin North Am* 1991;22(3): 401-418.
13. Canale ST, Harkness RM, Thomas PA, et al: Does aspiration of bones and joints affect results of later bone scanning? *J Pediatr Orthop* 1985;5:23-26.
14. Modic MT, Feiglin DH, Piraino DW, et al: Vertebral osteomyelitis: Assessment using MR. *Radiology* 1985;157:157-166.
15. Unger E, Moldofsky P, Gatenby R, et al: Diagnosis of osteomyelitis by MR imaging. *AJR Am J Roentgenol* 1988;150: 605-610.
16. Cohen MD, Cory DA, Kleiman M, et al: Magnetic resonance differentiation of acute and chronic osteomyelitis in children. *Clin Radiol* 1990;41:53-56.
17. Daoud A, Saighi-Bouaouina A: Treatment of sequestra, pseudarthroses, and defects in the long bones of children who have chronic hematogenous osteomyelitis. *J Bone Joint Surg Am* 1989;71:1448-1468.
18. Cole WG, Dalziel RE, Leitl S: Treatment of acute osteomyelitis in childhood. *J Bone Joint Surg Br* 1982; 64:218-223.
19. O'Brien T, McManus F, MacAuley PH, et al: Acute haematogenous osteomyelitis. *J Bone Joint Surg Br* 1982;64: 450-453.
20. Ogden JA, Lister G: The pathology of neonatal osteomyelitis. *Pediatrics* 1975;55:474-478.
21. Edwards MS, Baker CJ, Wagner ML, et al: An etiologic shift in infantile osteomyelitis: The emergence of the group B streptococcus. *J Pediatr* 1978;93:578-583.
22. Roberts JM, Drummond DS, Breed AL, et al: Subacute hematogenous osteomyelitis in children: A retrospective study. *J Pediatr Orthop* 1982;2: 249-254.
23. Gledhill RB, McIntyre JM: Various phases of pediatric osteomyelitis. *Instr Course Lect* 1973;22:245-269.
24. King DM, Mayo KM: Subacute haematogenous osteomyelitis. *J Bone Joint Surg Br* 1969;51:458-463.
25. Green NE, Beauchamp RD, Griffin PP: Primary subacute epiphyseal osteomyelitis. *J Bone Joint Surg Am* 1981; 63:107-114.