CHAPTER 22
Haematogenous Osteomyelitis and Septic Arthritis

Donald E. Meier
Bankole S. Rouma

Haematogenous Osteomyelitis

Haematogenous osteomyelitis (HO) is a common and devastating problem for children in less developed areas of the world[1] due to its frequent association with sickle cell disease (23–44%), delayed presentation, misdiagnosis, and undertreatment.[1–5] Sixty to eighty percent of children do not initially present until they have reached the stage of chronic osteomyelitis.[1–4] In more medically advanced areas, the spectrum of HO has changed significantly in the past few decades with decreased prevalence, earlier presentation, better nourished children, increased awareness of HO, improved diagnostic modalities for confirmation, precise laboratory techniques for microbial identification, and advanced antimicrobial agents for successful eradication of the infection. In locations without advanced technology (LWATs), however, little has changed in the past half century in the presentation or management of children with HO. Most children in advanced areas undergo successful *nonoperative* eradication of HO, many with nothing more invasive than a blood culture or bone aspiration.[6–8] Most current Western literature concerning HO—which concentrates on whether diagnostic aspiration of the bone marrow is really necessary and whether one powerful antibiotic is better than another in the eradication of HO—therefore has little relevance to practitioners in LWATs who are fortunate if they have materials for a gram stain and enough antibiotics for a week of oral treatment before the family runs out of funds. The purpose of this chapter is to provide a functional and practical approach to the classification and treatment of HO in children, taking into consideration the economic and technologic restraints that are inherent in any medical practice in LWATs.

Demographics

The exact incidence of osteomyelitis in Africa is unknown, but in reported series, children with osteomyelitis represent 7–20% of hospitalised children.[1–3] Data from the United Kingdom have shown an annual incidence of 2.9 per 100,000,[9] with boys affected more than girls, and half of osteomyelitis cases occurring in the first 5 years of life. In Africa, 56% of affected children are between 8 and 11 years of age.[1] In Cote d'Ivoire, the median age is 7.2 years.[2]

Aetiology/Pathology

Haematogenous osteomyelitis begins with entry of bacteria through a break in the skin or mucosa from otitis, pharyngitis, respiratory tract infections, or urinary tract infections. Most often the bacteria are *Staphylococcus*, but in sickle-cell children, both *Salmonella* and *Staphylococcus* are implicated. The bacteria are haematogenously disseminated and deposited in the trabecular bone or marrow, usually in the metaphysis of the proximal tibia or distal femur (Figure 22.1(A)). Sluggish blood flow in the metaphysis provides an ideal milieu for bacterial replication. Increasing pressure from the progressive, intramedullary purulent process results in destruction of the endosteal blood supply to the cortex. The pus under pressure escapes outward through Volkmann and Haversian canals (Figure 22.1(B)) and then spreads subperiosteally, stripping the cortex of its periosteal blood supply (Figure 22.1(C)). Without either endosteal or periosteal blood supply, the cortex becomes nonviable bone called sequestrum (Figure 22.1(D)). As the exudate

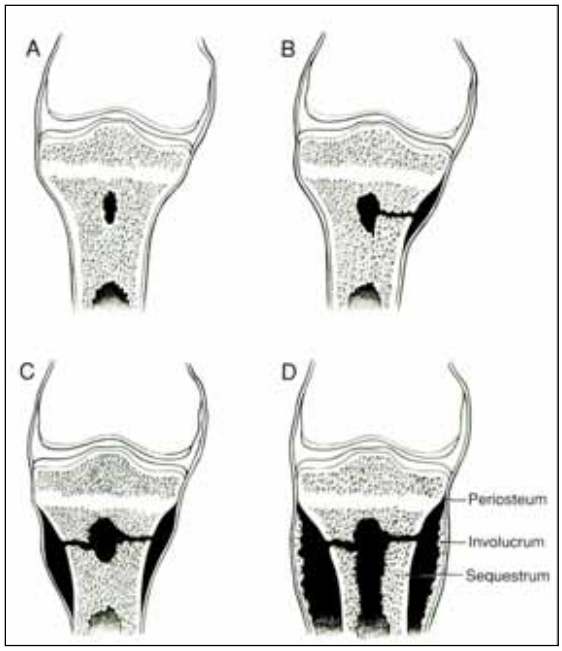

Figure 22.1: Pathologic development of haematogenous osteomyelitis.

increases in volume, it takes the path of least resistance. Sometimes, this involves circumferential stripping of the periosteum from one metaphysis to the other, resulting in a giant sequestrum consisting of the diaphysis and both metaphyses. At other times, the periosteum perforates under pressure, resulting in the spread of pus into muscles and along fascial planes. At this point, it is often confused with primary pyomyositis, and the bony origin is overlooked, resulting in inadequate decompression of the medullary canal. As the devascularised cortex is being absorbed, the inner surface of the periosteum produces new bone, called involucrum. The multiple areas of subperiosteal involucrum formation coalesce to form new bone on the inner surface of the periosteum but on the outside of the sequestrum. If the sequestrum totally resorbs, the patient may recover without a problem, but this rarely occurs. The nonviable, unresorbed sequestrum usually serves as a nidus for recurrent abscesses or chronically draining sinuses.

Clinical Presentation

Children may present with symptoms and signs of systemic sepsis, including fever, irritability, lethargy, or convulsions. Local symptoms include pain over the affected bone, which can be acute and overwhelming or insidious in onset, and unwillingness to use the affected extremity. The history may include a recent episode of impetigo, otitis, pharyngitis, or respiratory infection. Characteristically, there is tenderness, swelling, redness, or shininess over the affected bone. The adjacent joint usually appears normal.

Most laboratory values are nonspecific. The white blood count (WBC) may be elevated or normal. The erythrocyte sedimentation

rate (ESR) is elevated in 80–90% of cases, and the C-reactive protein (CRP) is elevated in 98% of cases. Blood cultures are often positive in children presenting with systemic sepsis. None of these tests, however, are specific for HO, and therefore must be used only to supplement the history and physical examination. Indeed, many African hospitals will not have these laboratory tests available. The most accurate method for determining whether osteomyelitis is present is a bone marrow aspiration with stains and cultures to determine the infecting organism.

Plain radiography, early in the clinical course, may show soft tissue swelling and obliteration of tissue planes. After 10–14 days from onset of the symptoms, bone resorption is demonstrated by irregular patches of radiolucency in the metaphysis, and periosteal elevation is demonstrated by an outside rim of reactive new bone formation. In chronic osteomyelitis, plain radiographs may demonstrate lytic areas of the bone, sequestrum formation, or pathologic fractures. The TA technetium 99m bone scan is a sensitive (84–100%) and specific (70–90%) test for acute osteomyelitis, and magnetic resonance imaging (MRI) is the best special imaging study. However these studies are not available in most African health care facilities.

Classification

Traditionally, the stages of HO have been described as: (1) acute, (2) chronic, and (3) subacute. This traditional classification, however, is not very practical for use in LWATs. As a result, African practitioners have developed alternative classification systems.[10–11] The classification system shown in Table 22.1 was developed in a Nigerian general medical practice hospital in 1993.[12]

This simplified and functional system classifies children at the time of initial diagnosis of HO into one of four stages based on symptoms, signs, and x-ray findings. In stage 1 HO (acute), there is pus in the medullary canal and perhaps subperiosteally. There are usually local and systemic signs, but no significant x-ray changes that would demonstrate bone destruction or the presence of sequestra. Bone destruction and sequestra formation do not usually result in significant x-ray changes for at least 2 weeks into the HO process. Therefore, stage 2 HO (acute with x-ray changes) begins around 2 weeks into the process and indicates that significant bone destruction has already taken place. Children with stage 3 HO (chronic localised) have usually suffered an acute bout of HO that has drained spontaneously or has been operatively drained at a health care facility. However the sequestrum, which has not resorbed nor been surgically removed, serves as a nidus for chronic draining sinuses or recurrent abscesses (Figure 22.2). If a significant abscess occurs around the sequestrum and does not spontaneously drain, the child becomes systemically septic and reaches stage 4 HO (chronic systemic). This Nigerian staging system will be used throughout this chapter due to its practicality in areas with limited diagnostic and therapeutic resources.

Differential Diagnosis

Bone infarction can be difficult to differentiate from infection in a child with sickle cell disease. In both situations, children present with fever and bone pain and have elevated inflammatory markers. Biopsy and culture of affected bone is often necessary to establish the diagnosis. Cellulitis of soft tissues may restrict movement and cause the child to limp, but in most cases swelling and erythema of the skin are obvious. A fracture also causes swelling, pain, tenderness, and increased warmth of an extremity and may be differentiated from HO only by the history (although most children with HO also have a history of trauma) and more definitively by an x-ray. Neoplasms, especially leukaemia and Ewing's sarcoma, may be confused with osteomyelitis and may require a biopsy for the correct diagnosis.

Treatment

All African hospitals are not equal. Many, particularly university hospitals in major African cities, have state-of-the-art facilities comparable to those in Western hospitals. As a result, in African hospitals with advanced technology, treatment for children with osteomyelitis is similar to that in Western hospitals. Early acute osteomyelitis is managed nonoperatively with a culture of the medullary contents and prolonged organism-appropriate intravenous antibiotics until the infection has been totally eradicated. Many other hospitals in Africa, however, are resource poor, and this section on treatment is directed more towards these hospitals.

Appropriate treatment, particularly in LWATs, depends on the stage of HO when the child presents and the resources (antibiotics, operative capabilities) physically and economically accessible in the particular location where the child presents. The best microbiological study available may be a gram stain. Surgical instruments, if available at all, are usually quite basic. Appropriate antistaphylococcal antibiotics may be totally unavailable or may be so expensive that parents are faced with the difficult decision of providing antibiotics for one child for a week or feeding the rest of the children in the family for the next several months. Health providers must therefore consider these painfully realistic socioeconomic factors in recommending appropriate practical treatment for a particular child with HO. The basic components of optimal treatment for HO are: (1) drainage of the pus under pressure, (2) acute antibiotics to treat systemic sepsis, (3) removal of nonviable bone, (4) sterilisation of the medullary contents with local or systemic techniques, and (5) wound closure. Unlike the current nonoperative treatment of HO in locations with advanced technology, operation is

Table 22.1: Classification and treatment system for haematogenous osteomyelitis in developing world children.

Classification	Characteristics	Treatment
Stage 1 **Acute**	Local and systemic signs. No bone changes on x-ray (less than 2-week history).	Incision and drainage. Antibiotics for 2–6 weeks.
Stage 2 **Acute with x-ray changes**	Undrained acute osteomyelitis (2–8 week history). Local and maybe systemic signs with bone destruction on x-ray and no clear sequestrum.	Surgical drainage and debridement of obviously dead bone only. Perioperative antibiotics.
Stage 3 **Chronic localised**	Long history of osteomyelitis, usually with persistent spontaneous drainage. No systemic symptoms.	Wide drainage and removal of sequestra. Antibiotics not required.
Stage 4 **Chronic systemic**	Chronic osteomyelitis with systemic manifestations.	Urgent wide drainage, removal of sequestra, and administration of antibiotics until systemic manifestations resolve.

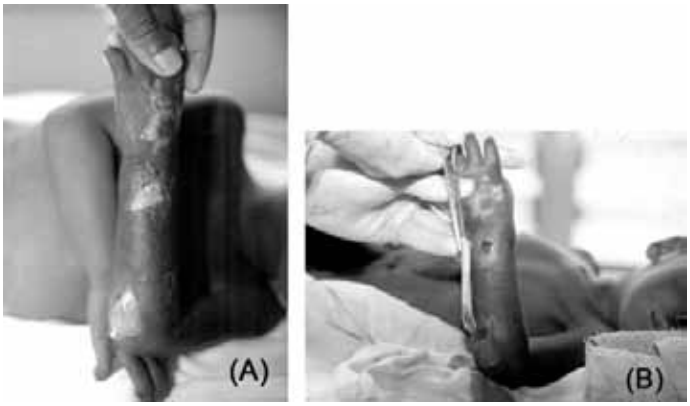

Figure 22.2: Neglected HO resulting in (A) chronic draining sinuses secondary to (B) a large sequestrum.

the essential modality in the treatment of all stages of osteomyelitis in LWATs.

Stage 1 HO is the most important stage for expeditious treatment, which may result in cure of the acute process and also prevent progression to chronic osteomyelitis. In acute HO, the operative goal is to decompress all pus under pressure. This involves decompression of both subperiosteal and intramedullary pus. Decompression must be adequate enough to prevent reaccumulation. Antibiotics are highly recommended in the initial treatment of stage 1 HO to manage the systemic sepsis. Antibiotics are also recommended for a 2–6 week period of treatment to prevent progression to chronic osteomyelitis.

Whenever HO has progressed to stage 2, x-ray changes already indicate nonviable bone while systemic and local sepsis remains. Treatment for stage 2 also involves prompt decompression and antibiotics to treat the systemic sepsis. Usually, the sequestrum is not well developed, and extensive debridement should be avoided because it is very difficult to differentiate viable from nonviable bone at this time. There is no documentation that prolonged antibiotics in stage 2 will prevent progression to chronic HO, but if antibiotics are available, they should be used for at least 2 weeks after resolution of the acute process.

Most children in LWATs present in stage 3 or 4. When HO has reached stage 3, antibiotics have little role in treatment. An x-ray serves as a "road map" (Figure 22.3) and is essential in planning proper treatment for both stages 3 and 4. With adequate debridement of sequestra, there is a chance of eventual healing of stage 3 HO even without antibiotics (Figures 22.4 and 22.5). The treatment for stage 4 HO (Figure 22.6) differs from the treatment for stage 3 only in the need for short-term antibiotics until the systemic sepsis has been controlled. Prolonged systemic antibiotic administration after total debridement of sequestra has not been proven effective in preventing further episodes of chronic HO.

Operative Techniques

This section considers in more detail the operative techniques used in the treatment of all stages of HO. An optimal basic instrument tray consists of the following instruments: soft tissue basic instruments (haemostat, scalpel, tissue forceps, needle holder, scissors); soft tissue

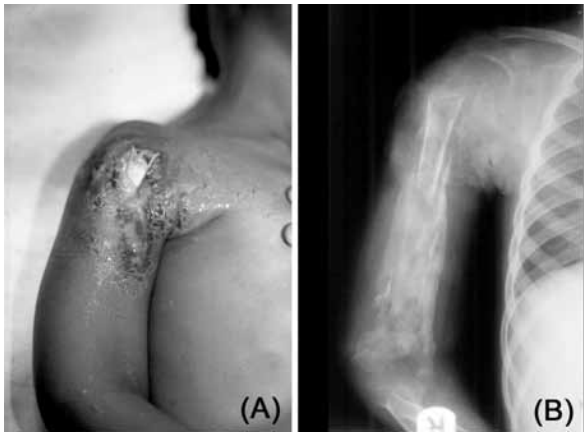

Figure 22.3: Stage 3 HO of the humerus in a 5-year-old child with (A) spontaneous extruding sequestrum, as seen on (B) a "road map" x-ray.

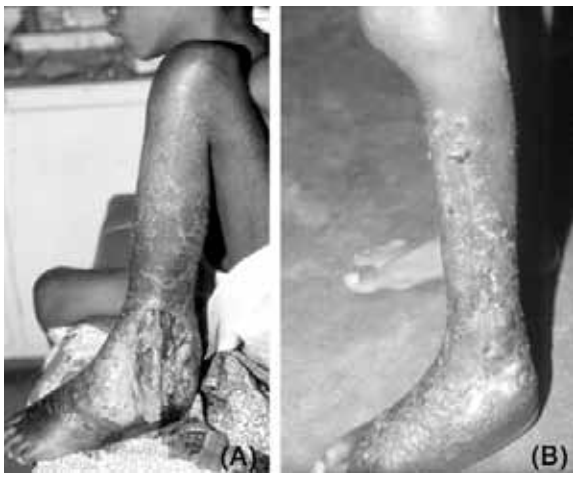

Figure 22.4: Stage 3 HO of the fibula with (A) spontaneous extrusion of the sequestrum. (B) After sequestrectomy, complete healing is achieved without antibiotics.

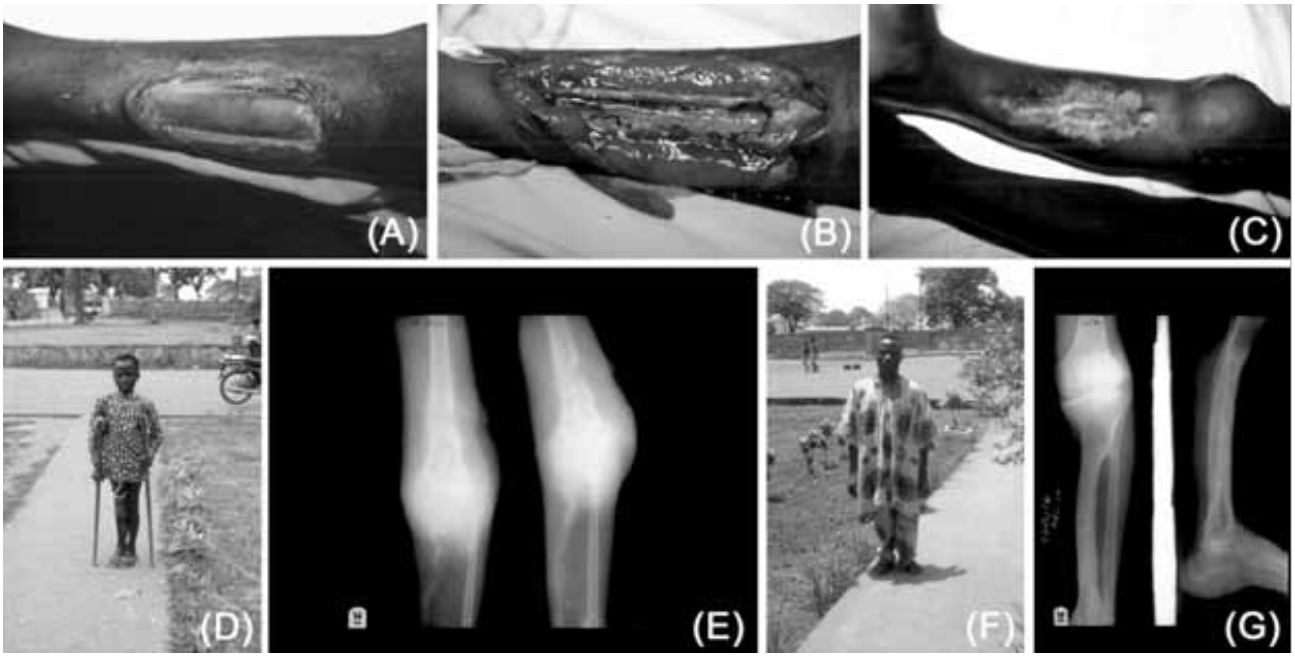

Figure 22.5: Stage 3 HO of the tibia in a 7-year-old male: (A) initial presentation; (B) tibia after removing a cortical trough, which showed that the tibial shaft was completely nonviable; (C) appearance after giant sequestrectomy and healing by secondary intention without using antibiotics; (D) child at time of discharge; (E) x-ray at time of discharge; (F) same patient 15 years later; (G) x-ray 15 years later.

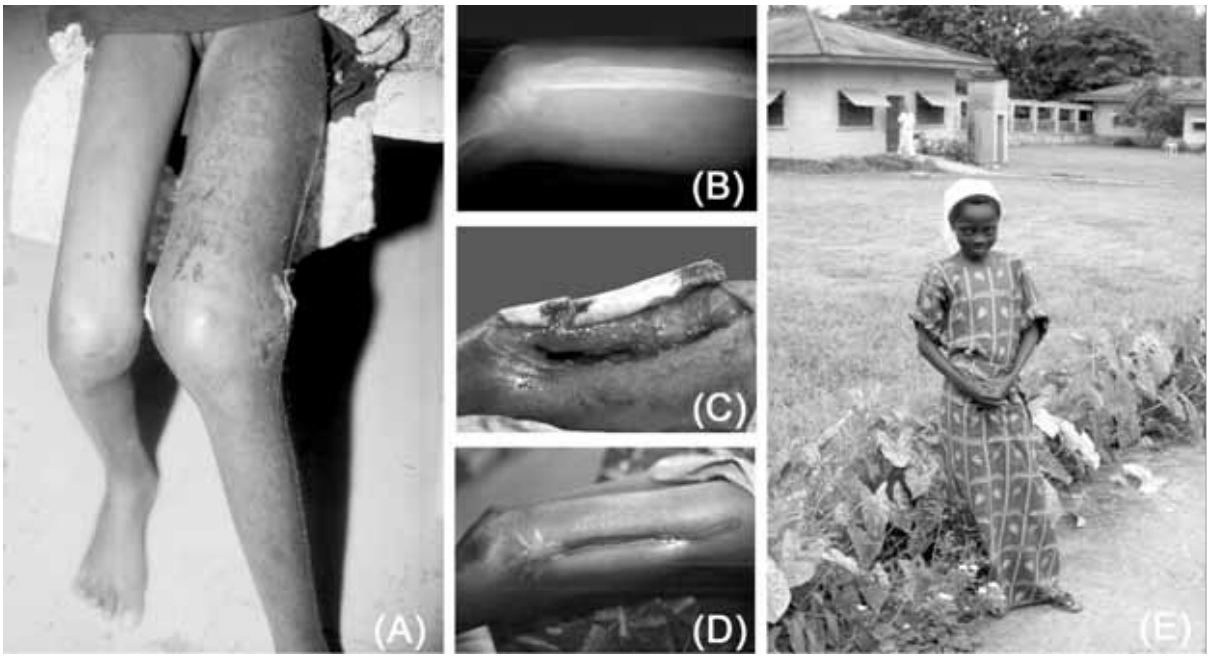

Figure 22.6: Stage 4 HO in a 12-year-old female with sickle-cell (SS) haemoglobin: (A) initial presentation with large abscess of left thigh, (B) x-ray showing giant sequestrum of femur, (C) drainage of abscess and sequestrectomy performed through a lateral thigh incision, (D) thigh wound, (E) child after complete healing by secondary intention with antibiotics used only to control the systemic sepsis.

retractors (self-retaining Gelpis are ideal); periosteal elevator; bone curette; bone rongeur; and bone drill. True orthopaedic bone drills are very expensive and justification of the cost is difficult for hospitals in LWATs. However, simple carpenter drills and bits can be used for orthopaedic purposes if proper sterilisation capabilities (ethylene oxide or formalin gas) are available. Cordless electric drills, commercially available in hardware stores, are relatively inexpensive and also can be effectively used for orthopaedic procedures if properly sterilised. They must, however, be used on a low speed because a high-speed mode will burn the bone. An orthopaedic exposure book[13] is a valuable asset in determining the safest approach for draining and debriding bones affected by HO. The cost of such books is prohibitive in most LWATs, however, and a basic anatomy book can be substituted to determine appropriate approaches to bones and joints in the least potentially destructive manner. The low-cost *Primary Surgery* textbook[14] presents good exposure techniques for the more commonly affected bones and joints. Ketamine anaesthesia is a very effective and safe technique in the operative management of children with HO in LWATs.[15] Using an extremity tourniquet significantly decreases the operative blood loss, but tourniquets should not be used in children with SS or SC haemoglobinopathies because this may precipitate a sickling crisis.

Treatment of acute HO (stages 1 and 2) begins with the soft tissue approach to the bone. The recommended approach to the proximal tibia (the most commonly affected bone) is from the medial or lateral aspect of the tibia so that there will be soft tissue remaining to cover the affected bone. For the health care provider unaccustomed to approaching the tibia in this manner, however, it is acceptable to incise the soft tissue directly over the tibia with as small a soft tissue incision as necessary. Usually the periosteum has already been elevated from the bone and needs to be incised longitudinally to drain the pus under pressure. If microbiological techniques (gram stain, culture) are available, a sample is taken. A periosteal elevator should not be used for this classic presentation because the increasing subperiosteal pressure has already stripped the periosteum from the cortex, and further periosteal elevation may impair blood flow to the remaining bone. After the periosteum is incised, a drill is used to enter the metaphyseal medullary canal. Usually pus drains from the drill hole. If so, other drill holes are placed in the area and a curette and bone rongeur are used to remove a 2-cm cortical window. This window serves to decompress the medullary canal and allows for irrigation of the canal. The medullary canal in acute HO should not be curetted for fear of damaging the precarious endosteal blood supply. The wound is left open and the patient brought back daily for irrigation of the medullary canal using ketamine anaesthesia. When there is no more purulent drainage, an attempt can be made to close the incision (this is often unsuccessful), or it can simply be left open to heal by secondary intention.

The treatment of chronic HO (stages 3 and 4) often requires a more extensive operative approach. There is rarely a total cure for chronic HO, but very long periods of remission can be achieved if all of the nonviable bone is removed. Sometimes the child with chronic HO has been neglected for so long that the sequestrum begins to spontaneously extrude (see Figures 22.3 and 22.4). When this happens, the child can be appropriately treated by simply removing the sequestrum, curetting the inner surface of the involucrum, and irrigating the medullary canal to remove any remaining smaller pieces of the sequestrum. Sometimes the sequestra are incarcerated by the involucrum, and removal requires a cortical trough to adequately visualise and remove all of the sequestra (Figure 22.7(A)). After the removal of the sequestra, advanced techniques for closure are available, including muscle and fascio-cutaneous flaps. Placement of antibiotic-impregnated beads can be used to decrease the number of relapses for chronic HO. Most of these advanced procedures are not commonly used in LWATs since in such locations the incidence of HO is so common as to be overwhelming for the resources of the hospital. In these instances, the large wounds can be left completely open, and they eventually will heal by secondary intention as long as all of the nonviable bone has been removed (Figure 22.7(B)).

Parents can manage the wounds with daily water irrigation and coverage with a bandage made from scrap cloth. In hospitals with adequate health care personnel and facilities, the wounds can be managed in a wound care clinic, but hospitals without such facilities can provide alternatives. For example, the Baptist Medical Center in Ogbomoso, Nigeria, provides a water hose so each day children and parents can use the handheld shower apparatus to wash any debris

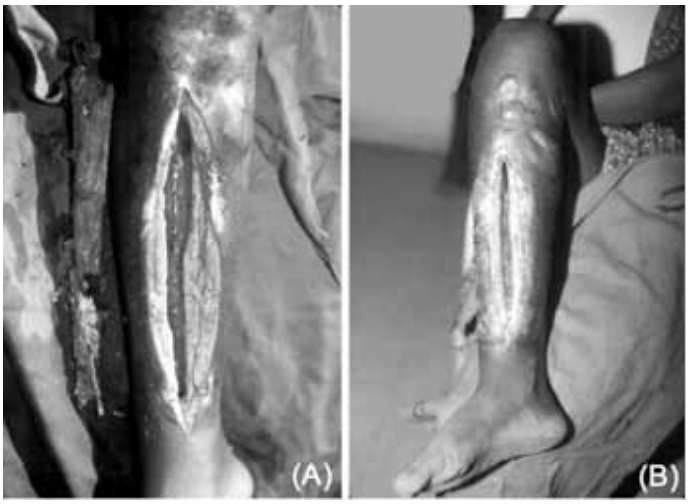

Figure 22.7: Stage 3 chronic HO of the tibia: (A) extensive sequestrectomy performed through a long cortical trough; (B) wound healing by secondary intention without antibiotics.

out of the cortical trough. It is not painful for the children, and the wound can be managed solely by a parent without using the services of hospital personnel.

The proper procedure is controversial for management of chronic osteomyelitis when the total bone from metaphysis to metaphysis has sequestered and there is not yet enough new involucrum to provide stability to the bone. Some surgeons prefer to proceed with removal of the giant sequestrum and splinting of the extremity to allow the involucrum to grow in a clean environment without the infected sequestrum interfering (Figure 22.5). Other practitioners believe that the best splint for the affected extremity is the sequestrum itself and that it should be left in situ until the involucrum has coalesced. There are obviously no prospective randomised studies to support either course of action.

Complications

Risk factors for development of complications of HO include delay in diagnosis, misdiagnosis, short duration of therapy, and a younger age at the time of initial illness. Recurrent bone infection is the most common complication after treatment for osteomyelitis followed by disturbance in bone growth, limb-length discrepancies, axial displacement of the limb, pathologic fractures, and abnormal gait.

Septic Arthritis

Pathogenesis

Although septic arthritis can be caused by joint trauma or extension of osteomyelitis into a joint, the most common aetiology in African children is haematogenous dissemination of *Staphylococcus* from an open skin or mucosal wound. Other offending organisms include *Streptococcus*, *Haemophilus influenza* (particularly in newborns), and *Salmonella* and *Escherichia coli* in sickle-cell children. Bacteria have an affinity for cartilage and directly attach to the chondral surface. An acute inflammatory response follows, resulting in migration of polymorphonuclear cells, production of proteolytic enzymes, and cytokine secretion by chondrocytes. Degradation of articular cartilage begins within 8 hours of onset of infection. The most commonly infected joints are the knee (41%), hip (20%), ankle (14%), elbow (12%), wrist (4%), and shoulder (4%).

Clinical Presentation

Any child with fever and reluctance to move an extremity should be considered to have osteomyelitis or septic arthritis until proven otherwise. The history should include any factors that may make the child more susceptible to the development of bacteraemia: recent systemic illness (chicken pox), respiratory or urinary infections, otitis media, indwelling intravenous catheters, immunosuppressive disorders, or sickle cell disease.[16] There is often the history of a traumatic event preceding the pain.

On examination, there is usually swelling and warmth over the joint, pain with mobilisation of the joint, and restricted range of motion of the joint. Tenderness over the metaphysis of a bone is more characteristic of osteomyelitis, whereas tenderness directly over a joint or pain with slight movement of a joint is characteristic of septic arthritis. The patient should also be evaluated for pharyngitis, rash, heart murmur, hepatosplenomegaly, and evidence of involvement of other bones or joints.

Diagnosis

Diagnosis must be made promptly to prevent damage to the articular cartilage. Blood and joint fluid should be obtained for cultures, and a gram stain and cell count should be performed on the joint fluid. A WBC count of 50,000/cu mm or greater with a predominance of polymorphonuclear cells is consistent with bacterial infection. Plain radiographs may show joint space widening, effusion, soft tissue swelling, or subluxation/dislocation of the joint. Radiographs are useful to rule out fracture, malignancy, or osteomyelitis as the cause of pain. Ultrasound is useful in determining whether fluid is present in the joint and is useful in guiding aspiration, but it cannot differentiate infected from noninfected fluid. The definitive diagnosis is made by either joint aspiration or operative identification of a purulent effusion.

Treatment

The three main therapeutic interventions are: (1) joint decompression and debridement, (2) antibiotics and initial joint immobilisation to decrease local irritation, followed by (3) mobilisation to decrease the development of fibrous adhesions and improve cartilage nutrition. Intravenous antibiotics (choice depends on availability in a given locale) should be started after the arthrocentesis and continued for 1–2 weeks, after which oral antibiotics are continued for another 2–6 weeks. Septic arthritis is a surgical emergency because prolonged elevated intracapsular pressure in the hip can tamponade blood flow to the femoral head and increase the possibility of developing avascular necrosis. A safe anatomical approach to the joint should be conducted.[13,14] The joint is opened and the pus drained. The joint is irrigated copiously with normal saline. After the effluent is clear, the joint is digitally palpated to determine how much of the cartilage has already been destroyed. If it is a superficial joint (knee), a drain is not necessary. For deeper joints (hip, shoulder), however, a Penrose or glove drain can be inserted to maintain the drainage tract between irrigations. The patient is placed at joint rest for at least 2 weeks. The joint undergoes repeat irrigation daily under anaesthesia until there is no more purulent drainage. It is important, particularly in joints with extensive cartilage destruction, that the joint be placed in a functional position because otherwise ankylosis may occur. If ankylosis does occur, reconstructive surgery will probably not be available in LWATs, and even if available, it will not be nearly as effective in providing function as would be a programme of splinting joints in a position of function before ankylosis. After the period of posterior plaster immobilisation, the joint is progressively mobilised to minimise ankylosis.

Key Summary Points

1. Haematogenous osteomyelitis (HO) is a common and devastating problem for children in developing countries.
2. Operation is the mainstay of treatment for HO in developing world children.
3. Acute stages of HO are best managed with subperiosteal and intramedullary decompression and antibiotics.
4. Chronic stages are best treated with extensive debridement to remove all sequestra, with antibiotics used to treat systemic sepsis.
5. In the absence of discharging sinuses the diagnosis of chronic osteomyelitis should be confirmed or excluded by biopsy.
6. Septic arthritis should be diagnosed and treated as an emergency because the longer intraarticular pus remains under pressure, the more likely there will be permanent destruction.
7. The African surgeon must be acquainted with a safe operative approach to all bones and joints commonly affected with osteomyelitis and pyarthrosis.

References

1. Tekou H, Foly A, Akue B. Oe profil actuel des osteomyelites hematogenes de l'enfant au Centre Hospitalier de Toioin, Lome, Togo a propos de 145 cas. Medecine Tropicale 2000; 60(4):365–368.
2. Kouame DB, Dick KR, Ouattara O, Gouli JC, Odehouri KT, Coulibaly C. Traitement des osteomyelites compliquees de l'enfant au CHU de Yopougon, Abidjan (Cote d'Ivoire). Cahiers Sante 2005; 15(2):99–104.
3. Nacoulma SI, Ouedraogo DD, Nacoulma EWC, Korsaga A, Drabo JY. Osteomyelite chroniques au CHU de Ouagadougou (Burkina Faso). Etude retrospective de 102 cas (1996-2000). Bull Soc Pathol Exot 2007; 100(4):264–268.
4. Oguachuba H. Mismanagement of acute haematogenous osteomyelitis by traditional medicine men (native doctors) in the Eastern and Northern Regions of Nigeria. Unfallchirurg 1985; 88:363–372.
5. Wong AL, Sakamoto KM, Johson EE. Differentiating osteomyelitis from bone infarction in sickle cell disease. Ped Emer Care 2001; 17(1):60–63.
6. Song KM, Sloboda JF. Acute haematogenous osteomyelitis in children. J Am Acad Orthop Surg 2001; 9(3);166–175.
7. Stanitski CL. Changes in pediatric acute haematogenous osteomyelitis management. J Pediatr Orthop 2004; 24(4):444–445.
8. Darville T, Jacobs RF. Management of acute haematogenous osteomyelitis in children. Pediatr Infect Dis J 2004; 23(3):255–258.
9. Marietta V. Osteomyelitis in children. Current Opinion in Ped 2002; 14:112–115.
10. Solagberu BA. A new classification of osteomyelitis for developing countries. East African Medical Journal 2003; 80(7):373–378.
11. Lauschke FH, Frey CT. Haematogenous osteomyelitis in infants and children in the northwestern region of Namibia: management and two-year results. J Bone Joint Surg Am 1994; 76(4):502–510.
12. Meier DE, Tarpley JL, OlaOlorun DA, Howard CR, Price CT. Haematogenous osteomyelitis in the developing world: a practical approach to classification and treatment with limited resources. Contemp Orthopaed 1993; 26(5):495–502.
13. Hoppenfeld S, de Boer P, eds. Surgical exposures in orthopaedics: the anatomic approach. Lippincott Williams & Wilkins, Philadelphia; 2003.
14. King M, Bewes PC, Cairns J, Thornton J, eds. Primary surgery, volume 1, non-trauma. Oxford University Press, Oxford, UK; 1993, Pp 83–103.
15. Meier DE, OlaOlorun DA, Nkor SK, Aasa D, Tarpley JL. Ketamine—a safe and effective anesthetic agent for children in the developing world. Pedi Surg Int 1996; 11:370–373.
16. Gutierrez K. Bone and joint infections in children. Ped Clin N Am 2005; 52:779–794.