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REVIEW ARTICLE

Osteomyelitis and septic arthritis in children

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Osteomyelitis is an infection in bone most frequently occurring in children. The current incidence is 1 in 5000. Septic arthritis is an infection of a synovial joint which may occur in all age groups in children but has a specific infantile form affecting the infant from birth to the first year of life. The majority of infections of bone or joint are caused by spread of bacteriae through the bloodstream or occasionally by entry of organisms through an open wound, by puncture or by extension of infection from adjacent tissue. The most common causative organism is *Staphylococcus aureus* but many other organisms may be responsible for a bone or joint infection. The treatment of both osteomyelitis and septic arthritis is based on antibiotic therapy in combination with surgical drainage if pus or infected tissue is present. Early diagnosis followed by adequate treatment gives good outcome. Inappropriate or delayed treatment may result in chronic osteomyelitis or irreversible joint destruction.

Keywords : osteomylitis ; septic arthritis ; children.

INTRODUCTION

Osteomyelitis and septic arthritis occur most commonly in children.

Osteomyelitis is an inflammation in the bone. The term osteomyelitis generally refers to a bacterial infection of bone. Osteomyelitis may be acute, subacute or chronic. Septic arthritis is a joint infection usually caused by bacteria. Bacteria can reach the bone and joint through several routes. Osteomyelitis and septic arthritis have a potential for life-long disability if treated insufficiently.

Osteomyelitis

In children, osteomyelitis is most often acute, with the bacteriae usually reaching the bone through the bloodstream ; it is commonly referred to as acute haematogenous osteomyelitis (AHO). Rarely an infection may spread to the bone from an adjacent infected focus or by direct inoculation through an open wound at the time of an open fracture or following surgery.

Acute haematogenous osteomyelitis

Acute haematogenous osteomyelitis is predominantly a disease in children. The overall prevalence of AHO is estimated at 1 case per 5000 children (*17*) but according to recent studies the incidence appears to be declining (*3, 8, 24*). AHO most often is monostotic, affecting nearly twice as many boys as girls (*8*). The clinical manifestation and the natural history of osteomyelitis depend on several factors including age of the patients, site of infection, viru-

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lence of the infecting organism and patient resistance. The pathogenesis and factors that predispose to AHO are poorly understood. The relationship of trauma to osteomyelitis is unclear. Trauma to the bone may cause local oedema and may alter the blood flow, and haematoma seems to provide a good local environment for bacterial proliferation (*30*). However children are continually subjected to injuries and AHO is relatively uncommon compared to trauma. In some instances there is a history of recent respiratory tract infection, otitis media or an infected wound but most often AHO begins spontaneously in a healthy child. AHO typically affects the most rapidly growing ends of long bones and is more common in the lower extremity, the metaphysis of the distal femur and of the proximal tibia being the most common sites of infection (*16, 22*). The metaphysis of the proximal humerus is less frequently affected. However AHO can develop in any bone of the skeleton. Multifocal sites of AHO may be present in neonates (*17*). The anatomical characteristics of the blood supply of the metaphysis of long bones seem to be the major factor in the predilection for infection of this area. The arterioles of the metaphysis terminate in a sharp loop before emptying into a wide venous sinusoid. The resultant sluggish blood flow creates an ideal environment for bacterial proliferation. In addition there are relatively few phagocytic cells in the metaphyseal vessels so that infection easily develops in this area. The bacteriae provoke an inflammatory process. The acute inflammation increases the vascular permeability, resulting in oedema and raised pressure in the venous sinusoids with thrombosis of the nutrient arteries. If untreated, the inflammatory process continues. Pressure within the metaphysis increases as pus collects. With increasing pressure, pus takes the path of lesser resistance and perforates the thin cortex of the metaphysis. It then spreads under the periosteum and strips it from the underlying bone, disrupting the cortex from its periosteal blood supply. The underlying bone becomes necrotic and then forms a *sequestrum*. The stripped periosteum on the other hand remains viable and will lay down new bone. The newly formed bone is known as an *involucrum*. The involucrum may completely encase the sequestrum. In some locations such as the proximal femur, the proximal humerus, the proximal radius and the lateral part of the distal tibia, the metaphysis lies within the joint capsule and the abscess may open into the joint resulting in a concomitant septic arthritis (*22*). Many different bacteriae can cause osteomyelitis (table I). The organism most commonly responsible for AHO is *Staphylococcus aureus* (*1, 3, 8, 11, 16, 17*). In neonates, next to *Staphylococcus aureus*, *Streptococcus* mainly group B and Gram-negative enteric bacteria are frequently found (*17*). Historically, *Haemophilus influenzae* was commonly encountered in children younger than 2 years but has now essentially been eliminated due to general vaccination against type B *Haemophilus influenzae* (*3, 8, 16, 24, 25*). In older children, *Staphylococcus aureus* is by far the most common causative organism. In patients with pre-existing diseases, uncommon organisms may be encountered. Children with sickle cell disease are at risk for *Salmonella* osteomyelitis (*21, 26*). A puncture wound through the shoe to the plantar surface of the foot may result in *Pseudomonas aeruginosa* osteomyelitis (*16*). Methicillin-resistant *Staphylococcus* had emerged as causative organism of osteo-articular infections in recent studies (*1, 8, 11, 25*). In immunocompromised patients, infections caused by fungi must be considered.

Table I. — Most common causative organisms by age group

Newborns (< 2 mo)	:	Staphylococcus aureus
		Streptococcus A and B
		Enterobacter
Children (2 mo - 4 y)	:	Staphylococcus aureus
		Streptococcus A
		Enterobacter
		Kingellae kingae
Children (5 y - Adult)	:	Staphylococcus aureus

The clinical response and symptoms of AHO are quite variable and depend on the age of the patient, the site of infection, the resistance of the child and the virulence of the affecting organism. The onset of AHO is sudden and is characterised by a well-localised bone tenderness, associated with high fever. The child looks sick and is often unwilling to move the affected extremity. If the infection is located in the lower limb, the patient may limp or

refuse to walk. Localised signs of inflammation are soon present : swelling, redness, warmth, local pain. Clinical signs however may be quite variable and the clinical presentation of AHO appears to be changing. Many patients now present with less florid illness than the classical presentation (*8*). In case of infection in a deep location such as the proximal femur or the pelvis, localised signs such as redness and swelling are not manifest in the early phase of the disease. The clinical presentation also differs in neonates. In the neonate there is often a lack of signs of systemic illness. Irritability and poor feeding are then the only findings. Pseudoparalysis and pain on attempt at passive motion of the affected limb are usually present but these signs may be overlooked or misinterpreted. The paucity of obvious clinical signs in the neonate is responsible for the frequent delay in diagnosis in this age group.

Laboratory studies include complete blood cell count, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). White blood cell count, with a leftward shift, is usually increased. ESR and CRP are typically elevated and the values return to normal after successful treatment. CRP declines and returns to normal values more rapidly (after 7 to 10 days) than ESR (after 3 to 4 weeks).

Radiographs show the inflammatory response of the infection. In the early phase of the infection, soft tissue swelling is the only radiological finding. This sign may be overlooked or neglected. In osteomyelitis there is swelling of the deep soft tissue next to the bone, while in cellulitis, the superficial soft tissue is swollen. Osseous changes on standard radiographs are not visible until 7 to 14 days after the onset of infection. Mottling of bone density is the first visible bone change (fig 1a). These bone changes should not be awaited to make the definitive diagnosis of AHO, because they occur too late to be of any help for an early diagnosis. If untreated, cortical erosion follows and the sequestrum and the involucrum become radiologically visible (fig 1a,c).

Technetium 99 m diphosphonate bone scan is a sensitive test (*11, 25, 28*). Bone scan however is non-specific and the differential diagnosis between infection, tumour or trauma is often impossible. It does not make the definitive diagnosis but it provides the location of the pathologic process (fig 1b ; 3c). A bone scan is especially helpful to detect multiple locations such as may occur in the neonate, or when the diagnosis is unclear, with infection in atypical locations such as the pelvis (*17*). Bone scans will show the site of osteomyelitis much sooner than plain radiographs. The images of a bone scan are not disturbed by a previous bone aspiration.

Computer tomography is not useful as a routine examination in the diagnosis of AHO but may be valuable in the diagnosis of chronic infection that may be confused with a tumorous process (*17*). Computer tomography is also useful in imaging difficult sites such as the pelvis (fig 2d).

Magnetic resonance imaging (MRI) should be reserved for diagnostic problems. MRI can provide additional information about the extent of the infection and the localisation of abscess formation (*8, 25*). MRI can also be valuable to detect and locate extra osseous soft tissue infections which may mimic bone or joint infection (*4, 5*).

The causative organism should be sought by blood cultures and by aspiration from the site of infection. Cultures should be obtained prior to starting antibiotics. Blood cultures are positive in 30% to 50% of cases (*17*). Bone aspiration should be performed at the site of maximum tenderness and swelling. Ultrasonography may be helpful to locate subperiosteal pus (*8, 10*). If aspiration does not yield pus, the needle should be inserted into the metaphyseal bone. All aspirated material should be cultured. A negative aspiration does not exclude an infection. When there is typical presentation in the early phase of the infection and good and rapid response to antibiotic therapy, a diagnostic bone aspiration is not necessary (*24*).

The most important differential diagnosis is a malignant process. The differentiation may be difficult. Malignant bone tumours such as Ewing sarcoma may give rise to fever in addition to pain in the affected extremity (*14*). Radiological signs of tumours such as osteosarcoma or Ewing sarcoma can be difficult to distinguish from the radiologic response to infection. MRI is more reliable to differentiate between a malignant process and an

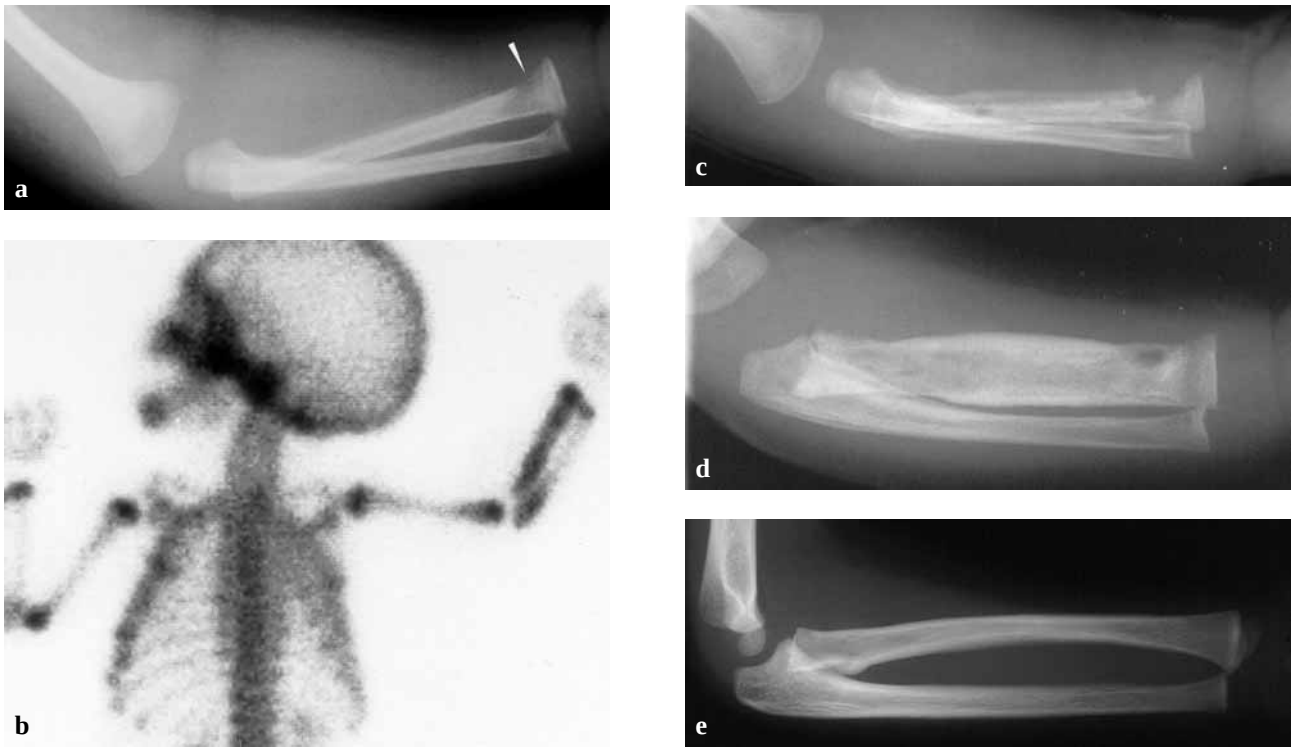


Fig. 1. — a) Lateral radiograph of the forearm of a 6-week-old boy. Note swelling of the deep soft tissue, mottling of the bone in the distal radius and perforation of the cortex ; b) Bone scan showing increased uptake in the whole radius and in the proximal ulna ; c) Lateral radiograph made 1 week after trepanation shows the sequestrum and the involucrum ; d) 4 weeks later there is resorption of the sequestrum ; e) Lateral radiograph made after 3 years shows remodelling of the bone. There was normal function.

infection. An appropriate aphorism is : "Culture all your biopsies, biopsy all your cultures". Other conditions that may be confused with osteomyelitis include cellulitis, septic arthritis, trauma and bone infarction.

If pus is obtained from the subperiosteal space or from the metaphysis, surgical drainage is mandatory (*3, 8*). The abscess should be decompressed, evacuated and washed-out under general anaesthesia. A suction drain should be left in place for forty-eight hours. Cultures should be obtained prior to starting antibiotics. While awaiting the microbiological results, parenteral antibiotics should be started empirically, based on the most likely organism (table II) taking into consideration the age of the patient. Recent studies recommend that flucloxacillin alone should be given as the empiric treatment of choice in children fully immunised against *Haemophilus influenza* (*3, 8*). Once the causative organism is identified, the antibiotics may be modified according to the sensitivity of the isolated bacteria. If the diagnosis is made in the early phase of the infection and before abscess formation, most children can be treated with antibiotics alone (*3*). However, if the patient does not respond to this regimen and has persisting pain and fever after 24 to 48 hours, surgical exploration should be undertaken (*8, 24*). A belief that no surgery is necessary may be inappropriate. There is no certain method of deciding how long antibiotics should be given. Traditionally parenteral antibiotics were prescribed for at least 6 weeks followed by 6 to 12 weeks of oral antibiotics (*24, 25*). Recent studies have demonstrated efficacy of shorter periods of antibiotic therapy (*1, 11, 18, 25, 29*). The most common treatment for the typical case in the preliminary inflammatory phase of AHO is 5 to 7 days of intravenous antibiotics followed by 3 to 4 weeks

Table II. — Commonly used antibiotics*

Newborns (< 2 mo)	:	Cefotaxim or Flucloxacillin and Gentamycin
Children (2 mo - 4 y)	:	Flucloxacillin or Ceftriaxon or Cefotaxim
Children (5 y - Adult)	:	Flucloxacillin
Sickle cell disease	:	Flucloxacillin and Ampicillin or Cefotaxim
Puncture wound (foot)	:	Flucloxacillin and Gentamycin or Ceftazidim
MRSA**	:	Clindamycin or Vancomycin

* These are only guidelines. Other antibiotics can be used.
** Methicillin-resistant Staphylococcus aureus.

of oral antibiotics (*27*). The treatment however should be individualised and the duration of antibiotic treatment depends on the severity of the infection, the time elapsed between the onset of the disease and the start of the treatment, the extent of bone involvement and the clinical and laboratory responses of the initial treatment (*18*).

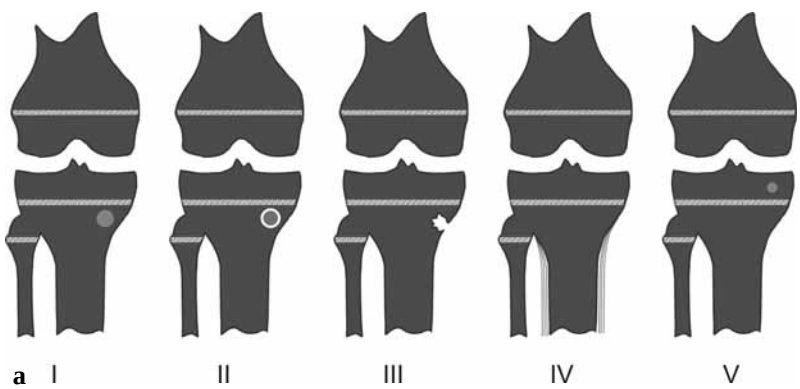
Subacute and chronic osteomyelitis

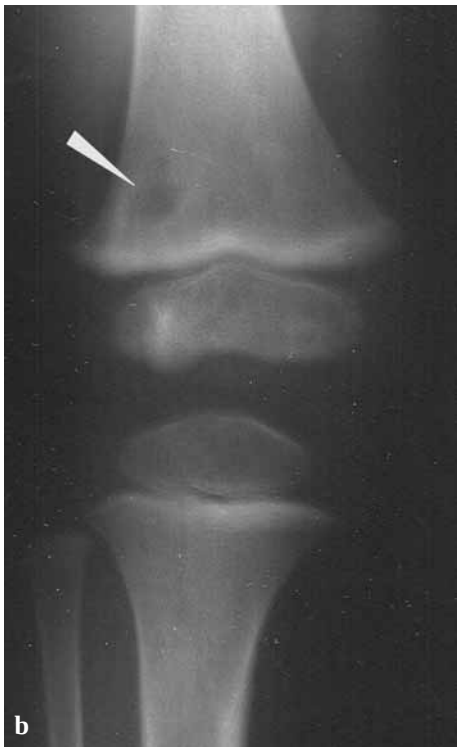
Although osteomyelitis generally presents as an acute disease, it may be subacute or chronic. Subacute osteomyelitis is considered as an infection with a duration longer than 3 weeks but many patients will present symptoms of one to several months duration (*7*). It is less common than acute osteomyelitis and is generally haematogenous. The patients are typically older than those with AHO, ranging in age from 2 to 16 years. The patient shows no or few signs of systemic illness but most often complains of mild, sometimes intermittent pain over a course of 3 or 4 weeks. There is local tenderness and if the lower limb is involved, limping may be the most obvious sign. Radiographic signs are usually visible at the time of presentation. Their appearance may be quite variable (fig 2 a-d). Subacute osteomyelitis can exceptionally occur in the epiphysis (*7*) or the diaphysis of the long bones (*7*). The radiologic lesions of subacute osteomyelitis should be differentiated from primary bone tumours (*14*). When in doubt, further investigations with computer tomography or MRI are indicated. Biopsy and cultures should be performed. Laboratory findings show normal or slightly elevated white blood cell count, ESR and CRP are usually elevated but not as high as in acute haematogenous osteomyelitis. The most common affecting organism is *Staphylococcus aureus* (*14, 17*). Most cases of subacute osteomyelitis can be treated by antibiotics without surgery. If however the symptoms persist after 2 weeks antibiotic treatment, drainage is indicated.

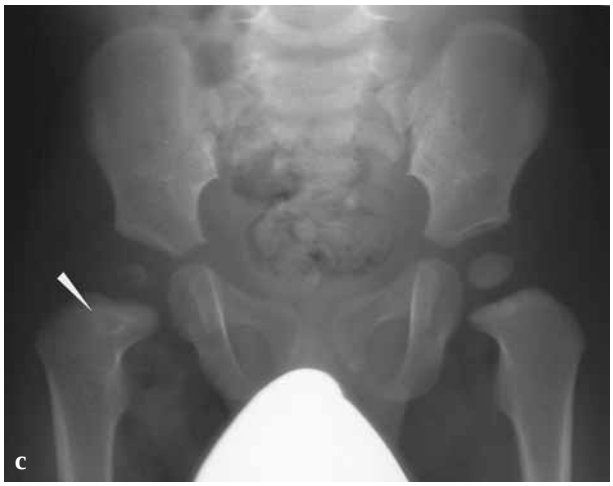
Chronic osteomyelitis is usually a sequel of an untreated or insufficiently treated osteomyelitis. The treatment consists of curettage and removal of nonviable tissue in combination with antibiotics. Pathologic fracture if present is a serious and often difficult to treat complication of long-standing osteomyelitis (fig 4).

Septic arthritis

Septic arthritis (SA) has many features in common with acute osteomyelitis. There is most often an acute onset. Infection may reach the joint through several routes. SA in children usually occurs through haematogenous dissemination of bacteria into the joint. Penetrating injuries especially of the knee joint may be responsible for SA. It may also result from contiguous spread of osteomyelitis. Direct extension of a metaphyseal abscess through the growth plate via vascular channels into the epiphysis and further in the joint space is possible in infants. Before the age of 18 months, small vessels cross the proximal growth plate of the femur from metaphysis to epiphysis (*19*). For this reason, a metaphyseal abscess, in this particularly age group, can easily extend directly into the joint (*2, 16, 20*). After about 18 months of age, the vascular channels have dropped out and the growth plate will from then on act as a barrier to the terminal vessels of the metaphysis. A second mechanism







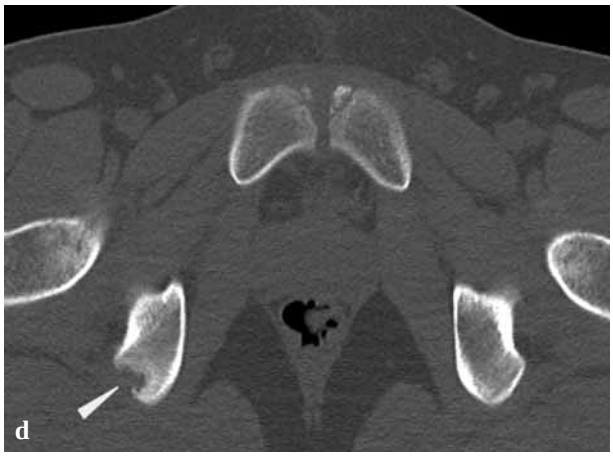


Fig. 2. — a) Different types of subacute osteomyelitis : I Punched-out lesion ; II Metaphyseal lesion with sclerosis ; III Metaphyseal erosion ; IV Periosteal reaction ; V Epiphyseal lesion ; b) Punched-out lesion ; c) Metaphyseal lesion with sclerosis ; d) Computer tomography of the pelvis showing subacute osteomyelitis with erosion.

of extension of a metaphyseal abscess into the joint is erosion of the cortex and spread of pus into the joint space. In certain locations the metaphysis lies within the joint capsule and therefore perforation of the thin metaphyseal cortex by the abscess may contaminate the joint (fig 3 a,b). This is most likely to occur in the hip joint but may also occur in the shoulder, the elbow (proximal radius) and the ankle joint (distal lateral tibia). Perlman *et al* (*22*) found a 33% incidence of adjacent septic joints in their patients who had osteomyelitis. This is the highest incidence reported in literature. Of these adjacent joint infections, 31% were located at the knee joint. Distribution of ages of their patients and high percentages of knee infections, suggest that spread of infection from the metaphysis across the growth plate is not the only mechanism, and probably other mechanisms may play a role in the distribution of pus from the metaphysis to the joint (*22*). In those cases of SA caused by contiguous spread of adjacent osteomyelitis, most patients present with symptoms of SA and the diagnosis of concomitant

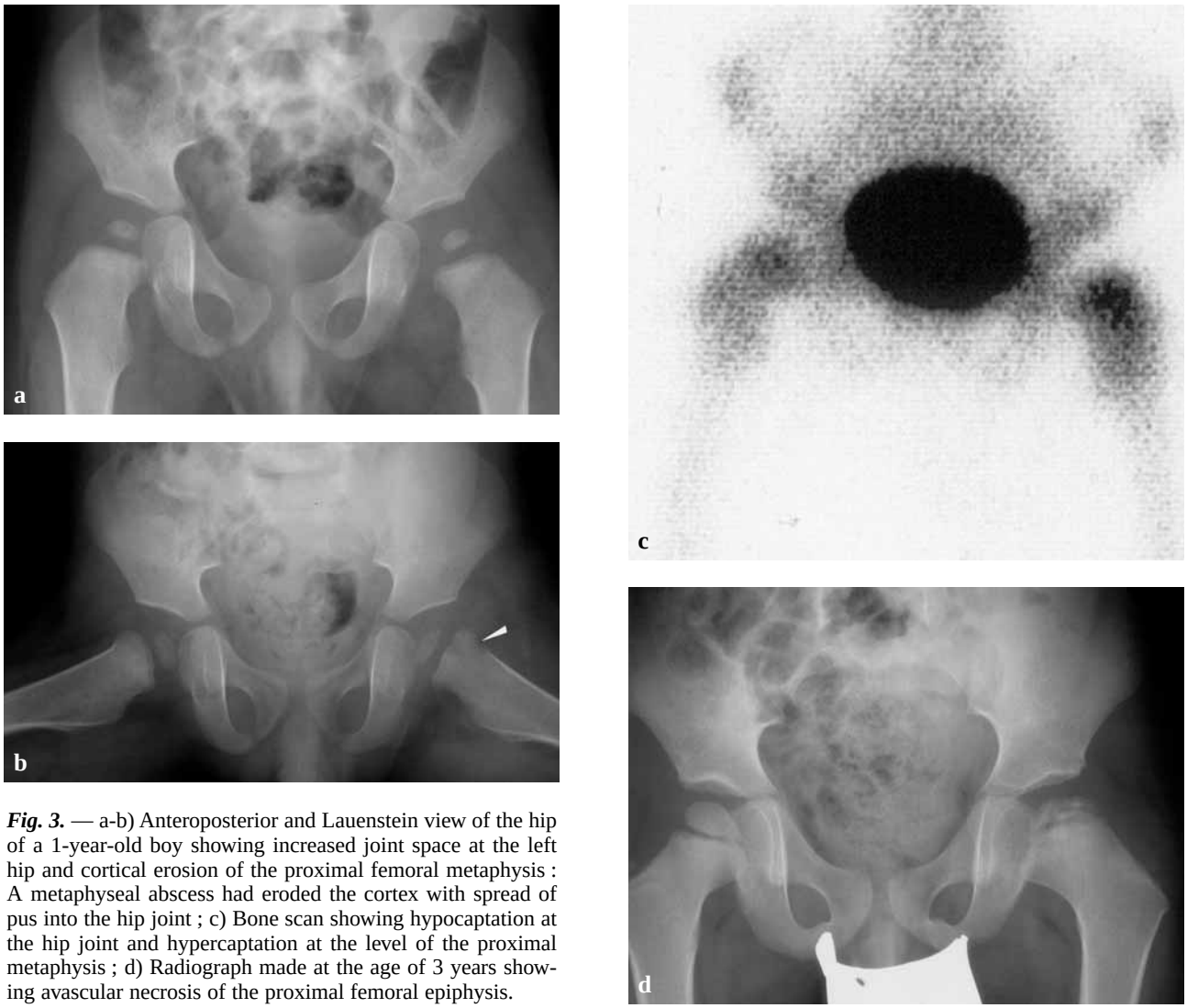


Fig. 3. — a-b) Anteroposterior and Lauenstein view of the hip of a 1-year-old boy showing increased joint space at the left hip and cortical erosion of the proximal femoral metaphysis : A metaphyseal abscess had eroded the cortex with spread of pus into the hip joint ; c) Bone scan showing hypocaptation at the hip joint and hypercaptation at the level of the proximal metaphysis ; d) Radiograph made at the age of 3 years showing avascular necrosis of the proximal femoral epiphysis.

osteomyelitis is often made late when radiologic signs of osteomyelitis become visible. Approximately 75% of cases of SA occur before the age of three years (*17*). Any joint may be infected but about 80% of cases are located in the lower extremity with the hip (in the young child) and the knee (in the older child) being the most common locations (*9, 11, 16*). SA rapidly provokes an inflammatory response. The synovial membrane responds with a hyperaemia followed by excessive production of fluid and pus. Pus contains a large variety of proteolytic enzymes which are chondrolytic and rapidly destroy the articular cartilage. The articular cartilage has virtually no reparative ability and once it is destroyed, irreversible joint damage and deformity is likely. This can lead to permanent joint destruction in as little as 3 days. In addition to this chemical reaction, the presence of pus within the articular cavity stops the normal nutritional function of synovial fluid. As the process of inflammation and pus formation continues, intraarticular pressure rises, which may lead to tamponade of the nutrient blood vessels, giving rise to avascular necrosis of the bone centre of the epiphysis. Increased intraarticular pressure stretches the capsule and may lead to pathologic dislocation. In the

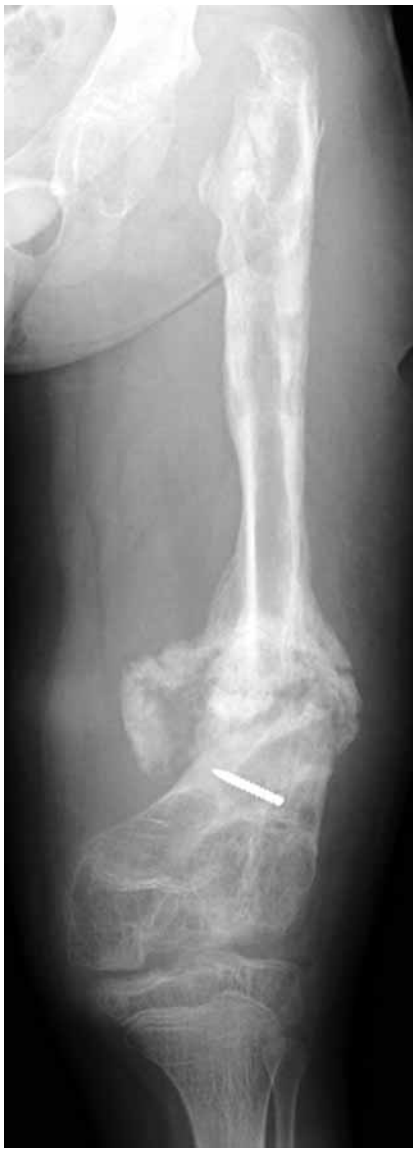


Fig. 4. — Sequelae of osteomyelitis and concomitant septic arthritis : Complete destruction of the hip joint and presence of a pathologic fracture.

hip joint, an inadequately treated infection may lead to destruction and disappearance of an already ossified epiphysis and may cause trochanteric overgrowth and leg length discrepancy when the growth plate is partially or totally damaged (fig 4). Fibrosis may occur later, resulting in joint stiffness. Virtually every organism can cause SA but the vast majority are similar to those seen in AHO (table I). The most common aetiologic agent is *Staphylococcus aureus* (*9, 11, 12, 16, 17, 23*). *Haemophilus influenzae* had virtually been eliminated due to vaccination (*16*). *Kingella kingae* infections, specifically in children younger than 2 years are reported more and more in recent studies (*1, 9, 17, 31, 32*). The incidence and spectrum of causative organism such as *Mycobacterium tuberculosis* and *Borrelia burgdorferi* appear to vary on a geographic basis (*8*). Fungal infections are rare in our country but seem to be endemic in some areas. While rare in our country, *Neisseria gonorrhoeae* is the most common cause of septic arthritis in the United States (*17, 23*).

Clinical manifestations of septic arthritis are comparable with AHO. The child looks sick, has fever, localised pain and does not move spontaneously the affected joint. In peripheral joints, swelling, redness, warmth and local tenderness are present early. For deep located joints and especially in the hip joint, these typical signs manifest late and delay in diagnosis is common. In neonates and infants the usual clinical signs of inflammation – even in peripheral joints – are usually lacking. Fever is absent and the neonate or infant may not appear sick. A high index of suspicion and careful observation are required to make the diagnosis and to determine the site of infection. The most common consistent finding is absence of spontaneous movements of the involved limb (pseudoparalysis). In this age group the hip is frequently affected. The infant will hold the leg flexed, abducted and externally rotated to decrease the intraarticular pressure. All movements of the hip joint cause pain. Swelling and redness in the groin are late signs in this particular joint infection (*6*).

Radiographs may show soft tissue swelling, capsular distension, joint space widening or radiologic signs of adjacent osteomyelitis (fig 3 a,b).

Ultrasonography is very useful to identify even small joint effusions and can guide joint aspiration.

Bone scan may show diffuse uptake within the joint in the early phase or a cold spot once the intraarticular pressure is raised (*28*) (fig 3c).

As in AHO, the peripheral white blood cell count, ESR and CRP are elevated but may be normal in the neonate with septic arthritis. Diagnosis

of SA is generally made by joint aspiration. *If joint infection is suspected, whatever the age of the patient, aspiration is mandatory*. This should be performed as an emergency and not delayed until obtaining the results of the laboratory studies. Aspirated fluid should be cultured for bacteria. The aspiration fluid in case of SA is cloudy. Demonstration of bacteria is diagnostic, however cultures will be negative in one third of cases but this does not exclude infection. Blood cultures will be positive in 30-50% of cases. Synovial white blood cell count will typically be greater than 50.000/mm^3. Drainage should be done promptly and intravenous antibiotics should be started immediately (*9, 12*). Drainage through an arthrotomy is mandatory for the hip and recommended for all other joints. Repeated aspiration for small joints can be an acceptable treatment. Arthroscopic drainage of the knee in older children allows thorough inspection and cleaning of the joint (*8*). As for AHO, antibiotics should be started instantly based on the most likely affecting organism (table II). Later, the antibiotics may be adjusted based on the bacterial identification and antibiotic sensitivity results. The optimal duration and route of administration are not well defined. There should be no strict general rules regarding the duration of antibiotic treatment in a disease with such a variable expression. A shorter duration than traditionally prescribed is now recommended (*11, 12, 29*). The recommended duration for an uncomplicated arthritis is 3 to 5 days parental antibiotics followed by 3 to 4 weeks oral therapy (*27*). However the treatment should be individualised and longer duration may be needed if the infection had not completely resolved. Intraarticular administration of antibiotics is not recommended because it seems useless and certain products may cause chemical synovitis.

Differential diagnosis

All types of non-infecting arthritis can mimic SA. The causes of arthritis in children are numerous.

Juvenile rheumatoid arthritis (JRA) may be acute in onset. Especially the monoarticular knee arthritis may be difficult to distinguish from SA.

Table III. — Kocher's criteria

• Non-weightbearing
• ESR > 40 mm/hr
• WBC > 12000/mm^3
• Fever

JRA differs from SA in that the child is usually less sick, has only little pain and no fever. Joint aspiration and cultures are indicated in case of doubt.

Acute joint pain associated with trauma is usually related to an obvious injury. A painful, swollen joint however is often too easily attributed to a trauma and the first signs of SA are frequently misdiagnosed as a traumatic event.

Reactive arthritis refers to sterile joint effusion associated with an extraarticular infection. Specifically following episodes of diarrhoea caused by *Shigella* species, *Salmonella* species, *Campylobacteria* species or *Yersinia* species (*23*).

Irritable hip is the principal differential diagnosis regarding septic arthritis of the hip. The differential diagnosis with SA can be difficult. Irritable hip is more common than infection of the hip joint. The child with an irritable hip most commonly presents with an acute onset of pain, limping or even refusal to bear weight. Most children between 3 and 7 years, who have a septic arthritis of the hip joint, have an irritable hip as their first diagnosis (*13*). Kocher *et al* (*13*) developed a clinical prediction algorithm to differentiate between SA and irritable hip based on 4 clinical variables (table III). According to these authors when four criteria were met there was a 99% chance that the child had a septic hip joint. When three criteria were met there was a 93% chance, a 40% chance when two criteria were met and 3% chance of having a SA when one criterion was met. Luhmann *et al* (*15*) applying Kocher's criteria found a predicted probability of having an infected hip joint of only 59% if 4 variables developed by Kocher *et al* (*13*) were present (*15*). Luhmann *et al* (*15*) identified three criteria having a predictive value for SA of the hip : a history of fever, a serum white blood cell count of > 12000/mm^3 and a previous health care visit. According to this last study, there was a predicted

probability of having a SA of the hip of 71% if all three variables were present. Although a clinical predictive algorithm can help making the correct diagnosis, in a child with a painful hip the diagnosis continues to be difficult. In fact there is no single analysis that can serve as a definitive test to diagnose a SA of the hip in children or to differentiate it from an irritable hip.

CONCLUSION

Osteomyelitis and septic arthritis in children are most often acute and most often secondary to haematogenous spread. The diagnosis can usually be made from the clinical signs but requires a high index of suspicion. In neonates and infants osteomyelitis and septic arthritis have certain peculiar features. The affected bone has a certain capacity for repair and remodelling : the sequestrum may be absorbed by enzymatic reactions, new bone may be formed and bone contours may return to normal (fig 1d, e). Joint infection destroys the articular cartilage and may cause permanent joint destruction (fig 3d and fig 4). This makes septic arthritis a more serious disease than osteomyelitis. Septic arthritis is a real emergency. In the neonates the diagnosis can be missed because there is often lack of systemic signs. The most susceptible joint for protracted disability is the hip.

Untreated osteomyelitis and septic arthritis has a dismal prognosis. Before the advent of antibiotics the mortality rate was close to 50% and the remaining patients usually were disabled for the rest of their lives. AHO and SA now are curable diseases if recognised early and adequately treated. Once the diagnosis is reasonably suspected, therapy should be started promptly. The basic treatment is antibiotics ; if pus is present, drainage is mandatory.

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