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Joint Pain in Children, Part VI: Bone Infections in Children

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Osteomyelitis is the generic term for bone infections. The great majority of bone and joint infections in North America are primary, developing as a consequence of direct contamination during trauma and or orthopedic surgery. The most common pathogens are pyogenic bacteria, mycobacteria, and fungi. These pathogenic microorganisms spread to bone by one of three routes: hematogenous spread; direct extension from a contiguous site of infection; and direct introduction. The most serious bone infections are pyogenic osteomyelitis and tuberculosis; there are also rare cases of syphilis and fungal infections.

Take a moment to review the anatomy of a tubular bone and its vascular supply. The vascular supply will vary depending on the age of the individual. In the infant (0-1 yrs.), diaphyseal and metaphyseal vessels may perforate the bone growth plate. In the child (1-16 yrs.), diaphyseal and metaphyseal vessels do not penetrate the open growth plate. Finally, in the adult (>16 yrs.) diaphyseal and metaphyseal vessels penetrate the closed growth plate.

Acute hematogenous osteomyelitis occurs predominately in children and before the age of epiphysial closure (>21 yrs). The infection typically originates in the metaphysis of the long bones, a highly vascular area. Common sites of involvement are the distal femur, proximal tibia, humerus and radius. Hematogenous osteomyelitis in children sometimes results from the blood-borne spread of infection to the bone from an extraskelatal foci, such as the skin, ear, and pharynx. Often the source of the infection is not readily apparent. An event that at the time seemed trivial may cause a bacteremia, such as an ordinary cut or bruise of the skin (or a human bite -- children have been known to bite each other), can be the source of the infection.

Hematogenous osteomyelitis of children is most often caused by *S. aureus*, which accounts for 60-90% of cases. Osteomyelitis of neonates is also frequently caused by group B streptococci and *E. coli*. Children with sickle cell disease are prone to acquire salmonella infections and to develop salmonella osteomyelitis.

Osteomyelitis of children usually begins in the metaphysis of long bones. The blood-borne bacteria are carried to the marrow space by way of the nutrient artery. The initial site of infection within a particular bone is determined by the vascular anatomy as related to the epiphysial growth plate. In children a year and older, the metaphyseal branches of the nutrient artery do not penetrate the growth plate. The vessels turn back upon themselves just proximal to the plate and enter venous sinusoids in the marrow space of the metaphysis.

The venous sinusoids are much larger than the arteries feeding them, have a slower blood flow, and provide a medium favorable for bacterial growth. The bacterial infection causes a fulminant acute inflammation of the marrow space, and an exudation of polymorphonuclear leukocytes. The presence of an inflammatory exudate within the rigid limits of the marrow space causes an increase in intramedullary pressure, reduced blood flow, local vascular occlusion, and thrombosis. Local ischemic injury and cell necrosis of marrow and osseous tissue occur and the bacteria, pus cells, and necrotic debris comprise a septic focus of purulent inflammation. At the early stage of the infection, no specific bone changes are seen radiographically.

The infection may then spread rapidly by way of vascular channels through the medullary cavity and the bone cortex, which is thin in the region of the metaphysis and provides easy access to the periosteum. The purulent material may elevate the periosteum and form abscesses beneath it or penetrate the periosteum as sinus tracts which drain into the soft tissue or extend to the skin surface.

The clinical characteristics of acute osteomyelitis are unique to each age group. Infants usually become abruptly and desperately ill with hectic fever, rapid pulse, vomiting, and occasionally convulsions. Less commonly, an infant will become gradually ill, showing the illness only by loss of appetite, lethargy, fretfulness, and variable fever. Older children will usually become rapidly ill, manifesting hectic fever, rapid pulse, and variable degrees of prostration. They will not appear as desperately ill as infants, however. Adults usually do not appear acutely ill and may complain of migrating arthralgias and myalgias prior to localization of pain in one extremity.

Eventually, all patients will develop signs of inflammation at the site of the infection. The signs at the site of infection usually develop in the following order:

1. Infants and young children manifest a flaccid extremity, at times so flaccid as to mimic a lower motor neuron paralytic illness. Older children and adults complain of pain and resist passive and active motion.

2. The area over the infected metaphysis becomes tender.
3. The area over the infected metaphysis becomes reddened, indurated, and swollen.
4. If the infection erodes into overlying soft tissue, the area will become fluctuant and may eventually develop a draining sinus.
5. The neighboring joint may develop an aseptic hydrarthrosis as it participates in the inflammation surrounding the bone infection.
6. If the infection erodes into a neighboring joint, the joint will develop all the signs of septic arthritis (joint will be very painful, hot, red and fluctuant; joint fluid when aspirated will be cloudy or frankly purulent).

Radiographic signs associated with osteomyelitis and joint infection will vary depending on the stage of development of the infection the films are taken. Radiographs are remarkably normal during the first 1-2 weeks of infection, although bone scans quickly demonstrate an increase uptake in the zone of infection. As the infection develops, the following changes will appear:

1. An initial break in the bony trabeculae of the metaphysis will appear and will progress to become larger areas of lytic destruction which appear as radiolucent areas in the metaphyseal region.
2. Periosteal new bone formation will appear at the epiphyseal end of the metaphysis.
3. The cartilage of the growth plate and/or joint surface may show multiple areas of scalloped erosion.

In children (>1 yr) who have an intact epiphysial plate without capillary penetration, a sterile "sympathetic" effusion may occur, indicating a barrier between the infection and the joint space. The pathological picture in hematogenous osteomyelitis may occasionally differ from the spreading and destructive pattern previously described. The initial focus of bone infection may become circumscribed by a fibrous capsule and bone sclerosis to form a localized abscess (Brodie's abscess), which may undergo sterilization or become a chronic focus of infection.

The course of acute hematogenous osteomyelitis is age-dependent. In neonates (<3 months), bone infections are often fulminant but rarely necrotizing, because the spongy bone and thin cortex adapt to increase intraosseous pressure without compromising the blood supply. In infants (3-12 months) and adults, capillaries extend from the metaphysis to the epiphysis and can spread the infection to the adjacent joint, causing suppurative arthritis and septic joint effusion.

Prompt clinical diagnosis and the institution of a potent and protracted regimen of antibiotic therapy have greatly decreased the mortality, which at one time was as high as 40% in the pre-antibiotic era. Even now, occasionally bone infections may be undiagnosed or inadequately treated. In chronic osteomyelitis, the avascular dead tissue, pus and bacteria may remain isolated within an area of bone fibrosis and sclerosis and give rise to recurrent episodes of acute osteomyelitis. The treatment of chronic bone infections usually requires, in addition to antimicrobial therapy, surgical intervention to drain abscesses and remove necrotic tissue.

Other less common fungal infections, mycobacterium tuberculosis, coccidioidomycosis, and actinomycosis, are likely to spread from antecedent lung and skin infections to bone and joint. Tuberculous osteomyelitis is almost always caused by the hematogenous spread of organisms from an active focus of tuberculosis elsewhere in the body, usually the lung and occasionally some other site. The bone infection may occur at any age but is most commonly seen in children. The vertebrae and long bones of the extremities are most frequently involved. In many cases the infection also spreads to contiguous joints such as the hip and knee or contiguous intervertebral joints.

Whenever an indolent arthritis fails to clear or a bone aches relentlessly without evidence of other causes, granulomatous infection must be included in the differential diagnosis along with chronic septic arthritis, rheumatoid arthritis and persistent synovial disorders.

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