Management of Acute Septic Arthritis of Hip in Children

Essay submitted for partial fulfillment of the master degree of orthopaedic surgery

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Introduction

Smith first described an ‘acute arthritis of infants’ in1874 and noted a mortality rate of 60% with severe disability in the survivors. The mortality rate did not improve until the advent of antibiotics in the1940s. Although the hip is the most common joint to be affected by septic arthritis in infants and young children, it is a rare disease in western countries. Today it is extremely uncommon for septic arthritis of the hip to be fatal and the prevention of damage to the developing hip joint is of much greater concern as it is the Joint which gives rise to the greatest disability after infection and thus early diagnosis and effective treatment are vital if serious long-term complications are to be avoided.[1-5]

The invading organism, most commonly Staphylococcus aureus, usually spreads from either an adjacent area of osteomyelitis or by haematogenous seeding.[1,3] The hip is typically held abducted, flexed and externally rotated. The child may be limping, with a pyrexia and raised erythrocyte sedimentation rate. If the diagnosis is doubtful, urgent ultrasound and aspiration can be helpful.[3-5]

Open arthrotomy drainage and thorough irrigation of the joint is considered the standard treatment for septic arthritis of the hip.[4,6] Hip arthroscopy, used as a diagnostic or therapeutic tool in certain indications, has some advantages over arthrotomy. It is less invasive and allows for quicker recovery and return to activities.[6]

Delayed diagnosis and ineffective treatment are associated with such complications as necrosis of the femoral head, osteomyelitis, chondrolysis, systemic sepsis, leg length discrepancy, and later osteoarthritis of the hip joint.[1-5]

Aim of the work

The aim of the work is to review literature about diagnosis and recent medical and surgical treatment protocols of acute septic arthritis of the hip in children.

Pathogenesis

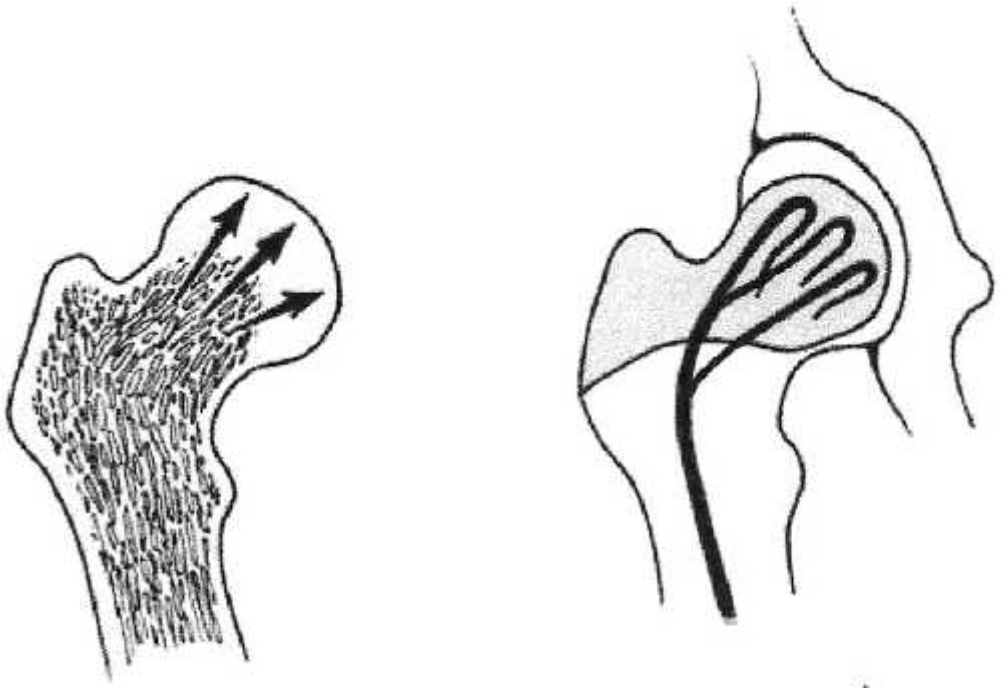
The pathogenesis of acute septic arthritis of hip in children is multifactorial and depends on the interaction of the host immune response and the invading pathogen. By taking into account the steps of bacterial colonization, infection and induction of the host inflammatory response, one may gain a greater understanding of this joint diseases.[1,2]

Predisposing factors

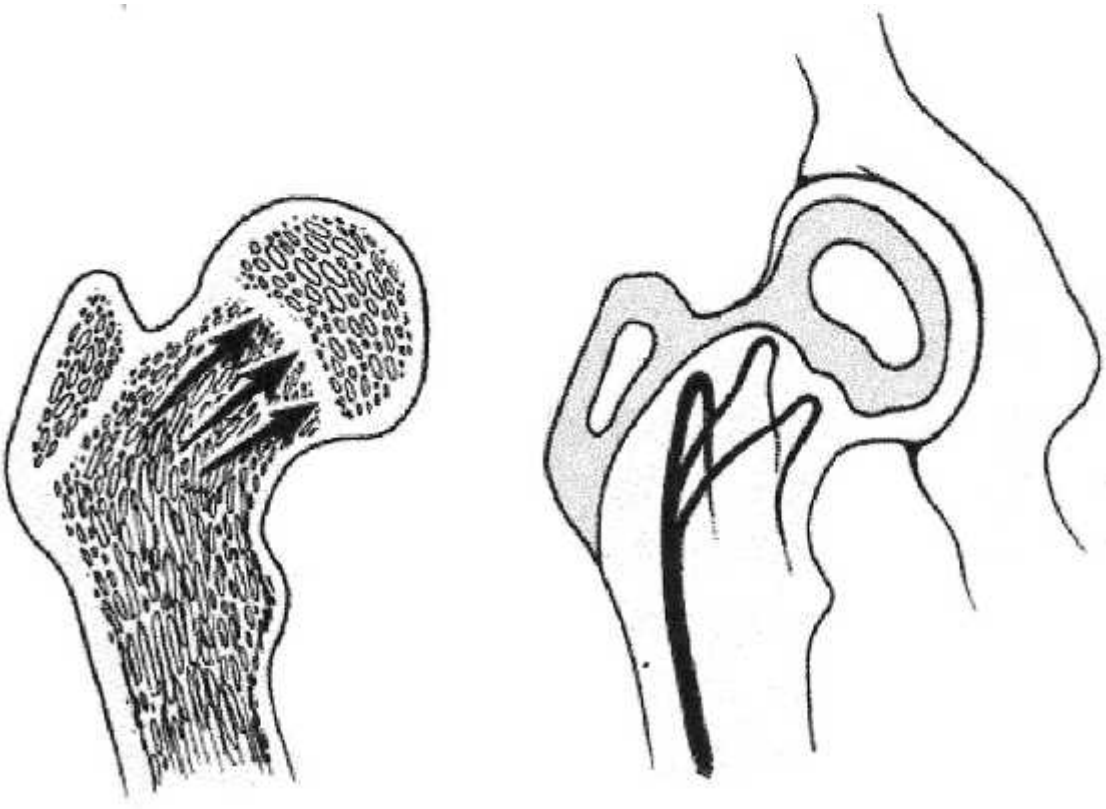
A variety of predisposing factors have been reported to be associated with acute septic arthritis of hip, including: neonates and infants, impaired host defense mechanism, pre-existing joint disease and presence of associated osteomyelitis.[1,3]

Neonates are special in that their immune systems still are immature. They are susceptible to a wide range of organisms that are unlikely pathogens in an older individual, and they are less capable of mounting an inflammatory response to infection.[4] Premature infants in the neonatal intensive care unit are particularly at risk. They often are debilitated with other illnesses, and they typically present with multiple ports for bacterial entry. Bone and joint infections in these patients commonly involve multiple sites.

The unique anatomy and circulation of the ends of long bones results in predilection for localization of bood-borne bacteria (Fig1). In the metaphysis, nutrient arteries branch into nonanastomosing capillaries under the physis, which make a sharp loop before entering venous sinusoids draining into the marrow. Blood flow in this area is sluggish and provides an ideal environment for bacterial seeding.[5]



(A)



(B)

Fig 1: The blood supply and spread of infection in the infant (A) and Children (B) .[4]

In the infant (from birth to 18 months), the infection may spread into the epiphysis through blood vessels that cross the cartilaginous physis. From the epiphysis, the infection may then break directly into the adjacent joint, resulting in septic arthritis (Fig.2a). This mechanism of spread directly into the epiphysis is unique to infancy.[6]

The blood vessels that cross the physis disappear by age 18 months, and the cartilaginous growth plate becomes a barrier to the spread of infection (Fig.2b). In the older child, hematogenous osteomyelitis spreads within the metaphysis until it breaks through the metaphyseal cortex. For most of the long bones, the bone infection breaks either into the subperiosteal space or through the periosteum into the adjacent soft tissues. Joint infection does not result, because the joint capsule is firmly anchored to the epiphysis. In the proximal femur, however, the joint capsules attach to the metaphyses; therefore, in this location, hematogenous osteomyelitis may decompress directly into the hip.[6,7]

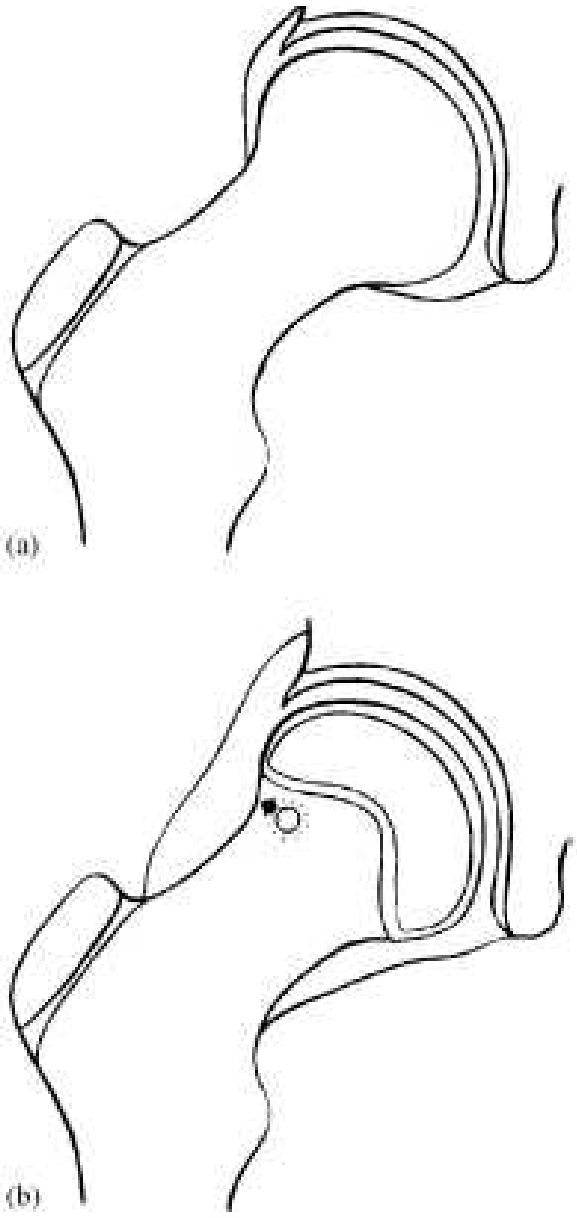


Fig 2a: The infant hip has vascular continuity between metaphysis and epiphysis and the capsule inserts close to the femoral head.[8]

Fig2b: In a child, the epiphyseal plate acts as a barrier to spread of infection from osteomyelitis within the metaphysis. The capsule inserts at the base of the femoral neck and infection is seen to discharge from the metaphysis into the hip joint, by passing the epiphysis.[8]

Bacteriology

In all age groups, the most common infecting organism for septic arthritis of hip is Staphylococcus aureus.[1,3,9] Other common pathogens include Streptococcus species, Pseudomonas aeruginosa, pneumococci [10], Neisseria meningitidis (with or without an associated meningitis), Escherichia coli, Klebsiella species, and Enterobacter species (table 1). Newborns can acquire Neisseria gonorrhoeae from an infected birth canal. Gonococcal arthritis is more common in sexually active teenagers, and it may be seen in younger children in association with sexual abuse.[11]

A neonate aged 5 weeks or younger is susceptible to infection as a result of a wide range of organisms that are unlikely pathogens in children with more developed immune systems. S aureus is still the most common pathogen in this age group and group B streptococcus is the next most common pathogen. Gram-negative organisms may be seen in as many as 15% of joint infections affecting neonates in a neonatal intensive care setting.[12,13]

In the past, H influenzae was the dominant pathogen causing septic arthritis in children younger than 4 years of age. A vaccine against this organism was introduced in 1985, and more effective conjugated vaccines against H influenzae are now extensively used

in the United States. As a consequence, H influenzae has nearly disappeared as a pathogen of osteoarticular infections in young children.[14]

Kingella kingae is a fastidious aerobic gram-negative coccobacillus that colonizes the oropharynx and upper respiratory tract[15], and was first described in 1960. As the incidence of H influenzae has decreased, the incidence of K. kingae as a pathogen of osteoarticular infection in children younger than 3 years of age has dramatically increased. This organism may be missed by routine synovial fluid culture onto solid media. Inoculation of joint fluid into blood culture bottles facilitates recovery of K. kingae.[16] It is now common enough that its presence must be suspected and investigated in children in this age group.[17,18]

Age	Common causative organism
Neonate	*Streptococcus* sp. Gram-negative organisms *Neisseria gonorrhoea*
Infant	*Staphylococcus aureus* *Kingella kingae* *Haemophilus influenza*
Child	*S. aureus* Salmonella (sickle cell)
Adolescent	*S. aureus* *N. gonorrhoea* (sexually active)

Table1: Common Causes of Septic Arthritis by Age[8] .

Portals of entery

The hip joint may become infected in one of three ways: (1) Direct inoculation (e.g. due to femoral venepuncture) is rare. (2) Haematogenous spread (e.g. in septicaemia in the premature infant) is more common. (3) Spread from an adjacent area of osteomyelitis. The osteomyelitis most commonly affects the proximal femoral metaphysis and/or epiphysis, although it may also arise primarily in the acetabulum.[8]

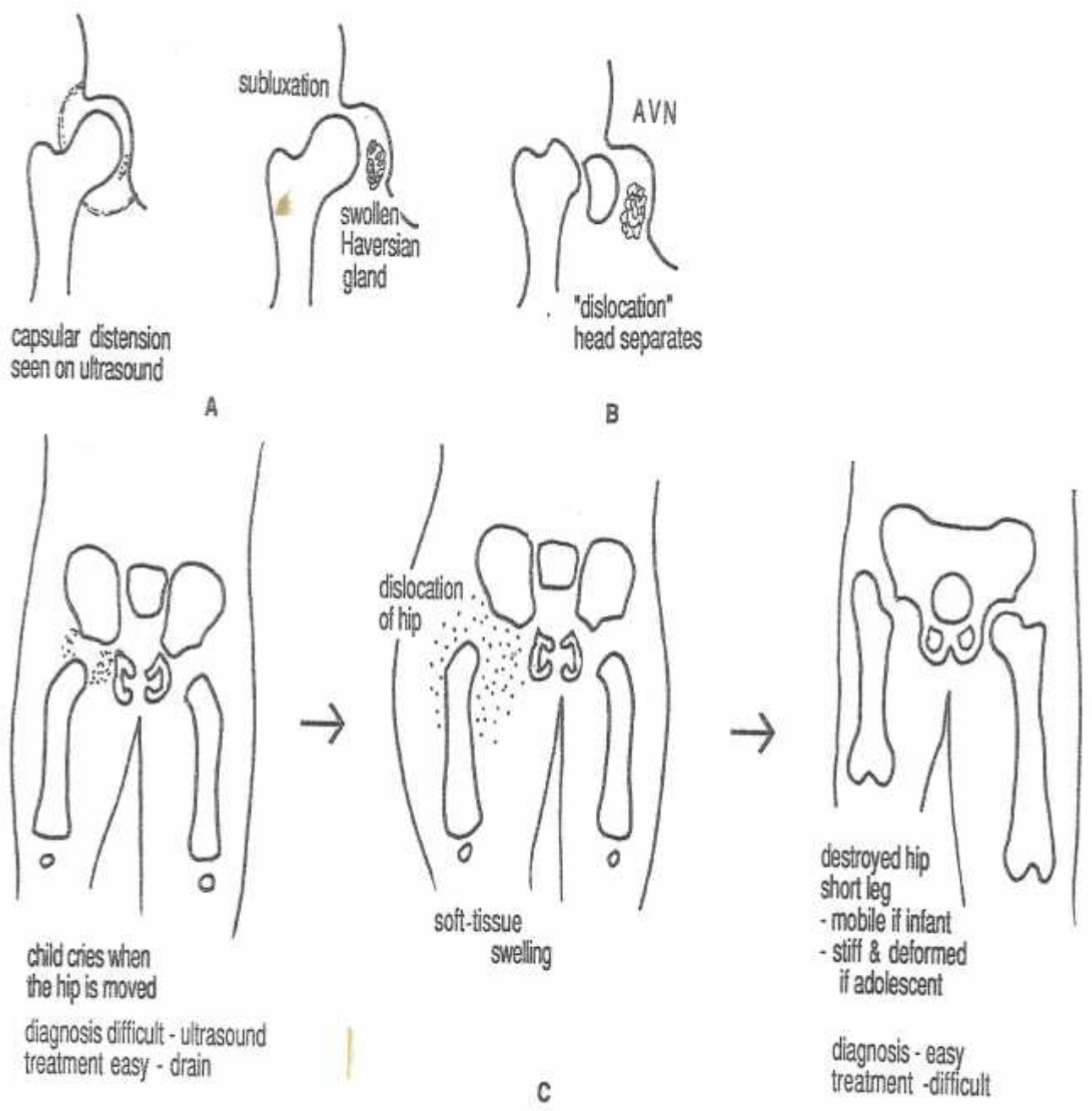
Pathological changes

Bacteria begin the destructive process. The purulent exudates from the white blood cells, as well as the enzymes and the lysozymes of both the white cells and bacteria, are chondrolytic and rapidly break down articular cartilage.[19-20] In addition to this, the presence pus within the joint stop the normal nutritive functions of synovial fluid.

A large variety of enzymes (e.g., proteases, peptidases, collagenases) are released from the leukocytes, the synovial cells, and the cartilage. These enzymes are capable of degrading the matrix and the collagen of articular cartilage. In addition, organisms

(e.g., *S. aureus*, several Gram-negative bacteria) liberate extracellular proteolytic enzymes.[19-20] These enzymes initiate the first measurable change in the articular cartilage, the loss of glycosaminoglycan. This can occur as early as 8 h in experimental models, and is not detectable by visual inspection.[21] It renders the cartilage less stiff and perhaps subject to increased wear. Collagen destruction occurs later in the process, and is responsible for the visual changes that may be seen. It is important to understand that these destructive mechanisms do not require the continued presence of live organisms to be sustained.

As septic arthritis progress, the intra-articular pressure from the presence of the exudates may rise, interfering with the venous blood flow from the femoral head portion and resulting in an avascular necrosis of the capital epiphysis.[5] The destruction may not be apparent for many months until the typical changes of growth failure and the secondary changes of increased density occur. As the acute destructive phase progress, capsular softening and stretching occur, possibly followed by a pathologic dislocation of hip. (Fig 3).



The natural history of septic hip treated by antibiotics alone. ***A:*** *Adolescent hip.* ***B:*** *Child.* ***C:*** *Infant hip. AVN, avascular necrosis.*

Fig. 3: The natural history of septic hip. A; Adolescent hip. B; Child hip C; Infant hip. AVN, avascluar necrosis.[22]