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REVIEW

A review of septic arthritis in children

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Abstract
Septic arthritis is a bacterial joint infection which most commonly occurs in children younger than five years of age. Diagnosing septic arthritis in children can be challenging as signs and symptoms can be non-specific. An accurate history, physical exam, laboratory investigations and imaging can contribute to the timely diagnosis of septic arthritis and limit chronic morbidity of joint dysfunction. The aim of this article is to review the epidemiology, clinical features, pathophysiology, differential diagnosis, special investigations, and management of septic arthritis in children.

Introduction

Septic arthritis (SA) is a bacterial synovial joint infection that most commonly occurs in young children with the highest incidence in those younger than five years of age.[1,2] This condition is an orthopaedic emergency and delay in the diagnosis and inappropriate management can lead to lifelong disability.[2] SA is typically a monoarticular condition with the most commonly affected joints being the knees, hips and ankles which account for up to 80% of the cases, but any joint can be affected.[3] A retrospective study done at Steve Biko Academic Hospital, Pretoria, South Africa between 2005 and 2009 described the phenotype of 44 children, less than 12 years of age with a suspected diagnosis of SA.[4] This study

found that the knee was the most commonly affected joint followed by the hip, shoulder, elbow and ankles.[4]

It is well known that diagnosing SA in children can be challenging, as signs and symptoms overlap with other joint pathologies. This is especially true for neonates and infants in whom non-specific signs can include fever, refusal to feed, crying and limitation of limb movements. Older children typically present with joint immobility in association with fever, malaise and pain. Around 20% of children have a history of injury to the affected limb or a non-specific fall before presentation.[3]

An accurate history, physical exam, laboratory investigations and imaging can contribute to the timely diagnosis of septic arthritis. Management of SA includes drainage of the affected joint and appropriate antibiotics. The aim of this article is to describe recent developments in the diagnosis and management of SA.

Epidemiology and risk factors

Overall incidence of SA is 4 to 10 per 100 000 children in well-resourced countries and 1 in 5000 children in sub-Saharan Africa.[3,5–7] The incidence of SA in South Africa is unknown due to lack of literature regarding its epidemiology.

A systematic review by Gigante et al. described the following risk factors for developing SA: young, male children (infants and toddlers) and presence of any immune suppressive condition such as prematurity, low birth weight, small size for age and sickle cell haemoglobinopathy.[7] Reveille reports that the incidence of SA in people living with HIV is similar to the incidence of SA in the general population, but that SA in the former group is usually caused by atypical organisms.[8]

Pathophysiology

Septic arthritis occurs from haematogenous spread, direct inoculation, spread from nearby osteomyelitis or surrounding soft tissue infections.[3] In the case of haematogenous spread, bacterial seeding occurs with subsequent lodging in highly vascular joint synovium.[9]

In neonates and children less than 18 months of age, transphyseal blood vessels allow communication between the growth plate and epiphyseal cartilage supplying a route for bacteria to spread from an osteomyelitic focus in the metaphysis to the epiphysis and subsequently to the joint lumen and vice versa.[9,10] Direct inoculation can occur through animal bites, penetrating injuries or medical procedures such intra articular injection of medicine.

Irreversible damage to the joint articular cartilage is caused by bacterial toxins, proteases from synovial cells and the increased pressure from pus formation within the joint capsule.[3,6,11] This increased intracapsular pressure can lead to avascular necrosis and is especially seen in the femoral head if SA is not promptly treated. Joint destruction happens within 8 hours after bacterial inoculation, emphasising the need for urgent diagnosis and management of SA.

Clinical features

Clinical features of SA are non-specific. Children can present with a warm, tender, red and swollen joint which fits the clinical manifestations of a number of other joint pathologies.[2] As the infection progresses, symptoms can rapidly progress over a few hours. Joint inflammation causes stretching of the joint capsule leading to pain with a limited range of movement in the affected joint as well as fever and malaise. SA of the lower limb can present with pseudo paralysis and refusal to weight bear. In neonates, signs and symptoms of SA can be absent. Neonatal patients can present with irritability, malaise and refusal to feed. Clinicians should thus keep a high index of suspicion in this age group.[5] In contrast to older children, neonatal patients with SA can have more than one joint involved.[5]

Differential diagnosis

Transient synovitis (coxitis fugax): this condition presents in a child between the ages of 3 to 8 years of age. It is a self-limiting condition that is managed without antibiotics and non-operatively. Patients present with acute onset of hip pain, refusal to weight bear in the absence of fever. Kocher criteria can help to differentiate this condition from SA. Transient synovitis is plausible when no predictors are found.[2]

Juvenile idiopathic arthritis: this condition is typically a polyarticular arthritis and has a gradual onset of symptoms. Joints are warm and swollen but not especially painful and the arthritis is symmetrical.[2]

Other differential diagnoses include slipped capital femoral epiphysis, malignancy, osteomyelitis, pyomyositis and cellulitis.

Causative Organisms

Staphylococcus aureus remains the most common cause of SA. In recent years, Panton-Valentine leucocidin (PVL) has been identified as a cytotoxin produced by some strains of *Staphylococcus aureus*. PVL induces pore formation in leukocyte cell membranes thus acting as a virulence factor. PVL producing *Staphylococcus aureus* SA are associated with longer hospital admissions, complicated infections with higher rates of septic shock, prolonged antibiotic use and a greater number of surgical interventions.[3,12])

Other common pathogens include *Kingella kingae*, *Streptococcus pneumoniae, Streptococcus pyogenes and Haemophilus influenzae* type B, Table 1.[2–4,13]

Atypical organisms that cause SA in immunosuppressed children in South Africa include Group B streptococci, *Escherichia Coli*, *Pseudomonas aeruginosa* and *Salmonella spp*.[2,4]

In cases of fastidious organisms, PCR can be performed on pus. This technology is not routinely available in routine laboratories.[2,5] In the South Africa context, pus should also be sent for tuberculosis diagnostic testing.

Table 1: Common bacterial pathogens associated with septic arthritis in children.[14]

Age group	**Bacterial pathogen**
Neonates	*Staphylococcus aureus* Group B streptococcus Gram-negative bacilli
Infants and toddlers (3 months to 3 years)	*Staphylococcus aureus* *Kingella kingae* Group A streptococcus *Streptococcus pneumoniae* *Hemophilus influenzae* (if not vaccinated)
Children (>3 years to 11 years)	*Staphylococcus aureus* *Kingella kingae*
Adolescents (>11 years to <18 years)	*Staphylococcus aureus* Gonococcal infections (in sexually active patients)

Laboratory studies

Initial studies should include a full blood count with differential, erythrocyte sedimentation rate (ESR), C reactive protein (CRP) and blood cultures (BC). These tests are helpful in making a diagnosis but alone cannot make a definitive diagnosis. Normal values of these tests do not exclude septic arthritis.

In 1999 the Kocher criteria were developed to aid in differentiating between SA and transient synovitis of the hip in children.
The criteria included the following four parameters:

a. history of fever,
b. non-weight bearing,
c. ESR of more than 40mm/h and
d. serum white blood cell count of more than 12 000 cells/mm^3.

According to the number of positive parameters present, patient probability of having SA of the hip is categorized into low or high probabilities.[2] A diagnosis of transient synovitis is plausible when no predictors are found.[2] In recent years, C reactive protein (CRP) levels of more than 20 mg/L have been added to the Kocher criteria as this laboratory test generally has a quicker turn-around time. The predicted probability of SA for the Kocher criteria ranges from 59 to 99.6% and remains similar when CRP is added, **Table 2**.[2]

Table 2: Number of Kocher criteria predictors present and predicted probability of septic arthritis.[15]

Number of predictors from Kocher criteria present	Predicted probability of Septic Arthritis (%)
0	<0.2
1	3.0
2	40.0
3	93.1
4	99.6

Imaging

Imaging of affected joint should commence with plain radiographs. This will aid in diagnosing osteomyelitis, fractures or neoplasms as the cause of a painful joint. Acute septic arthritis will most likely have normal radiographs aside from soft tissue swelling. In neglected SA, articular cartilage destruction will be evident by joint space narrowing and subchondral bone erosion.[3] It should be stressed that plain radiographs are not sufficiently sensitive to diagnose or exclude SA.[7]

Ultrasound is a rapid, non-invasive, non-irradiating test which is helpful in demonstrating joint effusions. It is especially helpful when assessing deep joints such as the shoulder and hip where palpation cannot reliably detect a joint effusion. An ultrasound where no joint effusion is detected within the first 24 hours after onset of symptoms, should be interpreted with caution. Ultrasound is unable to differentiate between SA and transient synovitis.[3,7]

Magnetic Resonance Imaging (MRI) is the gold standard imaging method in SA. It has a high sensitivity and specificity to detect SA, especially in early phases of the disease, **Figure 1**. MRI is also indicated when SA of more than one joint is suspected as it will aid the surgeon in planning surgical intervention. Furthermore, avascular necrosis may complicate SA of the hip and MRI can delineate early ischaemia. In SA of the shoulder and elbow there is a high risk of osteomyelitis and MRI can diagnose these complications.[3,7]

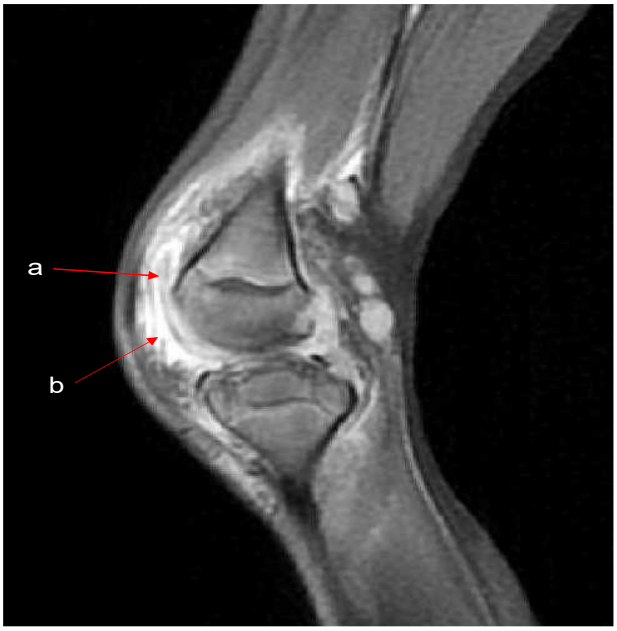

Figure 1. This image is an MRI scan of the right knee from a 6-year-old boy with septic arthritis. It shows (a) joint effusion with (b) extensive thickening and enhancement of the synovium.

Management

The mainstay of treatment involves prompt drainage and debridement of purulent material from the joint space and early treatment with antibiotics.[16]

Surgical management

Joint(s) affected by SA should be drained. There are three methods to achieve this: arthrocentesis, arthroscopy and arthrotomy. A systematic review done by Gigante et al. was unable to find consensus on the type of drainage procedure nor the timing of the intervention for SA for different joints. The authors did however find that surgical intervention becomes necessary when the clinical picture does not improve, and CRP does not decrease within 24 hours of antibiotic use.[7]

Medical management

In immune-competent hosts, empiric antibiotics that cover Gram-positive organisms should be selected based on local antibiogram patterns. Antibiotics should also cover *Staphylococcus aureus* SA until culture results become available. In immune-suppressed hosts, broad spectrum antibiotics which cover both Gram-positive and Gram-negative organisms should be selected based on local antibiogram patterns.

Evidence regarding the choice and duration of antibiotic treatment is sparse as no randomised control trials have been conducted thus far.[16] There is also no information available with regards to optimal duration of antibiotic use, timing of switching from intravenous to oral antibiotics and whether oral treatment is non inferior to intravenous treatment.

Donders et al. recommend that antibiotics should only be commenced once pus has been sampled for microbiology investigations unless a patient has signs of systemic sepsis, in which case antibiotics should be administered without delay.[2] The reason for this being that antibiotics administered before pus sampling decreases the culture yield and thus appropriate antibiotic therapy which prolongs a

patient's admission to hospital. Gigante et al. does not differentiate between the timing of empiric antibiotics, but states that antibiotics should be initiated immediately.[7]

Empiric antibiotic therapy should be changed to definitive antibiotics once sensitivity results become available. The duration of antibiotic therapy remains unsure, with authors agreeing that the patient response and normalisation of CRP should be the main indicator when selecting duration of therapy.

Conclusion

SA requires urgent recognition and treatment to avoid disability. The most common pathogen for SA in children remains *Staphylococcus aureus*. Treatment involves antibiotic therapy and surgical drainage.

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