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Paediatric Musculoskeletal Infection– A Review

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Abstract

Acute paediatric musculoskeletal infections (MSKI) include septic arthritis, acute osteomyelitis and pyomyositis. Prompt treatment is necessary to prevent long term disabilities in children. In this review, we discuss the etiopathogenesis, clinical features and management of MSKI. We also discuss about the role of new markers of inflammation and MRI in MSKI. The clinical presentation is variable, depending upon the age group, and difficult to distinguish from other pathologies. Diagnosis is therefore based on not only clinical presentation but also laboratory and radiological investigations. The mainstay of treatment includes antibiotic therapy, and surgical decompression.

Keywords: Septic arthritis, Musculoskeletal infections, Osteomyelitis, Pyomyositis

Introduction

Acute paediatric musculoskeletal infections (MSKI) comprise mainly septic arthritis, acute osteomyelitis and pyomyositis. The overall incidence of children suffering from MSKI is 5- 20 cases per 100,000 population [1].

Musculoskeletal infections, if not treated timely and appropriately can lead to severe long-term disability in children such as limb length discrepancies, angular deformities, arthritis, chronic osteomyelitis and pathological fractures. In presence of subtle clinical signs in children, clinicians find it difficult to diagnose these infections promptly. The diagnosis is therefore aided by radiological and laboratory investigations. Moreover, challenges are still present for the treating clinician in managing these children (especially neonates) as the evidence-based guidelines are not well defined. Therefore, in this article, we review the etiopathogenesis, clinical features and current practices for treating MSKI in the paediatric age group.

Epidemiology

Among all paediatric MSKI, acute osteomyelitis is probably the most common with an overall incidence of 3-100 per 100000 population (Table 1). These infections are more common in children less than 5 years of age [2-8]. Overall distribution of MSKI is given in Figure 1. Although, Staphylococcus aureus is responsible for approximately 65% and 50% cases of culture positive acute osteomyelitis and septic arthritis respectively, the overall incidence of Kingella kingae is on a rising trend [4, 5]. It is the most common reported organism in culture negative cases. A more detailed bacteriology profile of MSKI is given in Table 2.

Pathogenesis

The most common route of spread of MSKI is hematogenous. Joint synovium is highly vascularized and lacks a limiting basement membrane and, hence pathogen can seed the synovial space [9]. The vessels in the metaphysis are tortuous. Earlier, it was believed that the metaphysis had a hair pin arrangement of vessels which made the blood flow sluggish but now it is suggested that metaphysis is rich in many small

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Table 1: Incidence of MSKI in children

MSKI	Incidence
Osteomyelitis [4]	3-80 per 100000
Septic arthritis [6, 7]	3-30 per 100000
Pyomyositis [8]	10-20 per 100000

Table 2: Common pathogens causing MSKI with their associated risk factors

Common pathogens	Associated risk factors
S aureus	Most common across all age groups
K kingae	3 months to 4 years
Group B streptococcus	Most common in Infants
Streptococcus	More than 5 year
Salmonella enterica	Immunocompromised
Pseudomonas aeruginosa	Penetrating injury
Pasteurella canis	Animal bite
Salmonella typhi	Sickle cell disease
Neisseria gonorrhoeae	Sexually active adolescent/ sexual abuse
H influenza	Rare due to immunization

terminal vessels with leaky endothelium which makes it a favourable environment for the bacterial growth [10].

Rarely, it can also result from direct inoculation from the puncture wounds and intra-articular injections, or contiguous spread. Trauma to musculoskeletal tissue is also a significant risk factor [11]. For instance, hematoma can develop after a severe injury in the skeletal muscle, creating a focal point for infection leading to pyomyositis [8].

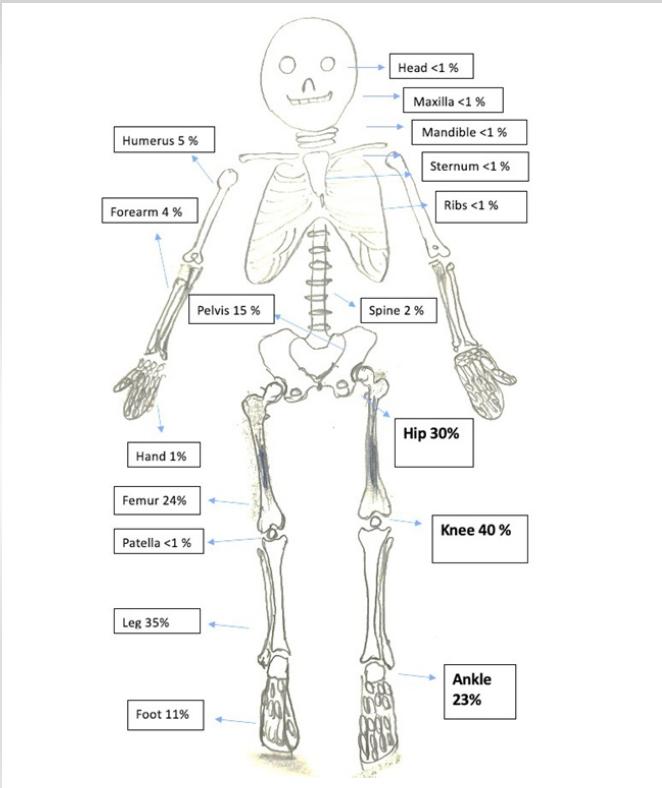

Figure 13: Anatomic distribution of musculoskeletal infections in children.

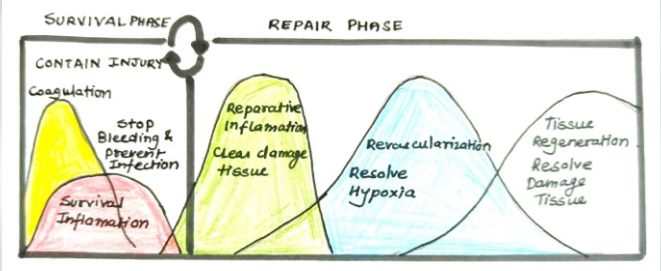

Figure 2: Acute phase reaction of the body in response to musculoskeletal infection.

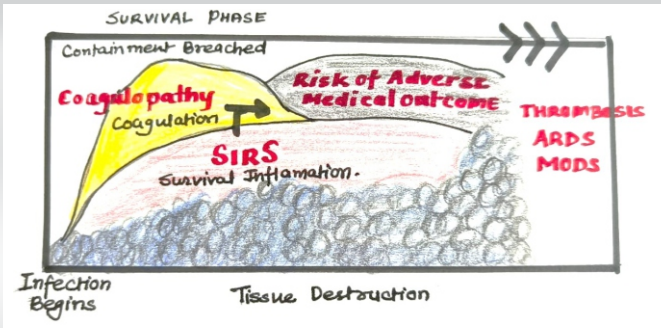

Figure 3: Hijacking of the acute phase reaction initiated by the body by the invading bacteria

- SIRS (Systemic inflammatory response syndrome)
- ARDS (Acute respiratory distress syndrome)
- MODS (Multiple organ dysfunction syndrome)

Recently, it was argued by Rosenfeld et al. that bacteria have preference for regenerating tissues or rapidly growing bone and muscle in children. This is also the possible explanation for infection being common in children during rapid growth years (<5 years) and why certain joints (hip and knee) and bones (femur and tibia) are more predisposed to MSKI [12].

After the bacteria has seeded the bone, joint or muscle, a cascade of inflammatory pathway known as acute phase response (APR) is initiated by a coordinated effort between coagulation cascade and survival inflammatory response (SIR) to seal the infection (Fig. 2). However, in MSKI, bacteria hijack this coordinated response by attacking the coagulation system leading to a continuous tissue damage by virtue of the virulence factors (Fig. 3) [12]. If the infection is contained by APR, it is followed by reparative phase in which necrotic tissue is removed by rapid revascularization [13]. After the necrotic material is removed, repair of the damaged tissue begins. However, if the infection is not contained, it can spread along the path of least resistance, eventually becoming chronic leading to catastrophic sequelae.

Clinical presentation

Irrespective of MSKI, children often present with pain, limited range of motion, difficulty in bearing weight, local signs of inflammation such as redness and swelling and systemic symptoms including fever. However, symptoms may vary according to the age and location of the infection. For example,

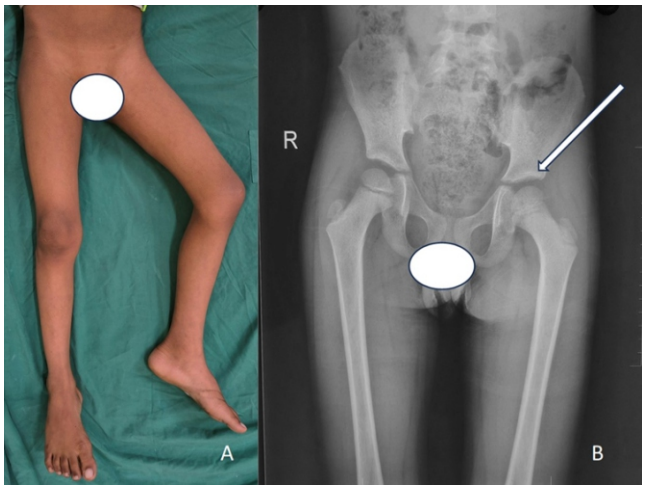

Figure 4: 5-year-old child was diagnosed with septic arthritis of the left hip joint. A. Clinical photograph shows the flexed, abducted and externally rotated position of the left hip joint. B. Unremarkable radiograph of the left hip joint early in the infection (marked by arrow).

deep infections may not show superficial signs. Similarly, neonates and infants have minimal systemic features and often present with hypothermia and refusal to feed. Parents usually come with complaints of excessive cry during diaper change and pseudo-paralysis of the limb. It is also not uncommon to see multifocal infections in this age group [14, 15]. The overall incidence of multifocal infection can be as high as 30% in the neonatal age group [16]. In septic arthritis, joints usually assume the position of comfort as the capsule is stretched by the accumulating pus. For instance, the hip assumes a flexed, abducted, and externally rotated posture when there is collection in the joint (Fig. 4a).

Laboratory measures of MSKI

These usually detect the host response to the MSKI. Serum biomarkers are of 2 types: plasma acute phase reactants [C-reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR), Interleukin-6 (IL-6), serum procalcitonin] and cellular acute phase reactants (complete blood count (CBC), neutrophil lymphocyte ratio (NLR), and platelet count) [17]. As per British Society for Children's Orthopaedic Surgery (BSCOS) Paediatric Musculoskeletal Infection Consensus Group (UK) (2023), CBC, CRP and ESR must be sent in every case of MSKI [18].

• CRP – It is one of the most sensitive markers for diagnosis of MSKI [19]. CRP level rises within 4-6 hours of onset of symptoms and peaks within 48 hours of symptom onset, and it starts decreasing within 6 to 7 hours of treatment initiation. CRP level at the time of admission also correlates with infection severity as its level increases proportional to the degree of tissue inflammation. Beyond severity, its serial measurement also guides the management of MSKI. Treatment failure is

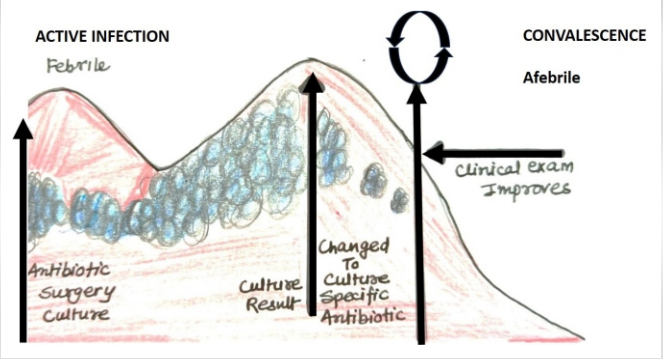

Figure 5: Trend of serial CRP level in musculoskeletal infections.

considered if there is no improvement in CRP levels within 48 hours of initiation of medical treatment indicating the need for surgical intervention [17]. It also plays a prognostic role, as increase in CRP levels by 10 units increases the probability of poor outcome by 30% [20].

• ESR – It is a surrogate marker of fibrinogen and estimates the host response mounted to contain the infection [21]. It is a less dynamic and a long-acting marker when compared to CRP (Fig. 5). ESR peaks at 3–5 days after the onset of infection and normalizes within 7–10 days after initiation of therapy [22].

• IL-6 – It is normally stored in musculoskeletal tissue, and is released into bloodstream very rapidly in MSKI [23]. It is not used currently as a marker for diagnosis of MSKI because of its short half-life.

• Serum procalcitonin – It is a very sensitive marker. Levels less than 0.3 ng/ml rules out infection and levels > 0.5 ng/ml strongly correlates with infection [24]. In MSKI, it can rise upto 1000 times the normal and starts decreasing within 72-96 hours after starting the treatment [25, 26].

• CBC - Raised TLC is seen with neutrophil predominance (left-shift). However, it is the least sensitive marker for MSKI as the counts may vary with age. Moreover, elevated TLC count may be found in only 25% cases [20]. Alternatively, differential leukocyte counts (DLC) can be used for MSKI. Neutrophils are the primary cellular player against the infection. However, neutrophils alone cannot detect and assess the severity of infection. Newer evidence suggests that instead of CBC or DLC, NLR is a superior marker of septicaemia. It indicates both, the severity and prognosis of MSKI [27]. Increase in NLR is associated with treatment failure and mortality. Lastly, platelet count can also be used to assess MSKI as they are the first line of defence in MSKI. Thrombocytopenia and thrombocytosis can be seen and is associated with poor outcomes [28, 29].

Culture

They not only help us to detect the offending organism but also provide antibiotic sensitivity. Hence, blood culture along with synovial fluid culture and/ or pus culture must be sent in all

cases of suspected MSKI. However, blood and pus culture may be negative in significant number of cases. This may be because of the antibiotic administration before collecting the sample. Secondly, routine culture techniques may not be able to detect fastidious organism like K. kingae. Recently, genomic analysis in the form of PCR and new generation sequencing (NGS) are introduced which are both highly sensitive and specific for diagnosis of MSKI [30]. These are much superior than traditional culture method as they can detect polymicrobial infection, even in patients already started on antibiotics. PCR also detects antibiotic resistant marker which helps choose the appropriate antibiotics. NGS includes transcriptomics which detects transcription proteins of the offending organism. Despite the multiple advantages, these new techniques are not used frequently as they are very expensive [30].

Synovial fluid analysis

A synovial fluid WBC count of 50,000/mm^3 with elevated polymorphonuclear cells (>90%) is highly suggestive of septic arthritis [31]. However, it is neither 100% sensitive nor 100% specific. Lactic acid levels in the synovial fluid increase in response to sepsis, but also in response to any type of joint inflammation. Synovial fluid lactic acid levels have not been shown to have any discernible value [32]. Recent research has also explored leukocyte esterase analysis of joint fluid as a new diagnostic technique for native joint infections [30]. The test takes 90 seconds to complete and has the potential to reduce the time to definitive therapy [30]. Blood contamination of the sample has been shown to cause a false-positive result. Further research on the test is ongoing.

Imaging

In MSKI, radiographs of the affected part should be obtained as the first line of radiological investigation for diagnosing MSKI. Radiographic imaging may be normal in the setting of acute infections (Fig. 4b). It is also used to rule out other bone pathologies such as fractures, and malignancies. The first radiographic sign of infection may emerge between days 4 and 10 after infection or even later [33]. Although radiographs are cheaper and are readily available, they underrepresent the extent of disease. Ultrasonography is appealing due to its low cost, relative availability, noninvasive nature, lack of ionizing radiation, and lack of anesthesia requirements. Pathological changes such as deep soft tissue oedema, periosteal elevation and circumferential soft tissue abscesses can be detected as early as 48 hours from the onset of infection [34, 35]. The yield of the aspirate may be increased by ultrasound-guided arthrocentesis. Follow-up scans also help in monitoring the results of intervention. Ultrasound's utility has been restricted due to its lack of specificity, reliance on operator expertise, and inability to image marrow or display cortical changes. Bone scan may

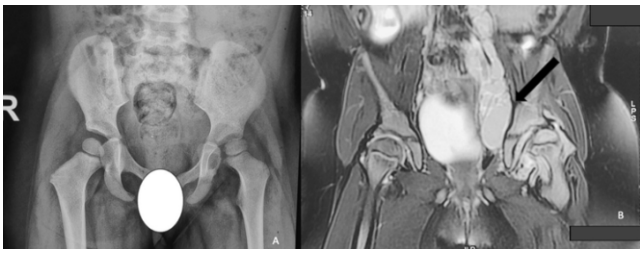

Figure 6: Two-year child came with complaints of pain and limitation at left hip joint for past 5 days. A. Subluxed hip joint left side due to septic effusion. B. MRI showed additional collection along the left psoas muscle extending into the left hip joint (marked by arrow).

exhibit widespread, faintly enhanced tracer absorption in the bone or joints. But it cannot distinguish between MSKI from inflammation. Pus and fluid within the joint, particularly in the hip raises intracapsular pressure and can impair epiphyseal perfusion, resulting in decreased epiphyseal tracer absorption. This may cause false negative results in 50% of the MSKI cases [36]. However, it is particularly helpful in diagnosis of multifocal infections. Computed tomography (CT) may assist in detecting bone and cartilage involvement. While CT scans can detect effusions and guide joint aspirations, they are regarded inferior to MRI in this setting as it gives excellent bony details but poor soft tissue visualization. MRI is now considered as the gold standard imaging modality for MSKI and should be done in every case of MSKI. It helps in detecting the extent of infection and is valuable in regions such as pelvis which are difficult to image (Fig. 6a and b). Although, high cost and its relative unavailability limits it use. MRI should be done wherever possible. However, if child's condition doesn't permit, USG of the affected part must be done. Another limitation of MRI is that child often requires sedation which may further delay the management. But with availability of fast sequencing MRI, sedation requirements can be omitted and scans can be done in a short span of 15-20 minutes [30].

Treatment Approaches for Paediatric Musculoskeletal Infections

The aim of treatment in MSKI is to eradicate the pathogen by either antibiotics or by both antibiotics and surgery in order to limit the tissue damage.

Antibiotic therapy

Determining the pathogenic organism in MSKI is critical for selecting the appropriate antibiotic, and managing the disease. There is considerable debate in the literature on the appropriate antibiotic to use, mode of antibiotic delivery - intravenous versus oral, the factors that influence the decision to switch to oral antibiotics, and the duration of antibiotic use. In neonates <3 months of age, combination of vancomycin and gentamycin is the recommended empirical treatment as this covers most

common pathogens of MSKI in this age group. In older children, a combination of first- generation cephalosporin along with vancomycin is preferred if MRSA prevalence in the community is ≥10%. This will also cover Kingella kingae [1]. Once the pathogen has been identified, treatment should be tailored according to the antibiotic sensitivity [37].

The duration of intravenous therapy is a point of contention, with recommendations ranging from two to seven weeks [38]. Prolonged antibiotic treatment is frequently required in disseminated infections. Injectable antibiotics are shifted to oral antibiotics when there is resolution of clinical symptoms and reduction in inflammatory markers. Efforts should be made to switch intravenous to oral antibiotics to reduce side effects of injectable antibiotics.

Surgical intervention

There is a considerable debate in literature about the surgical decision making in patients with MSKI. Surgery is indicated in osteomyelitis when despite injectable antibiotics, there is worsening of clinical features and inflammatory markers. WBC count more than 20,000 mg/dl and CRP greater than 20 mg/L warrant further localization of source of infection and possible surgical drainage [39]. The surgical procedure may include subperiosteal drainage of abscess with or without drainage of bone. The drainage of bone can be done by making multiple drill holes or by creating a window.

Arthrotomy is considered appropriate even in the presence of clinical suspicion. Swarup et al. in their review concluded that the surgical debridement whether open or arthroscopic, is effective in removing the infection [40]. Joint aspiration may be a viable alternative if the lesion is detected early. Superficial joints such as elbow and ankle joints can be aspirated percutaneously. Deeper joints like hip generally need open arthrotomy and debridement as repeated aspirations are difficult in deeply located joints. If aspiration/ drainage is not followed by a rapid resolution of clinical symptoms, then, this often indicates the presence of residual, multifocal or recurrent infection, and re-exploration must be considered. Delay in surgical intervention after 4–5 days is associated with poor outcomes and permanent destruction of joint is seen in majority of the cases after a delay of 5 weeks [40, 41]. Growth abnormalities due to physeal involvement can manifest up to 9 years after the acute event. Hence all children with MSKI should be monitored until skeletal maturity [42].

Indications for surgical intervention for pyomyositis are often based on the size of abscess and the existence of contiguous infection. Primary abscess usually responds to ultrasound or CT-guided percutaneous drainage and may obviate the need for open surgical drainage, while secondary abscesses require intervention directed at the primary pathology along with drainage.

Local antibiotic delivery systems

Local antibiotic delivery systems appear to offer an advantage over systemic therapy because higher levels of antibiotics can be delivered to the tissue while avoiding adverse side effects of systemic therapy. It can also be used as an adjunct to surgical debridement. Historically, beads made of poly methyl methacrylate (PMMA) and loaded with antibiotics were used to treat chronic infections and infected non-unions. But, a second surgery was often required for its removal. That is why, bone graft substitutes laden with antibiotics such as calcium sulphate, calcium phosphate, and combination of calcium sulphate and calcium phosphate gained popularity in the recent past because of their osteoconductive properties as well as in their ability to get reabsorbed.

The experience with local antibiotic delivery system to treat acute MSKI is limited. However, in a recent study by Zhang et al., 22 infants (age <12 months) with acute osteomyelitis were treated by debridement and resorbable calcium sulphate substitutes combined with 0.5 g of vancomycin. Complications included limb length deformity, avascular necrosis, and pathologic subluxation of the hip in seven out of 22 patients. Fifteen out of 22 patients had satisfactory outcomes. Furthermore, patients with concurrent septic arthritis were more likely to experience problems than those with isolated OM. The authors concluded that calcium sulphate is a viable option for treating acute hematogenous osteomyelitis in the presence of a bone lesion [43].

Conclusions

Early recognition and prompt control of infection using appropriate antibiotics with or without surgical decompression is essential in preventing long-term sequelae following MSKI. Close monitoring of the patient's clinical response and laboratory markers is crucial in assessing treatment efficacy and adjusting the therapy as needed.

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