Review Article

Indian Journal of Trauma and Emergency Pediatrics
Volume 9 Number 1, January - March 2017
DOI: http://dx.doi.org/10.21088/ijtep.2348.9987.9117.10

Septic Arthritis in Children: A Medical Emergency

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Abstract

Septic arthritis (SA) in children is considered as a medical emergency. If untread, it may destroy the joint in a period of days. The infection may also spread to other parts of the body. SA results from bacterial invasion of the joint space through the blood stream, from adjacent osteomyelitis, or through direct inoculation of the wound. Staphylococcus aureus is the most common cause of SA in all age group. Among those aged 15-50 years, Neisseria gonorrhoea runs a close second. Pain with an infected joint typically present with triad of fever (40-60% of cases) pain (75%of cases), and impaired range of motion. SA is a challenging clinical problem because: (1) signs and symptoms may be subtle and overlap with those found in other condition, (2) screening laboratory studies and synovial fluid cultures are relatively insensitive, and (3) optimal management, including duration of antibiotics therapy and surgical approach is not evidence based. Diagnosis of SA is based on a combination of clinical findings and results of synovial fluid analysis. Imaging studies is insensitive in the diagnosis of SA. All children presumed to have SA should be hospitalized for empiric intravenous antibiotic therapy. In general, 3-4 weeks to treat staphylococcus aureus, H. influenzae type B, or Strep. Pneumoniae infections, while gonococcal infections are treated for 7-10 days. Close follow up with physical examinations and laboratory tests must be done to make sure that patients remain afebrile, pain resolved, improved range of motion, and normalize laboratory values.

Keyword: Septic Arthritis; Microorganism; Synovial Fluid.

Introduction

Septic arthritis (SA) is the purulent invasion of a joint by an infectious agent which produces arthritis. A broader term is 'infectious arthritis', which describes arthritis caused by any infectious organism [1]. Approximately 20,000 cases of septic arthritis occur in the United States each year (7.8 cases per 100,000 persons each year), with a similar incidence occurring in Europe [2]. SA is more common in children than adults, but the actual incidence is unknown. Subgroups of children who are at risk for SA include neonates, individuals with hemophilia who are subjected to hemarthrosis, and individual who are immunocompromised, such as those with sickle cell anemia or human immunodeficiency virus (HIV) or those treated with chemotherapy. A higher incidence of SA is reported among boys than girls [3]. It is the most serious cause of joint inflammation and if not diagnosed and treated promptly can be linked with severe morbidity.

Etiology

SA is caused by bacteria, but may be caused viral, mycobacterial, and fungal pathogens as well [5]. In neonates, staphylococcus aureus is the most common cause of SA, but E. coli, group B streptococci, and other gram negative bacilli also cause the disease. In children aged 2 months to 5 years, Haemophilus influenzae type B was the most common cause of SA prior to the widespread use of vaccines; staph. aureus is now the most common [6].Other etiologies include group A streptococci and streptococcus pneumoniae. Community acquired methicillin resistant staph.

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Received on 31.10.2016, **Accepted on** 26.11.2016

aureus (MRSA-CA) is an increasingly common cause of SA in children. In adolescents, Neisseria gonorrhoea is the suspected cause for patients with either polyarticular or monoarticular disease. Group A streptococcus is reported in numerous children with active varicella- zoster infection. Salmonella is suspected in children with sickle cell anemia. Mycobacterium is a rare cause of chronic pyogenic arthritis. If identifiable risk factors are present, then a purified protein derivative (PPD) should be placed for the child with culture negative disease [7]. Pneumococcus is the causative agent of only 6% of cases of SA [8]. Septic arthritis caused by anaerobic organisms is a rare clinical entity. Anaerobic septic arthritis accounts for only 1% of all reported cases of bacterial arthritis in both children and adults [9].

Pathophysiology

Bacteria are carried by the blood stream from an infectious focus elsewhere, introduction by a skin lesion that penetrates the joint, or by extension from adjacent joint (bone or bursa) . Microorganisms must reach the synovial membrane of a joint which can happen in any of the following ways: (1) dissemination of pathogens via the blood, from abscesses or wound infections, or from an unknown focus; (2) dissemination from an acute osteomyelitic focus; (3) dissemination from adjacent soft tissue infection; (4) enter via iatrogenic trauma; (5) enter via penetrating trauma [10]. Previously damaged joints are the most susceptible to infection. The normal joint has several protective components. Healthy synovial cells possess significant phagocytic activity, and synovial fluid normally has significant bactericidal activity. Rheumatoid arthritis (JIA) and systemic lupus erythomatosus (SLE) and other pathology hamper these defensive function. Also patients with deficiencies of the terminal components of complement are susceptible to joint infections due to Neisserial bacteremia. The major consequence of bacterial invasion is damage to the articular cartilage. This may be due to the particular organism's pathologic properties, such as the chondrocyte proteases of staph. aureus, as well as to the host's polymorphonuclear leukocyte response. The cells stimulate synthesis of cytokines and other inflammatory products, resulting in hydrolysis of essential collagen and proteoglycans. Infection with Neisseria gonorrhea induces a relatively mild influx of white blood cells (WBC) into the joint, explaining in part, the minimal joint destruction observed with infection with this organism relative to destruction associated with staph. aureus infection. As the destructive process continues, pannus formation begins, an cartilage erosion occurs at the lateral margins of the joint. Later effusion impairs blood supply and result in aseptic necrosis of bone. These destructive processes are well advanced as early as 3 days in the case of untreated infection. Viral infections may cause direct invasion (rubella) or production of antigen-antibody complexes. Such immunologic mechanisms occur in infections with HBV, parvovirus B 19. Polymicrobial infections and infection with anaerobic organisms, fungi, mycobacteria,Lyme disease may produce non-suppurative joint infections [11,12].

Clinical Features

The most commonly involved joint in septic arthritis is the knee (50% of cases) followed by the hip (20%), shoulder (8%), ankle (7%), and wrist (7%). The elbow, interphalangeal, sternoclavicular, and sacroiliac joints each make up 1-4% of cases [12]. SA due to bacterial infection is commonly classified as either gonococcal or non-gonococcal [13]. Patients with an infected joint typically present with triad of fever (40-60% of cases), pain (76% of cases), and impaired range of motion. These symptoms may evolve over a period of days to a few weeks. Fever is usually low grade with rigor present in only 20% of cases. The pattern of joint involvement is an extremely important diagnostic feature of cases of non gonococcal septic arthritis, 85-90% are monoarticular. Polyarticular arthritis is usually observed in gonococcal disease, various viral diseases, reactive arthritis, and various noninfectious processes [12]. Acute joint inflammation marked by severe pain and swelling is the hallmark of SA. Joint pain results from stretching of the fibrous joint capsule. If lower extremity joints are involved, patients often report that children to bear weight and they resist all efforts to move the involved joint. Neonates are more likely to have infection in multiple joints. A septic joint is so painful that most children do not tolerate any range of movement, resulting in pseudoparalysis. If the hip or knee is involved, an ambulatory child refuses to walk or bear weight on the affected limb [14]. Children orient an affected joint in such a way to minimize the pain. The hip flexed, abducted and externally rotated. The knee, ankle, and elbow are partially flexed, whereas the shoulder is adducted and internally rotated [3]. Most of the patients have low grade fever, but a substantial number may be afebrile at presentation. Absence of fever should not spare the clinicians from the diagnosis. On the other hand, the presence or absence of fever may be helpful in distinguishing SA from transient synovitis as children with transient synovitis are usually afebrile. Swelling,

warmth and erythema are common physical sign of SA. However, because of the deep location of the hip joint, there may be no erythema or swelling noted [6].

Diagnosis

Septic arthritis (SA) should be considered wheneversome one is assessing a child with rapid onset of pain. Usually only one joint is affected, however in seeding arthritis, several joints can be affected at the same time; this is specially the case when the infection is caused by staphylococcus or gonooccus bacteria [15]. The diagnosis of SA can be difficult as no test is able to completely rule out the possibility. Because joint infections are uncommon, special attentions to be given on features of the patient's history that may indicate an infections process instead of a primary rheumatologic or orthopedic process. Attention to be paid to the following symptoms: acuteness of the joint pain; whether the pain is superimposed on chronic pain; previous history of joint disease or trauma; whether the process is monoarticular or polyarticular and which joints are involved; the presence of extraarticular symptoms; whether the patient has had vascular invasion due to catheterization or IV use. History to be obtained regarding the possible presence of sexually transmitted disease (STDs) or exposure to ticks (Lyme disease) [13]. When evaluating a child with suspected septic arthritis, also conditions such as primary rheumatologic disorders (e.g., vasculitis), drug induced arthritis and reactive arthritis (e.g., post infectious diarrhea syndrome), post meningococcal and post gonococcal arthritis, arthritis of intrinsic bowel disease to be considered [12]. Diagnosis of SA is established by a combination of history, clinical findings and results of synovial fluid analysis. Clinician should have a low threshold for performing arthocentesis, especially for children with a painful monoarthritis, significantly limited range of motion, and no plausible noninfectious explanation. When SA is suspected, synovial fluid should be obtained for a complete blood count, glucose, gram stain and culture. Synovial culture has poor sensitivity (60%-70%). A synovial fluid WBC count of more than 50,000/ml suggests SA, especially if the count exceeds 100,000/ml or if a predominance of polymorphonuclear cells is observed. The synovial fluid glucose conc. averages 30% of that in the serum, a finding unique to SA. ESR is typically elevated. C-reactive protein (CRP) is more sensitive marker for SA than is the peripheral WBC count [16,17]. A study evaluated the clinical utility of PCR as a supplemental diagnostic tool in the evaluation and treatment of children with SA. The study concluded that PCR provides supplemental information for diagnostic confirmation through an increased rate of detection of bacteria in the synovial fluid [18]. A lactate level in the synovial fluid of greater than 10mmol/1 makes the diagnosis very likely [18]. At least 2 sets of blood cultures should be obtained to rule out a bacteremic origin of septic arthritis. In the setting of possible gonnococcal infection, obtaining swabs for culture cultures from the patient's rectum, urethra, cervix and from any skin lesion is most helpful [20]. Plain X-ray is of limited value is evaluating a joint for infection; periarticular soft tissue swelling is the most common finding. This imaging mortality is most useful in ruling out underlying osteomyelitis or periarticular osteomyelitis caused by the joint infection itself. Ultrasonography may be used to diagnose effusion in chronically distorted joint (secondary to trauma or rheumatoid arthritis). Computed tomography(CT) scanning and magnetic resonance imaging(MRI) are more sensitive for distinguishing osteomyelitis, periarticular abscesses, and joint effusion [22].

Differential Diagnosis

The differential diagnosis of SA is rather extensive including juvenile idiopathic arthritis(JIA), Kawasaki disease, Lyme disease, serum sickness, rheumatoid fever, SLE, transient synovitis. In contrast to children with SA, children with transient synovitis appear well and usually afebrile with just a mild limp [23]. In adolescents, a slipped capital femoral epiphyses may manifest as a painful hip, thigh or knee. Most patients are afebrile and the onset of pain may be preceded by minor trauma. Legg-calve-perthes disease, which occurs mostly in boys, afflicts children aged 4-8 years. In contrast to SA, the pain is subacute, with a more indolent onset, and without fever [24].

Complications

Meningitis (10-30%), osteomyelitis, cellulitis (10-30%), and pneumonia (5%) are potential complications in young children with septic arthritis (SA) resulting from hematogenous spread of microbial agents. Osteonecrosis, growth arrest, and sepsis are potential complications from SA of any etiology. Because of the availability of antibiotics, children rarely die from SA or its complications. Although chronic arthritis is uncommon, the short-term morbidity and costs in terms of prolonged antibiotic therapy and hospitalization may be substantial [16].

Management

All children presumed to have septic arthritis (SA)

must be hospitalized. Medical management of SA focuses on adequate and timely drainage of the infected synovial fluid, administration of appropriate antimicrobial therapy, and immobilization of the joint to control pain. Initial antibiotic choices must be empirical, based on the sensitivity pattern of the pathogens in the community. The risk of resistance among potential bacteria must be considered when choosing an initial antibiotic regimen. Knowing the resistance patterns in the community, as well as in the hospital, is most important. Typically, a clinician chooses an antibiotic before synovial fluid results are known. Thus the child's age and risk factors, as well as gram stain results should influence initial antibiotic coverage. Neonates are at risk for infections with gram negative organisms, such as E.coli, and gram positive organisms, such as staph. aureus or group B streptococcus. For neonates without meningitis, a semi synthetic penicillin (eg-oxacillin) plus an aminoglycosides (e.g., gentamicin) may be used. Neonates with concomitant meningitis and SA present a therapeutic challenge in which case a combination of vancomycin and a third generation cephalosporin is a resonable choice for initial coverage. *Staph.aureus* is the most common cause of SA among non-neonates. Oxacillin alone should provide adequate coverage in children who are immunocompetent, assuming that they are immunized for H influenza type B. However, in communities in which methicillin resistant *staph.aureus* (MRSA) is prevalent, clindamycin is a better choice. A third generation cephalosporin is the initial therapy for an adolescent possibly infected with gonococcus. Once culture results and sensitivities are known, antibiotic selection can be more specific. The optimal duration of antibiotic therapy is not defined, recommendations vary from 1-6 weeks. Thus, 3-4 weeks of antibiotic therapy is used to treat *staph. aureus*, H influenzae type B, or strep pneumoniae infections, while gonococcal infections are treated for 7-10 days [26]. The affected joint should be splinted in a functional position for the first few days after a diagnosis of septic arthritis. Early passive range of motion to be encouraged to stretch tendons and prevent contractures. If the patient's condition responds adequately, gentle mobilization of the infected joint to be begun. Most patients require physical therapy to allow maximum post infection functioning of the joint. Urgent arthotomy and open drainage is usually performed in SA of the hip or shoulder, SA of other joints if no improvement occurs within 3 days of starting antimicrobial therapy, or if a large amount of pus or debris is aspirated during diagnostic arthrocentesis [27].

Prognosis

The primary morbidity of septic arthritis is significant dysfunction of the joint, even if treated properly [28]. Predictors of poor outcome in SA include the following: very young age, infection of hip or shoulder joint, positive findings on synovial fluid cultures after 7 days of appropriate therapy, delay of 7 days or longer in instituting therapy. The mortality rate depends primarily on the causative organism. Neisseria gonorrhoea septic arthritis carries an extremely low mortality rate, whereas that of staph.aureus approach significantly high level [29].

Conclusions

Septic arthritis in children is a medical emergency. High degree of suspicion and prompt, appropriate therapeutic intervention is needed in order to limit the morbity. Diagnostic investigations are limited and of low sensitivity. Early empirical antimicrobial administration based on suspected etiologies to be initiated. Physical therapy to be initiated as soon as the acute condition improves to avoid long term joint deformity.

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