Septic arthritis in children: diagnosis and treatment

Markus Pääkkönen1,2
1Department of Pediatric Orthopaedic Surgery, 2Department of Hand Surgery, Turku University Hospital and the University of Turku, Turku, Finland

Abstract: Acute septic arthritis in children is usually hematogenous. It is more common in boys, and it most often affects the large joints of the lower limb. Diagnosis is based on cultures obtained from the infected joint and is supported by C-reactive protein blood test or ultrasound imaging. *Staphylococcus aureus* is the most common causative agent and is the primary target for empiric treatment. First-generation cephalosporins and clindamycin are suitable antibiotics. Vancomycin is utilized in areas with high rates of clindamycin- and methicillin-resistant *S. aureus*. After a short intravenous administration of 2–4 days, a total course of 2 weeks is sufficient in uncomplicated cases. Early antibiotic treatment has significantly improved the prognosis in high-income settings, but uncomplicated recovery is compromised if the treatment is delayed. Complications such as symptomatic osteoarthritis or avascular necrosis of the femoral head develop slowly. A long follow-up of 1–2 years is required to detect all possible sequelae.

Keywords: child, septic arthritis, sepsis, *Staphylococcus aureus*

Introduction

Acute septic arthritis (SA) in children is most often a hematogenous infection.1,2 The sluggish blood flow in the metaphyseal capillaries makes growing bones susceptible to infection from hematogenous seeding from any trauma or infection. SA is more common in boys than girls with a ratio of 2:1.1,2 The incidence in developed countries is 4–5 cases per 100,000 children per year.1 Most commonly affected locations in the body are the large joints of the lower limb – hip, knee, and ankle joints. *Staphylococcus aureus* and respiratory pathogens are the most common causative agents. *Kingella kingae* is a regional pathogen that has increasingly been identified as a common causative agent especially in children aged 6–36 months.3 The role of trauma in the pathogenesis remains unclear.4 In neonates, bacteria such as *Escherichia coli* is frequently encountered in birth canal.

Diagnosis

Even though acute SA can present in a variety of ways, the classic presentation is an acutely swollen, red, painful joint with limited motion and fever. The child refuses weight-bearing if the lower limb is involved. Neonates with a septic hip assume a characteristic position with the hip joint flexed and abducted in internal rotation. Fever is especially high in cases caused by methicillin-resistant *S. aureus* (MRSA).5 Cases caused by *K. kingae* are milder, and fever may even be absent.6 The disease may be insidious, as any joint in the body may be affected, and immunocompromise,
immunosuppression, or recent use of antibiotics can alter the clinical and laboratory appearance of a septic joint. Differential diagnosis includes osteomyelitis, viral arthritides, and juvenile rheumatoid arthritis (Table 1). Serum C-reactive protein (CRP), white blood cell count (WBC), and erythrocyte sedimentation rate (ESR) are used to differentiate transient hip synovitis from septic hip arthritis, although overlap exists between the conditions.6,7

The diagnostic algorithm for SA is shown in Figure 1. If a child with acute symptoms has elevated CRP (>20 mg/L)8 or ESR (>20 mm/h) values, a joint puncture is performed to detect any purulent aspirate and obtain a sample for bacteriology. Blood cultures are obtained. Synovial fluid findings are unspecific, but a synovial fluid sample with WBC of >50,000/μL of which >75% are polymorphonuclear cells is considered to suggest SA.8 Cultures may remain negative even in 30–70% of the cases.1,9 In addition to agar plates, it is advisable to culture synovial fluid in aerobic blood culture bottles to improve the detection of K. kingae. Plain X-ray is not very useful in diagnosis, but it may detect joint swelling or chronic osteomyelitis. Ultrasound may be used to detect joint swelling and to guide joint puncture. Magnetic resonance imaging does show swelling and is particularly useful in diagnosing adjacent osteomyelitis or an abscess, but it does require anesthesia in infants.

### Treatment

Treatment is started without delay after synovial fluid and blood samples have been obtained. Empiric treatment primarily targets *S. aureus* and takes into account its resistance to antibiotics (Table 2). First-generation cephalosporins and clindamycin are both suitable, but these should be administered in large doses and 4 times a day as these are time-dependent antibiotics.10 In regions where prevalence of MRSA strains exceeds 10%, clindamycin is a valid option of treatment if prevalence of clindamycin-resistant strains remains <10%. If clindamycin resistance is common, vancomycin is the first option of treatment, despite concerns over poor bone penetration.11 Penicillin monotherapy is suitable for *Streptococcus pyogenes* and *S. pneumoniae*, provided that large doses are given.12 Children who have received a Haemophilus type b vaccination do not require adjuvant ampicillin or amoxicillin, which in the prevaccination era was often given to all children under 5 years of age.13 K. kingae is susceptible to most penicillins and cephalosporins.14 Fluoroquinolones or third-generation cephalosporins are valid options against *Salmonella* spp., but the cost may be a problem in low-income settings.15,16

In figure 2, it shows the optimal length of antibiotic treatment for acute SA. After a short 2–4 day intravenous course, the antibiotic may be administered orally if the patient is

### Table 1 Conditions to be considered in the differential diagnosis of septic arthritis of childhood

<table>
<thead>
<tr>
<th>Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aseptic synovitis of the hip</td>
</tr>
<tr>
<td>Viral arthritides</td>
</tr>
<tr>
<td>Osteomyelitis</td>
</tr>
<tr>
<td>Juvenile rheumatoid arthritis</td>
</tr>
<tr>
<td>Legg-Calve-Perthes disease</td>
</tr>
<tr>
<td>Lyme disease (borreliosis)</td>
</tr>
<tr>
<td>Slipped capital femoral epiphysis</td>
</tr>
<tr>
<td>Reactive arthritis</td>
</tr>
<tr>
<td>Sickle cell anemia</td>
</tr>
</tbody>
</table>

### Table 2 Recommended empiric antibiotic treatment

<table>
<thead>
<tr>
<th>Local resistance patterns of <em>Staphylococcus aureus</em></th>
<th>Choice of antibiotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;90% methicillin sensitive</td>
<td>First-generation cephalosporin or clindamycin</td>
</tr>
<tr>
<td>&lt;90% methicillin sensitive</td>
<td>Clindamycin</td>
</tr>
<tr>
<td>&lt;90% methicillin sensitive and &lt;90% clindamycin sensitive</td>
<td>Vancomycin</td>
</tr>
<tr>
<td>Vancomycin resistance</td>
<td>Linezolid</td>
</tr>
</tbody>
</table>

Figure 1 The diagnostic algorithm for childhood septic arthritis.
Abbreviations: CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.
recommended by most researchers. Also, the optimal length of a prolonged course of 3–4 weeks for MRSA SA is still recommended to last at least for 3 weeks. Dexamethasone can be administered to reduce inflammation and may lead to slightly shorter hospital stay. Nonsteroidal anti-inflammatory agents are administered for pain relief.

**Surgery**

Drainage of pus from an infected joint can be accomplished by joint puncture, arthroscopy, or open arthrotomy. Although traditionally open arthrotomy was recommended for hip arthritis, several publications have shown mini-invasive methods to be safe alternatives. Open arthrotomy can be reserved for cases that do not respond to repeated aspirations. Arthroscopy has gained popularity in knee, hip, and shoulder arthritis.

**Long-term sequelae**

Relapses or reinfections are rare if large-dose short-course antibiotic treatment has been administered. Fear of complication of septic hip arthritis is the avascular necrosis of the femoral head. Among infants, joint effusion can result in the dislocation of the hip joint and may lead to instability requiring hip spica casting. Cartilage destruction leads to symptomatic osteoarthritis in an affected joint. The complications may develop slowly, and a long follow-up of 1–2 years may be required to detect all possible sequelae.

**Conclusion**

All children presenting with symptomatic joint and fever should be suspected to have SA. Diagnosis is confirmed by a joint puncture, and a sample for bacteriology is obtained before intravenous antibiotics are administered. Intravenous course is continued for 2–4 days until recovery is observed. A total course of 2 weeks is sufficient in uncomplicated cases. Open arthrotomy is reserved for patients who do not respond to conventional therapy. Patients with delayed diagnosis or MRSA infection carry a poorer prognosis, as do non-privileged children in low-income settings.

**Disclosure**

The author reports no conflicts of interest in this work.

**References**


