Acute septic arthritis (SA) in children is almost often of hematogenous origin. In a Western setting, the annual incidence is around 4:100,000 children. Boys are more prone than girls. Hip, knee, and ankle joints are frequently affected. *Staphylococcus aureus* is the most common causative agent, followed by respiratory pathogens *Streptococcus pyogenes*, *Streptococcus pneumoniae* and *Haemophilus influenzae* type b (Hib). *Kingella kingae* and *Salmonella* spp. are regionally important agents.

**DIAGNOSIS**

An acutely swollen, red, painful joint combined with high fever signal a potential SA. Movement is limited in the affected joint and symptoms tend to increase progressively. The child refuses weight-bearing when the lower limb is involved. This classical pattern is often seen in cases caused by *S. aureus*, but in contrast SA caused by *K. kingae* may develop insidiously. The characteristic signs of SA may be difficult to detect in a child with a septic hip joint, but neonates often assume a characteristic position with the hip joint flexed and externally rotated. Serum C-reactive protein (CRP) and erythrocyte sedimentation rate are sensitive in diagnostics, but erythrocyte sedimentation rate alternates too slowly to be of much use in the follow-up. Procalcitonin challenges CRP, but the measurement requires more time (CRP only needs a few minutes) and is more expensive. Ultrasound detects joint effusion and can guide a diagnostic joint puncture, which should always be attempted. In younger children, the procedure is performed under anesthesia. A purulent joint discharge, positive Gram stain and/or bacterial culture confirm the diagnosis, whereas a traditional synovial fluid cytology is often difficult to interpret due to considerable overlap between different types of arthritis. In addition to conventional agar plates, aerobic blood culture bottles are required to detect *K. kingae*, and although special techniques are not required, an automated system with capability to detect this pathogen should be used.

**TREATMENT**

Intravenous antibiotic is instituted almost always before the causative agent has been identified by cultures. The role of *S. aureus* is overwhelming and its local resistance pattern dictates the choice of antibiotic. Large doses of clindamycin (≥400mg/kg/day divided in 4 equal doses, qid) or a first-generation cephalosporin (≥150mg/kg/day, qid) have been our choices against methicillin-sensitive *S. aureus* strains, but staphylococcal penicillins would likely work as well unless exceptionally large oral doses lead to diarrhea. The same dosing applies to both parenteral and oral administration. If a first-generation cephalosporin is not available for intravenous administration, a second-generation cephalosporin may also be used as a substitute.

Clindamycin continues to be effective against most methicillin-resistant *S. aureus* (MRSA) strains. Clindamycin and vancomycin are ineffective against *K. kingae* for which β-lactams are good choices. Interestingly, cheap trimethoprim-sulfamethoxazole is experiencing a renaissance in the treatment of MRSA. Vancomycin should be considered if resistance to clindamycin is common. An expensive alternative is linezolid. Pneumococcus and *S. pyogenes* do not usually cause problems in terms of resistance when high doses are used. Hib has largely vanished from the etiology of SA in regions with large-scale vaccinations. If this is not the case, unvaccinated children younger than 5 years should receive concomitant ampicillin/or amoxicillin until the agent is identified.

The optimal duration of intravenous and oral treatment has been disputed for decades. Severity of complications associated with SA has led many authors to recommend months’ long medications with an initial intravenous period even for several weeks. In a recent study, the majority of cases of SA were treated 3 to 5 days intravenously followed by 3 weeks of oral antibiotics. Current Infectious Diseases Society of America guideline for MRSA SA states that the exact duration of therapy should be individualized, but typically a minimum of 3 to 4 weeks is recommended. In our prospective, randomized trial, a total of 10 days of high-dose clindamycin or a first-generation cephalosporin was sufficient in uncomplicated cases caused by methicillin-sensitive *S. aureus*. The treatment was started intravenously, usually for no more than 2 to 4 days, and the course was completed orally to a total duration of 10 days.

One lesson learned from our largest-to-date trial was that CRP was of great value, not only in the diagnostics but also in monitoring the course of illness, and in the decision to discontinue antibiotics. Our cutoff level was set at 20mg/L; once this level was reached, the medication was stopped, provided most symptoms and signs had subsided. This occurred usually in a week or so. In cases of concomitant osteomyelitis, we routinely extended the total course to 20 days. Our treatment algorithm is depicted in Figure 1.

It should, however, be kept in mind that a 10-day treatment course is not an universally accepted standard of care, 1 of the reasons being that it has not yet been tested.
in MRSA cases. We therefore recommend, at least so far, our 10-day course only for previously healthy children beyond neonatal age whose SA is not due to agent such as *Salmonella* (uncommon in Western settings). These patients, as the neonates and those with an immunocompromise, likely need treatment to be individually tailored. Nonsteroidal anti-inflammatory agents are administered at the discretion of the attending physician. Adjuvant dexamethasone seems to slightly quicken recovery without reducing the frequency or extent of complications.

**COMPLICATIONS**

Because a dismal outcome—death, avascular necrosis of the femoral head, arthritis, an so on—may still be the ultimate outcome of complicated SA, some further aspects have to be taken into account. The prognosis worsens if a child presents late after significant cartilage destruction has developed. Already a delay of more than 5 days from the onset of symptoms seems to affect adversely on recovery, and such a wait is regrettably common in developing countries. In the United States, the MRSA-USA300 strain and Panton-Valentine leukocidin gene have associated with a severe disease with a longer duration of fever. Extended antibiotic treatment for MRSA has been recommended, but prospective trials are lacking. In our series, methicillin-sensitive *S. aureus* cases were treated as those caused by other agents, and there was no difference in outcome.

**OUR PRACTICAL APPROACH TO TREATMENT AND CONCLUSIONS**

Our approach to potential childhood SA is straightforward: a diagnostic joint aspiration (under anesthesia at least in younger children) is performed and if SA is diagnosed, clindamycin or a first-generation cephalosporin is instituted, first intravenously for a few days, then orally to a minimum of 10 days. However, exceptionally large doses (≥40 or 2150 mg/kg/day till a maximum daily dose of 2–4 g or ~3 g, respectively) and a qid regimen are used for these time-dependent antibiotics. Once CRP has declined below 20 mg/L, the antibiotic can usually be discontinued if most symptoms and signs are alleviated. Patients who have not responded uneventfully may benefit from prolonged treatment. Because sequelae may develop insidiously, a follow-up for at least a year is well-founded.

All this said, complications or deviations from the expected course of disease occur in medicine. However, the difficult-to-treat cases present a minority and can usually be detected by simple tools such as sequential CRP determinations.

**REFERENCES**