Received: 29.6.2006 **Accepted: 3.11.2006**

Case Report

Disseminated staphylococcal infection with polyarthritis in a 12 year old boy

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Abstract

Septic arthritis with polyarticular presentation is an uncommon clinical entity. A case of complicated staphylococcal infection with purulent and reactive polyarthritis, osteomyelitis, cutaneous and pleuropericardial involvement as well as transient blindness is presented here.

KEY WORDS: Septic arthritis, staphylococcal infection, toxic shock syndrome.

JRMS 2007; 12(1): 45-48

Staphylococcus aureus bacteremia, a relatively common infection, causes metastatic infections in up to one-third of patients, with joints and heart valves being the most commonly affected sites [1,2]. On the other hand, septic arthritis with polyarticular presentation is an uncommon clinical entity. There are few case reports of concomitant acute inflammatory arthritis and infection in the literature [3,4]. There have been other cases reported where patients have presented with an acute self-limiting sterile arthritis and a concomitant infection where there has been a dramatic improvement in arthritic manifestations only after the infection has been treated [3,5,6]. A case of complicated staphylococcal infection with purulent and also reactive polyarthritis and transient blindness is presented here, as the step by step decision making in such cases could be a clinical challenge to every physician.

Case Presentation

A 12-year-old boy was referred from Saveh (a city in Central Iran) to our hospital in Qom, with a seven-day history of fever, severe swelling and pain in the right knee joint and probable diagnosis of rheumatic fever. Three days before admission to a Saveh hospital, he had had a sore throat and symptoms of common cold. He received acetaminophen and benzathine penicillin for treatment. During the following three days the patient had developed additive polyarthritis in his knee joints, elbows and ankles. On the third day of admission in Saveh, aspirin was administered to him for presumptive diagnosis of acute rheumatic fever. On the sixth day he suddenly developed a macular rash, so aspirin was discontinued and corticosteroid was started. On the next day, he was referred to us for more evaluation and treatment. He did not have any history of dermatitis or chicken pox, past history of recurrent infections, consanguinity or relevant family history. The patient was toxic and asthenic on admission at our hospital. Vital signs were as follows: BP = 110/60, PR = 120, T = 39.2 and RR = 20. He was fully conscious. There were few lesions resembling rose spots on the abdomen. A smear and culture of these spots yielded no microorganisms. Apart from a

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macular rash and a mild functional grade2 systolic murmur at the apex without radiation, the examination of chest and abdomen was normal. The knees, elbows and ankles were swollen, tender, inflamed and extremely limited in motion.

Peripheral blood smear revealed toxic granulation in neutrophils with a shift towards the left. The patient also had elevated liver enzymes (ALT = 143, AST = 128), leukocytosis (WBC = 27000/µl), anemia (Hb = 9 g/dl), high ESR (130), coagulation defect (PT = 20", PTT = 73"), and electrolyte abnormalities (Na^+ = 130 and K^+ = 2.4 meq/l). A needle tap of the joints gave a clear low-viscosity fluid for the most part but there was evidently pus in the right and left knees. Multiple clusters of Gram-positive cocci were found in smears from the right and left knee fluids. The other joints gave sterile smear and culture. Antibiotic treatment with cloxacillin was initiated and three days later 3 sets of blood culture grew Staphylococcus aureus which was sensitive to cloxacillin and clindamycin and showed intermediate sensitivity to vancomycin. In the evening of the third day of admission to our hospital, the patient was sent to the operating room for joint drainage. Right and left knee joints were drained and elbow joints tapped by an orthopedic surgeon. The knees revealed purulent fluid with innumerable clusters of grapelike Gram-positive cocci. The elbows revealed a clear fluid with negative gram's stain. The immediate postoperative course was downhill with bradypnea (RR = 6/min), hypotension (BP= 80/50), and continuous drowsiness. Fluid therapy did not change hypotension but naloxone improved his consciousness and respiration. After an hour the patient was admitted to the ICU. A day later, a diffuse erythematous rash developed and fever associated with agitation, respiratory distress and decreased level of consciousness continued. Blood pressure did not increase despite administration of dopamine. Brain CT scan was normal, but chest radiography revealed cardiomegaly and discrete parenchymal infiltrations. Three days later, while symptoms of arthritis in the non-drained joints began to diminish, he opened his eyes, but vision had been totally lost. Pupils were normal and reactive to light, anterior and posterior chambers were normal, but the patient denied light perception. Fever, agitation and respiratory distress continued to persist. Serum creatinine began to rise from 0.8 to 3 mg/dl, peripheral WBC fluctuated from 3×10^3 to 26×10^3 /µl and ESR was between 110 and 130 mm/hour. He developed multiple cutaneous bullae that led to desquamation of the shoulder and upper arm areas. Clindamycin was added to cloxacillin with the diagnosis of toxic shock syndrome, and also 12 grams of IVIG was administered. After 12 hours, blood pressure rose to 110/70 mmHg but he still remained febrile. Another chest radiograph on the 6th postoperative day showed more pleural and pericardial effusion. He also had friction rub and coarse crackles in both lungs. Pleural tap was done and a large amount of bloody fluid was drained. Staining showed Gram-positive cocci. Echocardiography showed massive pericardial effusion. Findings of the pericardial tap were similar to that of the pleural fluid with positive smears. Pericardial fluid was drained repeatedly on a daily basis. Vancomycin was added to the prior combination of cloxacillin and clindamycin due to persistence and development of new infections despite continued antibiotic therapy.

On the 9th postoperative day, the patient suddenly recovered vision and fever and respiratory distress declined. During the next week serum creatinine decreased gradually and vital signs became stable. About the 25th day, fever increased again and he reported a severe right leg pain. Physical examination revealed swelling and tenderness on an area 12 cm below the knee. A plain film detected a focus of probable osteomyelitis that, upon surgical drainage, yielded about 300 ml of pus. A week later, fever and ESR diminished gradually, and one month later, he left the hospital in good condition. He completed a two-month course of antibiotics, and weekly follow-ups revealed continuous weight gain up to 5 kg during the first month and gradual normaliza-

tion of ESR. Other symptoms also resolved completely. Regular follow-up for two years did not reveal constrictive pericarditis or respiratory problems.

Albinism was not present and he did not have the blood characteristics of Chediak-Higashi Syndrome. Also we could not find any other defect in WBC function. We assumed the bullae caused on both palms following physical punishment by the patient's teacher a week before admission in Saveh as the route of Staphylococcus entry.

Discussion

Deep seated staphylococcal infection, if not treated promptly, will run a malignant course with grave complications [7,8]. Staphylococcus aureus is the most common cause of septic arthritis in children [9]. Recently, Wang has reported a case of a 10-year-old boy with multiple staphylococcal arthritis, deep vein thrombosis, pulmonary embolism, pericardial effusion and occlusion of the anterior parietal branch of the right middle cerebral artery [10]. In addition to septic complications, there have been case reports where patients have presented with an acute self-limiting sterile arthritis concomitant with a current infection [5,6]. The pathogenic mechanism is unclear but the prompt recovery of the patients following appropriate treatment of the primary focus indicates a direct relationship of the infection to the arthritic manifestations. One possibility is that the mechanism may be toxin-mediated. Staphylococcal enterotoxin B, as a superantigen, has been found to induce arthritis in female DBA/1 mice [11]. Moreover, antibodies against the toxic shock syndrome toxin-1 have been detected from serum and synovial fluid of a 31-yr old man with bilateral knee synovitis and effusion secondary to toxic shock syndrome due to Staphylococcus aureus bursitis [12].

Pleural effusion and empyema associated with pneumonia are relatively uncommon diseases and S. aureus has been the most common cause in some series [13]. On the other hand pulmonary involvement is as high as 82% in sepsis caused by S. aureus, and the pulmonary findings, including bronchopneumonic infiltration and lobar consolidation, were frequently seen in S. aureus pneumonia, causing a mortality rate of 18.75% [14]. Toxic shock syndrome is an acute illness characterized by fever, rash, and hypotension that can lead to multiple organ failure and lethal shock, as well as desquamation in patients that recover [15]. The disease is caused by bacterial superantigens secreted from Staphylococcus aureus and group A streptococci. Superantigens bypass normal antigen presentation, by binding to class II major histocompatibility complex molecules on antigen-presenting cells and to specific variable regions on the beta-chain of the T-cell antigen receptors. Through this interaction, superantigens activate T cells at orders of magnitude above the antigen-specific activation, resulting in massive cytokine release that is believed to be responsible for the most severe features of TSS [16].

Severity of illness at onset revealed by acute physiology and chronic health evaluation [APACHE III] >60 has been an important predictor of mortality[17]. Transient blindness in this patient happened postoperatively and may be due to shock that mostly has been reported with severe infections, trauma and systemic inflammatory response [18,19]. Wold and Leira for the first time reported temporary blindness after hemorrhagic shock [20]. Cardiac involvement is fairly high (20%) in S. aureus sepsis in childhood and is associated with high mortality. Pericardial effusion may progress to cardiac tamponade but usually resolves with supportive and antibiotic therapy by one to two weeks[21]. We come to the conclusion that, several complications can be associated with a severe infection such as staphylococcal diseases. They may have different mechanisms of action such as toxins, etc., and for suitable management several factors must be addressed.

Acknowledgments

The authors want to thanks Dr Amin Afzali and Dr Seyyed Hassan Adeli for their kind help to this work.

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