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Severe peripheral polyneuropathy in a child with infective endocarditis caused by *Staphylococcus* aureus

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Abstract

Although central nervous system complications such as stroke, encephalopathy and meningitis are commonly described in Staphylococcus aureus endocarditis, peripheral nervous system involvement is rarely reported in the literature. In this article we report on a 13-year-old boy with infective endocarditis caused by Staphylococcus aureus in whom severe polyneuropathy developed during hospitalization. To the best of our knowledge this is the first child case with infective endocarditis associated with peripheral polyneuropathy in the literature.

Key words : Endocarditis ; *Staphylococcus aureus* ; polyneuropathy.

Introduction

Staphylococcus aureus (*S. aureus*) is a major cause of infection in infants and children. It may cause pyogenic infection of the skin, osteomyelitis, septic arthritis, wound infection, abscesses, pneumonia, empyema, endocarditis, pericarditis, meningitis, toxin-mediated diseases and sepsis (Çaksen *et al.*, 2000 ; Çaksen *et al.*, 2000 ; Çaksen *et al.*, 2001 ; Çaksen *et al.*, 2001 ; Çaksen *et al.*, 2002 ; Çaksen *et al.*, 2002 ; Çaksen *et al.*, 2003). Although central nervous system complications and rarely peripheral nervous system involvement have been described in adults and children with *S. aureus* endocarditis, peripheral nervous system involvement has not been reported in childhood endocarditis (Pamphlett and Walsh, 1989 ; Lazzarino *et al.*, 1994 ; Corne *et al.*, 2001). In this article we report on a child with infective endocarditis caused by *S. aureus* in whom severe polyneuropathy developed.

Case report

A previously healthy 13-year-old boy was admitted with a 1-week history of fever, bloody diarrhea, and 1-day history of decreased consciousness, dyspnea and ecchymoses on the lower extremities. The personal and family histories were noncontributory. On admission, his general condition was very bad and he was on obtundation. He had the findings of severe shock. Necrosis was noted on the 1st, 2nd and 3rd toes on the right foot and on the 1st and 2nd toes on left foot. Deep tendon reflexes were normal on the upper extremities, but absent on the lower extremities. Plantar response was bilateral unresponsive. Muscle strength could not be determined during admission, but then it was normal on the upper extremities, but 3/5 on the lower extremities. Meningeal irritation signs were negative.

On laboratory investigation, erythrocyte sedimentation rate was 52 mm/hour. Hematological and biochemical analysis showed hyponatremia (118 mEq/L), hypercreatininemia (1.7 mg/dl), abnormal liver function tests, and consumption coagulopathy. The studies for vasculitis were found to be unremarkable. Cerebrospinal fluid (CSF) examination was normal. No microorganism was isolated from CSF and urine cultures. On the ninth day of admission *S. aureus* was isolated from blood culture. Echocardiographic examination performed on the 26th day of admission revealed 1st-2nd degree tricuspid insufficiency and valvular vegetations on the tricuspid valve, which were consistent with infective endocarditis (Fig. 1). Electromyography examined on the 32nd day of admission revealed no response of sensorial and motor conduction on the lower extremities, which were consistent with severe polyneuropathy, but normal on the upper extremities. Needle electromyography was unremarkable. A Tecnetium-99m bone examined on the 37th day of admission showed increased uptake in the soft tissue of upper legs bilaterally.

The patient was hospitalized with the diagnosis of septic shock and consumption coagulopathy. He was given antibiotherapy and intravenous heparin. On the second day of admission, peritoneal dialysis was performed for nine days because he was anuric and renal function tests were impaired. *S. aureus*

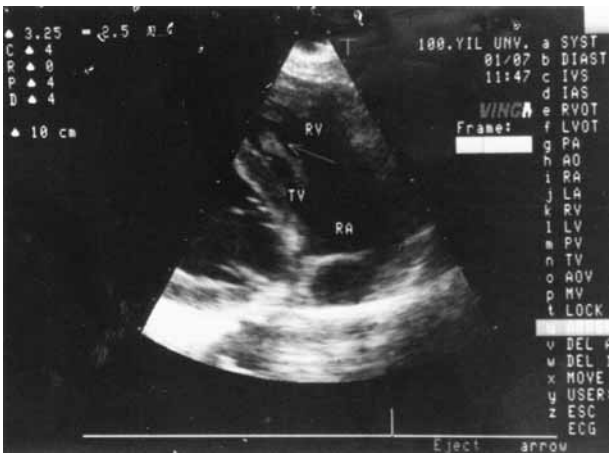

FIG. 1. — Echocardiography shows valvular vegetations on the tricuspid valve.

was isolated from blood culture on the ninth day of admission. On the 10th day of admission bilateral demarcation line on the region of necrosis of the toes were diagnosed and amputation was advised, but the parents did not give permission for operation. On the 10th hospital day *Pseudomonas aurigi-nosa* was isolated from peritoneal fluid culture and the antibiotics were rearranged according to the antibiogram. On the 12th hospital day bilateral pneumatoceles were diagnosed on the thorax radiography. On the 16th day of admission peritoneal dialysis catheter was extracted and hemodialysis program was initiated. *Stenotrophomonas maltophilia* was isolated from the peritoneal dialysis catheter culture. On the 24th day of admission it was diagnosed that muscle strength was normal on the upper extremities, but 1/5 on the lower extremities. Based on the clinical findings infective endocarditis was thought and it was confirmed by echocardiographic examination on the 26th hospital day. On the 35th day of admission hemodialysis was discounted because renal function tests became to normal ranges. On the 40th day of admission, echocardiogram showed markedly improvement of valvular vegetations on the tricuspid valve. On the 48th day of admission, a complete amputation of the 1st, 2nd and 3rd toes on right foot was performed, and only distal phalanxes of the 1st and 2nd on left foot were amputated because of severe necrosis. The patient was sent to the Department of Physical Medicine and Rehabilitation to intensive program on the 64th day of admission. Additionally, severe decubitus lesions on the buttocks required re-constructive operation developed during the hospitalization and they were successfully repaired later. On the 6th month of follow-up electromyographic examination revealed that no response of sensorial and motor conduction on the lower extremities, but normal on the upper extremities. Needle electromyography displayed chronic axonal degeneration on the distal region of lower extremities bilaterally. The findings were consisted with severe axonal and demyelinating (mix type) polyneuropathy. Now he is on the 13th month of follow up, he can walk with a crutch because of pain on the lower joints. Muscle strength was normal both on the upper and lower extremities, but severe pain was noted on the feet during touch and palpation. Severe atrophy on the muscles of buttocks, which were regions of decubitus was diagnosed. Deep tendon reflexes were normal on the upper, but decreased on the lower extremities. Markedly swelling and deformation were noted on the ankle joints bilaterally. Carbamazepine was given because he had symptoms of polyneuropathy including burning, pain and feel pins and needles.

Discussion

Neurologic complications remain a significant problem in bacterial endocarditis : they were reported between and 35% and 39% in the various series (Roder *et al.,* 1997 ; Pruitt *et al.,* 1978 ; Kanter and Hart, 1991). Roder *et al.,* 1997 reviewed a total of 260 cases with *S. aureus* endocarditis, and found 91 patients (35%) experienced neurologic manifestations : the most (45%) frequent neurologic manifestation was unilateral hemiparesis. In another series consisting 218 patients with endocarditis, 84 (39%) had a neurologic complication (cerebral embolism is the most frequent) and 58% of these 84 patients died. In contrast, the mortality rate was only 20% among those endocarditis patients without neurologic complications (Pruitt *et al.,* 1978). It was also reported that of 166 episodes of native valve endocarditis neurologic complications [stroke (55%) encephalopathy (29%), meningitis (17%), and death (48%)] occurred in 35% (58/166) of patients (Kanter and Hart, 1991). Although several neurologic complications, even death have been recorded in the series mentioned above peripheral neuropathy as in our case has not been reported.

Pamphlett and Walsh 1989 reported a 64-year-old woman developed septicemia and a generalized peripheral neuropathy. No cause for the neuropathy could be found during life. At autopsy she was found to have infective endocarditis and multifocal inflammatory lesions in the central and peripheral nervous systems, consistent with damage due to septic emboli. They recorded that infective endocarditis may be a cause of a generalized polyradiculoneuropathy and could be responsible for a proportion of cases of “critical illness polyneuropathy” (Pamphlett and Walsh 1989). Lazzarino *et al.,* 1994 reported that a case of acute endocarditis with mononeuritis multiplex. Mononeuropathy was the only detected neurological sign. The presence of muscular abscesses compressing the peripheral nervous system could lie at

the basis of the pathophysiological mechanism responsible for acute mononeuropathy (Lazzarino *et al.*, 1994). Recently, *Corne et al.,* 2001 reported that a case of a 65-year-old woman who had tetraparesia and aseptic meningitis revealing *S. aureus* endocarditis. Tetraparesia was due to an acute motor axonal neuropathy. Meningitis and tetraparesia were improved with antibiotic therapy. As a conclusion they noted that acute motor axonal neuropathy might be a presenting symptom of *S. aureus* endocarditis (Corne *et al.,* 2001).

Our patient was diagnosed with infective endocarditis on the tricuspid valve. We think that our patient also had mild mitral and/or aortic valve involvement, which was not detected on echocardiographic examination. Because it is well known that neither sensitivity nor specificity of echocardiography is 100%, and a negative echocardiogram does not rule out endocarditis, and M-mode echocardiography can detect valvular vegetations larger than 2-3 mm (Bernstein, 2000).

Toxic neuropathies of both endogenous such as diabetes mellitus, and uremia and exogenous origin including drugs, heavy metals, organic chemicals and biological toxins are uncommon in childhood at least in symptomatic form. Neuropathies may also be a manifestation of most vasculitides such as systemic lupus erythematosus, rheumatoid arthritis and polyarteritis nodosa. Additionally, neuropathy may occur in gravely ill patients with multiple visceral deficiency or following major surgery and burn. The nature of these “critically ill neuropathies” is obscure. They may entail compression, hypoxia and other mechanisms (Aicardi, 1998).

We thought that the most likely cause of the lower extremity neuropathy was ischemic/infarction from emboli or hypoxia due to low flow from shock. In conclusion we would like to emphasize that infective endocarditis caused by *S. aureus* may cause generalized peripheral neuropathy in childhood. Therefore, these children should be monitored for this unfortunate condition.

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