A CASE OF ACUTE STAPHYLOCOCCAL ENDOCARDITIS WITH MULTIPLE SEPTIC EMBOLIZATIONS

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ABSTRACT

Although it is mentioned as a rare cause of stroke, bacterial endocarditis must be considered in the etiologic diagnosis because its severe outcome. We present the case of an immunosupressed patient who developed staphylococcal acute bacterial endocarditis, whose first symptoms were neurological. Later she developed cutaneous, cerebral, pulmonary and splenic septic embolization.
Key words: endocarditis, Staphylococcus aureus, stroke, septic emboli, cerebral abscess

INTRODUCTION

Despite the availability of a wide range of antibiotics, infective endocarditis continues to be a major problem all over the world, the main reasons being the emergence of newer infective organisms and the presence of extra-cardiac complications. Among these, involvement of the central nervous system is possibly the most serious (2). The association of infective endocarditis and neurological complications has been recognised more than a century ago, when Osler described the triad of fever, heart murmur, and hemiplegia as being highly suggestive of endocarditis (8).

In 10-15% of patients with IE, the nervous system yields the first clinical signs and often before starting any treatment (8). The symptoms are varied, ranging from confusion to profound coma (5). Recent studies have emphasized a changing pattern of neurological complications due to advancing age of the patients, increase of nosocomial infections and of intravenous drug users, nowadays infective endocarditis being one of the major causes of stroke in young people (6).

Postmortem studies showed various types of brain lesions: cerebral infarction (65-68%), haemorrhage (35-57%), cerebral abscess (10-31%) and focal meningitis (5-14%) (2). In the pre-antibiotic era the mortality of infective endocarditis was nearly 100%. Currently it is 20-50%, often being the result of congestive heart failure or acute valvular insufficiency. Studies show that mortality is higher in patients with central nervous system involvement (58-74%) compared to its absence (20-56%) (7). Patients with IE have a risk of occurrence of stroke of 0.5% per day (8).

The incidence of neurological complications of acute endocarditis is dependent on the organism (Staphylococcus spp 54%, Enterococcus spp 20%, Streptococcus viridans 19%), and valvular location (the highest incidence is 87% with Staphylococcus aureus vegetations on the mitral valve) (8). Staphylococcal endocarditis occurs more often in patients with chronic renal failure, diabetes, alcoholism, cancer, immunosupression and drug abusers. (3). That is why its prognosis is more severe, with clinical signs appearing and evolving quickly, thus needing early hospitalization (4). The mortality in the acute phase is three times greater for Staphylococcus aureus compared to other microorganisms (34% versus 10%) (1), independent of age, co-morbidities or usual prognosis factors, which implies the need for an early instituted and aggressive treatment.

CASE PRESENTATION

It is presented the case of a 53 years old female patient, diagnosed with rheumatoid arthritis 7 months before the present admission, for which she was treated with methotrexate, sulphasalazine and ketoprofen. Five months after starting the above treatment she presented an episode of severe thrombocytopenic purpura (zero platelets at blood count), which initially improved under treatment with dexamethasone and metil-prednisolone and relapsed when dosage tapering was attempted.

Five days before admission she presented sudden onset of high fever accompanied by shivering, headache, balance disturbance and vomiting. Later that day she developed visual impairment and left hemiplegia.

On admission the clinical examination revealed a pyrexial, dyspneic and tachycardic patient, with a purpuric rash on limbs, thorax and oral mucosa. She was aware, bradylalic, with marked neck stiffness, left homonymous hemianopia, left hemiplegia and left superficial hypoesthesia. The fundoscopy revealed a large hemorrhage situated in the left optic disc. Blood tests showed thrombocytopenia (38000 platelets/mmc).

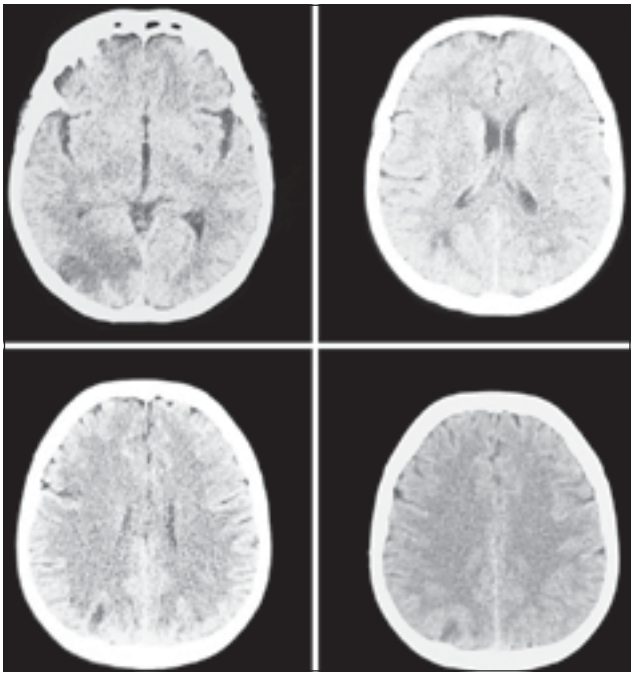


Figure 1

Hypodensities situated in both occipital lobes, corticosubcortical, parasagital, predominant on the right (area supplied by posterior cerebral artery) and in the right frontal lobe, corticosubcortical, paracentral (area supplied by middle cerebral artery)

The head CT scan done on admission (figure 1) showed hypodensities suggestive for recent ischemic stroke, but on the MRI (figure 2) the lesions appeared to be indicative of acute meningo-encephalitis.

In both blood cultures taken grew Staphylococcus aureus, thus sketching the picture of a staphylococcal septicemia.

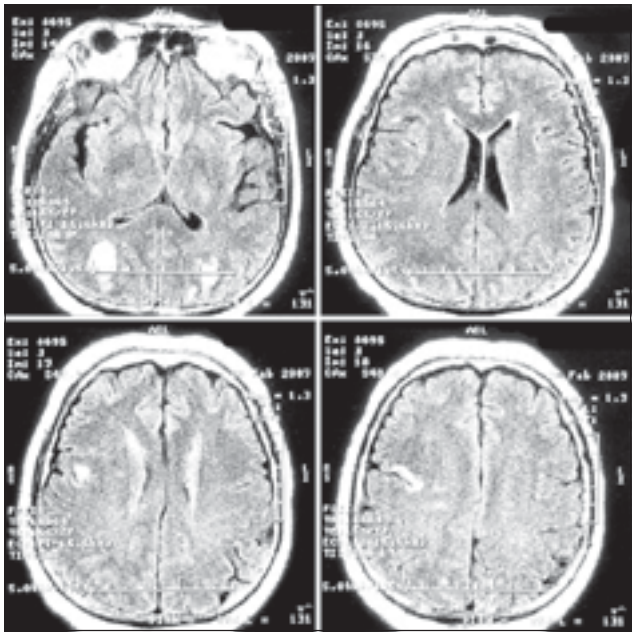
The cardiac ultrasound showed a vegetation of 1.7/1 cm situated on the posterior aspect of the mitral valve and causing mitral regurgitation. A first complete diagnosis was made, that of staphylococcal acute endocarditis causing cerebral septic embolization.

A CT scan of chest and abdomen (figure 3) showed bilateral pneumonia accompanied by pleural effusion and splenic infarct, all secondary to septic emboli.

The patient's condition steadily deteriorated despite a broad-spectrum potent antibiotic treatment (initially vancomicin, moxifloxacin and meropenem, later adding teicoplanin and linezolid) so that after a few days the patient developed multiple organ dysfunction syndrome. For the respiratory distress syndrome she required artificial ventilation and for maintaining a normal blood pressure she necessitated pressor support with dobutamine. The increased positive fluid balance demanded high doses of diuretic and total parenteral nutrition was instituted for digestive system dysfunction.

The head CT scan repeated at two weeks (figure 4) showed two round masses, suggestive for cerebral abscesses.

Four weeks after admission the patient died.



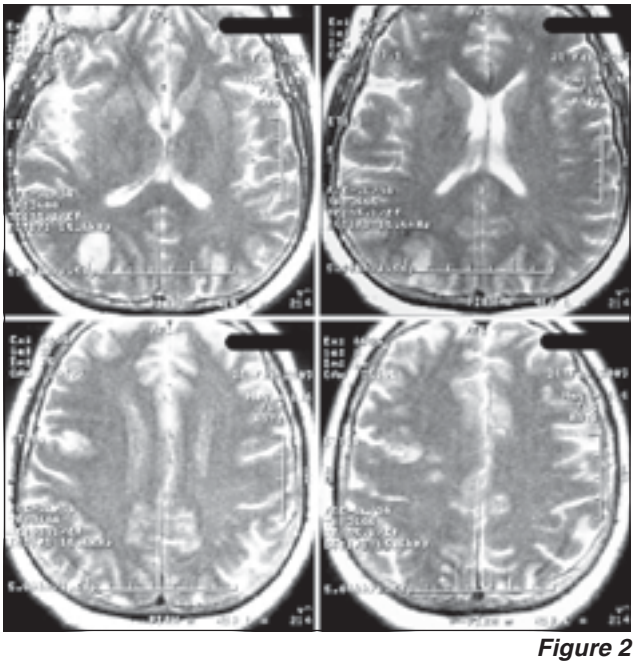


Figure 2

Diffuse areas of hypersignal on T2-weighted and Flair images, situated in the occipital lobes, corticosubcortical, more important on the right. The lesions lie along the cerebral sulci, predominant in the grey matter.

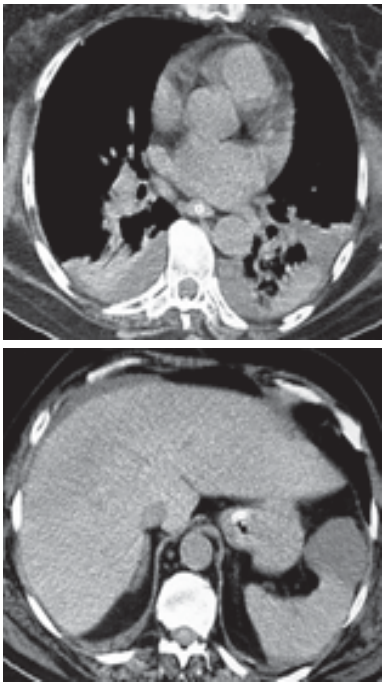


Figure 3
Bilateral basal lung collapse and bilateral lower lobe alveolo-interstitial consolidation. Small bilateral pleural effusion. A 5.5 cm diameter cystic mass situated between the stomach, spleen and diaphragm, displacing the spleen posteriorly.

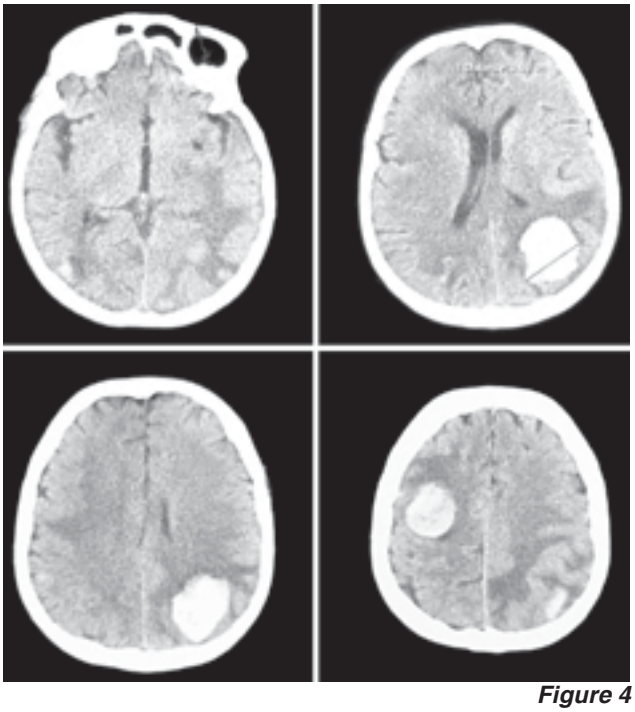


Figure 4
Two round hyperdensities, one of 3 cm diameter in the right frontal lobe, and the other temporo-parietal, of 3.6 cm diameter, both causing surrounding oedema.

COMMENTS

The peculiarity of this case was the presence of multiple septic emboli of infective endocarditis superimposed over the clinical manifestations of thrombocytopenic purpura. Besides the preexistent purpuric rash there were small macular lesions situated on the palms and soles, interpreted as cutaneous septic emboli (Janeway lesions). Also, the retinal hemorrhage seen at fundoscopy was the consequence of microemboli (Roth's spots).

The cerebral embolization followed a two-stages pattern. Initially the emboli caused ischemia, with typical clinical manifestations imaging features of acute ischemic stroke. In the second phase the cerebral infarcts presented hemorrhagic transformation and abscess formation.

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