Infective endocarditis complicated with cerebral and splenic infarction in a hemodialysis patient

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INTRODUCTION

Infective endocarditis (IE) is an infection of the endocardial surface of the heart that may or may not involve the heart valves, the mural endocardium, or a septal defect. IE has a high risk of morbidity and mortality, and prognosis is highly dependent on complications. The most common complication of IE is systemic embolization, which occurs in 22%–50% of IE patients. Emboli often involve major arterial beds, including the brain, lungs, coronary arteries, spleen, bowel, and extremities.

Here, we report the case of a patient with IE, who presented as a neurological emergency. Along with a cerebral infarction, the patient had a concomitant splenic infarction. The splenic infarction was due to systemic embolization, and was diagnosed incidentally in the emergency department (ED).

Case report

A 47-year-old man with altered mental status and fecal incontinence presented to our ED. The patient, who had a history of chronic renal failure and hemodialysis, had been complaining of high fever for 10 days, general poor health, and impaired oral intake.

On admission, the patient appeared generally ill, had a mild fever (38 °C), a Glasgow Coma Scale (GCS) score of 13, a blood pressure of 137/89 mmHg, a pulse rate of 129/min, a respiratory rate of 21/min, and a peripheral O₂ saturation of 97%. A very loud pansystolic murmur (grade VI) was audible at the cardiac apex. Neurologic examination revealed that the patient was in stupor with a left hemiparesis of 4/5. He did not have any aphasia, facial palsy, or dysphasia. There were no physical findings of Roth spots, Osler nodes, Janeway...
lesions, or conjunctival hemorrhage. His initial blood count revealed a leukocytosis of 9,500/mm$^3$ with 80.5% neutrophilia, hematocrit of 18, hemoglobin of 6.5 g/dL, and platelet count of 242,000/mm$^3$. The erythrocyte sedimentation rate was 76 mm/h, whereas the C-reactive protein level was 6 mg/dL (normal range 0 to 0.8).

Although the patient did not fulfill the criteria for a definitive diagnosis of IE according to the Duke Clinical Criteria for IE, he did meet one major [positive echocardiographic (ECHO) evidence, Figure 1] and two minor criteria (a fever of 38 °C and predisposing effect of hemodialysis) for the diagnosis.$^3$ The other major criterion (positive blood culture) was not met, as all three blood cultures, which were drawn one hour apart upon initial admission, were negative.

Because of the patient’s neurological findings, which caused us to consider cerebral emboli, a brain magnetic resonance imaging (MRI) was performed. Also, a contrast enhanced abdominal computed tomography (CT) was performed due to the fecal incontinence and subtle abdominal findings. Abdominal CT incidentally revealed encapsulated hypodense splenic lesions (–7 cm), which were suggestive of splenic infarction (Figure 2). Brain MRI revealed a lesion suggestive of an infarction.

The lesion had bright signal intensity on diffusion-weighted and contrast-enhanced T1-weighted images in the right fronto-parieto-occipital cerebral area (Figure 3).

The patient was admitted to the hospital with the diagnosis of IE complicated with cerebral and splenic infarction. The medical treatment included intravenous antibiotics (i.e. teicoplanin including gentamicin), subcutaneous low molecular weight heparin, and oral acetylsalicylic acid for four weeks. Mitral valve replacement surgery was performed after the patient improved clinically, and then he was discharged from the hospital.

**DISCUSSION**

IE represents the fourth leading cause of life-threatening infectious disease (after urosepsis, pneumonia, and intra-abdominal sepsis).$^{14}$ If complications occur and are left untreated, the mortality rates of IE can be very high.

In this report, we presented a case of IE complicated with cerebral infarction and concomitant splenic infarction. The frequency of specific complications depends on variables such as the infecting pathogen, the duration of disease before therapy, and the type of

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**Figure 1.** ECHO view of vegetative growth over the mitral posterior leaflet (arrow).

**Figure 2.** Abdominal CT view of encapsulated hypodense splenic lesion suggestive of splenic infarction (arrow).

**Figure 3.** Brain MRI view of lesion suggestive of brain infarction with bright signal intensity on diffusion-weighted (A) and contrast-enhanced T1-weighted (B) images in the right fronto-parieto-occipital cerebral area (arrows).

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treatment. However, it is often difficult to assess the true incidence of complications since published reviews are frequently based on retrospective chart reviews that use different diagnostic criteria. Systemic embolization rates range from 22% to 50% and neurological complications develop in 20% to 40% of patients with IE. These represent a dangerous subset of complications. Neurological complications of IE are almost always due to embolization, which include strokes, TIA, meningitis, intracranial hemorrhage, or encephalopathy. The central nervous system is the most common site for embolic events, with up to 65% occurring there.

In IE patients, some abnormalities that are less common pose a greater risk to the patient, and cause uncertainty in management for the clinician. For example, the spleen may become enlarged due to a hyperplastic response to persisting bacteremia, or splenic vasculature may be affected because of systemic embolization. These may result in splenic infarction or splenic abscess. The spleen is vulnerable to infarction because it lacks collateral circulation, and splenic infarction occurs in approximately 40% of left-sided IE cases. Localized findings are absent in 90% of splenic infaracts and abscesses. It has been estimated that approximately 5% of patients with splenic infarction will develop splenic abscesses. The current case had no physical findings for splenic infarction. If clinical suspicion had not the ED physician to perform an abdominal CT, it may have never been diagnosed in the ED.

Blood cultures are negative in less than 5% of patients with IE. This may be due to the use of inadequate microbiological techniques, the administration of antimicrobial agents before blood drawing, or difficulty in isolating some IE pathogens from blood cultures. We were unable to isolate any microorganisms from the blood cultures in our case.

Left-sided vegetations that are greater than 1 cm in diameter have higher embolic rates. The highest embolic rates (regardless of size) have been seen in mitral vegetations (25%), especially those that are attached to the anterior mitral leaflet.

In conclusion, it would have been expected if our patient was diagnosed with a single neurological complication of IE. However, the presence of another concomitant systemic complication of IE, and the absence of its clinical findings make our case report interesting for further reading. Finally, it is essential to maintain a high index of suspicion while managing patients with IE, and the threshold for diagnostic imaging modalities should be more flexible for all patients with IE.

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