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Aortic Valve Endocarditis with Splenic and Brain Abscesses: Difficult Management Issues

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ABSTRACT

Staphylococcus aureus endocarditis is associated with a high incidence of complications, a poor prognosis, and high operative mortality. We present a complex case of aortic valve endocarditis in a critically ill patient, highlight the difficult management issues, and report a good outcome.

INTRODUCTION

The in-hospital mortality rate of patients with infective endocarditis (IE) varies between 9% and 26%. The prognosis for IE is influenced by factors such as patient characteristics, the presence or absence of cardiac and noncardiac complications, the infecting organism, and echocardiographic findings. *Staphylococcus aureus* endocarditis (SAE) is associated with in-hospital mortality rates as high as 30% to 70%, especially without surgical intervention [Vlessis 1996; Remadi 2007]. A high incidence of embolic events (30%-40%) has also been reported with this condition, especially when the endocarditis affects the left side of the heart [Vilacosta 2002]. Consequently, early surgical intervention is indicated in SAE.

We present a case of aortic valve SAE complicated with hematoma of the brain and splenic abscesses, and we discuss the difficult steps involved in the management of this critically ill patient.

CASE PRESENTATION

A fit 43-year-old man presented to the emergency department with a 2-week history of flu-like symptoms. Subsequently, his Glasgow Coma Scale value dropped to 8, and he had to be intubated and ventilated. An examination revealed splinter hemorrhages and a loud diastolic murmur at the left lower sternal border. A computed tomography (CT) scan of the patient's brain showed a right-sided intracerebral hemorrhage (Figure 1A), and an echocardiogram revealed a bicuspid aortic valve with vegetations on both leaflets and severe regurgitation. The patient was started on an empirical course of antibiotics. *S aureus* was subsequently isolated from his blood cultures.

On the same day, the patient underwent an emergency right-sided occipital craniotomy and evacuation of the hematoma. Postoperatively, he developed a dense right-sided hemiparesis and remained pyrexial despite antibiotic therapy with benzylpenicillin and rifampicin. Further CT evaluation of the brain showed an intracerebral mycotic aneurysm, which was treated with embolization (Figure 1B). The patient remained septic with an increased white blood cell count (>30,000 cells/μL) and a high C-reactive protein concentration (>200 mg/L). A CT scan of his abdomen showed multiple hypodense lesions representing infarcts or abscesses within the spleen (Figure 1C). He was referred to our cardiothoracic center 2 weeks after presentation.

On arrival, the patient was pyrexial (temperature, 38.5°C), his systolic blood pressure was approximately 95 to 100 mm Hg, his heart rate was 110 beats/min, and his echocardiogram showed free aortic regurgitation. Discussion ensued regarding the timing of surgery. One option was to perform a splenectomy first because the splenic lesions might have represented abscesses and thus be a source of infection. The other option was to perform cardiac surgery first to prevent heart failure. Following discussions with the general surgeons and microbiologists and because the patient was becoming hemodynamically unstable, we proceeded to emergency cardiac surgery.

After a sternotomy and systemic heparinization, the aorta was opened to reveal a completely destroyed aortic valve. There was aortoventricular dissociation midway between the right and left coronary ostia, with a fistula communicating with the left atrium. The aortic valve was excised, and the area was washed with gentamicin. The fistula was closed with an autologous pericardial patch, and the valve was replaced with a 25-mm PERIMOUNT Magna Ease xenograft (Edwards Lifesciences, Irvine, CA, USA). The choice of a biological valve was to avoid warfarinization in this patient, who was likely to require abdominal surgery.

The patient did well and was extubated the following day. On the seventh postoperative day, however, he became hypotensive, with abdominal pain and distension requiring an emergency laparotomy and splenectomy for a ruptured spleen (Figure 2A).

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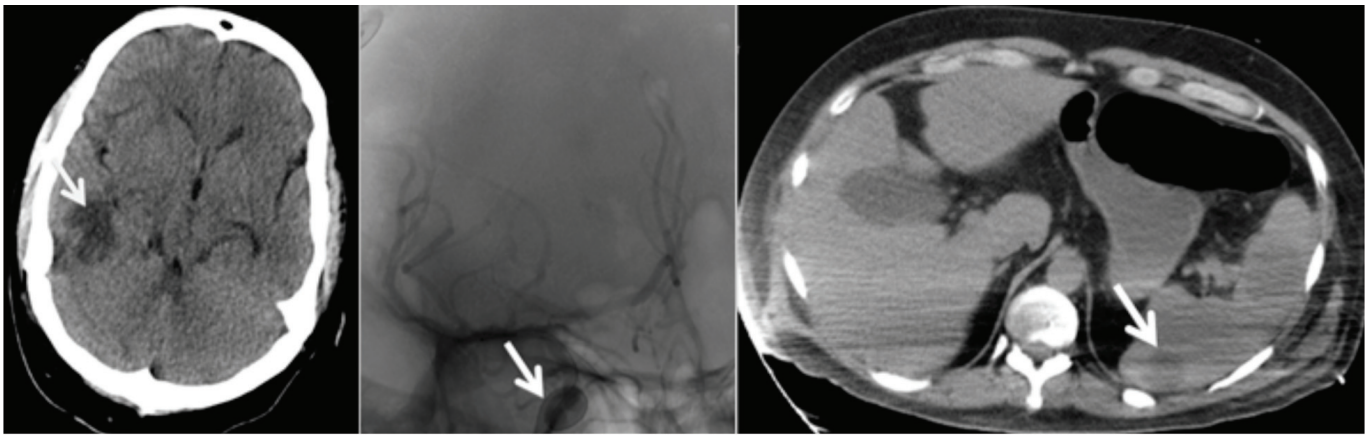
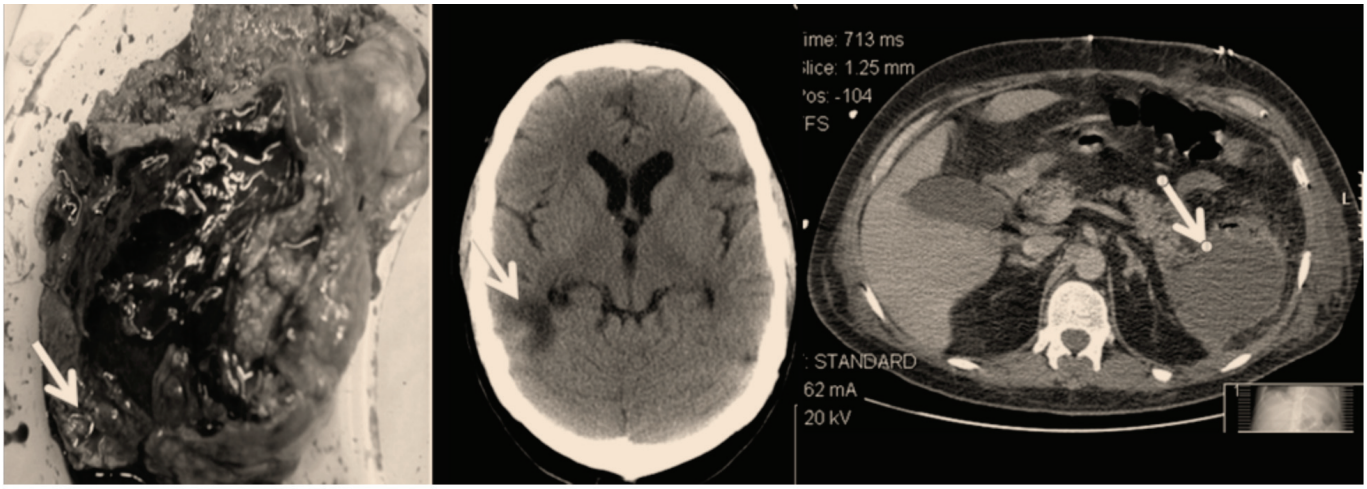


A B C

Figure 1. Hematoma in the brain (A), mycotic aneurysm (B), and hypodense area in the spleen (C).



A B C

Figure 2. Ruptured spleen (A), a computed tomography (CT) scan of the brain demonstrating a right cerebral abscess (B), and a CT scan of the abdomen demonstrating a collection in the splenic bed following splenectomy (C).

After the operation, the patient remained septic, and a CT scan of his brain and abdomen showed new intracerebral abscesses (Figure 2B) and a large collection in the splenic bed (Figure 2C). Both abscesses were drained. The patient made a good recovery and was discharged to a neurorehabilitation unit.

Two months later, a routine follow-up evaluation of the patient revealed a paraprosthetic leak with moderate aortic regurgitation. He was readmitted and underwent a second cardiac procedure. There was dehiscence of the xenograft at the site of the previous fistula. The dehiscence was closed with a bovine pericardial patch, and a new xenograft was inserted. Intraoperative bleeding from the aorta was controlled by supracoronary replacement of the ascending aorta with a Vascutek tube graft (Vascutek/Terumo, Inchinnan, Scotland, UK) with a proximal external extension. The results of tissue cultures were negative.

Postoperatively, the patient made a good recovery and was discharged back to his local hospital. At the 1-year follow-up, the patient was well and had made a tremendous neurologic recovery; he had a normally functioning xenograft and no paraprosthetic leakage.

COMMENTS

IE presents in variety of forms and carries a poor prognosis and a high mortality risk. S aureus infection is becoming the leading cause of IE [Habib 2009] and is responsible for an acute and destructive form of endocarditis.

The timing of surgery is crucial for patients with uncontrolled infection [Vilacosta 2002]. Reasons to consider early surgery in the active phase are to avoid the progressive heart failure and the irreversible structural damage caused by severe infection, and to prevent systemic embolism [Baddour 2005]. Delaying surgical treatment often increases the probability of complications and operative mortality and morbidity [Tirone 2007].

On the other hand, surgical therapy during the active phase of the disease is associated with significant risk, especially if other sources of infection, such as splenic and brain abscesses, are present. Although splenic emboli are common, splenic abscess is rare. Splenectomy may be considered for splenic rupture or large abscesses that respond poorly to antibiotics alone and should be performed before valvular surgery, unless the latter is urgent [Lerner 1984].

In addition, cardiac surgery is not contraindicated after an ischemic stroke unless the neurologic prognosis is poor. Evidence regarding the optimal time interval between stroke and cardiac surgery is conflicting, however, because of the lack of controlled studies [Thuny 2007]. Depending on the individual, surgery may be performed as an emergency or urgently, irrespective of the duration of antibiotic treatment. In some patients, surgery can be postponed to allow 1 to 2 weeks of antibiotic treatment under careful clinical observation [Habib 2009].

It is essential that a multidisciplinary team approach be adopted in the management of patients with IE, especially those infected with virulent organisms such as S aureus, to ensure the best outcome.

REFERENCES

Baddour LM, Wilson WR, Bayer AS, et al. 2005. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. Circulation 111:e394-434.

Habib G, Hoen B, Tornos P, et al. 2009. Guidelines on the prevention, diagnosis, and treatment of infective endocarditis (new version 2009): the Task Force on the Prevention, Diagnosis, and Treatment of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) and the International Society of Chemotherapy (ISC) for Infection and Cancer. Eur Heart J 30:2369-413.

Lerner RM, Spataro RF. 1984. Splenic abscess: percutaneous drainage. Radiology 153:643-5.

Remadi JP, Habib G, Nadji G, et al. 2007. Predictors of death and impact of surgery in *Staphylococcus aureus* infective endocarditis. Ann Thorac Surg 83:1295-302.

Thuny F, Avierinos JF, Tribouilloy C, et al. 2007. Impact of cerebrovascular complications on mortality and neurologic outcome during infective endocarditis: a prospective multicentre study. Eur Heart J 28:1155-61.

Tirone ED, Gheorghe G, Christopher MF, Tommaso R, Susan A, Manjula DM. 2007. Surgical treatment of active infective endocarditis: a continued challenge. J Thorac Cardiovasc Surg 133:144-9.

Vilacosta I, Graupner C, San Román JA, et al. 2002. Risk of embolization after institution of antibiotic therapy for infective endocarditis. J Am Coll Cardiol 39:1489-95.

Vlessis AA, Hovaguimian H, Jaggers J, Ahmad A, Starr A. 1996. Infective endocarditis: ten-year review of medical and surgical therapy. Ann Thorac Surg 61:1217-22.