Case Study

Intracranial complications of acute bacterial endocarditis

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INTRODUCTION

This review and case study focus on the presentation, diagnosis, treatment, and outcomes for patients with intracranial acute bacterial endocarditis (ABE) versus SBE. ABE may result in acute intracranial septic emboli and stroke and/or hemorrhagic infarcts, or ICH. SBE more classically produces septic microemboli and mycotic aneurysms that may leak, but rarely producing ICH. We also presented a patient with ABE attributed to *S. aureus* whose septic emboli/stroke was accompanied by a mycotic aneurysm; the ruptured resulting in a large right occipital ICH.

Key Words: Acute bacterial meningitis (ABM), mycotic aneurysms, SBE, acute bacterial endocarditis (ABE), intracranial hemorrhage (ICH)
bacterial endocarditis (SBE) is more classically characterized by septic microemboli and mycotic aneurysms that may leak, but rarely rupture (e.g. fewer ICHs). Here, we present a case of ABE where an infected embolus (e.g. *Staphylococcus aureus*) resulted in a mycotic aneurysm and acute ICH.

**CASE REPORT**

A 26-year-old male with a history of hypertrophic cardiomyopathy (HOCM) and intravenous drug abuse (IVDA) presented with generalized body ache, fever, and shaking chills. His fever was 103°F, heart rate was 112 beats/minute, he had no cardiac murmur, but he did exhibit tender scattered macular lesions of the palm and soles of the feet (Janeway lesions).

**Laboratory studies**

Laboratory studies revealed a white blood cell (WBC) count of 16.8K/µL (*n* = 3.9-11 K/µL) with 88% neutrophils, lymphocytes of 2% (*n* = 21%-51%), a platelet count of 90K/µL (*n* = 160-392K/µL), an erythrocyte sedimentation rate (ESR) of 77 mm/h, an elevated C-reactive protein (CRP) of 202 mg/L (*n* > 3 mg/L), and blood cultures positive for methicillin-sensitive *S. aureus* (MSSA; three or four bottles positive persisted for 4 days) [Table 1].

**Diagnostic studies**

The Chest X-ray (CXR) showed bilateral pulmonary nodules, while the chest computed tomography (CT) confirmed peripheral bilateral, nodular, cavitating septic emboli [Table 1]. The transthoracic echocardiogram (TTE) showed a 2.7 cm × 0.7 cm vegetation on the tricuspid valve and a 0.8 cm × 0.9 cm vegetation on the posterior mitral valve leaflets. The transesophageal echocardiogram (TEE) also showed a vegetation at the insertion of the papilla extending into the left ventricle (e.g., 1 cm × 0.4 cm) with mitral regurgitation [Table 1].

**Hospital course**

On the sixth hospital day, the patient became lethargic and developed blurred vision. The brain CT scan revealed an acute embolic infarction involving the right occipital lobe accompanied by marked mass effect [Figure 1]. When his cognitive function worsened over the next 24 h, a follow-up CT scan revealed a large 6-cm acute hemorrhagic stroke involving the right parietal/occipital lobe, nearly obliterating the right lateral ventricle [Figure 1].

**Treatment: Endovascular embolization**

The patient underwent successful endovascular embolization of the ruptured right occipital mycotic aneurysm; no craniotomy was required. After completing 6 weeks of nafcillin 2 g (IV) q4h (selected for its CNS penetration), he fully recovered.

**DISCUSSION**

There are significant differences between CNS findings for patients with SBE versus ABE [Table 1].

**SBE: Embolic strokes and mycotic aneurysms**

With the more indolent SBE, patients rarely present with seizures attributed to peripheral septic emboli resulting in mycotic aneurysms that may leak, but rarely rupture [Table 2].
ABE: Embolic strokes, ICH due to ruptured mycotic aneurysms, and microabscesses

Within 3 weeks of developing ABE, many patients initially present with intracranial embolic strokes. They may develop focal seizures, unexplained mental confusion, and/or retinal artery occlusion with accompanying visual loss.[1,6] With ABE, the vegetations are usually large and friable, predisposing patients to septic emboli, mycotic aneurysms (e.g. resulting in ICH), and/or microabscesses [Table 1].[3,5] Notably, mitral valve vegetations are more likely to become embolic versus aortic valve lesions.[7,8] In the case presented, the patient with ABE developed an acute ischemic infarct attributed to a septic embolus. This resulted in a mycotic aneurysm that ruptured creating a large right parieto/occipital ICH; the mycotic aneurysm was successfully occluded using endovascular techniques, and no craniotomy was performed. With 6 weeks of antibiotic therapy consisting of nafcillin 2 g (IV) q4h for MSSA, the patient fully resolved.

CONCLUSION

The cranial complications for patients with ABE versus SBE markedly differ. Those with ABE may develop unexplained seizures and cognitive dysfunction attributed to septic emboli/mycotic aneurysms, resulting in embolic strokes and massive ICH [Table 2]. As documented in this case, however, establishing the diagnosis of ABE early in the clinical course is critical to initiate appropriate antibiotic therapy (e.g., typically of *S. aureus*), endovascular treatment, and/or operative management (e.g., clipping) to maximize recovery and minimize morbidity.

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**Conflicts of interest**

There are no conflicts of interest.

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