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Case Study

Intracranial complications of acute bacterial endocarditis

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Abstract

Background: Infectious endocarditis (IE) clinically manifests as either subacute bacterial endocarditis (SBE) or acute bacterial endocarditis (ABE). Neurologic manifestations are markedly different for these two entities. ABE is caused by invasive, highly virulent pathogens (e.g., *Staphylococcus aureus*), whereas SBE is attributed to relatively avirulent, non-invasive organisms (e.g., *viridans streptococci*).

Methods: Here, we reviewed the clinical and radiographic presentations of a patient with cranial complications attributed to ABE. Such patients typically develop central nervous system (CNS) septic emboli resulting in stroke (with/without intracranial hemorrhage (ICH)) and/or mycotic aneurysms resulting in ICH bleeds.

Results: With ABE, cerebrospinal fluid (CSF) seeding may result in acute bacterial meningitis (ABM), documented by positive Gram stain and/or culture for *S. aureus*, decreased glucose, highly elevated lactose acid levels, or ICH. Alternatively, in SBE, the CSF profile reflects an aseptic (viral) meningitis (i.e., Gram stain and culture negative, a normal glucose, and lymphocytic pleocytosis), while septic microemboli to the vasa vasorum contribute to an inflammatory reaction in the adventitia/muscle layer that weakens the vessel wall and results in mycotic aneurysms that may leak but often do not rupture causing ICH.

Conclusion: Here, we reviewed the literature for intracranial pathology accompanying ABE versus SBE. ABE typically results in acute ischemia, septic emboli, stroke/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)

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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 infarct with/without accompanying mycotic aneurysms that may produce intracranial hemorrhage (ICH). Alternatively, subacute

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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 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.

Table 1: Summary of diagnostic tests

Variables	Results/Data
Laboratory tests	
WBC	16.8K/μL (PMNs=88%, lymphocytes=2%, stabs=21%)
ESR	77 mm/h
CRP	202 mg/L (n <3.0 mg/L)
Platelet count	90K/μL
UA	RBCs >182/hpf (n <3/hpf)
AST	49 IU/L (n=13-39 IU/L)
ALT	44 IU/L (n=4-36)
Cr	2.9 mg/dL (n=0.6-1.2 mg/L)
Blood cultures	4/4 methicillin-sensitive *Staphylococcus aureus*
Imaging	
CXR	Bilateral pulmonary cavity lesions
Chest CT scan	Bilateral cavitating septic emboli
TTE	Ill defined 2.7 cm × 0.7 cm vegetation on the tricuspid valve and 0.8 cm × 0.9 cm vegetation on the mitral valve
TEE	Vegetation at the insertion of the papillary muscle in the left ventricle measuring 1 cm × 0.4 cm with mitral regurgitation

WBC: White blood cell count, PMN: Polymorphonuclear neutrophil, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, UA: Urinalysis, AST: Aspartate aminotransferase, ALT: Alanine transaminase, Cr: Creatinine, CXR: Chest X-ray, CT: Computed tomography, TTE: Transthoracic echocardiogram, TEE: Transesophageal echocardiogram

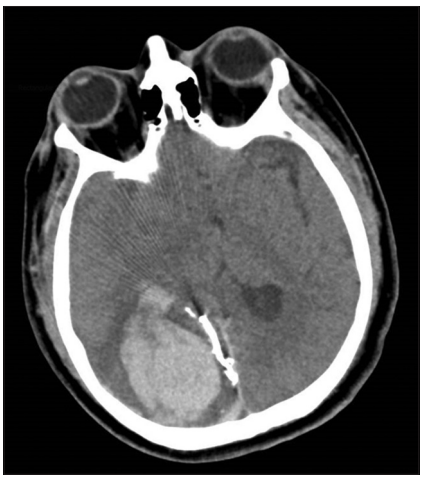

Figure 1: Head CT showing a large right parieto-occipital hemorrhage with edema secondary to the septic embolus. Note also the presence of marked mass effect as indicated by obliteration of the occipital pole of the right lateral ventricle

DISCUSSION

There are significant differences between CNS findings for patients with SBE versus ABE [Table 1].[2,4,7,8]

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].

Table 2: Neurologic complications of infective endocarditis

	SBE (*viridans streptococci*)	ABE (*S. aureus*)
Pathogen Virulence	Relatively avirulent	Highly virulent
Vegetation Location	MV > AV	MV>AV TV (in IVDAs)
Vegetation size	Small and nonfriable (low embolic potential)	Large and friable (high embolic potential)
Fever	<102°F (without chills)	>102°F (with chills)
Neurologic Complications/occurrence	Late (>3 weeks after presentation)	Early (<3 weeks after presentation)
Mental confusion (toxic encephalopathy)	−	+ (precedes CHF and/or ABM)
Embolic strokes†	+	+++
Mycotic aneurysms	+++	+
Microabscesses	+	+++
Macroabscesses	−	− (only with cyanotic heart disease or contiguous spread)
Focal seizures	+	+++*
CSF profile	Aseptic CSF profile	Purulent CSF profile
Gram stain	−	+ (Gram+cocci in clusters)
Lactic acid	Normal (<2.2 nmol/L)	(>6 nmol/L early with negative Gram stain (before CSF cultures become +)
Culture	Negative	Positive
Predominant WBC type	Lymphocytic response (initially may have mild PMN response)	Intense early PMN response
RBCs	− (+ with ruptured mycotic aneurysm)	− (+ with ruptured mycotic aneurysm)
Glucose	Normal	Decreased
Protein	Variably elevated	Variably elevated

SBE: Subacute bacterial endocarditis, ABE: Acute bacterial endocarditis, MV: Mitral valve, AV: Aortic valve, TV: Tricuspid valve, IVDA: Intravenous drug abuse, CHF: Congestive heart failure, ABM: Acute bacterial meningitis, CSF: Cerebrospinal fluid, WBC: White blood cell, PMN: Polymorphonuclear neutrophil, RBC: Red blood cell. *Generalized seizures should suggest a non-IE etiology. †Cerebral embolic events may occur alone or precede systemic emboli, but systemic emboli do not precede or predict cerebral emboli. Usually in the middle cerebral artery distribution with hemiparesis/hemisensory deficit. Retinal artery emboli, retinal artery hemorrhage, cortical blindness

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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