

Case Report

Ruptured mycotic cerebral aneurysm development from pseudoocclusion due to septic embolism

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Abstract

Background: Cerebral mycotic aneurysms are rare sequelae of systemic infections that can cause profound morbidity and mortality with rupture. Direct bacterial extension and vessel integrity compromise from septic emboli have been implicated as mechanisms for formation of these lesions. We report the 5-day development of a ruptured mycotic aneurysm arising from a septic embolism that caused a focal M1 pseudoocclusion.

Case Description: A 14-year-old girl developed acute left-sided hemiparesis while hospitalized for subacute bacterial endocarditis that was found after she presented with a 2-week history of fever, myalgia, shortness of breath, and lethargy. Mitral valve vegetations were confirmed in the setting of hemophilus bacteremia. Brain magnetic resonance (MR) imaging and angiography confirmed middle cerebral artery infarct with focal pseudoocclusion of the distal M1 segment. Given that further middle cerebral artery territory was at risk, a trial of heparin was attempted for revascularization but required discontinuation owing to hemorrhagic conversion. Decline of the patient's mental status necessitated craniectomy for decompression. Postoperatively, her mental status improved with residual left hemiparesis. On the third postoperative day (5 days after MR angiography), the patient's neurologic condition acutely declined, with development of right-sided mydriasis. Computed tomography (CT) angiography revealed a ruptured 19 × 16 mm pseudoaneurysm arising from the M1 site of the previous occlusion. Emergent coiling of aneurysm and parent vessel followed by hematoma evacuation ensued. At discharge, the patient had residual left hemiparesis but intact speech and cognition.

Conclusion: Focal occlusions due to septic emboli should be considered high-risk for mycotic aneurysm formation, prompting aggressive monitoring with neuroimaging and treatment when indicated.

Key Words: Cerebral aneurysm, infective endocarditis, mycotic aneurysm, septic emboli

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INTRODUCTION

Cerebral mycotic aneurysms are rare sequelae of systemic infections that can cause profound morbidity and mortality with rupture. Direct bacterial extension and vessel integrity compromise from septic emboli have been implicated as mechanisms for formation of these lesions. We report the 5-day development of a ruptured mycotic aneurysm arising from a septic embolism that caused a focal MI pseudoocclusion.

CASE REPORT

A 14-year-old girl with no major past medical history was sent to the emergency department for evaluation after her primary care doctor found a new heart murmur on physical examination in the setting of a 2-week history of fevers, myalgias, fatigue, and shortness of breath. She was admitted to the hospital for treatment of subacute endocarditis and was found to have blood cultures positive for nontypeable *Hemophilus influenzae*. Mitral valve vegetations were confirmed on transesophageal echocardiography. The patient's history was remarkable for a recent orthodontic appliance adjustment 1 month prior to admission.

Neurosurgical involvement began after the patient was found to have a right middle cerebral artery (MCA) territory infarct on computed tomographic (CT) imaging with symptoms starting 1 day earlier consisting of a left-sided hemiparesis and facial asymmetry. After removal of the patient's orthodontic appliance, magnetic resonance (MR) imaging and MR angiography of the brain confirmed completed infarction of approximately one-half of the right MCA territory due to a focal pseudoocclusion (high-grade stenosis causing near occlusion) in the distal M1 at the MCA bifurcation [Figure 1]. Endovascular revascularization was felt to be a poor option due to the >24-hour duration of symptoms prior to neurosurgical involvement, completed nature of the infarction, and large volume of MCA territory involved [Figure 2]. Because endovascular revascularization would not be pursued, in conjunction with the high-quality noninvasive imaging findings and the need to minimize pediatric exposure to ionizing radiation, diagnostic cerebral angiography was not pursued. A trial of heparin was initiated instead because the patient still had preserved speech, implying at-risk MCA territory. However, the heparin infusion was halted due to early signs of hemorrhagic transformation in the ischemic infarct. Subsequently, a decompressive craniectomy was performed due to increasing mass effect from the progression of stroke causing increased lethargy and left-sided hemiplegia [Figures 3 and 4]. After decompression, the patient's examination improved such that she was fluent with an age-appropriate level of consciousness and had moderate left-sided hemiparesis; her facial asymmetry persisted.

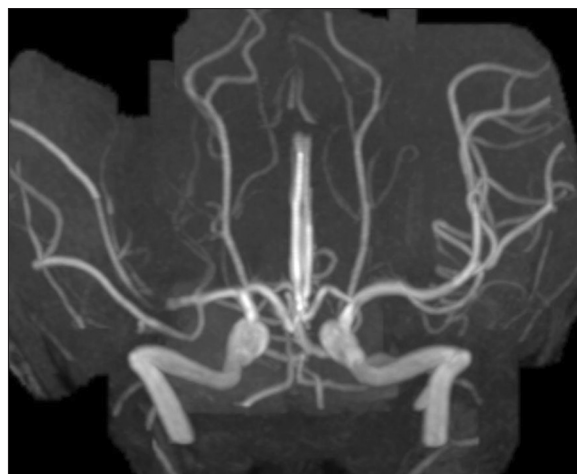


Figure 1: MR angiogram of the head reveals focal pseudoocclusion of the right M1

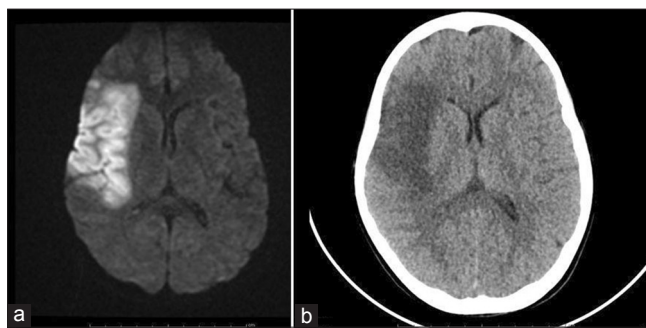


Figure 2: Diffusion-weighted magnetic resonance image (a) and noncontrast computed tomographic scan of the head (b) show completed infarct



Figure 3: Noncontrast CT scan of head shows hemorrhagic conversion of right MCA territory infarct

On postoperative day 3 after the decompressive craniectomy, 5 days after original presentation and MR angiography documenting the MI pseudoocclusion, the patient became acutely comatose with right-sided mydriasis. CT angiography of the head revealed a ruptured 19 × 16 mm mycotic aneurysm arising from the MI occlusion site,

associated with a large intracerebral hemorrhage and midline shift [Figures 5-8]. While the operating room was being prepared for a hematoma evacuation, the patient was taken to the endovascular suite for emergent coil embolization. Given completion of the M1 occlusion, extension of the MCA infarction, and rapid growth of the pseudoaneurysm, both parent vessel and pseudoaneurysm were rapidly coiled to complete obliteration [Figures 9-11]. From the endovascular suite, the patient was taken directly to the operating room for hematoma evacuation [Figure 12]. After emergency interventions were completed, pupil function was restored and standard intracranial pressure management ensued. Ultimately, she was discharged home with intact speech, cognitive function appropriate for age, and residual left hemiparesis.

DISCUSSION

Mycotic aneurysms account for 1.2-5.4% of all known cerebral aneurysms.^[1,3,8] The incidence of these aneurysms

in the pediatric population is approximately 2%.^[8,18] This incidence is likely an underestimation as mycotic aneurysms can go undiagnosed given their resolution after conservative medical therapy for microbial infection in patients without acute intracranial events or neuroimaging studies.^[7,13,15] The rupture rate is difficult to ascertain from current literature, ranging from <2% to 72%.^[5,9,11,17] Mortality rates have been reported as high as 30% for unruptured mycotic aneurysms and 80% ruptured mycotic aneurysms.^[5,8,9,19]

The majority of these pseudoaneurysms are formed after development of a septic embolism from infective endocarditis.^[4,6,12] This continues to hold true in the postantibiotic era.^[5,16] Less frequent causes include venous sinus thrombosis, bacterial meningitis, and intravenous drug abuse.^[2,5] The pathogenesis is related to the adherence of an embolus onto the vessel wall with subsequent direct bacterial extension and disruption of the underlying structures resulting in aneurysm



Figure 4: Noncontrast CT scan of head after decompressive craniectomy showing improvement of midline shift

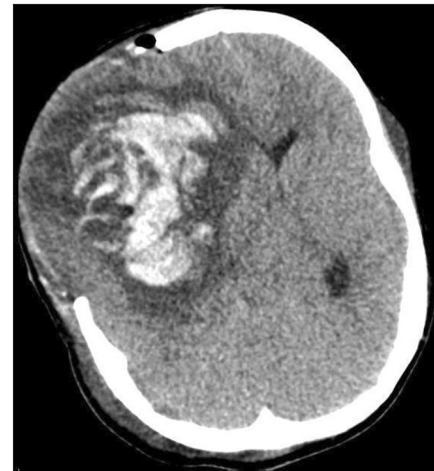


Figure 5: Noncontrast CT scan of head shows increased intracerebral hemorrhage with midline shift associated with the ruptured mycotic aneurysm



Figure 6: CT angiogram of head, thin-cut axial image, reveals right M1 ruptured mycotic aneurysm

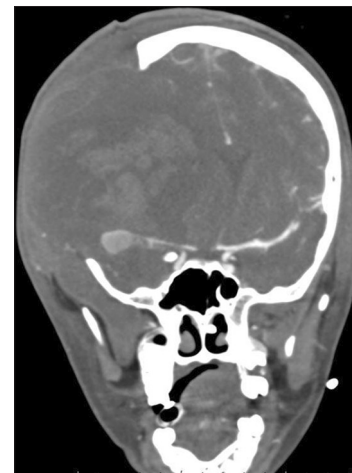


Figure 7: CT angiogram of head, coronal reconstruction, reveals right M1 ruptured mycotic aneurysm



Figure 8: CT angiogram of head, sagittal reconstruction, reveals right M1 ruptured mycotic aneurysm



Figure 9: Cerebral angiogram – early arterial phase, anteroposterior (AP) internal carotid artery injection – reveals right M1 mycotic aneurysm with occlusion of distal branches



Figure 10: Cerebral angiogram – mid-arterial phase; AP internal carotid artery injection – reveals right M1 mycotic aneurysm with occlusion of distal branches



Figure 11: Cerebral angiogram – early arterial phase, AP internal carotid injection – reveals coiled M1 mycotic aneurysm and parent vessel



Figure 12: Noncontrast CT scan of head status post hematoma evacuation showing improvement of mass effect

formation.^[6,12,14,20] This defect in the vessel wall is induced by cytotoxic effects from the septic embolus,

ischemic injury caused by occlusion of the vasa vasorum, and architectural breakdown of the media and adventitial layers caused by inflammatory response.^[6,10,12,14] Aneurysmal growth is promoted by repetitive arterial pulsation against the vulnerable, predisposed vessel wall.^[5,10] Molinari *et al.*^[14] demonstrated the pathogenesis of cerebral mycotic aneurysms using animal models. In this study, histological sections showed an acute inflammatory response to bacterial invasion of the muscularis layer with early aneurysmal dilatation as early as 24 hours after septic embolism development.

These mechanisms of mycotic aneurysm pathogenesis are relevant to the patient described above. Her imaging studies demonstrated a focal septic embolism from which a mycotic aneurysm developed, and subsequently ruptured within 5 days. This implies a hostile local milieu that compromised vessel integrity as described by the mechanisms above. Furthermore, one must ask if there is predictive value in identifying focal occlusions as a risk

factor for local mycotic aneurysm formation. Because the development of a mycotic aneurysm is such a rare occurrence, this question will be difficult to answer. We believe, given the profound morbidity and mortality associated with rupture of these lesions, focal occlusions due to septic emboli should be considered high risk for mycotic aneurysm formation, and as such, close surveillance and aggressive treatment are warranted.

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