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Surgical Management and Outcome of a Ruptured Aseptic Mycotic Cerebral Aneurysm: A Case Report

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Abstract

Background: Mycotic aneurysms are a rare subset of intracranial aneurysms caused by bacterial, viral or fungal sources. Rupture of an intracranial mycotic aneurysm is a life-threatening event untreated with a mortality range up to 80%.

Case description: Here, we report the case of a 21-year-old second year female medical student who suffered from a ruptured mycotic aneurysm of the middle cerebral artery with subarachnoid haemorrhage. After surgical resection of the aneurysm, intensive monitoring and treatment of cerebral vasospasm were performed. Further diagnostics for the focus of infection, including laboratory parameters, transoesophageal echocardiography and otolaryngoscopic diagnostic, showed normal findings without any sign of endocarditis, vasculitis or otolaryngitis. After 3 weeks, the patient was discharged in a very well condition.

Conclusion: Aseptic mycotic aneurysm is a rare subtype with posing significant therapeutic challenge. Surgical resection of those aneurysms might be safe and effective treatment.

Keywords: Mycotic aneurysm • Infectious intracranial aneurysm • Intracranial vasculitis • Ruptured aneurysm • Atraumatic subarachnoid hemorrhage • Aneurysm clipping

Introduction

Mycotic or Infectious intracranial aneurysms represent 2%-6% of all intracranial aneurysms [1]. These aneurysms develop due to a bacterial, rarely a viral or a fungal invasion of cerebral vessel walls, which causes local inflammation, subsequent vessel dilatation and aneurysm formation [2]. This may be attributed to a complication of hematogenous spread from septic emboli such as infective endocarditis [3,4], but also due to an extravascular cranial infection such as malignant otitis media, chronic mastoid infections or sinusitis. While there is no consensus regarding management of mycotic aneurysms, several reports suggest an aggressive approach even in unruptured cases. Despite the possibility of aneurysm regression by antimicrobial therapy, primary operative or endovascular treatment are favored due to its devastating outcome with mortality rates ranging from 30% in untreated unruptured to 80% in case of rupture [5-7]. Herein, we present a case of a successful neurosurgical treatment of a 21-year-old woman, who suffered from a ruptured mycotic aneurysm of the insular segment (M2 segment) of the middle cerebral artery (MCA) with subarachnoid hemorrhage (SAH).

Case Report

A 21-year-old woman presented with a sudden-onset headache combined with nausea and vomiting to the neurological department of the authors' institution. At admission, she had a Glasgow Coma Scale of 15 without focal neurological deficits or signs of meningism. The following Magnetic Resonance Imaging (MRI) revealed an atraumatic SAH over the right hemisphere and the patient was immediately transferred to our neurosurgical department. To localize the aneurysm precisely, computed tomography with angiography was performed which revealed an aneurysm of the M2 segment of the right MCA as the etiology of SAH (Figures 1 and 2). After obtaining interdisciplinary consensus, decision for surgical treatment was made due to limited access of endovascular approach. Right pterional craniotomy with frontal enhancement was performed. Intraoperatively, the brain was swollen with a sign of rerupture of the aneurysm showing a classical picture of an "angry brain". After microsurgical exposure of the M1 segment of the right MCA for proximal control, the aneurysm was dissected. Intraoperatively, the aneurysm had a fusiform configuration without a clearly distinguishable aneurysmal neck. Furthermore, the ruptured site on the dome was big as the aneurysm itself demonstrating active bleeding. In conclusion, the distal M2 segment had to be sacrificed to occlude the aneurysm. A clip was placed on the proximal side of aneurysm and the aneurysm itself was resected. Histological examination of the resected specimen showed a dilated segment of an artery, occluded by an adherent thrombus (Figure 3). The latter displayed focal accumulations of neutrophil granulocytes infiltrating the adjacent vessel wall (Figure 4). The findings were suggestive of a septic embolism, resulting in a weakening of the vessel wall, and were thus consistent with a so-called mycotic aneurysm. Granulomas, giant cells or fibrinoid segmental necrosis of the vessel wall indicating some form of autoimmune vasculitis were not found. However, the microbiological evaluation after 5 days of incubation revealed no bacteria or fungal infection. The blood culture test remained sterile as well.

After surgical treatment, the patient was treated in the intensive care unit according to the guideline of SAH treatment. Under constant hemodynamic monitoring, neurological examination and Transcranial Doppler Sonography (TCD) were performed every day up to day 14. Nimodipine 60 mg every 4 hours and caffeine 0.2 mg twice a day were administered. Immediately after surgery, the patient had a hemihypaesthesia and homonymous hemianopsia to the left side. The postoperative cCT revealed stable fronto-parietal SAH and a beginning demarcation of an infarction on the right hemisphere. The following MRI showed diffuse infarct demarcation on the right hemisphere with affection of the parietal lobe. One week after, digital subtraction angiography showed no aneurysm with mild sign of vasospasm (Figure 5). Ten days after

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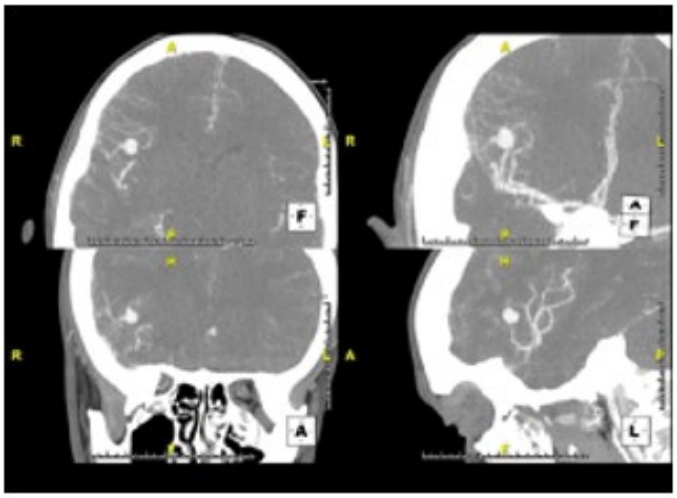

Figure 1. Demonstration of the aneurysm located in the M2 segment of the right MCA in a 2 × 2 Maximum Intensity Projection (MIP) overview of aneurysm reconstruction.

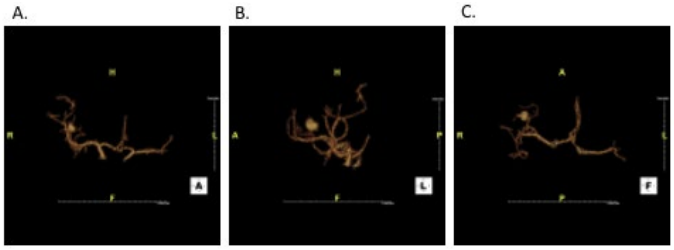

Figure 2. 3D reconstruction of angiographic cranial computer tomography. A: Anterior view; B: Lateral view; C: Frontal view.

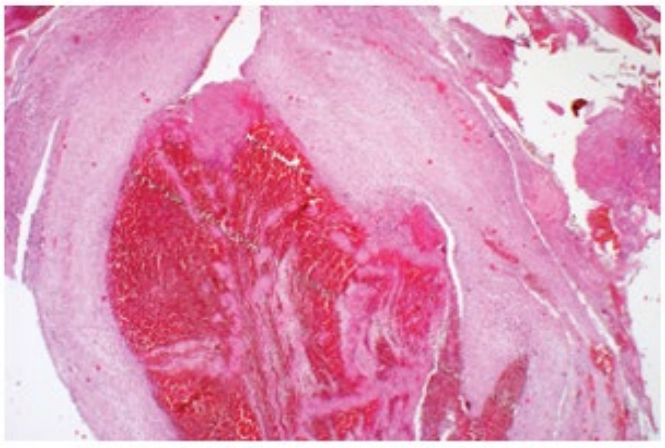

Figure 3. Cross section of the aneurysmatic artery occluded by an adherent thrombus. Hematoxylin and Eosin staining; magnification 20x.

the ictus, TCD controls showed manifest vasospasms on the right distal MCA with correlating clinical symptom of prominent hypesthesia of the left hand and face. For this reason, the patient remained on the ICU for monitoring and blood pressure regulation. After two weeks, the patients could be discharged from the ICU. Further diagnostics for the focus of infection, including laboratory parameters, transoesophageal echocardiography and otolaryngoscopic diagnostic were completed which showed normal findings without any sign of endocarditis or otolaryngitis. After 3 weeks, the patient could be discharged home in a very well condition with slight persisting hypesthesia on the left hand without presence of homonymous hemianopsia (modified Rankin Scale of 1). Three weeks after hospital discharge, the patient remains asymptomatic and has fully returned to her routine daily activities.

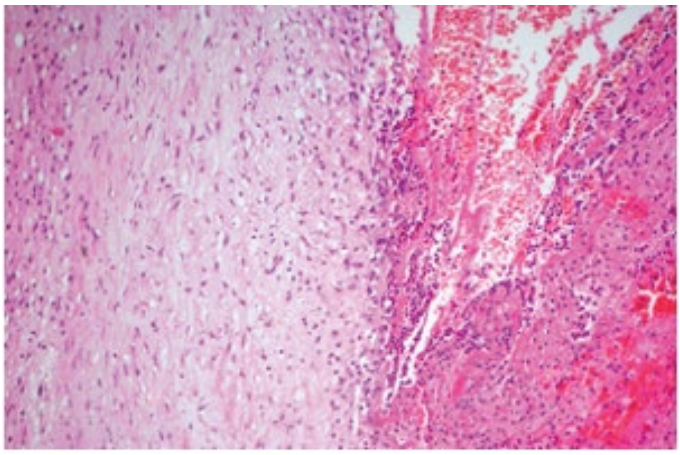

Figure 4. Detail of the thrombus and adjacent vessel wall, showing granulocytic infiltration and a microabscess. Hematoxylin and Eosin staining; magnification 100x.

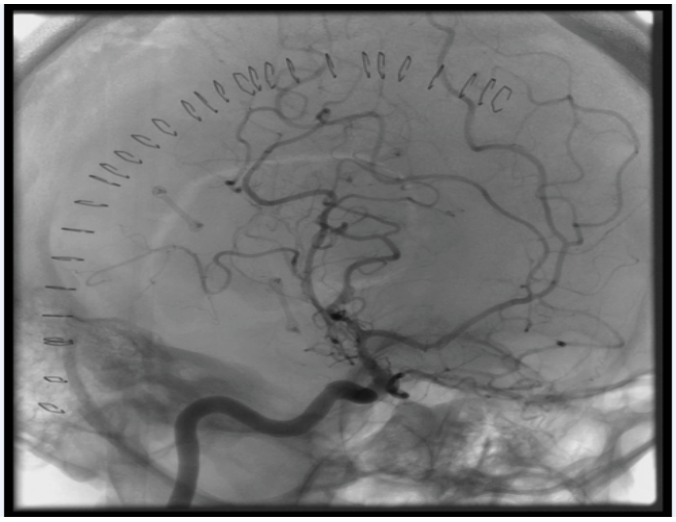

Figure 5. Post-operative angiography showing complete resection of the mycotic aneurysm.

Discussion

Mycotic aneurysms form a small fraction of all intracranial aneurysm that typically present in the periphery and are associated with a germ [8]. Previous studies reported heterogenous infection pathogens as its etiology and described challenging treatment of those subtypes of aneurysms [9-13]. Herein, we present a case of an aseptic ruptured mycotic aneurysm without a proof of microorganism successfully treated by microsurgical resection of the aneurysm. In general, classical aneurysms of non-infectious genesis present themselves centrally. In our case, the aneurysm was located in the periphery, which is considered as typical location of a mycotic aneurysm [1]. Besides infectious etiology, several studies describe autoimmune vasculitis as additional cause of development of mycotic aneurysm [14]. It is crucial to search for the focus of the mycotic aneurysm since the treatment of infectious genesis is different than those of autoimmune vasculitis. However, there was no clinical evidence of systematic infection nor vasculitis as part of an autoimmune process in our case. Both radiological imaging and histological examination ruled out systematic vasculitis, such that the etioogy of this mycotic aneurysm remains unclear. One of our hypotheses is that the presence of an asymptomatic previous infection might have caused a chronic artery wall inflammation resulting in the development of a mycotic aneurysm. In our case, we had to resect the aneurysm due to its fusiform configuration and big rupture site without clear neck border. In general, it seems that the mycotic aneurysm wall is thinner, more vulnerable and prone to rupture with tearing

down throughout the aneurysm itself. In view of those limitations, the treatment of ruptured mycotic aneurysm is challenging compared to the classical saccular aneurysm. Thus, an aggressive approach with primary surgical or endovascular treatment of mycotic aneurysm might be warranted to achieve better outcome for patients. Parallel to this, the diagnostic of focus should be done to prevent secondary risk of aneurysmal development.

Conclusion

Aseptic mycotic aneurysm is a rare subtype with posing significant therapeutic challenge. Primary surgical treatment of those aneurysms might be safe and effective.

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