Case Report

# A rare neurological infectious manifestation: Remedy accomplished exquisitely

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## Abstract

A 23-year-old Indian male with infective endocarditis with embolic infarct was on IV antibiotics. One month later, he presented with convulsions without any significant focal deficit, CT revealed large intraparenchymal bleed, which on Digital Subtraction Angiogram (DSA) showed a leaking intracranial Mycotic Aneurysm (MA). Aneurysm was embolized using 62% histoacryl glue under roadmap guidance following which the leakage was stopped successfully.

This case emphasizes the importance of considering the presence of MA in the differential diagnosis in the setting of suspected endocarditis and focal neurological deficits. Endovascular intervention and embolization with gluing is preferred for MA in view of friable nature of MA, thereby maintaining the stability of the patient.

**Key words:** DSA, gluing, histoacryl glue, infective endocarditis, Mycotic Aneurysm

### Introduction

An aneurysm is an abnormal focal arterial dilation. Pre-existing aneurysms can become secondarily infected, but aneurysmal degeneration of the arterial wall can also be the result of infection that may be due to bacteraemia or septic embolization, as in the case of mycotic aneurysm.[1]

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### Case report

A 23-year-old Indian male reported to the emergency department complaining of fever, headache and giddiness continuing since the past one month. At the time of presentation, he denied any visual changes, nausea, vomiting, numbness or weakness in the extremities. The patient was febrile at 102.3°F and his cardiovascular exam revealed a 3/6 holosystolic murmur prominent at the left midclavicular line with radiation to the axilla. The neurologic exam was normal and exhibited no deficits. Marked clubbing of his fingers on both hands was acknowledged.

The MRI signified a small acute non-haemorrhagic infarct in the left parietal lobe in white matter (Figure 1). The echocardiogram displayed large mobile, pedunculated vegetation on the posterior cusp of the mitral valve (Figure 2). Following this, intravenous ceftriaxone, linezolid and gentamicin were initiated immediately.

After 20 days of antibiotic therapy, the patient complained of a severe headache, vomiting and one episode of convulsion. A CT of the head revealed an intensely enhancing hypodense lesion (1.1x2.1x1.4cm) - mycotic lesion, with surrounding

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large intraparenchymal haemorrhage in the left parietotemporal lobe with significant surrounding edema, mass effect, midline shift of 8mm to the right and intraventricular extension of haemorrhage (Figure 3). Hence, anti-edema measures and anti-epileptics were initiated. The patient was neurologically stable with no deficits.

Subsequently, a DSA was done, which disclosed a ruptured fusosaccular mycotic (postinfectious) aneurysm measuring 11x22mm arising from the distal parietal branch of the left middle cerebral artery and distal parietal branch projecting posterolaterally (Figure 4).

In same sitting, using 6F Neuron as guiding catheter placed in left internal carotid artery (ICA) and using Headway Duo 167cm micro catheter and Traxcess 0.014 microguidewire, the left parietal branch feeding the mycotic aneurysm was cannulated and micro catheter placed at the neck of aneurysm and distal parent artery, and the aneurysm was embolized using 62% histoacryl glue under roadmap guidance. The angiogram check showed complete obliteration of distal parent artery, mycotic aneurysm and normal filling of rest of left ICA branches (Figure 5).

Due to the presence of intracranial haemorrhage and acute infarct, replacement of his mitral and aortic valves was deferred. The patient was transferred to a long-term nursing facility and was prescribed a six week course of IV antibiotics. On a subsequent visit to the OPD, the patient showed no neurological deficit.

**Discussion**

Cerebral mycotic aneurysms (CMA) are rare inflammatory neurovascular lesions. The patients are found to develop terminal arterial branches that rupture spontaneously causing subarachnoid haemorrhage and intracerebral haemorrhage. Significant morbidity and mortality have been reported in CMA. [1]

CMA uniquely develops in patients with infective endocarditis. The cardiac vegetations are friable and cause septic embolization. These emboli settle in the intracranial vessels leading to cerebral complications including vascular events and infections. Vasa vasorum, that has the emboli settled in them leads to inflammation of the adventitia. Consequently, the infection spreads.[2] As observed, the CMA forms more commonly in anterior circulation and enlarges into fusiform and eccentric aneurysms. [3]

Histologically, CMA shows acute neutrophilic infiltration, intimal proliferation and internal elastic lamina destruction. Many organisms have been isolated because of CMA, among them *Streptococcus viridians* and *Staphylococcus aureus* are found to be the commonest organisms.[1]

The patients of CMA mostly complain of fever, along with symptoms such as intracranial aneurysms such as headache, seizures, behavioral changes, ocular palsies etc.[4]

DSA has been the benchmark for detection of CMA. However, the multi detector CT angiographic imaging gives an increased resolution of the vascular tree and the aneurysm. In such cases, magnetic resonance angiography is an emerging modality. [5]

In this regard, the recommended treatments are not effectually defined. The medical treatment includes prolonged intravenous antibiotic administration for six weeks. However, the patient should be closely observed and followed with serial angiography to monitor the aneurysm size and resolution by spontaneous coagulation.[2] Also, endovascular therapy has a good safety profile, including cases with distal aneurysms. It may be safer as compared to open craniotomy with experienced hands. The patients, who develop intraparenchymal haemorrhage or need clot evacuation, are better treated with surgical therapy of open craniotomy and aneurysm clipping. Further, surgical measures also help preserve the distal circulation better than endovascular therapy.[6]

In spite of the fact that surgical therapy is usually preferred in cases of intraparenchymal bleeding for patients with CMA, glue embolization was recommended for our patient. Histoacryl glue embolization sticks to the walls and injecting it allows the collected mass to progress along and stick to the entire wall of aneurysm. This enables the complete filling of aneurysm and preserves the parent artery.[7]

Besides being friable in nature, surgical intervention may lead to further intraparenchymal bleeding. Probably, this would have determined a better outcome in case of our patient.

In case of unruptured aneurysms, cardiac surgery for valve repair is considered safe which reduces the subsequent risk of embolization and sepsis. Cardiopulmonary bypass and valve replacement are the options that can be offered to the patient. It is necessary that a ruptured CMA should be repaired first, unless the patient has left heart failure. [2, 8]

In comparison with the unruptured aneurysms, the ruptured CMA has a poorer prognosis with a higher likelihood for mortality and neurological decline despite intervention.[9]

**Conclusion**

Despite intervention, the morbidity and mortality of post CMA intraparenchymal bleeding is high. In spite of the fact that surgical therapy is mostly preferred in cases of intraparenchymal bleeding in patients of CMA, glue embolization was recommended for our patient in order to avoid the possibility of intracerebral haemorrhage and to ensure the preservation of the patent vessels.

In the case hereby discussed, the patient has recovered well and survived without any significant neurological deficit.

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