



CLINICAL IMAGES

Intracranial mycotic aneurysm due to infective endocarditis — successful NBCA glue embolisation

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Symptomatic intracranial mycotic aneurysms (ICMAs) are one of the most serious neurological sequelae of infective endocarditis. Changing cardiac murmurs and the sudden onset of neurological symptoms and signs are markers of this lethal combination. If computed tomography (CT) scan demonstrates an intracerebral haemorrhage in a patient with such a combination of findings, further imaging using cerebral angiography is essential. The primary treatment is antibiotics for at least 6 weeks. Transcatheter embolisation or surgical intervention may be required in some instances. We report on a patient with an intracerebral haematoma referred from a peripheral hospital to the neurosurgery department at our institution. The diagnosis of the cardiac condition, and probable mycotic basis for the intracranial aneurysm, were first suggested following peri-procedural examination during endovascular therapy in our angiography suite.

Case report

A 24-year-old right-handed male presented to a district hospital with acute onset of severe headache on rising that morning. This was associated with inability to move the left side of his body, slurred speech and involvement of the lower half of the left side of his face.

No preceding trauma, recent illness or the use of recreational drugs could be ascertained. Family members claimed that the patient had been well the previous day. On admission his general examination was recorded as normal. Blood pressure was 160/60 mmHg and pulse 76 beats per minute.

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The Glasgow coma score was low initially, recorded at 13/15. There were no stigmata of immunocompromise. Dense left hemiplegia and left upper motor neuron facial nerve palsy were identified. Meningeal irritation was noted with nuchal rigidity and positive Kernig's sign. A lumbar puncture was performed and this revealed uniformly bloodstained cerebrospinal fluid (CSF). Xanthochromasia was documented and no organisms were isolated on microscopy. With suspected subarachnoid haemorrhage, the patient was referred to the neurosurgeons at our hospital for further management.

A CT scan performed on admission (Figs 1 - 3) revealed a large right frontoparietal intracerebral haematoma straddling the sylvian fissure. A surrounding rim of vasogenic oedema and moderate mass effect were noted. Subarachnoid haemorrhage and a blood/CSF level were present in the

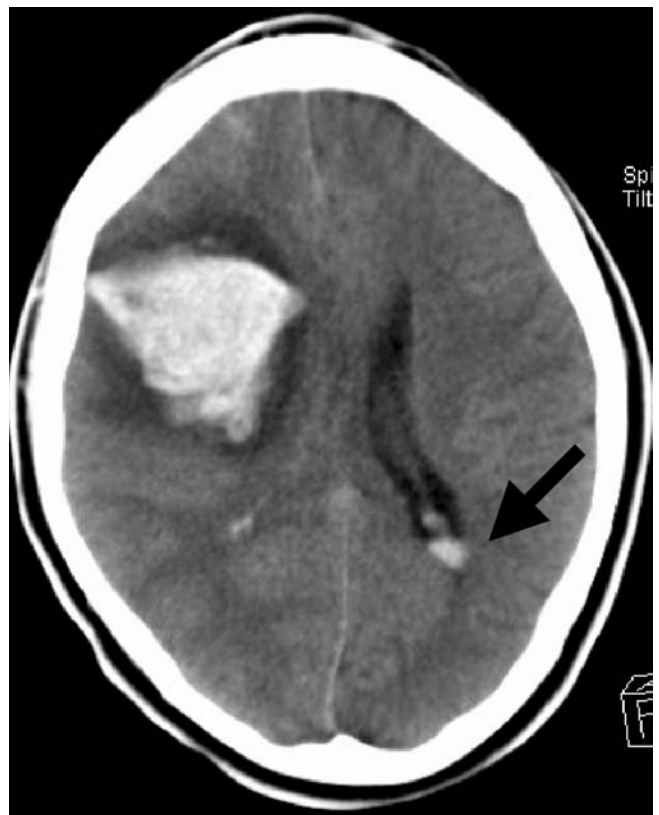


Fig. 1. Unenhanced axial CT brain scan demonstrating right frontoparietal intracerebral haematoma with effacement of the lateral ventricle and surrounding oedema. Note the blood/CSF level in the left occipital horn (arrow).



Fig. 2. Post-intravenous contrast CT brain scan demonstrating right cerebral gyriform enhancement and blood/CSF level in the left occipital horn (arrow).

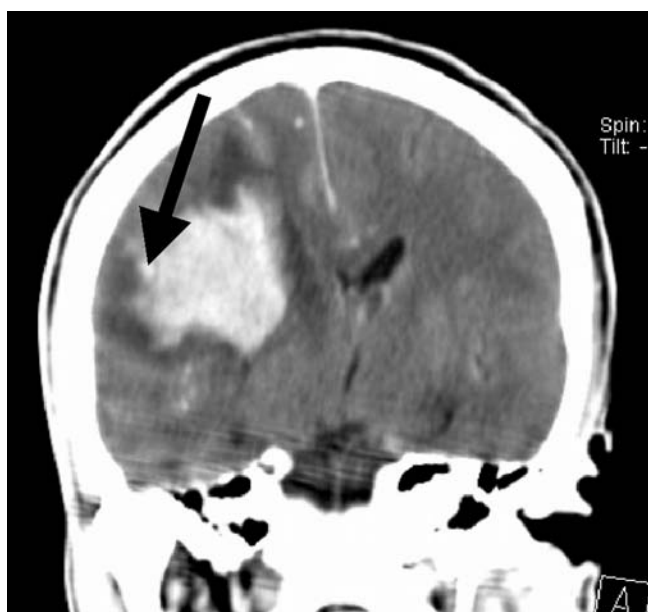


Fig. 3. Coronal reformat of the enhanced CT brain scan. The arrow indicates the aneurysm in the right MCA territory. Note the mass effect with subfalcine herniation and oedema.

occipital horn of the left lateral ventricle. No cranial vault fractures were identified. Following intravenous contrast administration a rounded enhancing focus was present lateral to the haematoma. In retrospect this represented the aneurysm.

The patient was admitted to the neurosurgical high-care ward with a clinical diagnosis of intracerebral haematoma and subarachnoid haemorrhage. The necessary medical care was immediately instituted and a cerebral angiogram was planned the following day.

Four-vessel angiography (Figs 4 - 6) identified a 5.0×3.5 mm pear-shaped aneurysm of the pre-central branch of the right middle cerebral artery (MCA). Moderate mass effect was present due to the haematoma. No other aneurysms or vasospasm was noted.

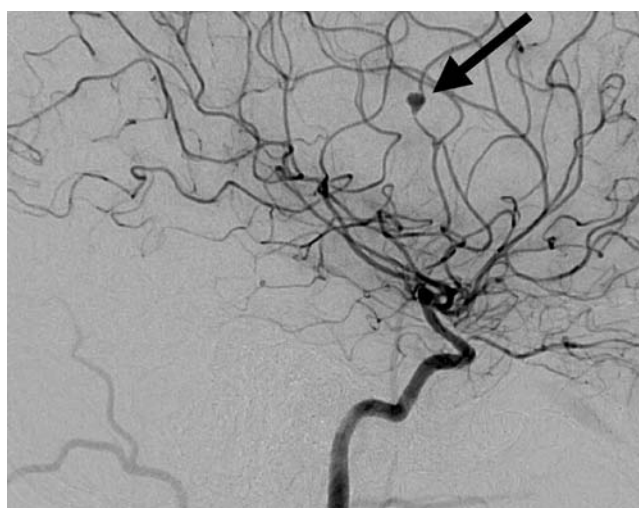


Fig. 4. Lateral projection of the right internal carotid artery angiogram. Pear-shaped aneurysm (arrow) noted of the precentral branch of the right MCA.

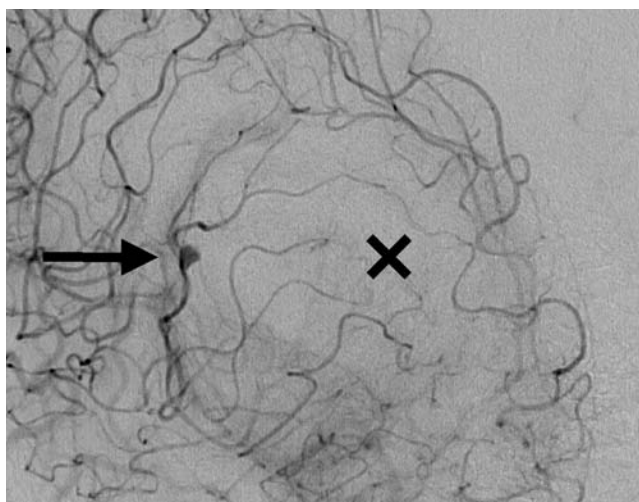


Fig. 5. Oblique view demonstrating aneurysm (arrow) and splaying of vessels (X) owing to the mass effect of the haematoma.

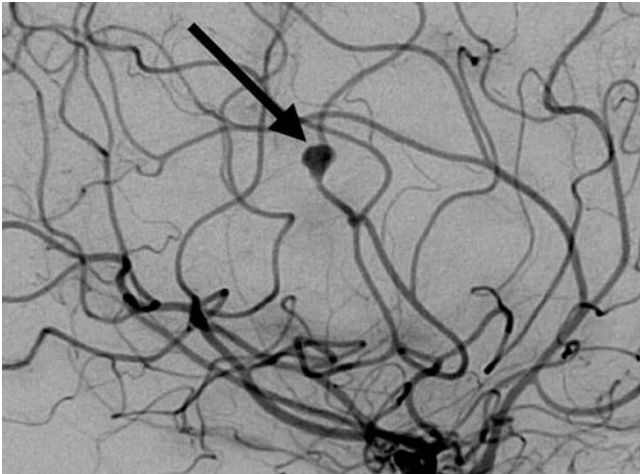


Fig. 6. Magnified view of the aneurysm (arrow).

There was no neurosurgical indication for evacuation of the aneurysm. We then proceeded to address the aneurysm by the endovascular route. Under general anaesthesia and heparinisation, a 6Fr Softip XF guide catheter was introduced into the distal right cervical internal carotid artery (ICA). An Ultraflow microcatheter and 0.10 Silverspeed guide wire were advanced into the right MCA (Fig. 7) by exchange. On reaching the neck of the aneurysm in the precentral branch, 0.4 ml of a 50 : 50 mixture of lipiodol and N-butyl-cyanoacrylate (NBCA) was slowly injected (Fig. 8). The aneurysm was completely embolised and there was glue penetration in the parent precentral branch, proximal and distal to the aneurysm neck. The proximal precentral and other right MCA branches were spared (Fig. 9).

The water hammer character of the right groin pulse, momentarily suspected at puncture, became more evident during groin compression. Intraprocedural monitoring documented a consistently wide pulse pressure and tachycardia of ± 100 beats per minute. We then undertook

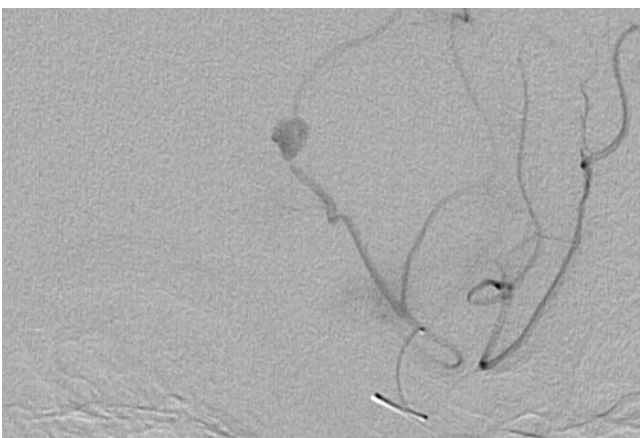


Fig. 7. Pre-embolisation contrast injection via microcatheter in the right MCA. Note the aneurysm (arrow).

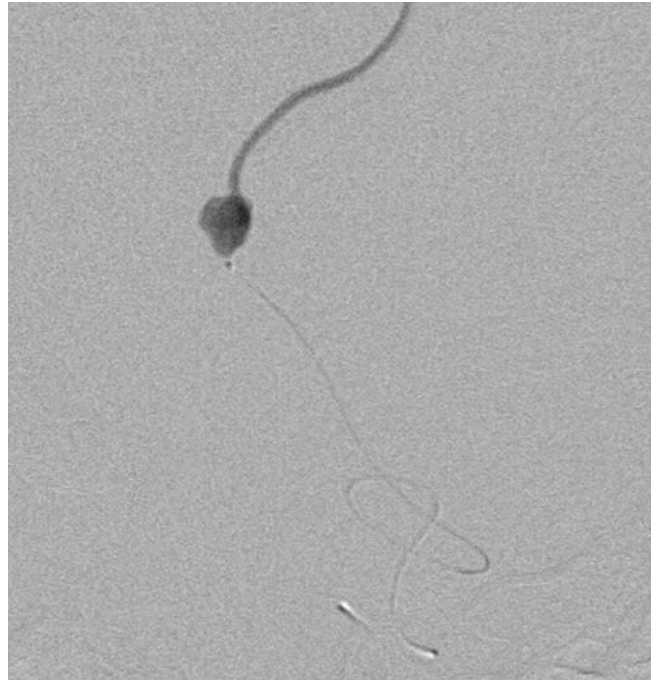


Fig. 8. Microcatheter advanced to the neck of the aneurysm in the precentral branch of the right MCA.

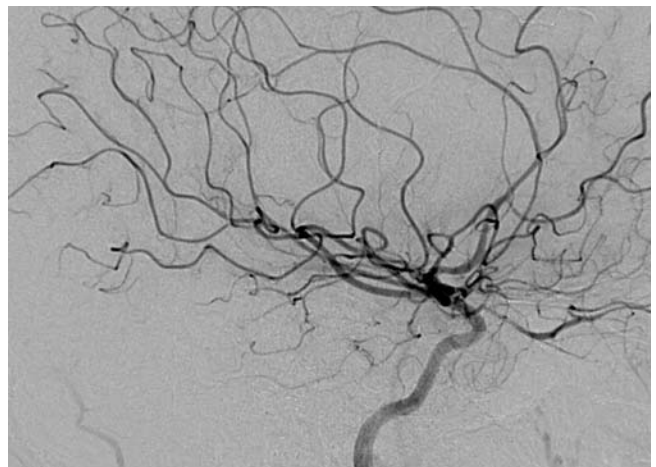


Fig. 9. Lateral view of the right ICA angiogram after glue embolisation demonstrating complete embolisation of the aneurysm sparing the parent artery.

careful precordial examination on the angiography table. The apex beat was displaced to the anterior axillary line in the fifth intercostal space. The first heart sound was soft. A $3/6$ pansystolic murmur was auscultated at the apex, radiating to the axilla. At the left sternal border we found a $2/4$ early diastolic murmur, and at the aortic area a $3/6$ ejection systolic murmur. Suspecting aortic valve infective endocarditis (causing mixed aortic valve disease with dominant aortic regurgitation) and associated mild left heart failure, we recommended an echocardiogram as well as blood cultures and activity bloods.



On echocardiography, multiple small vegetations were identified on the aortic valve, causing significant aortic regurgitation, with failure of coaptation of the aortic valve cusps. Left ventricular hypertrophy and moderate functional mitral regurgitation were noted. A posterior pericardial effusion measuring 1.6 cm was present.

Blood cultures were negative. The erythrocyte sedimentation rate was raised at 49 mm/hour.

A diagnosis was made of ruptured right MCA mycotic aneurysm complicating aortic valve infective endocarditis. Follow-up CT post-embolisation (Fig. 10) revealed a reduction in size of haematoma with lipiodol visualised in the embolised branch and aneurysm. The aneurysm remained occluded at check angiography 2 weeks later. No new aneurysms were demonstrated. Clinically, although hemiparetic, the patient had improved markedly and was capable of the activities of daily living. The endocarditis is still being treated. We intend doing a follow-up angiogram at 3 months.

Discussion

ICMAs have been reported in less than 10% of cases of infective endocarditis.¹ This figure is perhaps an underestimate as a fair number are asymptomatic and resolve with antibiotic therapy.² These aneurysms are usually located peripherally and involve the MCA or its branches in most cases. *Corr et al.*³



Fig. 10. Post-embolisation CT scan of the brain. Note lipiodol contrast in the embolised aneurysm (arrow).

reported a central location in one-third of the ICMAs in their study.

Pathogenetic mechanisms quoted in the literature note vasa vasorum micro-embolisation and vascular adventitial seeding, with subsequent localised necrotising arteritis and septic aneurysmal dilatation.⁴ The presence of vasa vasorum in the intracranial arteries is debatable; rather, the nutritive supply to these vessels is thought to be via surrounding CSF. In principle, the fundamental process involves the lodging of an infected embolus at some point in the vessel. Progressive intimo-medial weakening occurs with propagation of the infectious process to the outer layers. The arterial pulsation against a weakened wall leads to aneurysm formation. Pathological examination of these aneurysms reveals marked friability, which makes them difficult to clip surgically.

Clinically, patients with ICMAs present with non-specific neurological symptoms including acute headache, dizziness, seizures and altered sensorium. Long-tract signs are evident in patients with infarction or those with ruptured aneurysms resulting in intracerebral haematoma. Subarachnoid haemorrhage may be the dominant presentation in patients with ruptured ICMAs.

For unruptured ICMAs, the mortality rate is 30%. Following rupture the prognosis is poor, with an 80% mortality rate.⁵ Much controversy therefore exists regarding management of these lesions. The mainstay of treatment is immediate commencement of intravenous antibiotic therapy. It is generally accepted that endocarditis-related aneurysms will follow one of three possible outcomes. There is ample evidence that one-third resolve completely with antibiotic therapy

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alone.^{2,3,6} One-third will demonstrate no significant change in size.³ Of the remaining third, half enlarge on treatment while the other half reduce in dimensions.³ Unfortunately there is no way of predicting if a particular ICMA will regress or rupture on appropriate antibiotic treatment.

Traditionally, the neurosurgical management of ICMA involves evacuation of the haematoma with ligation of the parent artery.⁴ Surgery in the acute setting is technically difficult because of the friable nature of the necrotic tissue. It has been reported that once demonstrated, surgical treatment of a mycotic aneurysm is more deleterious than medical treatment alone.⁷ Peripherally located aneurysms are a greater challenge and frequently unnecessary brain damage is incurred in attempts to expose the aneurysm. The presence of surrounding clot also makes aneurysm identification particularly difficult.

Rapid improvements in technology have resulted in the endovascular approach coming into vogue. Distal intracranial vascular navigation, which is now increasingly possible, has made percutaneous obliteration of peripheral ICMA a feasible and effective therapeutic option.^{8,9} Techniques used include injection of NBCA glue into the affected vessel or deployment of short, detachable, platinum coils into the aneurysm.^{8,10} Generally, the cortico-cortical pial network provides collateral support to the distal circulation. Using NBCA glue to embolise the distal parent vessel prevents retrograde collateral filling of the aneurysm. The major advantage of the endovascular techniques is that they avoid craniotomy and surgical handling of the swollen brain. Post-intervention recovery is less complicated and usually quicker.

Currently there is general consensus regarding the

management of ICMA. Four to six weeks of antibiotic therapy is mandatory in all patients with suspected endocarditis-related ICMA. If there is evidence of aneurysmal rupture, the endovascular approach is the better option as it avoids surgical dissection in an oedematous brain with necrotic tissue and local clot. Follow-up of these patients is usually by repeat cerebral angiography to assess aneurysm response to treatment and to detect new aneurysms that may have developed.³ The use of CT angiography or magnetic resonance angiography in larger aneurysms (> 3 mm diameter) is theoretically possible, but still to be evaluated. For aneurysms that remain unchanged or increase in size on medical therapy, surgical or transcatheter intervention is required and may be individualised to the particular case.⁹ In our opinion, endovascular techniques should be the primary treatment option in ICMA requiring intervention after trial of medical therapy.

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