

Subarachnoid haemorrhage as a late complication of infective endocarditis: a case report

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Abstract

Rupture of an infectious intracranial aneurysm causing subarachnoid haemorrhage is a rare complication of infective endocarditis. We report a case of subarachnoid haemorrhage that occurred as a late complication, in an infective endocarditis patient, on the 30th day of successful treatment, which is an extremely rare occurrence. He was a mainline drug addict with a history of rheumatic valvular heart disease.

Key words: infective endocarditis, subarachnoid haemorrhage, rheumatic valvular heart disease

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Introduction

Despite advances in diagnostic modalities and treatment options, mortality of infective endocarditis (IE) has remained unchanged at 30% over the past few decades, as per the population-based registry study in Finland in 2021(1). This is a case of subarachnoid haemorrhage (SAH) that has occurred almost a month after successful intravenous antibiotic therapy for IE.

Case Presentation

A 36-year-old male presented with intermittent fever and generalised body weakness for two months and worsening exertional dyspnea for two weeks. He is an intravenous heroin addict with a background of rheumatic valvular heart disease. A

transthoracic echocardiogram (TTE) done 12 years ago revealed grade III mitral and grade II aortic regurgitation. He had defaulted clinic follow-up and antibiotic prophylaxis for the past 10 years.

On examination he was clinically stable with a temperature of 37.4 °C. Auscultation revealed bi-basal crepitations and a grade III pansystolic murmur at the apex. He had no peripheral stigmata of IE, focal neurological signs or evidence of meningeal irritation.

His investigations revealed leukocytosis with predominant neutrophils (WBC 15840 / μ L), a high C-reactive protein (CRP) level (160 mg/L) and microscopic hematuria in urine full report. Streptococcus species were isolated in three blood cultures with antibiotic sensitivity to Cefotaxime, Ceftriaxone and Vancomycin. Electrocardiogram demonstrated sinus tachycardia and possible left atrial enlargement.

His TTE revealed severe mitral regurgitation with a damaged anterior mitral valve leaflet and a restricted posterior mitral valve leaflet. He also had moderate to severe aortic regurgitation. There was a 1.39 cm x 0.384 cm size linear vegetation on the anterior mitral valve leaflet with valve leaflet perforation. Left atrium and left ventricle were enlarged. (Figures 1A and 1B)

Empirically intravenous Gentamicin and Ceftriaxone were started as per the guidelines.

Treatment response was monitored both clinically and biochemically with CRP levels and renal functions. As the CRP levels were rising, Ceftriaxone was changed to intravenous Cefotaxime on Day 11, based on microbiology opinion. His CRP continued to rise until day 20 and then gradually declined. Repeated 2D echo after three weeks of therapy, confirmed improvement with a small residual vegetation (0.28 x 0.29 cm). (Figure 2)

Figure 1 - Linear vegetation (Arrow) attached to anterior mitral valve leaflet in the modified parasternal long axis view of the 2D echocardiogram(A) and apical four chamber view(B)

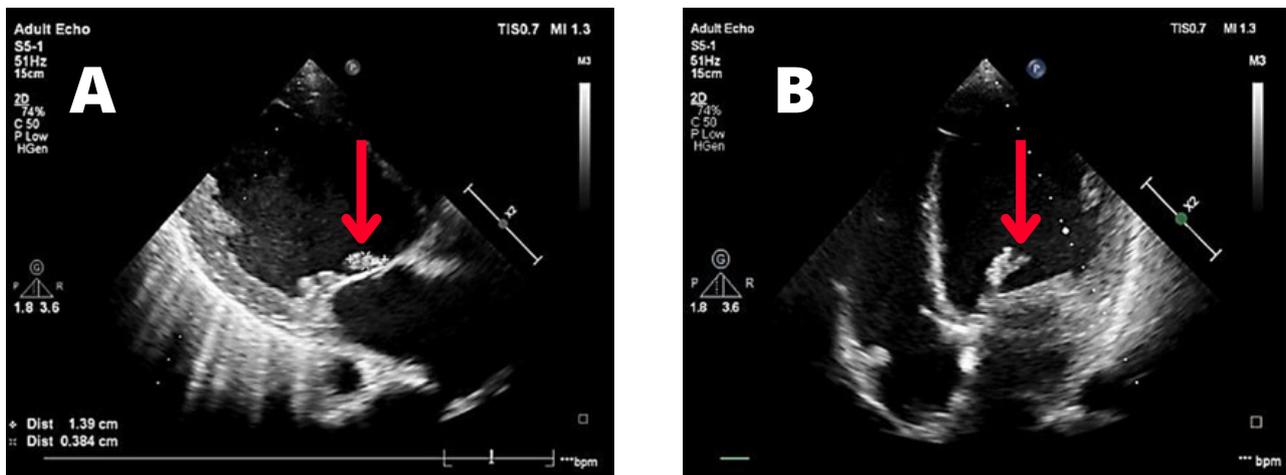


Figure 2 - Resolving small residual vegetation on the anterior mitral valve leaflet



Thirty days after admission, he developed a severe headache with short term memory loss. On examination his GCS was 15/15 without focal neurological signs. Non-contrast Computed Tomography (NCCT) brain demonstrated a subarachnoid haemorrhage with mild hydrocephalus. (Figure 3)

CT Angiogram of cerebral vasculature demonstrated an aneurysm in the M2 segment of

right middle cerebral artery (MCA) (Figure 4). Urgent surgical repair was done with clipping of the aneurysm. His postoperative period was complicated by a left sided upper and lower limb weakness which developed on day seven. Repeat NCCT of the brain showed a new infarction on the right side. He was managed appropriately and discharged with a plan to undergo dual valve replacement later.

Figure 3 - Horizontal planes at different levels of non-contrast CT brain

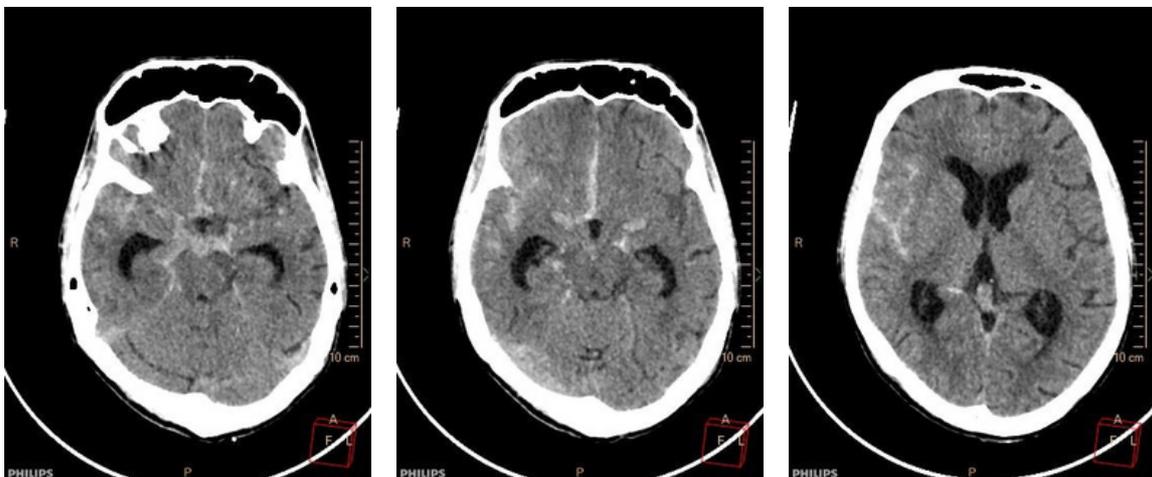
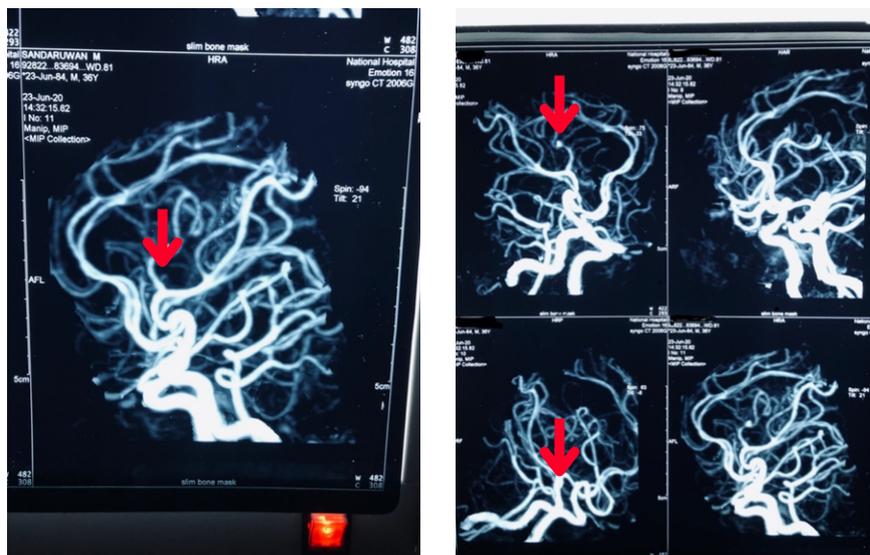


Figure 4 - CT angiogram showing right middle cerebral artery aneurysm (Arrows showing the aneurysm)



Discussion

Neurological complications occur in 30-40% of cases of IE which include strokes, transient ischaemic attacks, intra cerebral haemorrhage (ICH), cerebral abscesses, spinal epidural abscesses, infectious intracranial aneurysms, mononeuropathy, seizures, meningitis and toxic encephalopathy (2). While ischaemic stroke is the most common neurological complication, intra cerebral haemorrhage constitutes 12-30% of the complications (2,3) However, asymptomatic micro bleeds due to immunologic vasculitis are increasingly being recognised as the commonest neurological complication (2).

ICH following an embolus in IE, can occur by three methods; the most common method is infarction due to a sterile embolus resulting in secondary haemorrhage; secondly, septic embolus causing acute erosive arteritis and rupture; thirdly, septic embolus during effective antimicrobial therapy forming a subacute aneurysm leading to latent rupture as in our patient. However, the offending infected emboli may escape clinical recognition due to its small size, incomplete obstruction of the flow or collateral circulation which prevents infarction (4).

Aneurysms created by septic emboli are known as infectious intracranial aneurysms or mycotic aneurysms. Infectious intracranial aneurysms are a relatively rare complication of IE, found only in 2-4% of patients and accounts for 5-12% of patients with neurological complications due to IE. However, the actual incidence is probably higher, as they can be clinically silent and subsequently resolved with antibiotic therapy. Moreover, their uncertain risk of rupture also may influence the rate of detection (3).

SAH as a complication of IE is uncommon and is associated with rupture of an infectious intracranial aneurysm (5). Patients may present with a spectrum of non-specific constitutional symptoms or neurological symptoms such as seizures, focal neurological deficits, encephalopathy and ophthalmoplegia (2,3). Our

patient only had a severe headache and short-term memory loss to indicate an intracranial pathology in the setting of IE.

Imaging plays a major role in the management of a patient with suspected SAH. CT angiography (CTA), magnetic resonance angiography (MRA) or digital subtraction angiography are the imaging modalities used. Our patient's CTA revealed an aneurysm in the M2 segment of the right MCA. MCA and its branches are the most common sites that infectious intracranial aneurysms form and accounts for 78% of IE related aneurysms (6). Cerebral aneurysms that are ≥ 3 mm and ruptured aneurysms can be easily diagnosed using CTA and MRA, which have high sensitivity and specificity (2).

Management of infectious intracranial aneurysms depends on the size of the aneurysm, location, availability of resources and expertise, and most importantly, whether the aneurysm has ruptured or not. Although antibiotics have improved the outcome of IE, there is no significant reduction of neurological complications (5). An infectious intracranial aneurysm develops with a septic embolus that lodges in a cerebral artery which weakens the arterial wall. Aneurysms are more likely to develop before or within the first week of initiation of antibiotics (3). Treatment with sensitive antibiotics do not prevent aneurysm development, but may prevent their early rupture (5).

Recommendations include antibiotics and serial imaging for stable, small, unruptured aneurysms and endovascular repair or open surgical clipping for large or ruptured aneurysms (3). Surgery was encouraged for our patient as he developed neurological manifestations following rupture of the aneurysm.

Conclusions

This case report signifies the possibility of SAH late in the course of IE, even after antibiotic treatment and clinical, biochemical and echocardiographic improvement. Hence the

possibility of intracranial aneurysm should also be considered in patients without neurological manifestations as timely diagnosis, investigation and intervention are paramount for a favourable outcome.

Article Information

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