Infective Endocarditis - Rare Cause of Intracerebral Haemorrhage

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Abstract
Cerebral haemorrhage occurs rarely in infective endocarditis. Here, we present a case of young female with severe intracerebral haemorrhage. Later, she found to be a case of infective endocarditis with mitral valve prolapse and on investigation blood culture grew S. aureus.

Introduction
Intracerebral haemorrhage (ICH) occurs in about 5% of patients with infective endocarditis.1 These haemorrhages are usually attributed to ruptured mycotic aneurysms, even when no aneurysm is demonstrable.1 As mycotic aneurysms are sometimes obliterated by the haemorrhages they produce, their arteriographic and even pathologic demonstration is not always possible.2 However, ICH in infective endocarditis can also result from septic erosion of the arterial wall with rupture without a well-delineated aneurysm.3 Surgical intervention in this situation is usually not feasible, as the necrotic fusiform segment cannot be repaired but requires resection with or without pedicle bypass grafting.4

Case History
25-year old Asha w/o Laxminarayan admitted in hospital with sudden onset left hemiparesis and on evaluation patient had fever on and off last 4 months for which she took medicine from local practitioners. Patient also complained of headache since 1 month which was not associated with any projectile vomiting. Other than the history of fever the patient was negative for any recent sore throat or other symptoms of URI. There was no history of joint pain and palpitation, syncope, dyspnoea in the past. There was no history of smoking ,anticoagulation and IV drug abuse.

On Examination: The patient was averagely built female with fever and tachycardia. She was haemodynamically stable and did not appear to be in any apparent distress. On physical examination mild splenomegaly was there and on CVS examination systolic murmur in mitral area which was radiating to posterior axilla. On CNS examination pupil was normal in size reacting to light. and both left limb hypotonia and left sided plantar extensor.

Then CT Head done which was showing as acute focal intraparenchymal haematoma, particularly in the right parietal lobe (Figure 2).

On baseline investigation pt was moderately anaemic, leucocyte count was marginally raised with elevated ESR. Serum electrolytes, LFT, RFT were within normal limits. Coagulation studies and platelet count were normal.

CXR was normal and pt was found to be HIV negative. A 2 D Echo was performed which demonstrated a large mobile vegetation 6-7mm on the subvalvular region of mitral valve (Figure 1). Empirical treatment started for infective endocarditis and the blood sample sent for culture and sensitivity, later on culture was positive for Staph. aureus.

MRI was performed which showed large intra-parenchymal haematoma in the right high parietal region with perilesional oedema and mass effect as showing in Figure 4. No obvious evidence of vascular malformation on MR angiography. No obvious sinus thrombosis on MR angiography (Figure 3).
ICH complicating infective endocarditis is the result of a spectrum of arterial injury ranging from acute, pyogenic necrosis to large, aseptic aneurysms that may rupture weeks to months after bacteriologic cure. While the term “mycotic aneurysm” has usually been applied to both processes, the difference is not merely semantic.

Septic emboli appear to be a necessary substrate for ICH, although clinically recognised ipsilateral embolism precedes ICH in only 40% of cases.\(^1,5\) The offending infected emboli may escape clinical recognition by being small, by incompletely obstructing flow, or by preventing infarction by collateral circulation. Clearly, lack of antecedent clinical brain embolism does not eliminate the risk of ICH, as most haemorrhages occur without recognised, antecedent embolism.

Symptomatic ICH associated with S. aureus occurred within 48 hours of admission. This propensity for early haemorrhages in S.aureus endocarditis has been noted by others.\(^1,6\) ICH due to septic arteritis usually occurs during uncontrolled infection. Surgical treatment is difficult, requiring sacrifice of the involved artery, sometimes with microvascular pedicle/bypass surgery, as there is not a well-delineated aneurysmal neck that can be readily clipped.\(^5\) However, late rupture of mycotic aneurysm after otherwise successful treatment of infective endocarditis can occur. While ICH occurs in about 5% of patients during the acute course of infective endocarditis, proven ruptured mycotic aneurysm is reported in only about 1.7% (range 0.8-2.8%).\(^7\) We believe that brain emboli complicating infective endocarditis result in ICH by at least 3 different mechanisms: 1) sterile emboli can cause infarcts that undergo secondary haemorrhagic transformation that is usually mild and asymptomatic in the absence of anti-coagulation therapy; 2) septic emboli during uncontrolled infection, particularly with virulent organisms, can cause acute, erosive arteritis with rupture; and 3) septic emboli during effective antimicrobial therapy and/or associated with nonvirulent organisms can injure the arterial wall, leading to subacute development of aneurysms that are often aseptic at the time of rupture. S. aureus is the most common organism underlying symptomatic ICH; these haemorrhages usually occur early, during uncontrolled infection.\(^1,2\)

**References**

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