Intracranial mycotic aneurysms
A review of 9 cases

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Summary
Bacterial intracranial aneurysms are an important cause of mortality and morbidity in patients with infective endocarditis. Early recognition and appropriate treatment of these aneurysms can yield good results in an otherwise devastating and often fatal neurological illness.

William Osler first described the vegetations of infective endocarditis as 'mycotic' in 1885, and the term has since been used to describe infective vascular lesions. Intracranial aneurysms caused by micro-organisms usually present with distinctive features allowing differentiation from berry aneurysms. They may present with transient (and often bizarre) neurological signs and symptoms, or with meningitis or subarachnoid haemorrhage, usually about 18 days after the diagnosis of infective endocarditis has been made. However, in many patients the neurological features may precede the features of infective endocarditis. They often present as a peripherally situated spontaneous intracerebral haemorrhage in a young patient.

We report a series of 9 patients with bacterial intracranial aneurysms who presented to two neurosurgical units over an 18-month period with many of the features described.

Selected case reports

Case 1
A 52-year-old White housewife presented with anaemia, dyspnoea on effort, pyrexia and a macular rash. She was found to be in cardiac failure from gross aortic valvular incompetence complicated by bacterial endocarditis. Streptococcus faecalis was cultured from 6 consecutive blood cultures. Despite anti-failure therapy and antibiotics, the patient's haemodynamic function deteriorated and an aortic valve replacement was deemed necessary 16 days after commencement of therapy. Her immediate postoperative course was uneventful. On the 6th day after the operation she suddenly became deeply comatose and unresponsive to painful stimuli, and within an hour had developed bilaterally fixed dilated pupils. A computed tomographic (CT) scan revealed a left-sided occipital lobe intracerebral haematoma, complicated by massive intraventricular haemorrhage (Fig. 1). She died 4 hours after onset of the haemorrhage. At autopsy a 0.5 cm diameter aneurysm of the left posterior cerebral artery, which had ruptured into the occipital horn of the lateral ventricle, was found. Histological examination demonstrated features of a bacterial aneurysm.

Case 2
A 12-year-old Black boy who had undergone total correction of a Fallot's tetralogy and replacement of the aortic valve at the age of 8 years was brought to hospital because of progressive confusion, irritability and restlessness which had commenced during the previous night. He had complained of a severe headache and had had episodes of uncontrolled screaming. He was found to have no localizing neurological signs but was pyrexial and had marked neck stiffness. A systolic murmur was present over his aortic valve but there were no signs of cardiac failure. Lumbar puncture revealed turbid fluid, from which Staphylococcus aureus was subsequently cultured. He was treated for bacterial meningitis with intravenous antibiotics, but after 12 hours his condition deteriorated and he became deeply unconscious.

A CT scan was performed demonstrating a small left occipital haematoma (Fig. 2), which was not thought to be the sole cause...
TABLE I. SUMMARY OF CLINICAL FEATURES IN 9 CASES OF MYCOTIC INTRACRANIAL ANEURYSM

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yrs)</th>
<th>Source of endocarditis</th>
<th>Organism</th>
<th>Presenting symptoms</th>
<th>Site of aneurysm</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52</td>
<td>Aortic valve</td>
<td><em>Streptococcus faecalis</em></td>
<td>Sudden coma</td>
<td>Left posterior cerebral artery</td>
<td>Died, no surgery</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>Aortic valve</td>
<td><em>Staphylococcus aureus</em></td>
<td>Meningitis, subarachnoid bleeding</td>
<td>Left posterior cerebral artery</td>
<td>Died, no surgery</td>
</tr>
<tr>
<td>3</td>
<td>21</td>
<td>Aortic valve</td>
<td>No growth</td>
<td>Left hemiparesis, subarachnoid bleeding</td>
<td>Right middle cerebral artery</td>
<td>Surgical excision, good recovery</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>Mitral, aortic endocarditis</td>
<td><em>Staph. aureus</em></td>
<td>Right hemiparesis</td>
<td>Left middle cerebral artery</td>
<td>Surgical excision, good recovery</td>
</tr>
<tr>
<td>5</td>
<td>54</td>
<td>Black</td>
<td><em>Candida albicans</em></td>
<td>Left hemiparesis, meningitis</td>
<td>Right anterior cerebral artery</td>
<td>Died, no surgery</td>
</tr>
<tr>
<td>6</td>
<td>14</td>
<td>Mitral endocarditis</td>
<td><em>Escherichia coli</em></td>
<td>Left hemiplegia, headache</td>
<td>Right middle cerebral artery</td>
<td>Died in severe congestive cardiac failure after surgical excision</td>
</tr>
<tr>
<td>7</td>
<td>50</td>
<td>Mitral endocarditis, occult lung carcinoma</td>
<td>No growth</td>
<td>Headache, amnesia, multiple small cerebral infarcts</td>
<td>Left middle cerebral artery</td>
<td>Died, no surgery</td>
</tr>
<tr>
<td>8</td>
<td>12</td>
<td>Mitral endocarditis</td>
<td><em>Strept. viridans</em></td>
<td>Headache, left hemiparesis, subarachnoid bleeding</td>
<td>Right posterior cerebral artery</td>
<td>Surgical excision, good recovery</td>
</tr>
<tr>
<td>9</td>
<td>16</td>
<td>Aortic endocarditis</td>
<td><em>Staph. aureus</em></td>
<td>Focal fit, right hemiparesis</td>
<td>Left middle cerebral artery</td>
<td>Surgical excision, right hemiparesis</td>
</tr>
</tbody>
</table>

of his deterioration. Lumbar puncture was then repeated, revealing uniform heavily blood-stained fluid. Unfortunately the child became progressively unconscious and died shortly thereafter. At autopsy diffuse subarachnoid haemorrhage, an occipital haematoma and a ruptured fusiform aneurysm lying adherent to the dura were found. The aneurysm appeared to arise from a cortical branch of the left posterior cerebral artery. Histological examination revealed acute inflammatory changes and suppurative necrosis of the aneurysm wall.

Case 3
A 21-year-old Black man required an emergency aortic valve replacement because of deteriorating haemodynamic status due to infective endocarditis. Postoperatively he received high-dose antibiotic therapy for 1 month and was well on discharge. Six days after discharge he developed severe headache, vomiting, dysarthria and left-sided weakness. On readmission he was fully conscious, had marked neck stiffness and a dense left hemiparesis. He was afebrile and no signs of infective endocarditis were present. He was not in cardiac failure and his aortic xenograft valve was functioning normally. Lumbar puncture revealed blood-stained CSF and subsequent angiography demonstrated a large peripheral right-sided middle cerebral aneurysm (Fig. 3).

At craniotomy the aneurysm was found to be adherent to the dura; it was excised and its feeding vessel clipped. Histological examination confirmed a bacterial aneurysm, but no organisms were grown on blood culture. His recovery was uneventful and movement of the left hand returned over a 4-month period.

Case 6
An extremely ill 14-year-old Black child was brought to hospital unconscious and in severe cardiac failure on the 7th day of a pyrexial illness, during which the child had complained of breathlessness and headaches. On examination mixed mitral valve murmurs were present and bilateral papilloedema, neck stiffness and a left hemiplegia were demonstrated.

Carotid angiography demonstrated a large right parietal space-occupying lesion and an aneurysm of the middle cerebral artery complex (Fig. 4). An emergency craniotomy revealed a large intracerebral haematoma with intraventricular extension and an aneurysm arising from one of the middle cerebral vessels, which was actively bleeding. This was excised and histological examination confirmed its bacterial nature. *Escherichia coli* was cultured. Postoperatively, the child failed to recover consciousness and died on the 3rd day in severe cardiac failure.
Fig. 4. Right carotid angiogram demonstrating a mycotic aneurysm and parietal space-occupying lesion (haematoma).

Case 8

A 12-year-old Asian schoolboy with known rheumatic mitral valvular disease was taken to hospital from school where he had become irrational, irritable and had vomited twice. He complained of severe headache and a left-sided weakness. He was found to be afebrile, with severe neck stiffness, drowsiness and a left hemiparesis. No signs of cardiac failure were present, but the murmurs of mixed mitral valve disease were heard on auscultation. Lumbar puncture revealed blood-stained fluid and a subsequent CT scan demonstrated an intracerebral haematoma which on later angiography was shown to arise from the rupture of a large peripheral aneurysm of the right posterior cerebral artery (Fig. 5). At craniotomy an intracerebral haematoma was drained, the aneurysm excised and its feeding vessel clipped. Histological examination confirmed the mycotic nature of the aneurysm and Strept. viridans was cultured from 2 of 3 blood samples. The patient made a rapid recovery.

Fig. 5. Right vertebral angiogram demonstrating a peripheral mycotic aneurysm of the posterior cerebral artery.

Discussion

Arterial wall weakening and dilatation due to invasion by microorganisms may arise in 3 ways:2,6,6 (i) from embolization of septic thrombi arising from a cardiac source (80% of bacterial aneurysms); (ii) from adjacent sepsis in contact with the vessel wall, as in middle-ear disease or cavernous sinus infection and following meningitis; and (iii) from infection of atheromatous plaques in association with or following a transient bacteraemia (this usually involves the vessels of the circle of Willis in elderly individuals).

Although mycotic aneurysms have become less common as the incidence of rheumatic heart disease has declined in industrialized countries, they are still a significant and preventable cause of death in young people.7 It has been estimated that up to 15% of patients with infective endocarditis may develop mycotic aneurysms.4

In developing countries where the incidence of rheumatic heart disease and bacterial endocarditis remains high, the sequelae of septic cerebral embolization are an important cause of morbidity and mortality. Since the advent of valve replacement surgery for active endocarditis, the commonest cause of death in this condition has shifted from haemodynamic decompensation to neurological complications, which occur in 30-50% of cases.5,10,11 As the number of patients with prosthetic heart valves increases, the incidence of mycotic aneurysms may be expected to rise. In contrast to the cases reported by Roach and Drake,2 in which endocarditis was largely occult, 7 of our 9 patients presented with an obvious cardiac source of emboli, facilitating diagnosis. Infective endocarditis frequently runs a chronic smouldering course and may elude diagnosis until the patient presents with a catastrophic neurological event, which may be bland infarction, meningitis, cerebral abscess or rupture of a bacterial aneurysm.2,6 Under these circumstances the signs of infective endocarditis, such as splenomegaly, pyrexia, anaemia, Roth's spots, petechiae and Osler's nodes may be valuable aids in diagnosis, and serial blood cultures are mandatory.

The most striking radiological diagnostic feature of mycotic aneurysms is their peripheral situation in the cerebral vascular tree (Fig. 6).

SITES OF MYCOTIC ANEURYSMS OF INTRACRANIAL VESSELS

Whenever such peripheral aneurysms of non-traumatic origin are demonstrated, associated signs of infective endocarditis should be actively sought. The more peripheral situation of these lesions usually results in less destructive effects when rupture occurs than with berry aneurysms. Two of our cases proved to be exceptions (cases 1 and 6). In view of its peripheral situation, the aneurysm can usually be excised and the parent vessel safely clipped without severe infarction. Histological examination and culture of the aneurysm should always be performed.

The management of a ruptured aneurysm in a patient with severe cardiac failure may pose considerable problems, as demonstrated by case 6 of our series. A trephine procedure under local anaesthesia has been suggested, by means of which the haematoma and aneurysm may be dealt with.3

When mycotic aneurysms involve larger vessels of the circle of Willis, they are frequently fusiform and can present a major problem in management. The vessels are extremely friable and the aneurysms are often multiple.4 In these circumstances, Drake2 has recommended an interval of conservative treatment with high-dose antibiotics, in an attempt to allow the vessel wall to consolidate by fibrosis. A wrapping procedure is usually all...
that is possible, although it has been suggested that proximal excision followed by anastomosis of the superficial temporal artery to the distal middle cerebral artery may be a feasible solution to this problem. 4,5 With the wider application of CT scanning in the determination of the site and nature of spontaneous intracerebral bleeding, atypically situated intracerebral haematomas are being demonstrated more often. Angiography should always be considered in these patients, in order to exclude mycotic aneurysms and small arteriovenous malformations. The development of a bacterial aneurysm after septic embolism into the cerebral vascular tree may be delayed by the administration of antibiotics, 6,7 and may be preceded by an area of focal cortical inflammation, which may reveal itself as headache, transient weakness, or meningism. Several authors have therefore recommended that patients with infective endocarditis should undergo cerebral angiography even if only 'trivial' neurological signs or symptoms are present, to allow detection of unruptured aneurysms. 8-11 Angiography should be repeated about 10 days after management of bacterial aneurysms to ensure that subsequent aneurysms do not develop undetected.

Surgical excision of an unruptured cortial bacterial aneurysm should be a simple procedure with minimal morbidity and mortality, 4 unlike the clipping of a berry aneurysm of the circle of Willis. Only if an aggressive policy is followed in the management of infective endocarditis will the mortality associated with this condition be reduced still further.

REFERENCES


Pseudohyperkalaemia — a cause of diagnostic confusion

N. E. PARKER, P. JACOBS

Summary

Marked hyperkalaemia occurred on more than one occasion in 3 patients with thrombocytosis caused by the myeloproliferative syndrome. Initial failure to recognize that the elevation in the serum potassium level was an artefact resulted in unnecessary admissions to hospital and inappropriate investigations and treatment. Awareness of this entity and the use of suitable confirmatory tests should avoid such problems in management.


It is generally recognized that potassium is present in very high levels within cells, notably the erythrocytes. Furthermore, haemolysis with liberation of the intracellular cation readily comes to mind as a cause of hyperkalaemia. Less well appreciated is the fact that platelet destruction may have similar consequences on the serum potassium level. Experience with 3 patients with the myeloproliferative syndrome associated with marked thrombocytosis and spurious elevation of serum potassium levels is a reminder of this important clinical correlation, and a typical illustrative case report is presented.

Case report

A 65-year-old man had been attending a gastro-intestinal clinic for 10 years with a history of diarrhoea. Extensive investigations during this period had revealed no abnormality and the symptoms were attributed to an irritable bowel syndrome, for which he received antispasmodic therapy with good effect. In 1977 he was seen at another centre where a diagnosis of erythraemia was made. Subsequent to a myocardial infarction he was seen in the Department of Haematology at Groote Schuur Hospital, Cape Town, in 1979.