Cerebral Mycotic Aneurysm

A CASE REPORT

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SUMMARY

A case of *Streptococcus viridans* endocarditis with a subarachnoid haemorrhage, caused by rupture of a mycotic aneurysm of the right middle cerebral artery, is described. Treatment of the aneurysm was conservative. Postmortem findings are presented and discussed.

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CASE PRESENTATION

The patient, a 12-year-old Black girl, was admitted on 22 March 1973 complaining of progressive shortness of breath for one month. Her past history was unremarkable and there was no previous history of rheumatic fever.

On examination the patient was fully conscious, co-operative and intelligent. She was dyspnoeic at rest; her temperature was 39,0°C. There was clubbing of the fingers and slight ankle oedema. The pulse rate was 140 per minute, with regular rhythm and good volume. All the peripheral pulses were palpable. The blood pressure was 130/75 mmHg. The jugular venous pressure was 6 cm. The apex beat was heaving in the 5th intercostal space 3 cm outside the midclavicular line and there was a systolic thrill. A grade 4/6 pansystolic murmur was heard at the apex, radiating to the left axilla. There were no other murmurs. Coarse crepitations and rhonchi were heard over the right base of the chest. The spleen was enlarged to 4 cm below the left costal margin. The fundi and central nervous system were normal.

The clinical impression was that the patient had infective endocarditis on a rheumatic mitral valve.

Investigations

The patient's haemoglobin concentration was 10 g/100 ml, the ESR 58 mm/first hour (Westergren) and the white cell count 7 000/mm³. Urinalysis showed no trace of albumin, while serum electrolytes and blood urea were normal. The rheumatoid factor was positive. Three blood cultures grew *Streptococcus viridans* sensitive to penicillin. X-ray examination of the chest revealed right lower lobe pneumonia. An ECG showed sinus rhythm, rate 65/min, a normal QRS axis, and inverted T waves V2 to V4.

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Course in Hospital

The patient was digitalised, blood was taken for culture and she was given 10 million units of soluble penicillin intravenously daily and probenecid 0,5 g three times a day. Two days later the patient was afebrile and clinically much improved. Her improvement continued until, 2 weeks later, she developed a severe occipital headache and became stuporous. On examination she had marked neck stiffness and a left hemiplegia. The fundi were normal.

A lumbar puncture showed uniformly blood-stained fluid; the supernatant was xanthochromic. A right carotid angiogram showed a large aneurysm situated on the right middle cerebral artery at its bifurcation (Fig. 1) and a

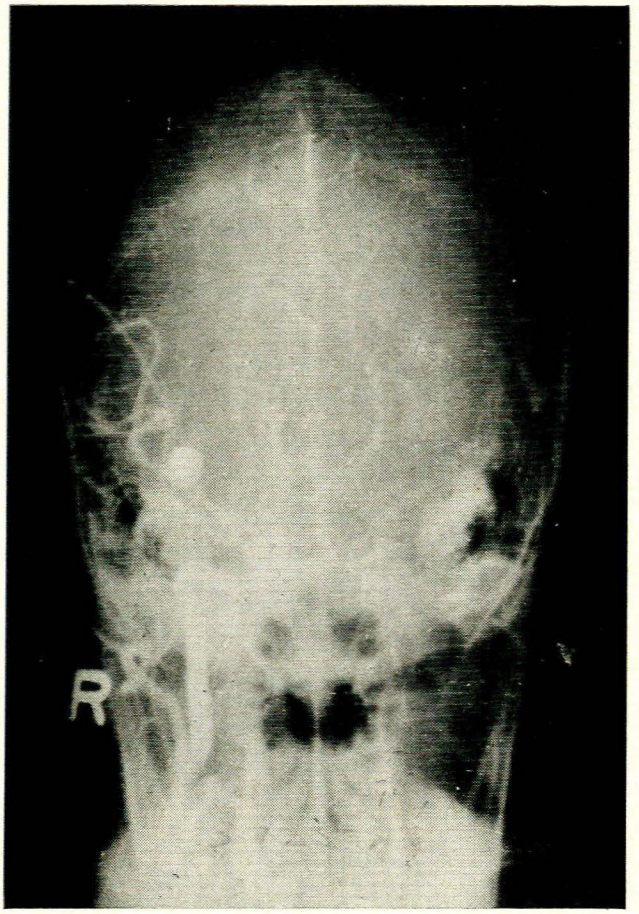

Fig. 1. A large aneurysm is situated on the right middle cerebral artery at its bifurcation.

space-occupying lesion in the right temporoparietal region. The left carotid angiogram was normal.

Neurosurgical intervention was not undertaken, since it was felt that the patient was unfit for operation. Intravenous penicillin was continued; the patient's mental state improved and she became lucid and regained some power of the left limbs. Ten days later she died suddenly.

Postmortem Examination Findings

The body was that of a slightly wasted 12-year-old Black female, weighing 32 kg. There were small bilateral serous pleural effusions and a small serous pericardial effusion, but no ascites or cardiac oedema. The tonsils were prominent and contained purulent material in the crypts. The lungs were oedematous. The liver showed slight autolysis but no sign of chronic venous congestion. The left kidney was normal; the right kidney showed polycystic disease of the lower pole. The spleen was enlarged, weighing 210 g (expected weight 100 g). There was a small healing infarct in one area. The heart was enlarged, weighing 275 g (expected weight 125 g). The right ventricle and atrium appeared normal. The left atrium was dilated. The left ventricle showed dilation and hypertrophy. The aortic, tricuspid and pulmonary valves were normal. The mitral valve was dilated and admitted 3 fingers. The cusps were thickened and the atrial surfaces roughened; a small vegetation was attached to the anterior leaflet. Sections confirmed myocardial hypertrophy of the left ventricle, but no evidence of rheumatic carditis. Sections of the mitral valve and vegetation showed nodular fibrosis of the cusp, but no active rheumatic lesions. The vegetation was composed of recent thrombus with calcified material and connective tissue at the centre. Gram staining of the vegetation did not show bacterial colonies.

The brain was normal in weight (1 300 g). The right hemisphere was swollen. A large, partly clotted haemorrhage which was present in the right temporoparietal area had caused cerebral destruction in the region of the right lateral sulcus. The right middle cerebral artery was embedded in the thrombus and could not be dissected out. Sections of the brain were taken in the region of the haemorrhage.

The right middle artery (Fig. 2) showed the following features: (*i*) the lumen was occluded by calcified vegetation and thrombus; (*ii*) there was purulent inflammation of the wall between the intima and the media, the accumulation of pus giving the appearance of a dissecting aneurysm (at deeper levels the media was necrotic at one point but neither the site of vessel rupture nor the aneurysmal sac was located); (*iii*) there was periarterial suppuration and haemorrhage with fragments of vegetation in the pus around the vessel, some of them containing colonies of Gram-positive cocci.

DISCUSSION

Neurological complications are found in one-third of patients with infective endocarditis.[1] However, cerebral mycotic aneurysm is one of the less common manifestations. Its incidence in infective endocarditis varies from series to series. Of Harrison and Hampton's[3] 116 patients with infective endocarditis only 2 developed mycotic aneurysms, whereas 16 of 442 patients reviewed by Cates and

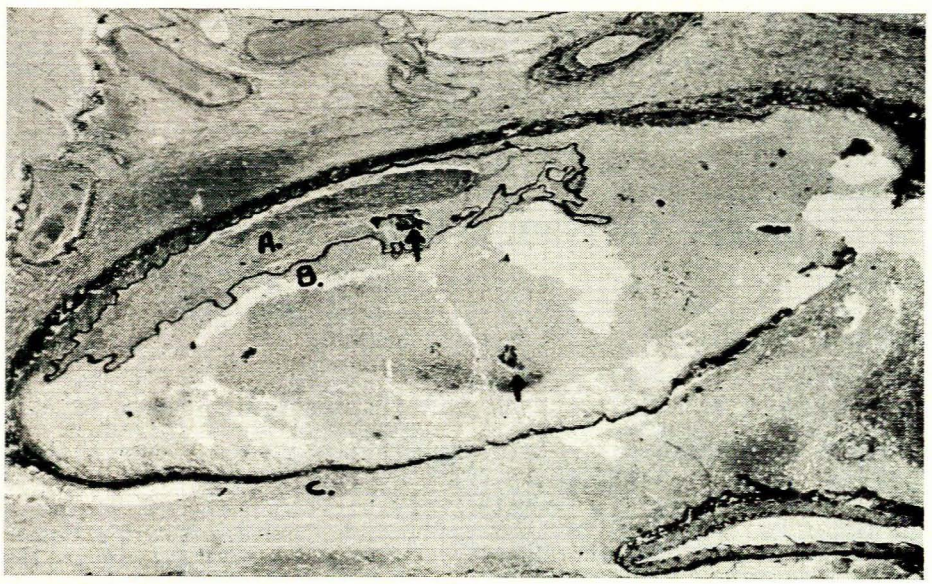

Fig. 2. Section of right middle cerebral artery (elastic van Gieson stain × 25): A—lumen of vessel; B—internal elastic layer; C—external elastic layer. Arrows point to fragments of vegetations in the vessel lumen and in the vessel wall. The media is expanded between the 2 elastic layers by purulent haemorrhagic material, and there is also extensive perivascular suppuration and haemorrhage. Vegetations contained Gram-positive cocci.

Christie[2] died of cerebral haemorrhage from ruptured mycotic aneurysm. The true incidence of cerebral mycotic aneurysms may have been underestimated, for angiography is seldom done in the absence of a subarachnoid haemorrhage.[3] Of 191 patients with proved intracranial aneurysms, 5 had mycotic aneurysms due to bacterial endocarditis[4] but in none of them was the diagnosis of endocarditis made before rupture of the aneurysm.

Mycotic infective aneurysms develop when infective emboli reach the vessel wall as a result either of lodgement of small emboli in the vasa vasorum or of impaction of larger emboli in the arterial lumen.[5] In this case, although either process may have caused purulent arteritis of the middle cerebral artery, it seems that the second alternative was demonstrated in the lumen of the middle cerebral artery.

The treatment of mycotic aneurysm according to Roach and Drake[4] depends on the size, the site and whether a haematoma is present. If the aneurysm is situated on a peripheral vessel, or if there is an associated haematoma, immediate surgery is recommended.

When the aneurysm is large and situated on a major vessel, as in our case, a delay in operation is advisable since surgery on an inflamed, friable vessel is dangerous. In this case, the two neurological episodes suggested that the aneurysm leaked about 2 weeks after admission and ruptured about 10 days later, causing death. It is possible that surgical intervention during the period of recovery from the first episode might have saved the patient's life.

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