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# **Endovascular treatment** of bacterial intracranial aneurysms

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# Introduction

Bacterial intracranial aneurysms, frequently caused by blood spread or direct involvement of the vessel wall by organisms in endocarditis, meningitis or infection of the paranasal sinuses, are not real aneurysms, but the sequelae of infection, necrosis and inflammation of the vessel wall [1–6]. Treatment of these aneurysms is very important because of the high mortality of rupture, with subarachnoid haemorrhage and intracerebral haematoma; optimal management of these patients depends on early diagnosis. Sequential cerebral angiography may demonstrate rapid change in the size and shape of these aneurysms. Whether the most effective treatment is appropriate high doses of antibiotic alone or both medical and surgical management is controversial [2, 7, 8]. Surgical management is not always possible in cases of dis-

**Abstract** We report three patients with bacterial intracranial aneurysms treated by the endovascular approach: two presented with sudden severe neurological deficits after a diagnosis of endocarditis; the other had suspected vasculitis. CT showed an intracerebral haematoma in all cases; angiography revealed bacterial aneurysms of distal branches of the middle cerebral artery in two cases and posterior cerebral artery in one. Because of the patients' condition and the location of the aneurysms, endovascular treatment was considered the fastest and safest treatment. Hyperselective catheterisation of the parent branch, close to the aneurysm, was performed with a microcatheter. A small amount of glue was injected to occlude both

the aneurysm and a short segment of the diseased vessel. Follow-up angiography revealed occlusion of the aneurysm in all cases. One patient recovered completely; one recovered over some months, with neurological deficit due to the haematoma. The third patient suddenly worsened and died 9 days after treatment for a contralateral haematoma, due to rupture of a new bacterial aneurysm of the middle cerebral artery. Endovascular occlusion of the aneurysm and parent vessel may be an alternative to surgery in selected, severe cases of deep or distal bacterial intracranial aneurysms.

Key words Bacterial intracranial aneurysm · Endovascular treatment

tal or deep location [2, 9], and carries a risk of intra- and postoperative complications [8]. Few patients undergoing endovascular therapy have been described [9, 10]. The aim of treatment is completely different from that of berry aneurysms with a neck, since bacterial aneurysms are in fact focal dilatations of the vessel itself. We report three cases of bacterial intracerebral aneurysm treated with the endovascular technique.

# Methods

The decision to use an endovascular approach was taken after multidisciplinary consultation. Each patient underwent complete angiographic investigation of the intracranial circulation via the femoral route; a complete medical assessment was performed. Because of the patients' condition and the location and characteristics of the aneurysms, the endovascular approach was judged the Fig.1 a CT: left temporo-parietal intracerebral and intraventricular haemorrhage. b Left common carotid arteriogram: aneurysm of the posterior temporal branch of the left posterior cerebral artery, with a filling defect due to clot within it.

c Superselective injection of glue into the posterior temporal branch, with cast within aneurysm and parent vessel. d Postembolisation angiogram: complete occlusion of the aneurysm; filling of distal branches via corticocortical anastomoses



safest and fastest. A routine coaxial technique via the femoral route was used; a microcatheter was superselectively positioned very close to the aneurysm, within the parent vessel. Isobutyl *N*-cyanoacrylate (NBCA) was injected with the aim of occluding the aneurysm and a short segment of the artery bearing it. Follow-up angiography, CT and MRI were performed at different intervals.

# **Case reports**

#### Case 1 (Fig. 1)

A 25-year-old woman had a sudden headache, with progressive neurological deterioration, 15 min after take-off of a 1.5 h flight. On admission, she was comatose, Glasgow Coma Scale 5. CT showed a left parietotemporal haematoma with intraventricular haemorrhage, and four-vessel angiography demonstrated an aneurysm on the posterior temporal branch of the left posterior cerebral artery with leakage of contrast medium out of the vascular dilatation. Embolisation was then performed using 0.2 ml NBCA, 75 % glue, 25 % Lipiodol. The aneurysm and the artery at the level of leakage were both occluded. Angiography immediately after embolisation showed revascularisation of the branches distal to the occlusion via corticocortical anastomoses. CT and MRI 2 weeks after the procedure showed glue within the vessel and the aneurysm, the original haemorrhage, and no infarct. Arteriography showed exclusion of the aneurysm. The patient was discharged 3 weeks later without sequelae and resumed full activity in the following months. In hospital she had high anticardiolipin antibodies levels in serum, which subsequently returned to normal. She had a tentative final diagnosis of vasculitis.

#### Case 2 (Fig. 2)

A 56-year-old man was diagnosed as having aortic valve endocarditis after 4 months with septic fever; antibiotic therapy was initiated. After 1 month of treatment, he suddenly had right temporal headache, rapidly became confused and developed a left hemiparesis. CT showed a right temporoparietal haematoma, with subarachnoid haemorrhage. Four-vessel angiography revealed a right middle cerebral artery (angular branch) aneurysm. Embolisation was performed using 0.6 ml NBCA, 50 % glue, 50 % Lipiodol; angiography after embolisation showed total occlusion of both aneurysm and parent vessel. One day later the right occipital haematoma was removed surgically. The patient's motor functions and level of consciousness improved after the embolisation and surgery. Seven days after the procedure he suddenly became comatose; CT showed a new left temporal haematoma. Angiography revealed a left middle cerebral artery aneurysm not present 1 week previously, with marked mass effect. Because of the patient's very poor condition no treatment was given and he died 2 days later.

### Case 3 (Fig. 3)

A 39-year-old woman had a history of arthralgia and a suspicion of collagen disease because of high inflammation indexes in the serum for 8 months. An echocardiogram revealed mitral valvulopathy, suggesting endocarditis. Antibiotic therapy was instituted, but



**Fig.2 a** Internal carotid arteriogram: bacterial aneurysm of the angular branch of the right middle cerebral artery. **b** Superselective injection of glue into the angular artery; cast within aneurysm and parent vessel. **c** Postembolisation angiogram: complete occlusion of the aneurysm

after 1 week she had sudden headache, right hemiplegia and coma. CT showed a large haematoma in the right basal ganglia. Fourvessel angiography, carried out immediately, demonstrated an aneurysm on the frontal branch of the right middle cerebral artery. Embolisation was performed using 0.3 ml NBCA, 50 % glue, 50 % Lipiodol. Complete occlusion of both the aneurysm and a small portion of the precentral branch was shown on angiography. The patient deteriorated progressively in the following days because of increase in the mass effect due to the haematoma; the latter was then removed surgically. The patient had an episode of pneumonia and diffuse intravascular coagulation in the following months. She is now slowly recovering.

# Discussion

Bacterial intracranial aneurysms occur most frequently on the middle cerebral artery or its distal branches, less commonly on the posterior and anterior cerebral arteries; large arteries are rarely affected [3, 5]. They constitute 2.5-6.2% of all intracranial aneurysms [11], appearing clinically in 2% of cases of bacterial endocarditis and 5–15 % at autopsy [8, 12, 13]. The pathogenesis of bacterial aneurysms is believed to start when an infected embolus lodges at some point in the vessel wall; the wall is weakened by spread of infection, involving the media and adventitia and pulsation against the necrotic, weakened wall leads to aneurysmal dilatation and rupture [14, 15]. These aneurysms can be divided into three types: embolic origin aneurysms secondary to bacterial endocarditis, affecting mainly the middle cerebral artery; extravascular origin aneurysms secondary to extension of infection from a neighbouring septic focus affecting mainly large arteries such as the intracavernous carotid artery; and primary bacterial aneurysms,

occurring in the absence of an obvious inflammatory focus elsewhere in the body [5, 12].

In an experimental study in dogs, bacterial aneurysms formed in 1–3 days after lodging of an infected embolus in a cerebral vessel without antibiotic treatment; with treatment, aneurysms developed in 7–10 days [15].

Aneurysms may resolve or decrease in size in some patients with medical treatment alone. However, Bohmfalk et al. [12] pointed out the high mortality (44 %) in patients with bacterial intracranial aneurysms treated by antibiotics alone and the lower mortality (23 %) in those who had both antibiotics and elective surgery.

The aim of therapy should be both to treat the underlying disease medically and any threatening intracranial condition surgically. Evacuation of a haematoma with a bleeding or fragile infected vessel may cause rebleeding. Treatment of the aneurysm itself may be difficult both because of its possibly deep position and because these aneurysms do not usually have a neck; they are in fact a focal dilatation of a segment of what should be considered the parent artery. Surgery involves treatment of a diseased fragile, vessel wall that will probably be sacrificed. Intravascular treatment may be proposed as an alternative in particularly difficult cases, because of either the characteristics and position of the aneurysm or the patient's condition.

The aim of intravascular treatment of berry aneurysms is to occlude the aneurysm and preserve the parent artery. Bacterial aneurysms are a totally different disease and occlusion of the parent vessel may be justified, particularly if collateral circulation may be foreseen and provided that the aneurysm is sufficiently distal.

Our experience in these cases indicated that occlusion of the aneurysm and parent vessel may be performed, when needed, without significant ischaemic brain damage; closure of the aneurysm, reducing the risk of rebleeding, may allow more aggressive subsequent medical or surgical treatment. The severity of the underlying disease may however be a dramatic challenge and represent a negative prognostic factor. Fig. 3 a Right internal carotid angiogram, oblique view: bacterial aneurysm of a frontal branch of the middle cerebral artery and stenosis of the parent vessel.

b Superselective injection of glue: cast of the aneurysm and part of the parent vessel.
c Postembolisation angiogram: complete occlusion of the aneurysm.

**d** Postembolisation CT: glue within aneurysm, anterolateral to the haematoma



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