Endovascular Treatment for a Unusually Large Mycotic Aneurysm Manifesting as Intracerebral Hemorrhage

—Case Report—

Yoshie HARA, Kohkichi HOSODA, Taro WADA, Hidehito KIMURA, and Eiji KOHMURA*

Department of Neurosurgery, Hyogo Emergency Medical Center and Kobe Red Cross Hospital, Kobe, Hyogo; *Department of Neurosurgery, Kobe University Graduate School of Medicine, Kobe, Hyogo

Abstract

A 62-year-old male presented with an unusually large mycotic aneurysm mimicking a saccular aneurysm manifesting as coma and hypotension. Computed tomography showed intracerebral and intraventricular hemorrhage. He was in septic shock due to acute infectious endocarditis. Cerebral angiography disclosed a large distal anterior cerebral artery aneurysm. The diagnosis was mycotic aneurysm based on the morphological features and associated endocarditis. The aneurysm and the parent artery were successfully occluded by endovascular embolization. High-dose antibiotic therapy in the following 6 weeks resulted in resolution of the infectious endocarditis. Early exclusion of ruptured mycotic aneurysm is mandatory because of the high risk of rerupture. Endovascular treatment is an effective alternative for mycotic aneurysms, especially if the patient's general condition is poor. Parent artery occlusion can be safely tolerated if the aneurysm is located distally.

Key words: mycotic cerebral aneurysm, endovascular treatment, endocarditis

Introduction

Mycotic cerebral aneurysm associated with infectious endocarditis is very likely to rupture and the prognosis is unfavorable with high mortality. Management of this disease remains controversial due to the technical problems associated with obliteration of the lesion as well as the frequently unreliable response to antibiotic therapy. Most reported mycotic aneurysms were small, reflecting their nature as a false aneurysm susceptible to rupture. We report a case with an unusually large mycotic aneurysm associated with infectious endocarditis which was successfully treated through the endovascular approach.

Case Report

A 62-year-old man presented with sudden loss of consciousness and was transferred to our center. Two months before presentation, he had transient left hemiparesis. One month later his regular laboratory check up found increased white blood cell count. Further examination identified no specific cause for the inflammatory reaction. Cardiac sonography was not performed.

On admission he was comatose with bilateral decerebrate posture and agonal breathing. He was immediately intubated and ventilated. His systolic blood pressure was 180 mmHg on arrival, which dropped to 80 mmHg 30 minutes later. Immediate computed tomography (CT) disclosed a right frontal intracerebral hematoma, intraventricular hemorrhage, and marked ventriculomegaly (Fig. 1). Subarachnoid hemorrhage was not apparent. CT angiography showed poor filling of the cerebral vasculature probably due to the increased intracranial pressure. Emergent ventriculostomy was performed. The initial intraventricular pressure was as high as 600 mmH2O. After the procedure, he remained unconscious and was admitted to the intensive care unit.

The hypotension persisted despite continuous catecholamine infusion. Cardiac sonography was
Endovascular Treatment for a Mycotic Aneurysm

performed to search for the cause of hypotension, and revealed vegetation and partial distraction of the mitral valve, and resultant regurgitation. His serum white blood cell count was 13600/mm³, C-reactive protein level was 5.9 mg/dl, and platelet count was $18.1 \times 10^4$/mm³. The diagnosis was inflammatory endocarditis manifesting as septic shock based on his clinical condition and laboratory findings. Arterial blood culture was positive for *Streptococcus mitis*. We suspected that the intracerebral hematoma might be related to the endocarditis, and decided to repeat CT angiography on the same day.

The second CT angiography disclosed a large cerebral aneurysm at the $A_3$ portion of the left anterior cerebral artery (ACA). The aneurysm seemed to originate from the ACA-orbitofrontal artery bifurcation. Immediate transarterial cerebral digital subtraction angiography by the Seldinger method via the right femoral artery revealed a large aneurysm that originated from the side wall of the orbitofrontal branch of the left ACA (Fig. 2). The maximum diameter of the aneurysm was 11 mm and the neck diameter was 3.5 mm. There was a marked stenosis on the orbitofrontal artery proximal to the aneurysm neck. We decided that this aneurysm was responsible for the intracerebral and intraventricular hemorrhage.

The patient remained in septic shock under continuous catecholamine infusion, and his systolic blood pressure was no more than 90 mmHg at the time of the angiography. Based on his poor general condition, we decided to occlude the aneurysm by endovascular coiling to prevent rerupture. The procedure was carried out under local anesthesia. A 6Fr, 90 cm guiding catheter was introduced into the left internal carotid artery. After systemic heparinization, a microcatheter was introduced over a microguidewire through the guiding catheter into the right ACA, and then into the orbitofrontal branch. Because occlusion of the orbitofrontal artery usually does not lead to a significant neurological deficit, we planned to occlude both the aneurysm and the parent artery. The microcatheter was introduced past the stenotic parent artery into the aneurysm sac. A Guglielmi detachable coil (GDC) was introduced into the aneurysm sac through the microcatheter. A total of 19 GDCs were inserted into the aneurysm sac. The aneurysm and the orbitofrontal artery were completely occluded with preservation of the ACA main trunk (Fig. 3).

After the procedure, continuous cerebrospinal fluid drainage was performed via a ventriculostomy and high-dose (24,000,000 U/day) penicillin infusion instituted for 6 weeks. The patient’s blood pressure gradually reached the normal range without catecholamine infusion in the 2 days following the procedure, and arterial blood culture 4 days later was negative. Follow-up angiography 6 weeks after GDC embolization showed the aneurysm and the left orbitofrontal artery were completely excluded from the circulation (Fig. 3). Cardiac sonography revealed remission of the mitral valve vegetation and regurgitation. Ten weeks after the onset, the patient underwent ventriculoperitoneal shunting for persistent hydrocephalus. CT showed a low density area in the right frontal lobe, but not in the right orbitofrontal artery territory (Fig. 4). The patient regained consciousness but is confined to a wheelchair and totally dependent.
Discussion

The present diagnosis of mycotic aneurysm was based on the presence of infectious endocarditis and the morphologic features of the aneurysm, not on the histological evaluation.1) Previously reported criteria for mycotic aneurysm include arterial stenosis or occlusion close to the aneurysm, the presence of multiple aneurysms, and rapid morphologic changes.20) In our case, the aneurysm neck was on the side wall of a relatively distal branch of ACA with nearby arterial stenosis, in contrast to most berry aneurysms. We considered the diagnosis was clear, especially with the associated vegetation on the mitral valve and bacteremia.

Mycotic cerebral aneurysms are notorious for high rerupture rates, especially if the size is increasing.7,13,16) We consider surgical excision to be the treatment of choice for ruptured mycotic aneurysms to provide both permanent exclusion of the aneurysm from circulation and histological confirmation. Some authors recommend stereotaxic surgery for distally located aneurysms.2,8,19) However, our patient had hypotension refractory to high-dose catecholamine, so that open surgery under general anesthesia carried unpredictable risks. Therefore, we decided to occlude the aneurysm and the parent artery with endovascular coils under local anesthesia. In the previous report, three patients with infectious cerebral aneurysms were treated with endovascular embolization.18) The endovascular approach was proposed as an alternative in particularly difficult cases because of either the characteristics or the position of the aneurysm, or the patient’s condition.

Induction of foreign material into an infected vessel may lead to prolonged infection and abscess formation. Previous findings of endovascular treatment for mycotic aneurysms using coils and liquid materials have not reported deterioration of infection or abscess formation.3,10) Such patients are always receiving antibiotics for infectious endocarditis, so the concurrent use of antibiotics and the biologically inert nature of the platinum coils may prevent this complication.

Parent artery occlusion and/or intra-aneurysmal packing are both endovascular treatments for mycotic aneurysms.6,10,12) We occluded both the aneurysm sac and the parent artery with GDCs. During surgery small vessels around a large cerebral aneurysm, as in our case, are often found to feed the aneurysm wall, especially if the area is infected and fibrosis has developed. Coil occlusion of only the parent artery might lead to refilling of the aneurysm through such small vessels. Tight packing of the aneurysm sac with parent artery preservation was one option. Mycotic aneurysms are basically false aneurysms, so the wall is fragile and tight intrasaccular packing may cause rupture.14) Additional parent artery occlusion enforces aneurysm exclusion from the circulation even if the intrasaccular packing is not very tight. The parent artery was already stenosed near the aneurysm neck in our case.

Therapeutic occlusion of a vessel at the site of a mycotic aneurysm is unlikely to induce ischemic

Fig. 3 Follow-up digital subtraction angiogram 6 weeks after coil embolization of the aneurysm sac and the parent artery showing the aneurysm was completely excluded from the circulation.

Fig. 4 Computed tomography scan 3 months after treatment showing a low density area in the right frontal lobe due to the initial hemorrhage, but no abnormality in the right orbitofrontal artery territory.
stroke. The first event in the creation of mycotic aneurysms is septic embolus migration into the vessel.\(^{11}\) Local spread of the bacterial agent leads to vessel wall necrosis and development of a false aneurysm. At the same time arterial occlusion may induce ischemic stroke, which may cause clinical symptoms or remain silent. Ischemic stroke is avoided if retrograde filling of the occluded artery occurs through leptomeningeal anastomoses. In our case, the left frontal lobe fed by the occluded orbitofrontal artery did not become infarcted.

After shunt surgery, CT showed satisfactory resolution of the hydrocephalus with a low density area in the right frontal lobe resulting from the initial hemorrhage. In spite of the relatively benign CT findings, our patient remained in a vegetative state. Whole brain hypoperfusion at the onset due to increased intracranial pressure concurrent with systemic hypotension may have caused his poor outcome.

The present case of ruptured large mycotic cerebral aneurysm manifesting as intracerebral and intraventricular hemorrhage showed that the endovascular approach can achieve successful exclusion of the aneurysm from the circulation in a patient in poor general condition with acute infectious endocarditis.

References