Dissecting Basilar Artery Aneurysm Growing During Long-term Follow Up

-Case Report-

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Abstract

A 58-year-old male presented with a dissecting aneurysm of the basilar artery manifesting as dysarthria, left hemiparesis, and numbness of the left side. Angiography revealed a double lumen at the midportion of the basilar artery which was consistent with a diagnosis of dissecting basilar artery aneurysm. The patient was treated conservatively, and remained neurologically stable for a 5-year period following initial presentation, but serial magnetic resonance imaging revealed growth of the aneurysm compressing the brain stem. His condition then worsened. Computed tomography revealed obstructive hydrocephalus. Ventriculoperitoneal shunting was performed and the patient's symptoms improved. However, he died of subarachnoid hemorrhage. Autopsy showed the patient had had a type 3 "dolichoectatic dissecting aneurysm." Surgical treatment should be seriously considered for treating the patients with dissecting basilar artery aneurysm causing brain stem ischemia, especially if the aneurysm is growing. High-flow bypass and proximal occlusion may be the choice in patients with poor collateral circulations.

Key words: basilar artery, dissecting aneurysm, dolichoectatic aneurysm

Introduction

Recent developments in neuroimaging tools, such as magnetic resonance (MR) imaging and digital subtraction angiography, now allow the accurate diagnosis of dissecting vertebrobasilar artery aneurysms. Recurrence of bleeding is the most important prognostic factor for patients presenting with subarachnoid hemorrhage (SAH), and surgical treatment in the acute stage is recommended.²⁾ Most reviews focus on cases of dissecting vertebral artery aneurysms, and considerably fewer reports describe dissecting aneurysms of the basilar artery. We treated a patient with a dissecting basilar artery aneurysm which initially presented with brain stem ischemia but subsequently caused fatal SAH.

Case Report

A 58-year-old male with a history of hypertension visited our hospital following sudden onset of dysarthria, left hemiparesis, numbness of the left side. Computed tomography (CT) revealed a high density area in the prepontine cistern (Fig. 1A). Left vertebral angiography revealed a double lumen at the midportion of the basilar artery (Fig. 1B, C). These findings were consistent with a diagnosis of dissecting basilar artery aneurysm. The patient was treated conservatively to control his blood pressure. The left hemiparesis gradually improved over the next 2 weeks and he was discharged to a rehabilitation facility. Two days later, he showed neurological deterioration. He had alalia and quadriplegia, but he was alert and ocular movement was unaffected, namely the "locked-in syndrome." The patient was again treated conservatively and his condition gradually improved. MR imaging in 1995 revealed an infarction on the right side of the pons. The patient

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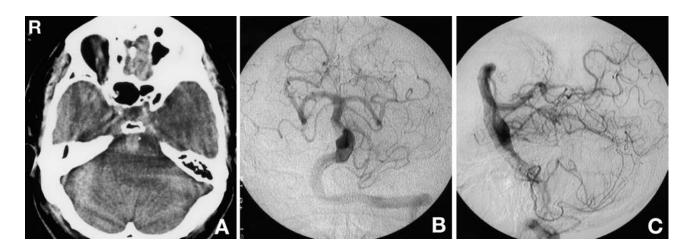


Fig. 1 A: Computed tomography scan showing an area of high density in the prepontine cistern. B, C: Left vertebral angiograms, frontal (B) and lateral views (C), showing a double lumen at the midportion of the basilar artery, consistent with a diagnosis of dissecting basilar artery aneurysm.

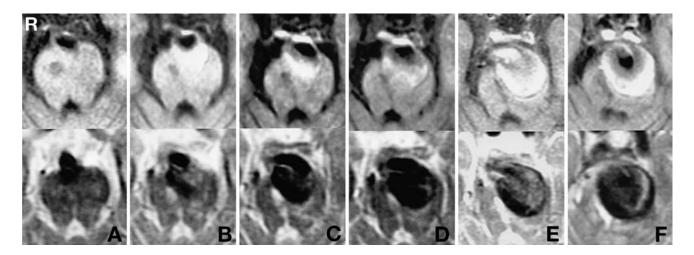


Fig. 2 Magnetic resonance (MR) images in 1995 (A) showing an infarction on the right side of the pons. Serial MR images at yearly intervals (B-F: 1996-2000) illustrate the growth of the dissecting aneurysm which was starting to compress the brain stem.

became ambulatory and was discharged with mild neurological deficit. Follow-up MR imaging revealed that the aneurysm was growing and had started to compress the brain stem (Fig. 2). However, the patient remained neurologically stable. Six years after the initial attack, the patient was readmitted with gait disturbance, and 5 months later his consciousness and dysarthria worsened. CT revealed obstructive hydrocephalus. Ventriculoperitoneal shunting was performed and the patient's condition transiently improved. However, one month later he died of SAH.

Autopsy revealed a giant fusiform aneurysm,

 $4.5 \times 3.5 \times 3.0$ cm, arising from the basilar artery. The aneurysm had caused compression necrosis in the brain stem (Fig. 3A). The ruptured site had a diameter of about 3 mm (Fig. 3B). We could not identify the perforating arteries from the basilar artery. The hemorrhage had extended into and completely obliterated the third and fourth ventricles. The axial section of the aneurysm showed a fresh hematoma in most parts of the small true lumen, which had a diameter of 1.0 cm (Fig. 4A). The aneurysm had a very thin wall with partial fibrillary hypertrophy. The aneurysm lacked intactness of the internal elastic lamina (IEL), with only a few fragmented area of

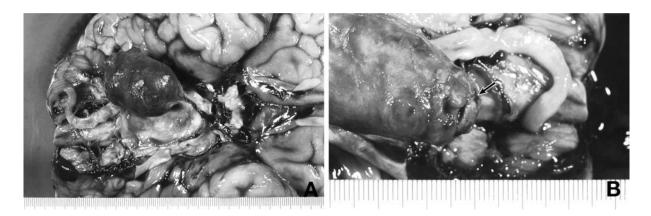


Fig. 3 A: Postmortem photograph showing a giant fusiform aneurysm, $4.5 \times 3.5 \times 3.0$ cm, arising from the basilar artery, and causing compression necrosis at the brain stem. The hemorrhage had extended into and completely obliterated the third and the fourth ventricles. B: Postmortem photograph showing the rupture site (arrow) at the lower end of the aneurysm and subarachnoid hemorrhage.

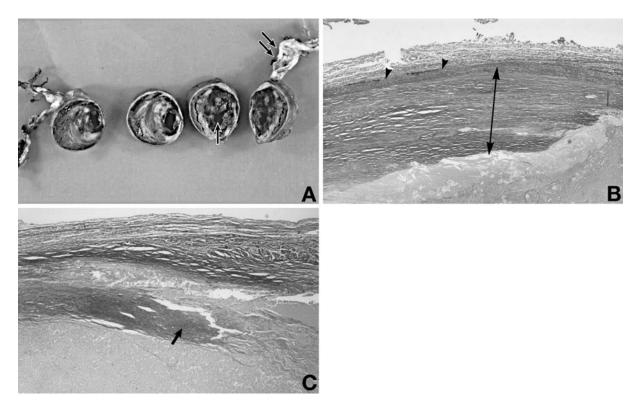


Fig. 4 A: Photograph of the axial section of the aneurysm showing a fresh hematoma in most parts of the small true lumen (arrow). double arrow: Left vertebral artery. B: Photomicrograph showing the aneurysm lacked intactness of the internal elastic lamina (IEL), but had a few fragment area of IEL (arrowheads) and thickened intima (arrow). Elastica-van Gieson (EV) stain, ×4. C: Photomicrograph of the dissection of the thickened intima showing an intimal flap (arrow). EV stain, ×4.

IEL (Fig. 4B). Dissection of the thickened intima had begun in the basilar artery aneurysm and formed an intimal flap (Fig. 4C). No thrombus had formed in and around the dissected intima. Atherosclerosis was observed in the thickened intima of the basilar artery except in the region of the aneurysm.

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Discussion

Sixty-four cases of dissecting basilar arterv aneurysm have been reported.^{1-5,7,8,10,13,14,17,18,21}) The patients were aged from 15 to 77 years (median 42.8 years). Males slightly outnumbered females (29:23), but 12 were of unknown sex. Thirty patients were admitted with SAH, 24 were admitted with brain stem ischemia, eight were asymptomatic, and two had an unknown diagnosis. Most of intracranial dissections result in ischemia as the cause of death,⁶⁾ but we found that SAH slightly outnumbered ischemia as the main cause of death.^{4,17} Recurrence of bleeding in the acute stage accounted for eight cases (26.1%), which is similar to that reported for dissecting vertebral artery aneurysms (28%).^{23,24)} Further hemorrhaging tended to occur by the subacute stage.¹⁶⁾ The risk of second hemorrhage in the chronic stage is very low.¹⁶ The mortality was quite high at 38.3% among these 64 patients, compared to the mortality of 8% among patients with dissecting vertebral artery aneurysm.²⁴⁾ However, the mortality was lower among patients who underwent surgery than among those treated conservatively (24% vs. 44%).

Further bleeding of the aneurysm is the most important prognostic factor for patients with ruptured dissecting aneurysms of the vertebral artery.¹⁵⁾ Therefore, treatments for preventing recurrence of SAH are vigorously performed in such cases. Reported surgical treatments for dissecting aneurysms of the vertebral artery include proximal occlusion (clipping), trapping, wrapping, clipping, and reconstruction of arteries. However, the optimal treatment for dissecting aneurysm of the basilar artery is not well established. We compared the treatment of patients that presented with SAH with those that presented with ischemia among the 64 previously published cases.

Thirty patients presented with SAH, and 15 of these underwent surgical treatment including proximal occlusion, neck clipping, and wrapping. Seven cases among these 15 cases achieved good clinical outcomes. Very recently, successful endovascular embolization of a dissecting basilar artery aneurysm which had caused repeated SAH was reported.¹² Prophylactic vascular reconstruction of the rostral brain stem before the direct surgical treatment of aneurysm is recommended.⁸ In particular, highflow bypass is suggested to enable collateral flow to reach the brain stem after basilar artery occlusion. Cases of serpentine basilar artery aneurysms have been clipped under deep hypothermia with cardiac arrest.^{9,20}

Some patients treated conservatively following

basilar artery dissection causing brain stem ischemia have achieved good outcomes.^{17,18)} However, most patients treated conservatively had poor outcomes. Twenty-one of the 24 patients who presented with ischemia were treated conservatively. Three patients had a good outcome and 12 patients died. Therefore, aggressive treatment should be considered even in patients with ischemia. Especially, in patients with progressive dissection at radiographic follow up, further treatment seems to be necessary to prevent subsequent bleeding¹³ as in our own case.

In our case, the right vertebral artery was aplastic and there was no flow from the posterior communicating artery. Therefore, simple proximal occlusion of basilar artery was not a choice for treatment.

Recently, high-flow bypass and proximal occlusion of the basilar artery have been reported for large aneurysms in the posterior circulation.^{8,12,19,22} Radial artery bypass grafts were used between the intracranial vertebral artery and posterior cerebral artery to treat large basilar artery aneurysms and achieved good results.⁸ Therefore, high-flow bypass surgery and proximal occlusion might have been the only option to rescue our patient. However, we should keep in mind that there is a risk of occlusion of the perforating arteries and the growing aneurysm.

Fusiform and dissecting aneurysms can be classified into four categories, type 1 to type 4.¹¹ Damage to the IEL is often an associated factor in the pathogenesis of aneurysm formation and there is a strong relationship between the pathological features of the aneurysm and the clinical course.¹¹⁾ In the present case, intimal thickening compensated for the weakness of the arterial wall caused by the damaged IEL. In addition, adaptive and multiple dissections had occurred in the thickened intima, with a thrombus forming in and around the dissected intima. This type of aneurysm is pathologically characterized by fragmentation of the IEL, multiple dissections of the thickened intima, and organized thrombus in the lumen. According to the classification by Mizutani et al.,¹¹⁾ our patient had a type 3 dolichoectatic dissecting aneurysm. This type of aneurysm is exclusively located on the basilar artery and appears angiographically as a tortuous fusiform aneurysm with irregular contrast as seen in our case. Patients with such aneurysms usually present with non-SAH symptoms and aneurysm growth to giant size. The patients may die of SAH, three of eight in Mizutani's series. The present case also suggests that the natural course of angiographical type 3 basilar artery aneurysm is ominous and intensive treatment should be performed.

Surgery should be seriously considered for treating a patient with dissecting basilar artery aneurysm causing brain stem ischemia, especially if the aneurysm is growing. High-flow bypass and proximal occlusion should be the choice in patients with poor collateral circulation.

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