Basilar Artery Occlusion Caused by Thrombosis of Atherosclerotic Fusiform Aneurysm of the Basilar Artery

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Background Basilar artery occlusion caused by thrombosis of atherosclerotic fusiform aneurysm of the basilar artery is rare. We present a case confirmed by 6 years of follow-up and autopsy.

Case Description A 63-year-old man suffered from brain stem infarction. Computed tomography and angiography showed a fusiform aneurysm on the proximal portion of the basilar artery. At the time of the third attack 5 years later, complete thrombosis of the aneurysm was found, and the proximal basilar artery was occluded. On autopsy, an atherosclerotic aneurysm 15 mm in diameter was found.

Conclusions A large atherosclerotic fusiform aneurysm of the basilar artery often causes brain stem infarction and involves a risk of complete thrombosis of the aneurysm and subsequent parent artery occlusion. (Stroke. 1994;25:1068-1070.)

Key Words • basilar artery • brain stem • cerebral aneurysm

The patient was a 63-year-old man with a history of hypertension, hypertensive thalamic hemorrhage, and radical operation for colonic cancer. The present disorder began with vertigo, vomiting, and headache on October 22, 1984, and the patient was hospitalized at the Yonezawa City Hospital on the same day. Computed tomographic scan on admission showed a space-occupying lesion of isodensity at the right lateral side of the pons (Fig 1A).

Cerebral angiography revealed a tortuous and ectatic left vertebral artery (VA) intracranially; the proximal portion of the basilar artery (BA) was also fusiform and dilated. The dilated portion of the BA showed retention of contrast medium in the venous phase, but there was no double lumen sign (Fig 2A and 2B). The right VA was hypoplastic and ended in the posterior inferior cerebellar artery. The bilateral intracranial internal carotid arteries (ICAs) were also dilated. The bilateral posterior communicating arteries were of fetal type, and the bilateral posterior cerebral arteries were visualized on carotid angiography.

On the basis of the above findings, a diagnosis of a fusiform aneurysm of the BA and vertebrobasilar insufficiency was made. Symptoms disappeared after medication, and the patient was discharged on August 10, 1985, able to return to normal life. However, on May 3, 1987, he experienced tetraplegia and dyspnea and was again hospitalized. Computed tomographic scans revealed an area of slightly high density at the BA aneurysm and showed contrast enhancement (Fig 1B and 1C). No low-density areas in the brain stem were found. The second angiographic study showed further dilatation of the BA aneurysm (Fig 2C), and there was again retention of contrast medium in the venous phase. After tracheostomy, medication, and rehabilitation, the symptoms improved, and the patient was discharged on May 29, 1988, able to walk with the aid of a cane.

However, on September 3, 1989, the patient again suffered tetraplegia, dysphagia, dyspnea, and ocular bobbing; became comatose; and was again hospitalized. The BA aneurysm and proximal BA were found to still be of a higher density, and diffuse low-density areas were seen in the cerebellum and brain stem (Fig 1D). Magnetic resonance imaging showed disappearance of the signal void at the BA aneurysm and proximal BA and VA, high signal intensity in the BA aneurysm (Fig 3), and equal signal intensity in the proximal BA and VA in both T1- and T2-weighted images. Left vertebral angiography was performed, but the intracranial part of the left VA and the BA were not depicted (Fig 2D).

The patient died approximately 18 months later after a prolonged period of disturbance of consciousness. On autopsy, a fusiform cerebral aneurysm 15 mm in diameter on the proximal BA was found in the region of the right lateral side of the pons (Fig 4). Pathological examination of the aneurysm showed severe atherosclerotic changes. Old and new thrombi were found within the aneurysm. Examination of serial slices of the aneu-
FIG 1. Computed tomographic (CT) scans on the first, second, and third admissions. A, Plain CT scan on the first admission demonstrates an isodensity area in the right parapontine region (arrow). B, Rain CT scan on the second admission still demonstrates a high-density area in the same region (arrow). C, Contrast-enhanced CT scan demonstrates homogeneous enhancement of the lesion (arrow). D, Plain CT scan on the third admission demonstrates a high-density area in the parapontine region (arrow) and low-density areas in the pons and cerebellum (arrowheads).

FIG 2. Vertebral angiograms on the first, second, and third admissions. A, Left vertebral angiogram (arterial phase) on the first admission shows elongation and fusiform dilatation of the proximal portion of the basilar artery (arrow). B, Left vertebral angiogram (venous phase) on the first admission shows retention of contrast medium in the dilated portion of the basilar artery (arrow). C, Left vertebral angiogram on the second admission shows further dilatation of the basilar artery (arrow). D, Left vertebral angiogram shows nonfilling of the contrast medium in the intracranial vertebral artery and basilar artery (arrow).

FIG 3. Axial T-weighted magnetic resonance image on the third admission demonstrates an area of high signal intensity in the right parapontine region (arrow) and areas of low signal intensity in the pons (arrowheads).

Watanabe et al Basilar Artery Occlusion Caused by Aneurysm

rysm, the BA, and the intracranial VA provided no findings indicative of dissecting aneurysm except marked atherosclerosis of the proximal BA and VA with intraluminal thrombi. The origin of the anterior inferior cerebellar arteries arising from the aneurysm was occluded by thrombi, but the distal BA was patent without intraluminal thrombi. Ischemic necrosis was found in the bilateral pyramids of the medulla oblongata, bilateral pontine tegmentum and basis, and bilateral anteroinferior portion of the cerebellum.
Discussion

More than half of large cerebral aneurysms with a diameter of 20 mm or more are known to have thrombi, and in cases of giant aneurysms in which the thrombosis has progressed, it is often found that bizarre serpentine vascular channels have formed and blood flow is maintained from the parent artery to peripheral arteries. Complete thrombosis of the aneurysm is estimated to occur with a frequency of 1% to 20%, but it is only in extremely rare cases that the parent artery becomes completely occluded. There have been several reports of such cases. These include the complete thrombosis of a giant aneurysm of the cavernous portion of the ICA and occlusion of the proximal portion of the ICA, reported by Whittle et al. and Sato et al.; the complete thrombosis of a giant middle cerebral artery (MCA) aneurysm and occlusion of the proximal MCA, reported by O'Neill et al.; and one case of complete MCA occlusion and one of ICA occlusion due to thrombosis of giant aneurysms of the MCA, reported by Endo et al. Nonetheless, there have been no previous reports of complete thrombosis of an atherosclerotic fusiform aneurysm arising on the basilar artery in which the occlusion of the proximal basilar artery was diagnosed using angiography, computed tomography, and magnetic resonance imaging and confirmed at autopsy.

Occlusion of the basilar artery due to a dissecting aneurysm has been reported, and differential diagnosis with the present disorder is important. In cerebral angiograms, a dissecting aneurysm will sometimes show characteristic features such as a double lumen or pearl and string sign, but such classic signs are in fact somewhat rare, and a differential diagnosis solely on the basis of angiography is difficult. Normally, pathological study of the aneurysm is also required.

In a study of 86 cases of vertebral artery aneurysm reported by Yamaura, approximately 60% were saccular aneurysms and 28% were dissecting aneurysms, whereas a relatively low percentage (13%) were atherosclerotic aneurysms. Thrombus in the fusiform aneurysm in the vertebrobasilar system is found in approximately 60%, and death after brain stem infarction is common as a result of this thrombus. The mortality rate of such cases once the basilar artery has become occluded is approximately 70%. Therefore, therapy should be undertaken immediately if these findings of progressing thrombosis are found: (1) the appearance of ischemic symptoms, (2) an increase in density of the aneurysm and the vertebrobasilar artery on computed tomographic scans, and (3) the loss of the flow void phenomenon and high signal intensity of the aneurysm and vertebrobasilar artery on T1- and T2-weighted magnetic resonance imaging.

References

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