

# SPONTANEOUS OCCLUSION OF A GIANT BASILAR TIP ANEURYSM AND A BASILAR ARTERY DUE TO THE DISSECTION OF BOTH STRUCTURES: CASE REPORT

Keiichiro Maeda, M.D.,\* Masaaki Usui, M.D.,† Kazuo Tsutsumi, M.D.,\* and Akira Iijima, M.D.\*

\*Department of Neurosurgery, Aizu Central Hospital, Fukushima, Japan; and †Department of Neurosurgery, Toranomon Hospital, Tokyo, Japan

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## BACKGROUND

Spontaneous occlusion of a giant aneurysm with its parent artery is relatively rare. Complete occlusion of a giant aneurysm at the basilar bifurcation and a basilar artery due to the dissection of the basilar artery has never been reported.

## CASE DESCRIPTION

This 62-year-old man presented with left hemiparesis and right oculomotor palsy. Radiographic study showed a giant aneurysm at the basilar artery bifurcation with hemorrhage in its wall and an ischemic area in the right midbrain. Subsequent study revealed that thrombosis of the aneurysm rapidly progressed and that the parent basilar artery caused the dissection. Finally the giant aneurysm and the basilar artery were completely thrombosed.

## CONCLUSION

The dissection was considered to occur in the aneurysm wall first by the hemorrhage in it and progress proximally along the basilar artery. Intramural hemorrhage in the wall of a giant aneurysm can be a cause of dissection of its parent artery. This seems to be one of the mechanisms by which a giant aneurysm and its parent artery are spontaneously thrombosed. © 1997 by Elsevier Science Inc.

## KEY WORDS

*Spontaneous occlusion, giant aneurysm, dissection, basilar artery.*

Some giant aneurysms are known to be obliterated spontaneously. But spontaneous occlusion of the lumen of a giant aneurysm together with

the parent artery is very uncommon [8,10,12,14,15,17]. We report an extremely rare case in which a giant basilar tip aneurysm and a basilar artery were completely thrombosed in a short period, probably due to the dissection of both structures. The mechanism of the occlusion and the dissection is discussed.

## CASE REPORT

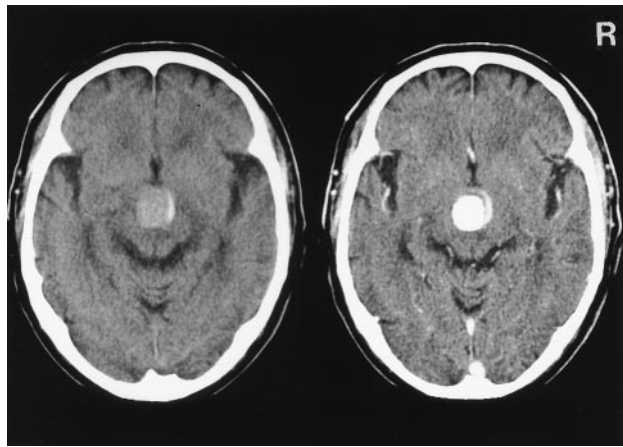
This 62-year-old man was admitted to another hospital complaining of sudden onset gait disturbance. Computed tomography (CT) showed a mass in the third ventricle, and the same day he was transferred to our hospital for further evaluation.

On admission, he was slightly disoriented, and had mild left hemiparesis. Right cerebellar sign and right oculomotor palsy were also noted. CT scan demonstrated a round mass in the third ventricle with a diameter of about 3 cm in lower part of the third ventricle. After the administration of a contrast medium, the mass was homogeneously enhanced except for a hyperdense crescent part on the right side (Figure 1). Magnetic resonance imaging (MRI) showed characteristic findings of a partially thrombosed saccular giant aneurysm. The crescent part, which was hyperdense on CT scan, was almost isointense on T<sub>1</sub> and hyperintense on T<sub>2</sub>-weighted images (Figure 2). This finding was considered to be hemorrhage in the aneurysmal wall. On T<sub>2</sub>-weighted images, an ischemic area in the right midbrain was also noted.

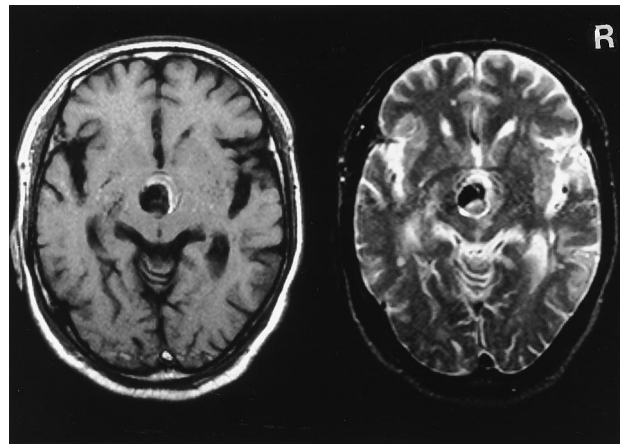
Six days after the admission, the patient suddenly became comatose. CT scan revealed neither hemorrhage nor new infarction, but the density of the aneurysm increased. MRI showed evident decrease

Address reprint requests to: Keiichiro Maeda, M.D., Department of Neurosurgery, University of Tokyo Hospital, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113, Japan.

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**1** (Left) Plain CT scan reveals a round mass in the third ventricle, and a crescent part on the right side is hyperdense. (Right) The mass is almost homogeneously enhanced after the administration of a contrast medium.

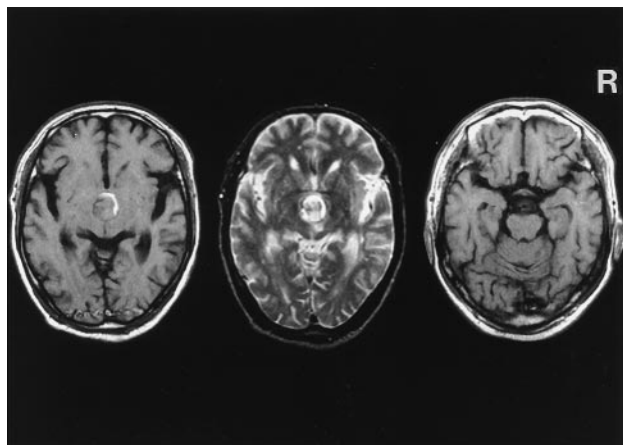


**3** MRI scan on 6 days after the admission. (Left) an axial T<sub>1</sub>-weighted image (TR 500/TE 30); (center) an axial T<sub>2</sub> image (TR 2000/TE 120). Signal flow void in the mass is clearly diminished and the thrombosis is progressing; (right) an axial T<sub>1</sub> image at the level of upper pons. The basilar artery has a double lumen and the right dorsal side of its wall is slightly hyperdense.

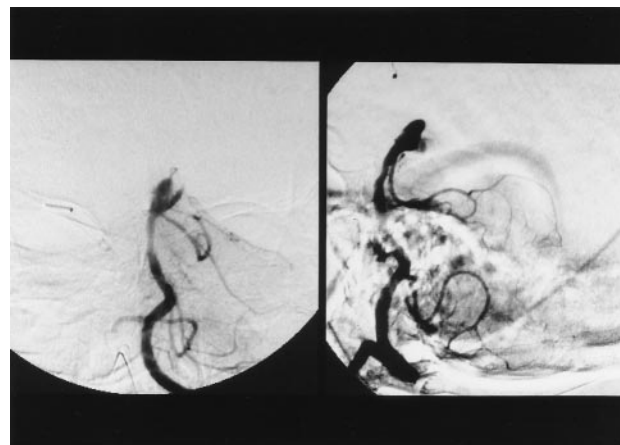
of signal flow void in the aneurysm and rapid progress of thrombosis in the lumen. It also showed a previously undemonstrated double lumen of the basilar artery and the hematoma in the aneurysm wall that was slightly hyperintense on T<sub>1</sub>-weighted MRI. This finding strongly suggested the dissection of the basilar artery (Figure 3). A left vertebral angiogram on the same day showed a partially thrombosed saccular aneurysm on the basilar bifurcation. It also disclosed abnormal narrowing of upper basilar artery, complete occlusion of bilateral P1 segments of the posterior cerebral arteries (PCAs), and near occlusion of the right superior cerebellar ar-

tery (SCA) (Figure 4). These findings indicated an isolated basilar artery dissection.

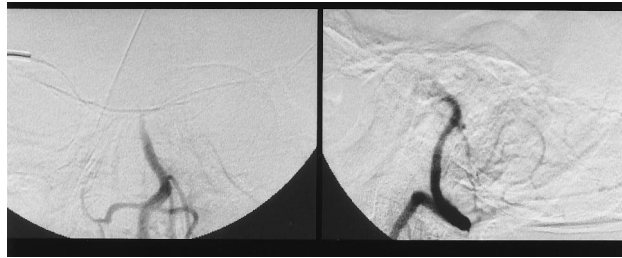
A repeat angiogram done 14 days later disclosed that the basilar artery was completely occluded between the SCA and the anterior inferior cerebellar artery (Figure 5). MRI showed a completely thrombosed aneurysm. A newly developed ischemic area appeared in bilateral upper cerebellum and right occipital lobe, and the territory of brain stem infarction enlarged. The patient gradually recovered, but mild consciousness disturbance,



**2** MRI scan on admission. (Left) an axial T<sub>1</sub>-weighted image (TR 500/TE 30); (right) an axial T<sub>2</sub> image (TR 2000/TE 120). The mass demonstrates a signal flow void and thrombus in it. The crescent part demonstrated on a CT scan is isointense on T<sub>1</sub>-weighted image and hyperintense on T<sub>2</sub>-weighted image.



**4** A left vertebral angiogram showing a partially thrombosed giant aneurysm at the basilar artery bifurcation. Bilateral posterior cerebral arteries are occluded and the origin of the right superior cerebellar artery (SCA) is narrow. Abnormal tapered narrowing of the basilar artery is also demonstrated.



**5** A left vertebral angiogram showing complete occlusion of the basilar artery and the aneurysm.

slight weakness of extremities, and right oculomotor palsy still remained 2 months after the onset.

## DISCUSSION

It is well known that giant aneurysms frequently thrombose completely; the incidence is reported to be from 13%–20% [1,13]. But there are few reports of thrombosis of both the giant aneurysm itself and the parent artery [8,10,12,14,15,17]. As a mechanism of occlusion of an internal carotid artery and its aneurysm, Mikabe et al. and Sato et al. postulated the stretching and compression of a parent artery by its giant aneurysm against the surrounding structure such as an anterior clinoid [10,15]. A parent artery might be simply occluded by the retrograde development of thrombus within a giant aneurysm [15]. However, these mechanisms are unlikely in our case.

The wall of a giant aneurysm is known to be highly vascularized, and hemorrhage in the aneurysmal wall is considered to occur frequently and to play some role in its growth [7,16]. In recent years, MRI has demonstrated this phenomenon [2,16]. Intramural hemorrhage is also known to be a causative factor for intracranial dissecting aneurysm, and this hemorrhage is considered to be caused by the rupture of the vasa vasorum or of the newly formed vessels in the arterial wall [5].

Considering the radiographic findings and the way the symptoms progressed, the following is supposed about the pathogenesis in our case. The hemorrhage in the aneurysmal wall, which corresponded to the crescent part located on the right side of the aneurysmal wall, caused the dissection of the aneurysmal wall first. The dissection progressed down to the parent artery proximally as a case of retrograde dissection of the basilar artery (reported elsewhere), and the perforating artery to the midbrain arising from the right side of the aneurysmal neck was damaged [4,6]. The thrombosis of the aneurysm was rapidly accelerated by the

decrease of blood flow in it. The basilar artery dissection advanced proximally beyond the SCA and caused the occlusion of some arteries, which resulted in brain stem ischemia. Finally, the basilar artery itself was completely thrombosed as well as the aneurysm. It seems unlikely that the thrombus in the aneurysm grew retrograde and occluded the basilar artery, because this theory cannot explain the stenosis of the origin of the right SCA and the abnormal narrowing of the basilar artery. The stretching and compression mechanism described above is also unlikely.

The treatment in this case is controversial. Prompt anticoagulation therapy might prevent occlusion of the basilar artery. However, anticoagulation therapy for intracranial dissecting aneurysms has not been widely advocated because it may aggravate the risk of rupture [3,9]. Moreover, in this case, it also aggravates the risk of the coexisting giant aneurysm rupturing [11]. Surgical intervention such as wrapping is not indicated for an isolated basilar artery dissection causing ischemic stroke. More information about this disorder is needed to decide the best treatment.

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#### COMMENTARY

This is a very unique case that has been nicely documented, both clinically and radiologically, by Dr. Maeda and his colleagues. We have seen this circumstance on a number of occasions when we have deliberately occluded the basilar artery in the treatment of a giant basilar aneurysm with the clip and more often, using the tourniquet. Dissection of the vessel certainly hastens the thrombosis of the sac, but has been a feared complication in our series because it usually means branches and perforators coming off the dissected segment are also occluded, resulting in brain stem ischemia and infarction. If this patient survived with good quality neurologic function, it is yet another example of the remarkable potential for collateral flow to the brain stem and cerebellum.

**S. J. Peerless, M.D., F.R.C.S.C.**

*Director, Mercy Neuroscience Institute  
Miami, Florida*

**I**N THE UNITED STATES THERE'S A PURITAN ETHIC AND A MYTHOLOGY OF SUCCESS. HE WHO IS SUCCESSFUL IS GOOD. IN LATIN COUNTRIES, IN CATHOLIC COUNTRIES, A SUCCESSFUL PERSON IS A SINNER.

—UMBERTO ECO (B. 1932)  
"INTERNATIONAL HERALD TRIBUNE" (PARIS), DEC. 14, 1988