

Two Cases of Spontaneous Internal Carotid Artery Occlusion Due to Giant Intracranial Carotid Artery Aneurysm

Kiyotaka Sato, MD, Satoru Fujiwara, MD,
Takashi Yoshimoto, MD, and Takehide Onuma, MD

Although spontaneous thrombosis of a giant intracranial aneurysm is relatively common, occlusion of its parent artery is rare. We describe two recent patients in whom the parent artery spontaneously occluded. One patient had severe stenosis of the left internal carotid artery, with delayed appearance of a faint shadow of vascular widening near the posterior clinoid process. One month later, complete occlusion of the left internal carotid artery was shown angiographically. The second patient had dysarthria and left hemiparesis, resulting in the diagnosis of a left internal carotid artery giant aneurysm. He had suffered an episode of visual disturbance of the right eye 5 years before. Angiography showed the right cervical internal carotid artery to be occluded. We believe the mechanism of parent artery occlusion in our two patients to be due first to stretching of the internal carotid artery by the enlarged aneurysm, followed by compression of the internal carotid artery by the aneurysm itself. Next, the anterior clinoid process and the optic nerve are involved, and, finally, thrombosis of the aneurysmal cavity extends into the internal carotid artery itself. (*Stroke* 1990;21:1506–1509)

Spontaneous occlusion of the lumen of a giant cerebral aneurysm has been infrequently reported.¹ Whittle et al² have argued that such occlusion occurs in 13–20% of giant aneurysm cases. We describe two patients in whom spontaneous thrombosis of an intracranial giant aneurysm of the internal carotid artery occurred together with thrombosis of the parent artery.

Case Reports

Case 1

The patient was a 49-year-old man complaining of headache and esotropia whose medical history was otherwise negative. He had gone to sleep at about 1 AM on January 9, 1985, but was later awakened by nausea and vomiting. The next morning he had double vision and esotropia of the left eye but did not seek medical help. The diplopia had worsened by the following day, and he noticed a sharp pain in the left forehead region. On January 16, he went to a local ophthalmologist, who diagnosed the esotropia. Following computed tomography (CT) on January 21, he

was admitted to our hospital with a diagnosis of suspected brain tumor.

The patient was conscious on admission. We found left oculomotor and abducens palsy and disturbance of the first branch of the left trigeminal nerve, but the ocular fundus was normal. On the CT scan obtained at the ophthalmologic clinic on January 17, a round enhanced area 3 cm in diameter was evident in the left middle fossa. However, the CT scan obtained on admission to our clinic showed an enhanced area of somewhat higher density surrounding the previously seen area, which was found to be normodense (Figure 1).

During the arterial phase of left carotid angiography, we detected severe stenosis 3 cm from the bifurcation of the cervical internal carotid artery and no further flow of blood intracranially. However, the stenotic C1 and C2 portions were visualized via the ophthalmic artery and the middle meningeal artery (Figure 2). During the delayed phase (approximately 14 seconds after the injection of contrast medium) there was accumulation of the medium in the vicinity of the posterior clinoid process (Figure 3). In contrast, on a right carotid angiogram the left anterior cerebral artery was visualized via the anterior communicating artery, and elevation of the left M1 portion was seen. Using T2-enhanced magnetic resonance imaging with a repeat time of 3 seconds, a round, high-intensity mass in the left middle fossa was

From the Division of Neurosurgery (K.S., S.F., T.Y.), Tohoku University School of Medicine and the Department of Neurosurgery (T.O.), Sendai City Hospital, Sendai, Japan.

Address for reprints: Kiyotaka Sato, MD, Division of Neurosurgery, Tohoku University School of Medicine, 1-1 Seiryō-cho, Sendai 980, Japan.

Received December 18, 1989; accepted May 23, 1990.



FIGURE 1. Cranial computed tomogram of case 1 on admission. Ring-enhanced lesion is depicted in left middle fossa.

revealed (Figure 4). A similar signal was obtained on T1-enhanced images with a repeat time of 1 second. Within the posterior portion of the high-intensity mass, we also detected a small, round, low-intensity region.

The patient's headache persisted for 10 days following admission, but the disturbances of the oculomotor and trigeminal nerves worsened. There were no further incidents of nausea or vomiting, and thereafter the symptoms gradually improved. Left

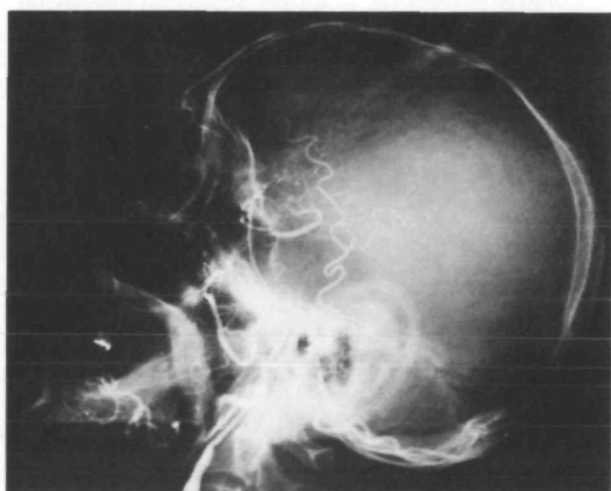


FIGURE 2. Left carotid angiogram (arterial phase) of case 1 on admission. Severe stenosis of cervical portion of left internal carotid artery and retrograde filling of stenotic C1 and C2 portions via ophthalmic artery.

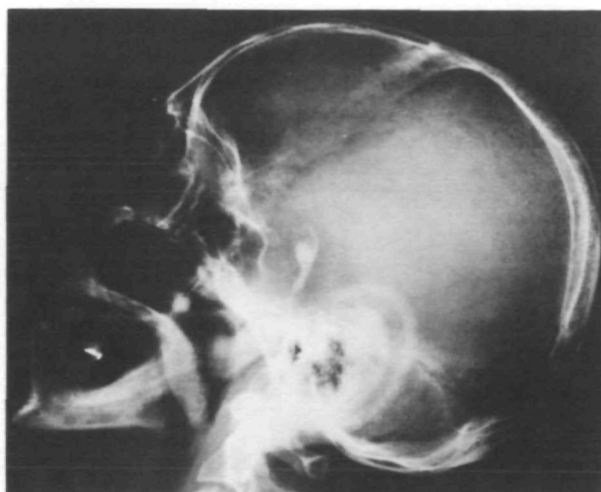


FIGURE 3. Left carotid angiogram (aeriyéa phase) of case 1 on admission. Faint shadow near posterior clinoid process.

carotid angiography at this time showed complete occlusion of the cervical internal carotid artery immediately distal to its bifurcation from the external carotid artery (Figure 5). No accumulation of contrast medium was seen during the delayed phase. The patient was discharged on March 2, and he returned to his previous employment.

Case 2

This patient was a 21-year-old man complaining of left hemiparesis and dysarthria whose history was unremarkable. He noticed deterioration of right visual acuity while playing tennis in August of 1976. In October of that year he went to a local neurosurgical unit, where he was diagnosed as having a giant aneurysm of the right internal carotid artery and was treated conservatively. For 5 years thereafter he had no symptoms other than acuity disturbance of his right eye, but on May 13, 1981, he experienced sudden nausea and vomiting, followed by dysarthria 2

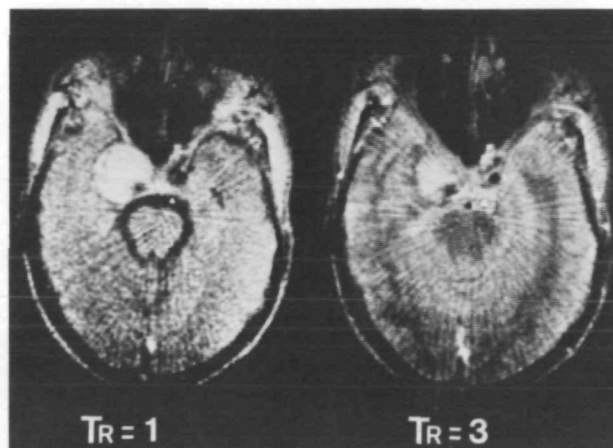


FIGURE 4. T2-enhanced magnetic resonance imaging scans of case 1, with repeat times (TR) of 1 second (left) and 3 seconds (right). High-intensity mass with flow void and posterior low-intensity area are depicted.



FIGURE 5. Left carotid angiogram of case 1 performed 1 month after admission. Complete occlusion of cervical internal carotid artery immediately distal to bifurcation from external carotid artery.

hours later. Following the onset of left hemiparesis, he was admitted to our clinic.

On admission, he was conscious but showed incomplete left hemiparesis and dysarthria. He could distinguish fingers at 30 cm with his right eye, but atrophy of the right optic nerve was evident at the fundus. His left eye was normal.

Cranial CT revealed an oval high-density area in contact with the Turkish saddle and extending to its right. In a rostral slice at the level of the third ventricle, a round high-density area 3 cm in diameter was seen approximately on the midline. The caudally located oval region showed strong enhancement.

Right carotid angiography 5 years earlier had shown an abnormally dilated vessel extending peripherally from the cavernous portion of the internal carotid artery, together with elevation of the M1 region. On a subsequent carotid angiogram, the internal carotid artery was completely occluded at the cavernous sinus. On a left carotid angiogram obtained during this hospitalization, the right anterior cerebral artery was visualized via the anterior communicating artery, and the right A1 portion was found to be markedly elevated. However, the right middle cerebral artery was not seen.

The patient's left-sided hemiparesis improved following admission but reappeared during vascular

compression following angiography. Since no low-density region was seen on CT, the right superficial temporal artery and the middle cerebral artery were anastomosed under the administration of brain-protective substances^{3,4} 40 hours after the onset of symptoms. Three days postoperatively the patient could walk with the aid of a cane, and he was discharged with only mild hemiparesis.

Discussion

Reports in the literature suggest that thrombosis occurs in 13–20% of all giant aneurysm cases,² but we have found only five previous reports of thrombosis of the parent artery. These reports include one case of middle cerebral artery thrombosis,⁵ two cases of posterior cerebral artery thrombosis,^{6,7} and two cases involving internal carotid artery thrombosis.^{2,8}

The high incidence of internal carotid artery thrombosis among the seven cases reported, including ours, is thought to be due not only to the fact that giant aneurysms frequently occur in this vessel,⁹ but also because the anatomic features of the internal carotid artery are believed to facilitate vascular occlusion. Five of the seven cases showed no symptoms of occlusion, suggesting that the parent artery may occlude only gradually.

There are several published reports on the mechanism of internal carotid artery occlusion. Mikabe et al⁸ argue that occlusion is due to pressure exerted on the artery by the aneurysm itself. Sarwar et al,⁵ Kowada et al,⁶ and Pinto et al⁷ maintain that occlusion of the parent artery in their cases was caused by retrograde development of a thrombus originating within the aneurysm. Since the M1 portion was elevated in both of our cases, we believe that the internal carotid artery became occluded due to growth of the aneurysm, stretching the vessel, to compression of the internal carotid artery and the optic nerve against the anterior clinoid process by the aneurysm itself, and finally, to development of a thrombus within the aneurysm.

In case 1, symptoms of occlusion did not occur because of collateral blood flow through the anterior communicating artery from the contralateral internal carotid artery and increased blood flow through the ipsilateral external carotid artery system. In case 2, however, symptoms emerged because the parent artery occluded quickly and there were insufficient collateral pathways.

References

1. Fukawa O, Fukada N, Aihara H, Wakasa H: Spontaneous thrombosis of an azygos anterior cerebral artery aneurysm. *Neurol Surg* 1982;10:105–111
2. Whittle IR, Williams DB, Halmagyi GM, Besser M: Spontaneous thrombosis of giant intracranial aneurysm and ipsilateral internal carotid artery, case report. *J Neurosurg* 1982;56:287–289
3. Suzuki J, Fujimoto S, Mizoi K, Oba M: The protective effect of combined administration of anti-oxidants and perfluorochemicals on cerebral ischemia. *Stroke* 1984;15:672–679

4. Suzuki J, Yoshimoto T, Kodama N, Sakurai Y, Ogawa A: Development of a new therapeutic method for the acute period of brain infarction: Revascularization following the administration of mannitol and perfluorochemicals. *Surg Neurol* 1982; 17:332-352
5. Sarwar M, Bantnitzky S, Schechter MM: Tumorous aneurysms. *Neuroradiology* 1976;12:79-97
6. Kowada M, Hirayama A, Tsukuda F, Gito Y: Calcified giant aneurysm of posterior cerebral artery. *Diagnosis and treatment* 1973;61:666-668
7. Pinto ES, Kvicheff II, Butler AR, Murali R: Correlation of computed tomographic, angiographic and neuropathologic change in giant cerebral aneurysms. *Radiology* 1979;132:85-92
8. Mikabe T, Ogihara R, Tomita S, Kin H, Karasawa H, Watanabe S, Aiba T: Giant intracranial aneurysm visualized by prolonged injection angiography. *Neurol Surg* 1980;8:749-753
9. Fox JL: Giant intracranial aneurysm, in *Intracranial Aneurysm*. New York, Springer-Verlag, 1983, vol 1, p 527

KEY WORDS • carotid artery diseases • cerebral aneurysm