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

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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)
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 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 neurological 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...
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 34 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, but we have found only five previous reports of thrombosis of the parent artery. These reports include one case of middle cerebral artery thrombosis, two cases of posterior cerebral artery thrombosis, and two cases involving internal carotid artery thrombosis.

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, 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 al argue that occlusion is due to pressure exerted on the artery by the aneurysm itself. Sarwar et al, Kowada et al, and Pinto et al 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


KEY WORDS • carotid artery diseases • cerebral aneurysm
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Stroke. 1990;21:1506-1509
doi: 10.1161/01.STR.21.10.1506

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