Late Detection of Supraclinoid Carotid Artery Aneurysm After Traumatic Subarachnoid Hemorrhage and Occlusion of the Ipsilateral Cervical Internal Carotid Artery

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Background and Purpose—We report the first case of traumatic aneurysm of the supraclinoid internal carotid artery (ICA), which we speculate may have developed or grown after traumatic occlusion of the ipsilateral cervical ICA.

Case Description—A 26-year-old man presented with severe traumatic subarachnoid hemorrhage (SAH) and occlusion of the right cervical ICA after a motor vehicle accident. Three-dimensional CT angiography on admission showed no aneurysm. However, cerebral angiography 3 weeks after the injury showed a large aneurysm of the right supraclinoid ICA. The aneurysm was trapped, and pathological examination showed that it was a traumatic aneurysm.

Conclusions—In this case we cannot be sure that the aneurysm was not present on admission. In view of the significant SAH, a lesson of this case may be to suspect such an aneurysm early on and perform early diagnostic cerebral angiography. (Stroke. 2001;32:2203-2205.)

Key Words: carotid artery occlusion • cerebral aneurysm • cerebral infarction • head injury • subarachnoid hemorrhage

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rauma is the most frequent cause of subarachnoid hem-

orrhage (SAH). However, SAH associated with trauma may also rarely be associated with either traumatic or nontraumatic aneurysm1–3: a rupture of a preexisting aneurysm as either a cause or a result of head trauma, or acute or late development of a traumatic aneurysm (TA). We report a case of TA of the supraclinoid internal carotid artery (ICA) detected at 3 weeks after SAH and occlusion of the ipsilateral cervical ICA.

Case Report

A 26-year-old man was admitted to our hospital 40 minutes after being involved in a single motor-vehicle accident, in which he was a drunken driver. He was comatose but had no lateralizing signs. Radiographic studies showed pulmonary contusion and pneumothorax on the right side, a fracture of the mandible, and a normal cervical spine and skull. CT of the brain and the skull base revealed diffuse, thick SAH and only mild right hemispheric swelling but excluded fractures through the carotid canal (Figure 1). Three-dimensional CT angiography (3D-CTA) showed no visualization of the right ICA just proximal to the posterior communicating artery (PCoA), which suggested occlusion of the right cervical ICA (Figure 2). No aneurysm was shown.

Treatment using moderate hypothermia was performed, and rectal temperature was decreased to 34°C. However, anisocoria (right>left) and left hemiparesis developed 12 hours after the injury. An emergency brain CT revealed a pronounced increase in the right hemispheric swelling (Figure 3). A right frontotemporoparietal decompressive craniectomy with a dura patch and right frontal and temporal lobectomies were urgently performed, followed by tracheotomy and stabilization of the mandibular fracture. Postoperatively, the anisocoria disappeared. The disturbance of consciousness gradually improved, and the patient opened his eyes and could sometimes respond to simple orders 2 weeks after the injury.

Three-vessel angiography performed at 3 weeks after the injury showed occlusion of the right cervical ICA, vasospasm of the left ICA and the sphenoid segment of the left middle cerebral artery (MCA), and a large aneurysm of the right supraclinoid ICA, which showed delayed filling and emptying (Figure 4). The right supraclinoid ICA and the right MCA were anterogradely visualized through the right PCoA. A repeat 3D-CTA also showed the aneurysm (Figure 5), but MRI and angiography failed to reveal the aneurysm, probably due to turbulent or irregular flow in the right supraclinoid ICA. One week later, the aneurysm was trapped in combination with a right superficial temporal-to-MCA anastomosis and cranioplasty. The aneurysm with a fragile wall and a broad-based neck, similar to a dilatation of the ICA itself, involved the right ICA between the anterior choroidal artery

Received February 2, 2001; final revision received April 9, 2001; accepted June 8, 2001.

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Stroke is available at http://www.strokeaha.org

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and the carotid summit bifurcation. Therefore, the aneurysm excision required sacrifice of the distal right ICA segment. Pathological examination showed that the wall of the aneurysm consisted of granulation, including traces of disrupted elastic fibers and smooth muscle layer, which suggested a TA (Figure 6). Right ventriculoperitoneal shunting was employed for hydrocephalus at 7 weeks after the injury. Three months after the injury, the patient could give his name and age, but had a moderate left hemiparesis. He was discharged to a rehabilitation center for further therapy.

**Discussion**

TAs of the supraclinoid ICA are very rare lesions.\(^3\) Rupture of these aneurysms usually occurs within 2 months, with peaks on the first day and between the second and third weeks.\(^3\) In this patient who presented with severe traumatic SAH, no aneurysm was noted on the first 3D-CTA study performed on admission, but an aneurysm was noted on conventional angiography and the second 3D-CTA study performed at 3 weeks after the injury. However, considering the potential mechanism of severe SAH, we cannot exclude the possibility that an aneurysmal traumatic disruption of the supraclinoid ICA was present acutely and caused the SAH, because of the

**Figure 1.** CT on admission showing subarachnoid hemorrhage.

**Figure 2.** 3D-CTA on admission showing no visualization of the proximal right ICA (→). A, Anteroposterior view; B, superoinferior view.

**Figure 3.** CT at 12 hours after the injury showing right hemispheric swelling.

**Figure 4.** Angiograms performed at 3 weeks after the injury showing the right carotid occlusion (A), no cross-flow to the right MCA through the anterior communicating artery (B), and an aneurysm of the right supraclinoid ICA (C). A, Right carotid angiogram, lateral view; B, left carotid angiogram, anteroposterior view; and C, left vertebral angiogram, lateral view.
absence of conventional angiography on admission. Therefore, the lesion might represent a growth of TA rather than the genesis of de novo aneurysm in the interim.

Another specific aspect of this case is that the aneurysm was associated with traumatic occlusion of the ipsilateral cervical ICA, which might be caused by direct blunt trauma associated with the mandibular fracture. In such a situation, an aneurysm is unlikely to develop or grow in the ICA distal to the occlusion site, as the hemodynamic factors are important in aneurysmal formation. In fact, to our knowledge, a TA of the supraclinoid ICA associated with occlusion of the ipsilateral cervical ICA has not been previously reported.

Possible mechanisms for the supraclinoid ICA injuries after closed head trauma are as follows: direct injury by a basal skull fracture, overstretching or torsion by movement of the brain following impact, tear by nearby prominent bony structures, and avulsion of a perforator. In this case, it was most likely that overstretching or torsion caused the ICA injury, according to the CT, intraoperative, and pathological findings. The fragile wall of the severely injured ICA could not withstand the hemodynamic stress due to collateral circulation across the PCoA, which we speculate may have caused aneurysmal formation. Vasospasm of the contralateral ICA might increase the blood flow across the PCoA and contribute to the development or growth of the aneurysm.

For early diagnosis of TAs, a high index of suspicion and the importance of repeat angiography have been reported. However, the timing of screening angiography, such as after stabilization or the third week after trauma, remains controversial. Because conventional angiography has some risks, the use of the noninvasive tests such as 3D-CTA and MR angiography may be beneficial for the repeat screening for the TAs. On the other hand, such noninvasive tests may miss the aneurysms, and severe SAH may warrant early diagnostic cerebral angiography.

The high mortality and morbidity rates for TAs merit immediate emergency treatment after diagnosis. Most of the reported TAs of the supraclinoid ICA were treated by trapping or proximal ICA occlusion with or without distal revascularization. Direct clipping of TAs is rarely successful, and endovascular obliteration of TAs presents extreme danger, because most of them are pseudoaneurysms and have broad-based, fragile, and obscure necks. Treatment with stents or direct clipping by use of an encircling clip, eg, Sundt clip-graft, may be reasonable alternatives to the sacrifice of a potentially important conduit.

References
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*Stroke*. 2001;32:2203-2205
*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/32/9/2203

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