Subarachnoid hemorrhage possibly caused by a saccular carotid artery aneurysm within the cavernous sinus

Case report

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Unlike aneurysms at other locations, aneurysms arising from the intracavernous portion of the internal carotid artery (ICA) very rarely cause subarachnoid hemorrhage (SAH). This is partly because they are surrounded by the confining dural walls of the cavernous sinus and the sphenoid bone. Small aneurysms may rupture and usually result in a carotid-cavernous fistula. We report a case of SAH caused by rupture of a wholly intracavernous carotid artery aneurysm, which was successfully clipped by a direct surgical approach. The possible pathogenetic mechanisms of this unusual case are discussed.

**KEY WORDS** - intracavernous lesion • aneurysm • subarachnoid hemorrhage • internal carotid artery

**Case Report**

This 50-year-old previously healthy woman was admitted on October 13, 1987, following the sudden onset of headache and nausea.

**Examination.** Examination revealed mild confusion, slight neck stiffness, and moderate hypertension (blood pressure 160/102 mm Hg). Neither Horner's syndrome nor paralysis of the extraocular muscles was evident. Laboratory results were all within the normal range. Plain skull x-ray films did not demonstrate bone erosion. Computerized tomography (CT) scans showed diffuse SAH in the suprasellar, insular, and ambient cisterns, predominantly on the right side (Fig. 1). On the same day, four-vessel cerebral angiography was performed. The right carotid injection demonstrated a saccular aneurysm arising from the lateral aspect of the C_{4} segment of the ICA and projecting laterally and inferiorly (Fig. 2). No engorged veins or other vascular anomalies were seen. The remainder of the angiogram was normal.

**Operation.** On October 19, the patient was operated on under general anesthesia without hypotension or

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**FIG. 1.** Computerized tomography scans revealing subarachnoid clots in the interhemispheric fissure, suprasellar cistern, interpeduncular cistern (left), and right insular cistern (right). Note the effacement of the anterior horn of the right lateral ventricle.
hypothermia. She was placed in the supine position with a 45° rotation of the head, and a right frontotemporal craniotomy was performed. The cavernous sinus was exposed by opening the sylvian fissure and gently elevating the temporal and frontal lobes. The anterior clinoid process was drilled away and a moderate amount of clot was found under the process, but an aneurysm was not seen. The external wall of the sinus seemed to be intact. After the sinus in Parkinson's triangle was opened, a semicircular-shaped aneurysm was found between the third cranial nerve and the first division of the trigeminal nerve, displacing the fourth cranial nerve laterally (Fig. 3). Apparently, the aneurysm was completely embedded in the sinus and was not in contact with or adherent to the lateral wall of the sinus. Venous bleeding from the sinus was controlled by packing with Surgicel (oxidized cellulose). The wall of the aneurysmal dome was very thin and blood could be seen flowing through the membrane. The dural wall of the sinus medial to the third cranial nerve was incised for further exposure of the C4 segment of the ICA. The neck of the aneurysm was dissected and clipping was carried out with an L-shaped Sugita clip applied through the space between the fourth cranial nerve and the first division of the trigeminal nerve. Thereafter, the ICA was carefully explored near the junction of the posterior communicating artery and no other aneurysms were found. No aneurysm was seen at the anterior communicating artery complex.

**Postoperative Course.** Postoperatively, the patient was well, except for a right ophthalmoplegia which recovered completely after 3 months. Postoperative four-vessel angiography was performed on October 29. The right carotid injection outlined a small residual portion of neck of the aneurysm (Fig. 4). Examination of the other vessels disclosed no additional aneurysms.

**Discussion**

Aneurysms in the cavernous portion of the ICA represent from 3% to 11% of all intracranial arterial aneurysms. Symptoms may be caused either by local mass effect on adjacent structures or by rupture. Small aneurysms at this site usually give rise to a carotid-cavernous fistula instead of producing SAH when they rupture. Due to the support of the overlying dura, many of these aneurysms otherwise remain asymptomatic until reaching a certain critical size. Rupture of such aneurysms which have eroded into the sphenoid air cells may cause severe and often fatal epistaxis.

In our case, neither neuroradiological nor operative findings could demonstrate any vascular abnormality other than an intracavernous saccular aneurysm, and therefore rupture of this aneurysm appeared to be responsible for the SAH. Subarachnoid hemorrhage from intracavernous aneurysms is said to occur in less than 10% of cases. When hemorrhage does occur, intracavernous aneurysms must be distinguished from "paraclinoid" aneurysms. The latter type of aneurysm sometimes arises partly within the subarachnoid space and projects secondarily into the cavernous sinus. To our knowledge, only two substantiated cases of intracranial rupture from a wholly intracavernous carotid artery aneurysm have been reported. Both cases showed a common clinical course; the patients were symptomatic for many years, developed cavernous sinus symptoms over several days, and died. Both aneurysms were large enough to erode the dural wall of the sinus, through which intracranial rupture had taken place. According to Umansky and Nathan, the lateral wall of the sinus in the area of the window between the third cranial nerve and the first division of the trigeminal nerve is incomplete and is formed only by the
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superficial layer, so this part of the wall seems to be physiologically weaker than other parts of the wall. In our case, however, the operative findings showed that the dural wall of the sinus was not disrupted even at this point.

Several cases have been reported in which a carotid-cavernous fistula was complicated by subarachnoid, intraventricular, and/or intraparenchymal hemorrhage. In these cases, the hemorrhage was attributed to combined arterial ischemia and venous hypertension. When an intracavernous aneurysm ruptures, the venous pressure within the cavernous sinus is transiently elevated and high-pressure venous reflux may occur. In our case, however, angiographic studies performed on the day of onset did not show any signs of venous hypertension and this mechanism appears to be ruled out.

The intracavernous portion of the ICA is covered in its trajectory by a sheath of connective tissue. In these cases, the hemorrhage was attributed to combined arterial ischemia and venous hypertension. When an intracavernous aneurysm ruptures, the venous pressure within the cavernous sinus is transiently elevated and high-pressure venous reflux may occur. In our case, however, angiographic studies performed on the day of onset did not show any signs of venous hypertension and this mechanism appears to be ruled out.

The intracavernous portion of the ICA is covered in its trajectory by a sheath of connective tissue. At the entrance of the artery into the subarachnoid space there is a very dense fibrous ring fixing the artery wall at the roof of the cavernous sinus. This fibrous ring at the dural transition is not yet clearly formed in fetal and infant specimens. If this fibrous ring remains loose, an intracavernous aneurysm can easily develop into the intradural space. Even if it remains completely embedded in the cavernous sinus, blood can course along the carotid artery and erupt into the subarachnoid space through the fibrous ring when it ruptures. Unfortunately, we neither observed nor opened the fibrous ring at operation, but the above-described mechanism is the most likely one by which SAH took place in our patient.

According to recent reports, the cavernous sinus is a plexus of veins of various sizes, dividing and anastomosing with each other, that incompletely surrounds the ICA. A direct shunt from the intracavernous ICA system can theoretically connect with two different structures: the veins of the plexus or the perivascular bare spaces between the veins. In addition, we suggest the possibility that the type of anatomical connection discussed here can also occur.

**Acknowledgment**

The authors thank Dr. Hideyuki Ohnishi, Department of Neurosurgery, Osaka Minami National Hospital, for his helpful advice.
References


Manuscript received September 19, 1989.
Accepted in final form February 5, 1990.
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