False Traumatic Aneurysm of the Dorsal Wall of the Supraclinoid Internal Carotid Artery

—Case Report—

Kiyoshi SAITO, Mustafa K. BASKAYA*, Masato SHIBUYA, Yoshio SUZUKI, and Kenichiro SUGITA

Department of Neurosurgery, Nagoya University School of Medicine, Nagoya; *Department of Neurosurgery, University of Kentucky Medical Center, Lexington, Kentucky, U.S.A.

Abstract

A 32-year-old male presented with subarachnoid hemorrhage following head trauma. Initial carotid angiography revealed an aneurysm protruding from the dorsal wall of the supraclinoid internal carotid artery. Two weeks later, following two additional hemorrhagic episodes, repeat carotid angiography showed severe vasospasm and dramatic expansion of the aneurysm. He died 17 days after the accident. Autopsy and histological examination confirmed the diagnosis of a traumatic, false aneurysm of the internal carotid artery. Improved prognosis requires early recognition and surgical obliteration of such aneurysms.

Key words: false aneurysm, head trauma, internal carotid artery, traumatic aneurysm, vasospasm

Introduction

Traumatic intracranial aneurysms are rare complications of head trauma that account for less than 1% of intracranial aneurysms and are usually located on distal cerebral arteries. In one study of 73 traumatic aneurysms, 27% were located on the meningeal, 40% on the middle cerebral, and 18% on the anterior cerebral arteries. Traumatic aneurysms of the internal carotid artery (ICA) are unusual, mostly occurring in the petrous and cavernous segments. Previous cases of traumatic aneurysms of the supraclinoid ICA include only one with an aneurysm originating from the dorsal wall of the ICA. This lesion was attributed to iatrogenic trauma during removal of a tuberculum sellae meningioma. Saccular aneurysms protruding from the dorsal wall of the ICA are also rare, and are associated with fragile necks and a higher incidence of premature bleeding. This type of aneurysm is difficult to treat.

We report a patient with a false, traumatic aneurysm originating from the dorsal wall of the supraclinoid ICA.

Case Report

A 32-year-old male was involved in a motor vehicle accident and was semicomatose when admitted to an affiliated hospital. Computed tomography (CT) demonstrated diffuse subarachnoid hemorrhage (SAH) (Fig. 1). Right carotid angiography revealed an aneurysm protruding from the dorsal wall of the supraclinoid ICA (Fig. 2). His condition gradually improved until Day 6, when he developed left hemiparesis. Repeat CT showed new SAH and a region of hypodensity around the right internal capsule which suggested an ischemic process. He was subsequently transferred to our institution.

On admission, he was drowsy and disoriented. Neurological examination revealed blindness in the right eye and mild left hemiparesis. Since the findings indicated an ischemic process from vasospasm, surgery was postponed. His condition improved until Day 13, when CT revealed an increase in the
ischemic area in his right hemisphere. On Day 15, his consciousness deteriorated to semicomatose. Repeat right carotid angiography showed marked growth of the aneurysm with delayed filling and emptying, in addition to poor filling of the distal cerebral arteries due to severe vasospasm (Fig. 3). CT revealed a third hemorrhagic episode (Fig. 4). He died 17 days following the accident.

Autopsy revealed a traumatic aneurysm originating from the dorsal wall of the ICA and fractures in the anterior cranial base to the planum sphenoidale and right optic canal. Histological examination showed the wall of the aneurysm consisted of fibrous tissue and fibrin-like material, which suggested a false aneurysm (Fig. 5).

Discussion

The 32 reported cases of traumatic aneurysms of the supraclinoid ICA\(^1\) include only one located on the dorsal wall\(^1\) (Table 1). Saccular aneurysms of the dorsal wall of the ICA are also rare.\(^13,22\) The pathogenesis of these saccular...
aneurysms, which are unrelated to the various arterial divisions, is controversial. Some investigators have hypothesized that such aneurysms may be caused by hemodynamic stress, arteriosclerosis, or trauma. Since most dorsal ICA aneurysms have wide fragile necks and a tendency to rupture prematurely, patients with traumatic aneurysms of this location are at high risk for morbidity.

Traumatic aneurysms are classified into three groups according to their pathogenesis: dissecting, true, and false. A dissecting aneurysm is formed by disruption of the intima and internal elastica that allows blood to penetrate between these layers and the muscularis. A true aneurysm develops secondary to an incomplete rupture of an arterial wall which leaves the adventitia intact. False aneurysms are the result of complete interruption of the arterial wall with the formation of a perivascular hematoma which is subsequently organized into a fibrous wall. Hemodynamic excavation and/or repeated hemorrhage will increase the false aneurysm size. A mixed aneurysm occurs when a true aneurysm ruptures and produces a secondary, false aneurysm. Only nine of the previously reported cases of traumatic supraclinoid ICA aneurysms were either operatively or histologically diagnosed, finding eight false aneurysms and one true aneurysm (Table 1).

Eighteen of the previously reported traumatic aneurysms of the supraclinoid ICA (56%) involved closed head trauma, including six patients with basal skull fractures (Table 1). A traumatic aneurysm of the supraclinoid ICA occurring after closed head trauma is probably due to one of three mechanisms: 1) A basal skull fracture may cause direct injury to the ICA, 2) overstretching or torsion of the ICA wall may occur due to movement of the brain following impact, and 3) nearby prominent bony structures such as the anterior or posterior clinoid processes may tear the ICA. In our patient, postmortem examination suggested that the basal skull fracture had injured the ICA. Other causes of injury included penetrating stab wounds in nine patients (28%), missile wounds in one patient (3%), and iatrogenic trauma during intracranial surgery in four patients (13%) (Table 1).

Twelve of the previous patients presented with intracranial hemorrhages, including seven with SAH and four with intracerebral hemorrhage. The second most common presentation involved was progressive neurological deficits in seven patients. The initial presentation of 10 patients is unknown (Table 1). Most traumatic aneurysms rupture after an interval

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Fig. 4 CT scan taken 15 days after the head trauma, showing a third hemorrhagic episode and regions of low density in the right hemisphere.

Fig. 5 Photomicrographs at autopsy, showing a hole in the wall of the ICA (asterisk). The wall of the aneurysm (arrows) consisted of fibrous tissue and fibrin-like material, suggesting that the aneurysm was a false traumatic aneurysm. Elastica Van Gieson stain, upper: ×10, lower: ×40.
Table 1 Summary of previously reported patients with traumatic aneurysms of the supraclinoid ICA

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age/Sex</th>
<th>Type of injury</th>
<th>Basal skull fracture</th>
<th>Presentation</th>
<th>Type of aneurysm</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alexander et al. (1963)</td>
<td>56/M</td>
<td>iatrogenic</td>
<td>−</td>
<td>asymptomatic</td>
<td>unknown</td>
<td>surgical treatment</td>
<td>unknown</td>
</tr>
<tr>
<td>Salmon and Blatt (1968)</td>
<td>24/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>EDH 7 days later</td>
<td>unknown</td>
<td>surgical treatment</td>
<td>unknown</td>
</tr>
<tr>
<td>Dechaume (1969)*</td>
<td>15/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>asymptomatic</td>
<td>unknown</td>
<td>wrapping</td>
<td>improved</td>
</tr>
<tr>
<td>Benoit and Wortzman (1973)</td>
<td>45/M</td>
<td>closed head trauma</td>
<td>+</td>
<td>SAH 11 days later</td>
<td>unknown</td>
<td>trapping</td>
<td>unknown</td>
</tr>
<tr>
<td>Dharker (1975)*</td>
<td>17/M</td>
<td>closed head trauma</td>
<td>+</td>
<td>hemiparesis 5 yrs later</td>
<td>unknown</td>
<td>—</td>
<td>death</td>
</tr>
<tr>
<td>Funahashi et al. (1977)*</td>
<td>15/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>SAH 9 days later</td>
<td>unknown</td>
<td>aneurysmal neck ligation</td>
<td>improved</td>
</tr>
<tr>
<td>Parkinson and West (1980)*</td>
<td>11/M</td>
<td>closed head trauma</td>
<td>+</td>
<td>decerebrate within 1 day</td>
<td>false</td>
<td>—</td>
<td>death</td>
</tr>
<tr>
<td>Yonas and Dujovny (1980)*</td>
<td>27/F</td>
<td>closed head trauma</td>
<td>−</td>
<td>SAH within 1 day</td>
<td>false</td>
<td>—</td>
<td>death</td>
</tr>
<tr>
<td>Reddy and Sundt (1981)*</td>
<td>43/M</td>
<td>iatrogenic</td>
<td>−</td>
<td>asymptomatic</td>
<td>false</td>
<td>trapping</td>
<td>death</td>
</tr>
<tr>
<td>Pozzati et al. (1982)*</td>
<td>45/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>ICH 9 days later</td>
<td>true</td>
<td>trapping</td>
<td>death</td>
</tr>
<tr>
<td>Shigemori et al. (1982)*</td>
<td>14/M</td>
<td>visual loss 5 days later</td>
<td>−</td>
<td>ICH 9 days later</td>
<td>unknown</td>
<td>trapping</td>
<td>death</td>
</tr>
<tr>
<td>Enomoto et al. (1984)*</td>
<td>17/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>visual loss 5 days later</td>
<td>false</td>
<td>—</td>
<td>improved</td>
</tr>
<tr>
<td>Jakobsson et al. (1984)*</td>
<td>16/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>VIth cranial nerve palsy 5 days later</td>
<td>unknown</td>
<td>ICA ligation</td>
<td>improved</td>
</tr>
<tr>
<td>Kieck and de Villiers (1984)*</td>
<td>22/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>visual loss 5 days later</td>
<td>unknown</td>
<td>—</td>
<td>improved</td>
</tr>
<tr>
<td>Salcman et al. (1985)*</td>
<td>45/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>SAH, IVH 17 days later</td>
<td>unknown</td>
<td>ICA ligation</td>
<td>unchange</td>
</tr>
<tr>
<td>Steinmetz et al. (1985)*</td>
<td>25/M</td>
<td>closed head trauma</td>
<td>+</td>
<td>SAH within 1 day</td>
<td>false</td>
<td>trapping</td>
<td>death</td>
</tr>
<tr>
<td>Bousquet et al. (1989)*</td>
<td>42/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>ICH 4 days later</td>
<td>unknown</td>
<td>surgical treatment</td>
<td>improved</td>
</tr>
<tr>
<td>Haddad et al. (1991)*</td>
<td>unknown</td>
<td>stab wound</td>
<td>unknown</td>
<td>unknown</td>
<td>unknown</td>
<td>surgical treatment</td>
<td>improved</td>
</tr>
<tr>
<td>Saito et al. (1992)*</td>
<td>26/M</td>
<td>closed head trauma</td>
<td>−</td>
<td>SAH, IVH 1 day later</td>
<td>unknown</td>
<td>ICA ligation</td>
<td>improved</td>
</tr>
<tr>
<td>Salz et al. (1993)*</td>
<td>35/F</td>
<td>closed head trauma</td>
<td>−</td>
<td>hemiparesis 5 yrs later</td>
<td>unknown</td>
<td>ICA ligation</td>
<td>unknown</td>
</tr>
<tr>
<td>Bousquet et al. (1993)*</td>
<td>35/M</td>
<td>iatrogenic</td>
<td>−</td>
<td>ICH, IVH 2 mos later</td>
<td>unknown</td>
<td>—</td>
<td>improved</td>
</tr>
<tr>
<td>du Toreau and van Dellen (1993)**</td>
<td>26/M</td>
<td>missile wound</td>
<td>unknown</td>
<td>unknown</td>
<td>unknown</td>
<td>no surgical treatment</td>
<td>unknown</td>
</tr>
<tr>
<td></td>
<td>35/F</td>
<td>iatrogenic</td>
<td>−</td>
<td>ICH, IVH 2 mos later</td>
<td>unknown</td>
<td>—</td>
<td>improved</td>
</tr>
</tbody>
</table>

*From Buckingham et al., Neurosurgery 22: 398-408, 1988.4) **In this series, there were seven patients who had traumatic aneurysms of the supraclinoid ICA due to penetrating stab wounds. Further data not described. *Patient with an aneurysm of the dorsal wall of the supraclinoid ICA. EDH: epidural hematoma, ICH: intracerebral hemorrhage, IVH: intraventricular hemorrhage.

following head injury, with a peak at 2–3 weeks.13,6,7,13b The intervals reported for traumatic supraclinoid ICA aneurysms range from several hours to 5 years. Rupture of these aneurysms usually

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occurred within 2 months, with peaks on the 1st day and between the 2nd and 3rd weeks (Table 1). In our patient, there were three hemorrhagic episodes including SAH that occurred immediately following the injury.

Radiological diagnosis of traumatic aneurysms is based on angiography. In contrast to congenital aneurysms, traumatic aneurysms are located remote from arterial divisions, often have irregular outlines, and demonstrate slow hemodynamics (i.e. delayed filling and emptying of the aneurysmal sac) and frequent increases in size. Sometimes the necks of these aneurysms cannot be detected easily.

Yonas and Dujovny have stated that early, direct surgical treatment of traumatic aneurysms of the supraclinoid ICA should be avoided until the maturation process (i.e. thickening, thrombosis, or calcification of the aneurysmal wall) is completed. However, the patient may die during this waiting period since traumatic aneurysms at this location have high rebleeding rates. Repeated bleeding may cause vasospasm, possibly resulting in enlargement and rupture of the aneurysm due to the increased peripheral vascular resistance. An improved prognosis requires early recognition and surgical obliteration.

Excision of the aneurysm followed by microsurgical repair of the ICA and ligation of the aneurysmal neck have both been successfully used, but direct surgery on these aneurysms is difficult because of their fragile, broad-based, and obscure necks. Saito et al. have suggested that balloon occlusion of the carotid artery is relatively easy and effective for the treatment of traumatic aneurysms in this location. Eleven of the previous patients with aneurysms of the supraclinoid ICA were treated by trapping or carotid ligation in the neck. Two of these patients died, and the aneurysm of one patient persisted following cervical ICA ligation.

The surgical outcome for the 20 patients reported included six (30%) deaths. We believe that effective therapy for this type of lesion requires extracranial-intracranial bypass followed by direct surgery on the aneurysm such as complete wrapping and clipping of the aneurysm, excision of the aneurysm followed by ICA repair, or trapping of the aneurysm.

References

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Address reprint requests to: K. Saito, M.D., Department of Neurosurgery, Nagoya University School of Medicine, 65 Tsurumai, Showa-ku, Nagoya 466, Japan.