Traumatic aneurysm of the internal carotid artery: a late finding presenting as a mass in the sphenoid sinus.

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Traumatic Aneurysm of the Internal Carotid Artery: A Late Finding Presenting as a Mass in the Sphenoid Sinus

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Summary: We describe a case of a traumatic aneurysm of the cavernous portion of the internal carotid artery in a patient who had had craniofacial trauma 12 years before. MR and CT revealed a mass in the sphenoid sinus thought to be unrelated to the patient's symptoms. Carotid angiography gave the correct diagnosis.

Index terms: Aneurysm; Paranasal sinuses

Traumatic aneurysms of the intracranial portion of the internal carotid artery are rare. These lesions may appear in the petrous (1), cavernous (2–4), or supraclinoid (5) portions of the internal carotid artery. Most frequently, they are located in the cavernous portion. A traumatic aneurysm of the cavernous portion of the internal carotid artery must be suspected in a patient presenting with the clinical triad of severe craniofacial trauma (typically a frontal facial fracture), late epistaxis, and monocular blindness (3–7). Carotid angiography will lead to the correct diagnosis in most cases.

Case Report

A 32-year-old man presented to our hospital with a history of episodic dizziness and visual deficits that recurred over a period of 8 years. These episodes were associated with hemicranial pain that was relieved by common analgesics. More recently, over the period of the last 2 to 3 months, episodes of somnolence developed.

History revealed severe craniofacial trauma 12 years before admission that resulted in a severe hemiparesis. This gradually resolved and at the time of admission, neurologic examination revealed mild right hemiparesis. Admitting laboratory tests were unremarkable. Electroencephalography showed no abnormalities.

Diagnostic examinations included magnetic resonance (MR) imaging and computed tomography (CT). The spin-echo T1-weighted MR sequences were obtained with and without gadolinium administration. In addition, T2-weighted and intermediate-weighted images were obtained. A mass was seen in the sphenoid sinus, with curved layers of different signal intensities on all sequences (Fig 1A–C). After contrast administration, there was heterogeneous enhancement, more intense in the center of the mass. Some inflammatory pathology was seen in the ethmoid sinuses. The carotid arteries produced a normal signal void at the level of the cavernous sinuses. The CT scan revealed a mass that occupied the entire sphenoid sinus, with erosion of the superior wall (Fig 1D–F). There also was a small defect on the right lateral wall. After contrast administration, there was intense enhancement in some portions of the mass.

Initial biopsy of the mass by nasal endoscopy failed to obtain any significant tissue. Eventual transsphenoidal surgery was performed, during which a well-encapsulated mass that bled profusely when pierced was identified. Local hemostasis was performed, and the patient then was referred for angiography.

Selective carotid angiography revealed a giant post-traumatic aneurysm with an irregular wall that originated from the cavernous portion of the right internal carotid artery (Fig 1G). Embolization of the internal carotid artery with two 8-mm silicone balloons was performed 2 weeks later. One balloon was placed immediately next to the aneurysm. The patient tolerated the expansion of the initial balloon without deficit after 30 minutes of evaluation. A second balloon then was placed in the petrous portion of the carotid artery. The patient recovered uneventfully, and a later control CT scan demonstrated absence of contrast enhancement at the level of the sphenoid sinus, indicating complete thrombosis of the aneurysm. The carotid artery also was thrombosed in an angiographic control.

Discussion

One half of all intracranial traumatic aneurysms affect the internal carotid artery. The most frequent location is the cavernous portion (60%), whereas the petrous portion is rarely affected (6). Other traumatic lesions of the ca-

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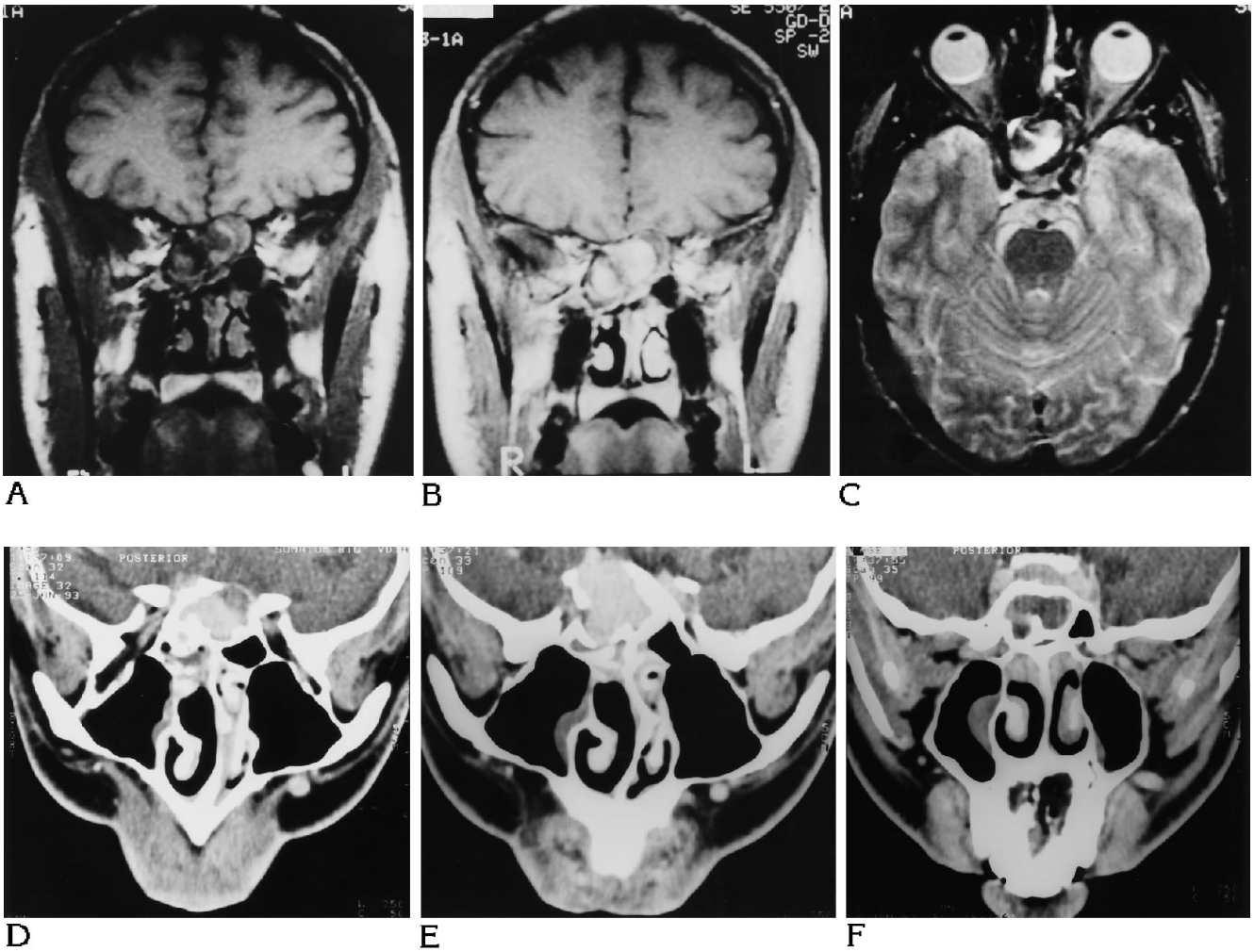


Fig 1. *A–C*, Spin-echo MR. *A*, Coronal (550/30 [repetition time/echo time]): a mass occupies the entire sphenoid sinus. It shows curved layers of different signal intensity (from *left* to *right*, isointense with gray substance-hypointense-isointense-hypointense).

B, Coronal with gadopentetate dimeglumine (550/20): there is contrast enhancement in the hypointense central area, representing the lumen within the aneurysm.

C, Axial (2500/90): hypointense periphery and central area with high and low signal intensity. The signal void of the cavernous portion of both carotids presents a normal caliber.

D–F, Coronal contrast-enhanced CT front to back.

D, A mass occupies the entire sphenoid sinus and has broken through the roof. Intense contrast enhancement of the *lower portion* with no enhancement of the *upper*. The enhancement represents the lumen of the aneurysm.

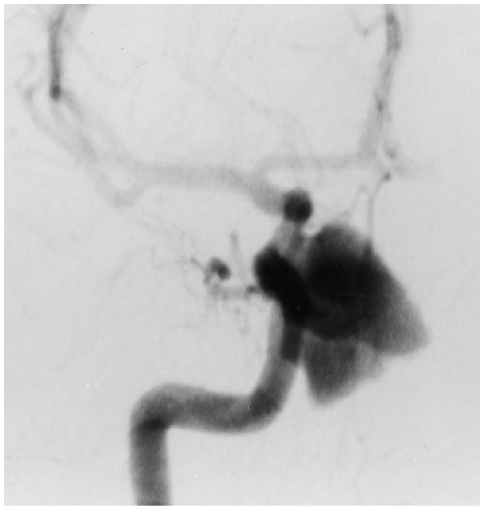
E, Notice a small defect on the right lateral wall and floor; intense enhancement of the whole mass.

F, The floor is intact, and there is no enhancement in this posterior cut. (*Figure continues.*)

rotid artery include dissection with thrombosis, carotid cavernous fistula, and complete blood vessel rupture (3).

Although symptoms can be variable, traumatic aneurysm in the sphenoid sinus must be suspected in patients presenting with the clinical triad of facial fractures, monocular blindness, and late epistaxis. If a diagnostic angiogram is performed early, it may or may not reveal the presence of an aneurysm. Some have indicated that a second angiogram, which must be performed in 2 to 3 weeks, then may show the traumatic aneurysm (3).

Of the classic clinical triad, epistaxis was a constant finding in a previous review of 100 cases from the literature (3). Unilateral blindness appeared in 73 patients, whereas a history of fracture occurred in 77 of 88 patients. Occasionally, other cranial nerves were affected, although much less frequently than the optic nerve. Epistaxis appeared at variable times after the trauma, usually less than 6 months after



G

Fig 1, *continued.*

G, Carotid angiography: a large aneurysm with an irregular wall and no neck originates from the cavernous portion of the right internal carotid artery.

injury. In seven cases, the epistaxis appeared later than 6 months. Several cases of long-delayed epistaxis have been reported (8–10). In some cases, recurrent episodes of epistaxis have occurred before the diagnosis is reached.

The loss of unilateral vision that occurs immediately after trauma often is the result of either a direct lesion to the optic nerve, injury to the ophthalmic artery, or compression by a lesion such as an aneurysm. In some cases, the loss of vision may appear late and may even be bilateral (11). Traumatic aneurysms of the cavernous portion of the carotid artery also may produce symptoms related to compression of the cranial nerves in the cavernous sinus or symptoms secondary to a carotid cavernous fistula (proptosis, loss of visual acuity, or homonomous hemianopsia) (8).

Our case, which appeared as an asymptomatic mass in the sphenoid sinus, is similar to another case in which a mass was discovered by CT (10). The vascular lesion occupied the posterior ethmoid air cells and the anterior portion of the sphenoid sinus. This asymptomatic female patient was evaluated approximately 6 years after a serious injury. As in our case, angiography was performed after a bleeding mass was encountered during transnasal surgery.

The MR images of giant partially thrombosed aneurysms produce a variety of signal intensities, depending on the flow velocity, the amount of existing thrombus, and the possible presence of associated blood products (12, 13). The easiest recognizable form is the presence of a signal void in the central zone, caused by rapid flow, surrounded by layers of high- and low-signal intensity that represent a mixture of methemoglobin and hemosiderin (12). Next to the aneurysm, the artery from which it originates can be present as an area of signal void. Our case did not show a definite flow void within the aneurysm. Curved layers of mixed signal intensity represented the various components of the thrombus. Slow flow within the lumen of the aneurysm gave a variable signal. This image with no internal signal void sometimes can be seen in large aneurysms (12, 13).

An interesting question that arises with the discovery of an aneurysm long after craniofacial trauma is whether the lesion is a pseudoaneurysm secondary to the trauma or an independent preexisting lesion. In our case, the angiographic characteristics of irregular walls, no true neck, late filling and emptying, and the characteristic intrasphenoid location all are indications of a posttraumatic lesion (3.5).

Before the development of MR imaging and CT, patients with severe craniofacial trauma often were examined with angiographic procedures. This sometimes allowed the early detection of traumatic pseudoaneurysms. Because less invasive techniques are more commonly used today, it is possible to foresee a growing frequency of late diagnosis of traumatic aneurysms. This delay in diagnosis may be aggravated whenever there are no symptoms present, even if there is a history of a serious craniofacial injury. In our case, as in a similar case in the literature, the findings led to the performance of a diagnostic biopsy that could have had catastrophic consequences for the patient. If an isolated mass is seen on imaging studies within the sphenoid sinus, it seems appropriate that an aneurysm always should be considered, even if the injury occurred many years earlier. Aneurysm must always be considered if an enhancing lesion close to the carotid artery is seen with CT or MR or a mass with curved layers of mixed signal intensity is see with MR. If there is any question in the differential diagnosis, angiography may be necessary to exclude the presence of a traumatic aneurysm.

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