Are your MRI contrast agents cost-effective?
Learn more about generic Gadolinium-Based Contrast Agents.
FRESENIUS KABI
caring for life

AJNR

Carotid-cavernous fistula due to traumatic dissection of the extracranial internal carotid artery.

J M Tucci, C G Maitland, D W Pcsolyar, J R Thomas and J L Black

AJNR Am J Neuroradiol 1984, 5 (6) 828-829
http://www.ajnr.org/content/5/6/828.citation

This information is current as of April 20, 2024.

Carotid-Cavernous Fistula Due to Traumatic Dissection of the Extracranial Internal Carotid Artery

James M. Tucci,[1] Charles G. Maitland,[2] Dale W. Pcsolyar,[1] John R. Thomas,[3] and Jeffrey L. Black[4]

Carotid-cavernous fistulae are usually associated with significant head injury and presumably result from direct intracavernous carotid artery damage [1]. Less common spontaneous fistulae may arise from ruptured intracavernous aneurysms [2]. Recent angiographic evidence suggests that more often they represent dural arteriovenous shunts established by rupture of thin-walled dural arteries in or about the cavernous sinus [3]. Minor head trauma or straining in the presence of predisposing, acquired vascular disease are suggested causes [3]. We report a patient with angiographic features of a dissecting aneurysm of the cervical part of the internal carotid artery with intracranial extension causing carotid-cavernous fistula formation.

Case Report

A 40-year-old man developed severe, throbbing, right-sided neck pain that extended within hours to involve the right side of his face and head. Twelve hours earlier he had been struck high in the right side of the neck by a fist. He had neither fallen nor lost consciousness. Within 1 day he complained of brief episodes of tingling and numbness in the left arm and face, occasional visual blurring, and a sense of constriction of the visual fields in both eyes. He was admitted 10 days after the symptoms appeared.

The neurologic and neuroophthalmologic examinations were normal except for extinction on the left arm with bilateral, simultaneous, tactile stimulation. There were no carotid, cranial, orbital, or subclavian bruits. Brachial blood pressure was 125/80 mm Hg in each arm. Ophthalmoplethysmographic pressure was 65 mm Hg in the right eye and 104 mm Hg in the left eye. Skull and cervical spine radiographs, CT scans of the head, blood cell counts, routine blood chemistries, electrocardiography, and chest radiographs were normal. Transfemoral bilateral carotid and vertebral angiograms showed a dissecting aneurysm of the right internal carotid artery, proximal to the base of the skull, that extended into the carotid canal. In addition to a pseudoaneurysm of the proximal petrous part of the artery, there was severe tapered stenosis of the precavernous internal carotid artery. Within the cavernous sinus, a carotid-cavernous fistula drained primarily through the inferior petrosal sinus and pterygoid venous plexus, through the contralateral cavernous sinus, and to some extent through the ophthalmic veins (fig. 1).

A left carotid angiogram showed that the right anterior and middle cerebral arteries were supplied exclusively via the anterior communicating artery. Anticoagulation therapy with heparin, instituted before arteriography, was unsuccessful in relieving the patient's symptoms and was discontinued. Transient sensory symptoms resolved within 6 weeks. However, right-sided retroorbital pain persisted, and after a 2 month period, the patient noted the gradual appearance of a right-sided bruit in the head associated with intermittent lid swelling and scleral injection of the right eye. Examination showed a loud pulse-synchronous bruit over the right orbit. The right eye was mildly chemotic with congested conjunctival vessels; it protruded 5 mm more than the left eye. Repeat angiographic study demonstrated a patent right internal carotid artery with enlargement of the fistula (fig. 2).

An attempt to occlude the fistula via a transfemoral approach with a detachable Silastic balloon (Pevsner, Becton-Dickinson) was unsuccessful. The lumen of the fistula was judged to be too small to allow passage of the balloon into the cavernous sinus. Proptosis and intraocular pressure increased and visual acuity diminished to 20/40 in the right eye with funduscopic signs of an ischemic retinopathy. Another similar attempt to close the fistula selectively was unsuccessful. Subsequently, both the right internal carotid artery and fistula were occluded simultaneously with a single balloon. Postoperatively, there was evidence of a mild, transient left hemiparesis. Vision improved to 20/20, and all signs of orbital venous congestion resolved over a 2-week period. Follow-up examination 6 weeks later was normal.

Discussion

Blunt, nonpenetrating neck trauma causes a variety of lesions of the carotid artery [4]. Stretching of the artery across the bony projection of the upper cervical vertebrae lying posterolateral to the vessel may cause intimal disruption with secondary dissection of blood into the media. Hyperextension and lateral flexion neck maneuvers seem the most likely cause of such injury.

Received April 26, 1983; accepted after revision September 13, 1983.

This material has been reviewed by the Uniformed Services University of the Health Sciences, and there is no objection to its presentation and/or publication. The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Uniformed Services University of the Health Sciences or the Department of Defense.

[1] Department of Neurology, National Naval Medical Center, Bethesda, MD 20814.

[2] Department of Neurology, Neuro-ophthalmology Unit, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Rd., Bethesda, MD 20814. Address reprint requests to C. G. Maitland.

[3] Department of Radiology, National Naval Medical Center, Bethesda, MD 20814.

[4] Department of Radiology, Walter Reed Army Medical Center, Washington, DC 20012.

AJNR 5:828–829, November/December 1984 0195–6108/84/0506–0828 $00.00 © American Roentgen Ray Society

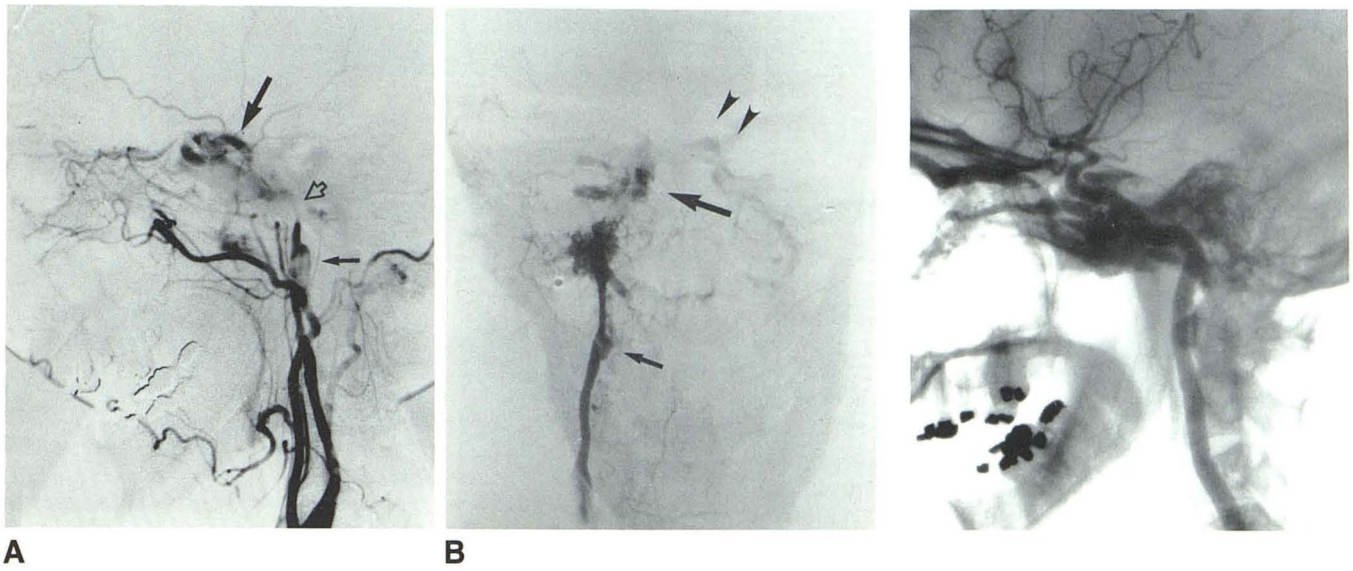

Fig. 1.—Right common carotid arteriogram, lateral (**A**) and AP (**B**) views. Dissection with false aneurysm formation (*small arrows*). Segment distal to aneurysm is narrowed (*open arrow*). Carotid-cavernous fistula (*large arrows*) drains primarily into pterygoid plexus and contralateral cavernous sinus (*arrowheads*).

Fig. 2.—Right internal carotid arteriogram. Caliber of vessel is now relatively normal. Fistula persists; significant drainage is present through dilated superior and inferior ophthalmic veins.

The exact mechanism of carotid-cavernous fistula formation remains unknown. These fistulae usually result from head trauma, often with associated basal skull fracture [1]. Some investigators suggest that fistula formation might require simultaneous injury to both artery and venous channels [5]. Cahill et al. [6] reported the angiographic association of a carotid-cavernous fistula with an ipsilateral, internal carotid occlusion in a patient with severe head trauma. They speculated that an intimal tear just proximal to the cavernous part of the carotid artery may have produced both a proximal carotid occlusion and a dissection distally. Collateral retrograde flow into the carotid artery may have resulted in intracavernous carotid rupture with fistula formation in their case.

Our case demonstrates that carotid dissection can be associated with a carotid-cavernous fistula without associated head trauma. Furthermore, the origin of the dissection may be remote from the cavernous sinus. Initially, patency of the fistula seemed to be maintained partially by collateral circulation through the ophthalmic artery and through the severely narrowed right internal carotid artery; the fistula displayed low flow characteristics similar to dural arteriovenous shunts [3]. Ultimately, with normalization of the patency of the internal carotid artery, a high flow state with attendant signs of orbital congestion developed.

Spontaneous resolution of the luminal narrowing of the internal carotid artery in this case supports the tendency toward conservative management of traumatic dissections unless there are signs of imminent catastrophic ischemia [7]. The additional findings of a carotid-cavernous fistula made management problematic. However, neurologic sequelae are seldom encountered as a direct result of carotid-cavernous fistulae [6]. Therapeutic intervention was accomplished only after the development of symptoms and signs of retinal ischemia. This case confirms the utility of the balloon catheterization technique, with its attendent low morbidity [8], as a means to accurately locate and shut off blood flow to control a carotid-cavernous fistula in the presence of a more diffuse carotid disease process, providing an alternative to more direct surgical intervention.

REFERENCES

1. Hamby WB. *Carotid-cavernous fistulae*. Springfield, IL: Thomas, **1966**
2. Jefferson G. On the saccular aneurysms of the internal carotid artery in the cavernous sinus. *Br J Surg* **1938**;26:267–302
3. Newton TH, Hoyt WF. Dural arteriovenous shunts in the region of the cavernous sinus. *Neuroradiology* **1970**;1:71–81
4. Krajewski KP, Hertzen NR. Blunt carotid artery trauma. *Ann Surg* **1980**;191:341–346
5. Taptas JN. Arteriovenous aneurysm in pulsating exophthalmos: a new conception of mechanism. In: *Excerpta Medica International Congress, series 60*. Amsterdam: Excerpta Medica, **1963**:98–99
6. Cahill DW, Rao KCVG, Ducker TB. Delayed carotid-cavernous fistula and multiple cranial neuropathy following basal skull fracture. *Surg Neurol* **1980**;16:17–22
7. Stringer WL, Kelly DL. Traumatic dissection of the extracranial internal carotid artery. *Neurosurgery* **1980**;6:123–130
8. Berenstein A, Kricheff I, Ransohoff J. Carotid-cavernous fistula: intraarterial treatment. *AJNR* **1980**;1:449–457