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# Iatrogenic Carotid-Cavernous Fistula Occurring after Embolization of a Cavernous Sinus Meningioma

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**Summary: A carotid-cavernous fistula developed in a 62-year-old woman during an attempt at embolization of a skull base meningioma. The cause is thought to be perforation by the guide wire during catheterization of the meningohypophyseal trunk at the sharp bend at its origin.**

**Index terms: Catheters and catheterization, complications; Iatrogenic disease or disorder; Interventional neuroradiology, complications**

Preoperative embolization of meningiomas is routinely performed with relatively few reported complications. There are rare reports of subdural and subarachnoid hemorrhage associated with embolization of meningiomas. We report the creation of a direct carotid-cavernous fistula during embolization of a skull base meningioma.

## Case Report

A 63-year-old woman presented with a large skull base meningioma with extension into both cavernous sinuses (Fig 1A). She had a long history of decreased vision in the left eye (light perception only) and a progressive right temporal field cut was developing from tumor encroachment on the optic chiasm. Arteriography demonstrated a hypertrophied left meningohypophyseal trunk supplying most of the tumor (Fig 1B and C), with the remainder supplied by branches of the left middle meningeal artery.

Selective catheterization of the meningohypophyseal trunk was attempted with a Tracker-18 catheter and 0.016-in Taper guide wire (Target Therapeutics, Fremont, Calif); the guide wire could enter the proximal artery, but the catheter tip was too large to follow into the vessel. A Tracker-10 catheter and 0.010-in Seeker Lite guide wire were then used to catheterize the meningohypophyseal trunk. With the Tracker-10 catheter in place, there was slow antegrade flow within the meningohypophyseal trunk and only limited contrast opacification of the tumor could be achieved without reflux into the internal carotid artery. The angiographic appearance was not that of injection through a wedged catheter into a closed system; rather, it was consistent with occlusion of the dominant arterial supply with continued supply of unopacified blood by tiny, angiographically invisible cavernous internal carotid artery and parasitized pial branches. Provocative pharmacologic testing with 25 mg of lidocaine produced no neurologic changes, so tumor embolization with a dilute Avitene (Alcon, Inc, Humacao, Puerto Rico) slurry (1 gm/500 mL Ioversol 320) was begun. Because of the unfavorable flow characteristics, it was not possible to produce a significant angiographic change in the tumor without reflux into the internal carotid artery. Because the patient had experienced considerable pain and some nausea during catheterization, the procedure was discontinued. A follow-up internal carotid artery arteriogram showed no appreciable change in the tumor blush after partial embolization. The patient had mild headache and nausea, which improved overnight.

The patient returned the next day for proximal occlusion of the meningohypophyseal trunk with platinum microcoils, embolization of the left middle meningeal artery and left internal carotid artery balloon test occlusion. A left internal carotid artery arteriogram (Fig 1D) now showed a new direct carotid-cavernous fistula via the meningohypophyseal trunk. Selective catheterization of the meningohypophyseal trunk was performed with a Tracker-10 catheter and this was advanced into the venous side of the fistula. Five circular 7-mm long $\times$ 3-mm diameter platinum microcoils (Target Therapeutics, Fremont, Calif) were placed into the venous outflow producing occlusion of the fistula and the proximal meningohypophyseal trunk. A follow-up internal carotid artery arteriogram (Fig 1E and F) showed persistent tumor blush both from pial branches and from minuscule cavernous internal carotid artery branches, but not from the meningohypophyseal trunk. Embolization of the left middle meningeal artery and a left internal carotid artery balloon test occlusion were then completed uneventfully.

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Fig 1. *A*, MR (coronal postgadolinium T1-weighted image) shows an enhancing parasellar mass encasing the left cavernous internal carotid artery. The hypertrophied meningohypophyseal trunk is visible (*arrow*).

*B* and *C*, A left internal carotid artery arteriogram demonstrates the hypertrophied meningohypophyseal trunk in the arterial phase (*B, arrow*) and the dense tumor blush in the capillary phase (*C*).

*D*, A left internal carotid artery arteriogram obtained 1 day after partial embolization of the meningohypophyseal trunk shows a new direct carotid-cavernous fistula via the meningohypophyseal trunk (*straight arrow*) with drainage into the inferior petrosal sinus (*curved arrow*).

*E* and *F*, A left internal carotid artery arteriogram obtained after embolization of the carotid-cavernous fistula with platinum microcoils (*E, arrow*) confirms complete occlusion of the fistula. There is persistent tumor blush from both pial branches and minuscule cavernous carotid branches (*F*), but the meningohypophyseal trunk is no longer filled.

## Discussion

Embolization of small cavernous internal carotid artery branches may be necessary to treat vascular malformations, fistulas (1), or skull base tumors. Catheterization and embolization of these arteries is usually much more difficult than external carotid branches. Even when hypertrophied, these internal carotid artery branches may be smaller than a Tracker-10 catheter; the origin and tortuosity of these branches also may prevent successful catheterization. The meningohypophyseal trunk usually has a sharp turn just beyond its origin. We suspect the apex of this turn to be particularly vulnerable to injury by the guide wire, because this was the site of the fistula in our patient. We believe perforation of this artery was caused by the 0.016-in Taper wire during our unsuccessful attempt to introduce the Tracker-18 catheter into the meningohypophyseal trunk. High pressure generated during injection of a wedged catheter into a closed system may also produce arterial rupture, but the angiographic appearance of such a closed system was not present in this case. Arterial perforation by both mechanisms during neurointerventional procedures has been described (2, 3).

We elected to perform transarterial, rather than transvenous, embolization of the carotid-cavernous fistula, because we already had a catheter positioned in the internal carotid artery, a Tracker-10 catheter had been prepared for immediate use, and catheterization of the meningohypophyseal trunk with the Tracker-10 catheter had been easily performed the previous day. Although transvenous embolization has a potentially lower morbidity, the transarterial route was more expedient in this case. Had we encountered any difficulty catheterizing the meningohypophyseal trunk, a transvenous ap-

proach via the inferior petrosal sinus would have been used.

We have been unable to locate any report of creation of a carotid-cavernous fistula during embolization of a skull base neoplasm. It is interesting that the fistula was not immediately apparent. The risk of arterial perforation is probably significantly increased during catheterization of tortuous tumor vessels. Both subarachnoid and subdural hemorrhages have been reported during meningioma embolization (4–7); these may have occurred from arterial perforation or tumor necrosis with subsequent hemorrhage. It is important for the interventionalist to be aware of these possibilities and to respond to them if they occur.

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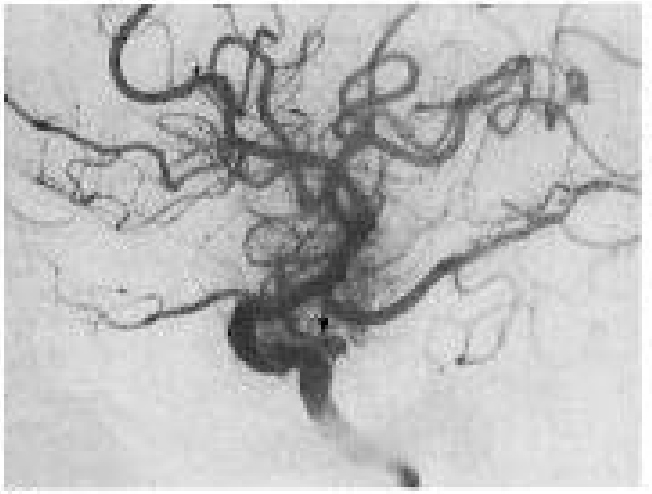
A



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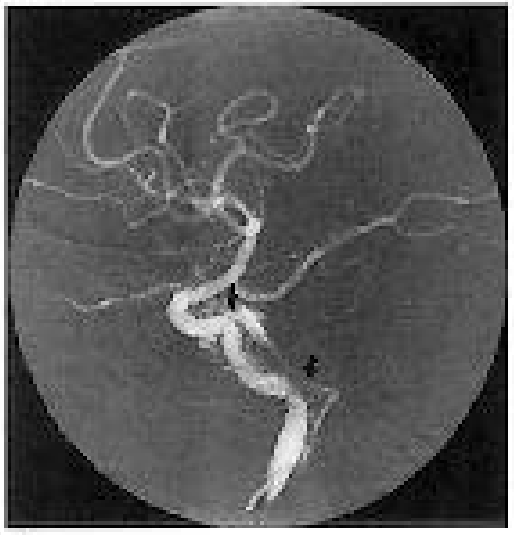

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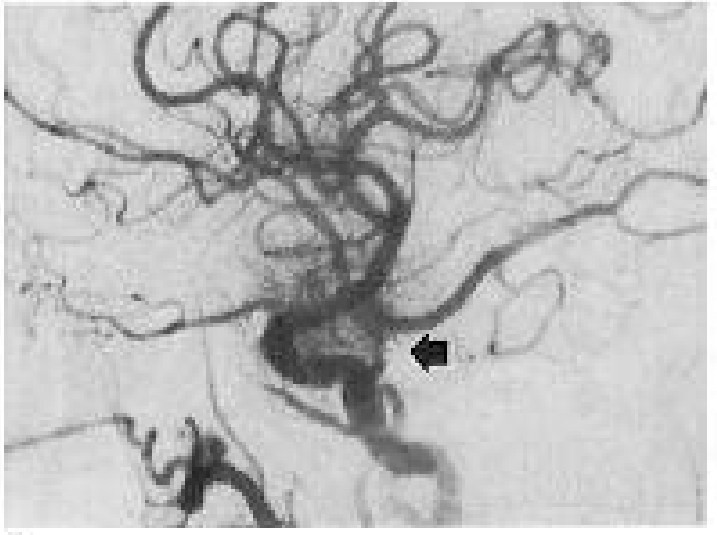

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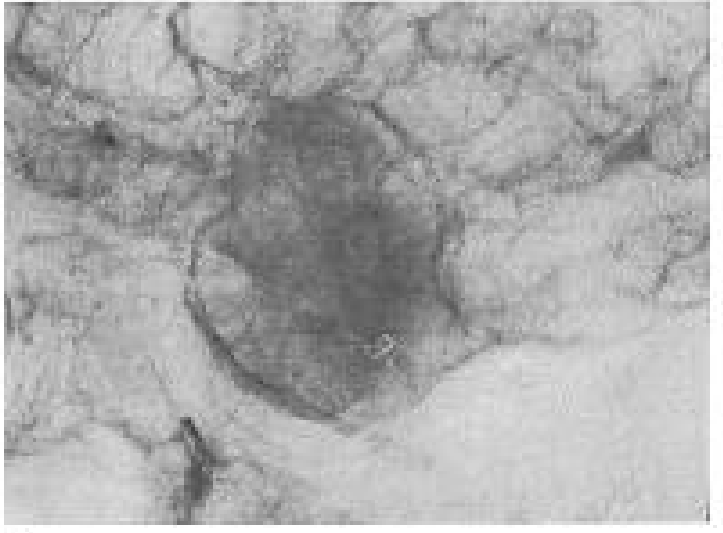

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