

Cerebellar hemorrhage and subsequent venous infarction followed by incomplete transvenous embolization of dural carotid cavernous fistulas: a rare complication

Case report

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✓ Endovascular therapy for dural carotid cavernous fistulas (CCFs) is generally accepted to be safe and effective. The authors report a rare complication of hemorrhage and subsequent venous infarcts of the pons and cerebellum after transvenous embolization.

This 41-year-old man presented with a severe left frontal headache, congestion of the left conjunctiva, blurred vision, and photophobia. Cerebral angiography demonstrated a right dural CCF. The patient underwent transvenous embolization of the cavernous sinus but had the initial complication of cerebellar hemorrhage. One month later, he developed progressive dizziness, ataxia, and right-sided weakness. Magnetic resonance imaging revealed severe cerebellar and pontine edema. The cause was a residual fistula combined with delayed occlusion of the inferior petrosal sinus. The fistula was obliterated after repeated embolizations. The patient's symptoms gradually resolved, and there was no evidence of recurrence during the 4-year follow-up period.

Incomplete transvenous embolization of a dural CCF can result in life-threatening vascular complications due to redistribution of shunt flow. Early recognition of redistributed drainage and preventive placement of coils at the origin of draining veins during the procedure could avert this rare complication. (DOI: 10.3171/JNS.2008.108.6.1245)

KEY WORDS • brainstem ischemia • dural arteriovenous fistula •
postoperative complications • therapeutic embolization • venous infarction

DURAL arteriovenous malformations account for 10–15% of all intracranial arteriovenous malformations¹² and most commonly involve the cavernous, transverse, and sigmoid sinuses.⁹ A dural CCF consists of arteriovenous shunting between the carotid artery and the cavernous sinus. In recent years, transvenous embolization of dural CCFs has become widely accepted as a safe and effective therapeutic method. We report the case of a patient with a dural CCF who received transvenous embolization and who experienced the rare complications of intraprocedural cerebellar hemorrhage and late-onset venous infarcts.

Case Report

History and Examination. This 41-year-old man presented with left-sided visual disturbances and diplopia accompanied by a 3-month history of nausea and vomiting. He

Abbreviations used in this paper: CCF = carotid cavernous fistula; ICA = internal carotid artery; IPS = inferior petrosal sinus; MR = magnetic resonance; NBCA = *N*-butyl-2-cyanoacrylate; SPS = superior petrosal sinus.

also had a severe left frontal headache, photophobia, and a sensation of bruit behind the eyeball, which developed over 1 month. On examination, a neurologist observed conjunctival chemosis, ptosis, anisocoria, and abducent nerve palsy. Magnetic resonance angiography was performed, and the findings suggested a CCF. Digital subtraction cerebral angiography then showed a dural CCF. The dural branches of the left ICA (Fig. 1 *left*) and the external carotid artery were connected to the cavernous sinus. Also visualized were a retrograde shunt with an engorged left inferior ophthalmic vein, the left sphenoparietal sinus, and the SPS.

First Embolization. Transvenous embolization of the cavernous sinus was performed through the left internal jugular vein and the IPS with the patient in a state of general anesthesia. Embolization was achieved using a microcatheter (FasTracker 18, Target Therapeutics/Boston Scientific) and 16 fibered microcoils (Vortex, Target Therapeutics/Boston Scientific). Follow-up angiography showed substantially reduced shunt flow, but residual flow was present.

To completely occlude the shunts, NBCA was added through the microcatheter. Immediately after NBCA injection, the patient's heart rate and blood pressure transiently rose from 70 to 90 bpm and from 100/50 to 140/70 mm Hg,

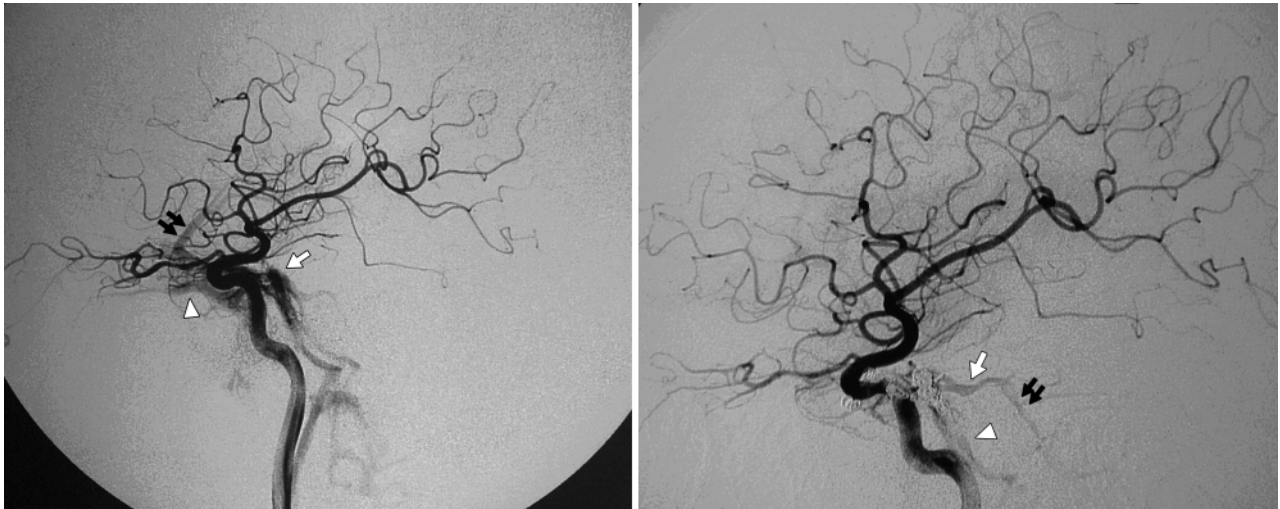


FIG. 1. *Left*: Lateral left ICA angiogram demonstrating an early opacifying left cavernous sinus (*arrow*), bilateral IPSs, the left inferior ophthalmic vein (*arrowhead*), and the left sphenoparietal sinus (*double arrows*). *Right*: Left ICA angiogram, obtained after the fibered microcoils and NBCA deployment, showing a substantial reduction in shunt flow. The residual shunt flow was drained by the IPS (*arrowhead*), SPS (*arrow*), and deep cerebellar veins (*double arrows*).

respectively. Postembolization angiography demonstrated residual shunt flow draining to the left IPS, the SPS, and the deep cerebellar veins (veins of the lateral recess) (Fig. 1 *right*). The procedure was then halted.

The patient's headache and eye pain resolved after embolization, but dilation of the left pupil and the abducent nerve palsy persisted. On the 2nd day, MR imaging revealed an acute hemorrhagic lesion of the left cerebellum with peripheral edema (Fig. 2).

Second Embolization. One month after embolization, the patient suffered progressive dizziness, ataxia, and right-sided weakness. An MR imaging study revealed severe edema in the left pons and cerebellum (Fig. 3 *left*). Subsequent angiography showed occlusion of the left IPS with progressive engorgement of the SPS and deep cerebellar veins, which were mainly supplied by the left ICA through residual shunts (Fig. 3 *right*).

Because of the occlusion of the left IPS, repeated attempts at transvenous embolization failed. Transarterial embolization through the left ICA obliterated only part of the fistulous flow. For complete occlusion of the fistula, trapping of the left ICA was planned after a balloon occlusion test. The patient well tolerated the test occlusion for 30 minutes. Four detachable balloons were then used to occlude the left ICA and the fistula (Fig. 4).

Postembolization Course. After embolization, the patient's symptoms gradually resolved, and he was discharged home after rehabilitation. At 2-year follow-up, MR imaging showed regression of the edema and cerebellar atrophy (Fig. 5). During the 4-year clinical follow-up period, the patient exhibited mild weakness of the right limb. Ataxia and dysmetria remained and were stable.

Discussion

For patients with dural CCF and mild symptoms, observation alone or carotid jugular compression⁴ provides an opportunity for the fistula to close. For patients with severe symptoms, various treatment options are available. Sur-

gical obliteration of the involved dural sinus is not favored because severe complications have been observed.¹⁴ Transarterial particle-based embolization of the feeding arteries may be associated with a high recurrence rate. Transarterial application of NBCA¹⁰ and transvenous embolization^{6,11} have been good alternatives for the treatment of dural fistulas. Direct transvenous NBCA injection combined with coil deployment, as in our case, can achieve complete oc-

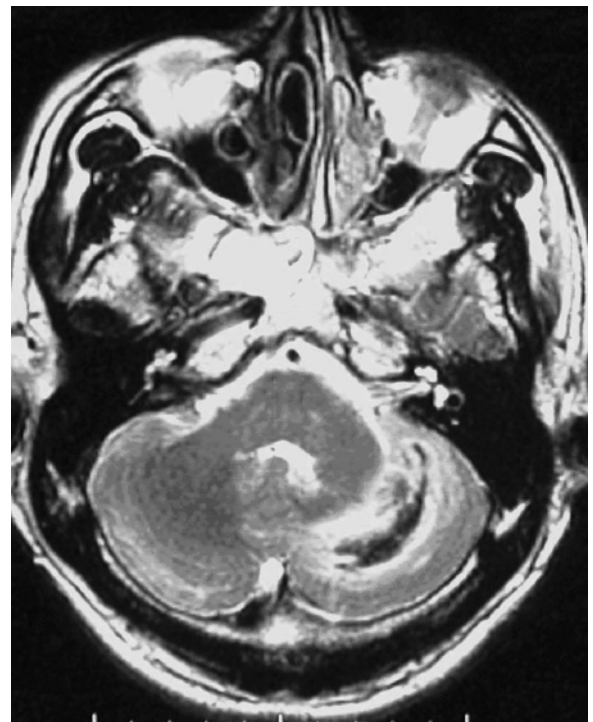


FIG. 2. Axial T2-weighted MR image, obtained 1 day after embolization, demonstrating acute hemorrhage in the left cerebellum with peripheral edema.

Cerebellar venous infarcts following dural fistula embolization

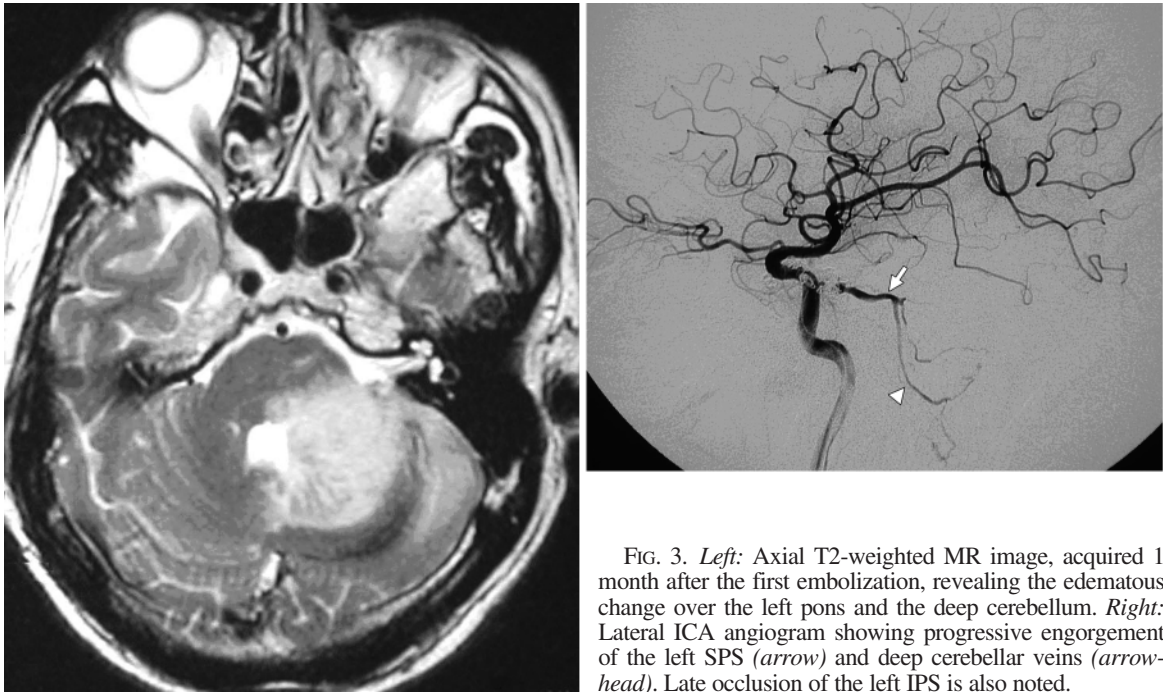


FIG. 3. *Left:* Axial T2-weighted MR image, acquired 1 month after the first embolization, revealing the edematous change over the left pons and the deep cerebellum. *Right:* Lateral ICA angiogram showing progressive engorgement of the left SPS (arrow) and deep cerebellar veins (arrow-head). Late occlusion of the left IPS is also noted.

clusion of complex dural fistulas.¹⁵ Radiosurgery offers another choice for patients who are hesitant to receive endovascular therapy.²

Reported complications after transvenous embolization of a dural CCF include deterioration of ocular motor dysfunction,^{1,13} transient visual loss,³ embolic stroke,³ and metachronous dural arteriovenous fistula.⁷ We report the rare postembolization complication of a posterior fossa hemorrhage and delayed venous infarcts.

In our case, venous infarcts and hemorrhage of the left cerebellum and pons were closely related to the venous drainage pathways. Huang⁵ and Lasjaunias and Berenstein⁸ have illustrated anatomical variations of venous drainage in the posterior cranial fossa. They described 2 main channels that drain the deep cerebellar hemispheres. The first group of channels drains to the hemispheric or vermian veins and



FIG. 4. Right common carotid artery angiogram, obtained after the left ICA was trapped, showing good collateral flow into the left cerebral hemisphere and the absence of fistulas.

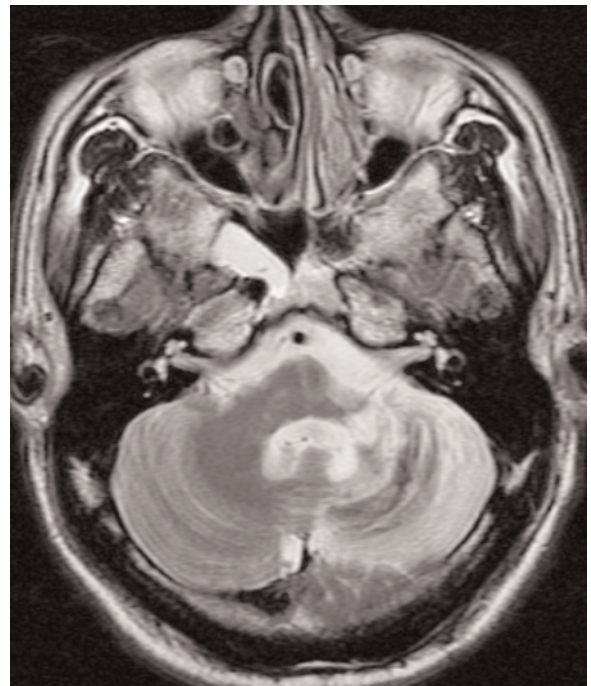


FIG. 5. Two-year follow-up axial T2-weighted MR image revealing reduced volume of the left pons and the cerebellum due to the regression of edema.

later to the torcula herophili or tentorial sinus. The second group drains to the veins of the lateral recess, the petrosal vein, and later to the SPS. The present case fits the second drainage pathway.

The cerebellar hemorrhage that occurred immediately after NBCA-assisted embolization could have been due to high pressure in the cavernous sinus. Aihara et al.¹ have described high intracavernous sinus pressure during embolization, which resulted in cranial nerve palsy. In our first embolization procedure, occlusion of the main venous drainage outlets, such as the inferior ophthalmic vein, the sphenoparietal sinus, and the contralateral cavernous sinus, was achieved. At the same time, the microcatheter partially occluded the left IPS. Residual shunt flow might then have been redirected to the SPS and the deep cerebellar veins, causing cerebellar hemorrhage.

High intrasinus pressure after partial obliteration of the drainage pathways can be dangerous and can lead to subsequent complications.⁷ In our patient, the same mechanism may also account for the delayed onset of the cerebellar and pontine venous infarcts. Nonobliterated shunt flow combined with late occlusion of the draining IPS resulted in focal cerebellar congestion and edema 1 month later.

To prevent such complications, it might be helpful to recognize redistributed venous channels on serial angiograms during the procedure. In our case, selective placement of coils at the SPS origin could avoid this rare complication. Detachable coils such as Guglielmi detachable coils are preferable because they are retrievable and easy to advance to the desired position.

Conclusions

We report the rare complication of transvenous embolization in association with a dural CCF. Redistributed residual blood flow through the fistula can result in cerebellar hemorrhage and venous infarcts. To avoid this complication, early recognition of redistributed shunt flow and selective placement of coils at the origin of draining veins during the procedure could be helpful.

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