

# Direct Carotid-Cavernous Fistula Causing Brainstem Venous Congestion

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**Abstract:** A 39-year-old man who presented with unilateral proptosis and periocular pain rapidly developed reduced consciousness, facial numbness, dysarthria, and gait ataxia from a direct carotid-cavernous fistula (CCF) with drainage into posterior fossa veins. Brain MRI revealed abnormal signal throughout the brainstem, indicative of venous hypertension and edema. Closure of the fistula by detachable balloon eliminated the clinical and imaging abnormalities. This is the fifth reported case of brainstem complications of a direct CCF. It highlights potentially serious complications of this condition and their reversibility with prompt treatment.

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Carotid cavernous fistulas (CCFs) are acquired lesions caused by aberrant communication between the internal or external carotid arteries and the cavernous sinus (1,2). Patients classically present with the clinical triad of proptosis, chemosis, and pulsatile bruits and less commonly with ophthalmoplegia, visual loss, and facial pain. We describe a patient with a CCF who presented with signs of anterior (orbital) venous congestion and who went on to develop neurologic signs of posterior (brainstem) drainage.

## CASE REPORT

A 39-year-old man developed sudden left-sided headache and pulsatile tinnitus followed by left periocular pain, left eye proptosis, and horizontal diplopia. There was no history of trauma. Our examination disclosed visual acuities of 20/20 in the right eye and 20/150 in the left eye. Intraocular pressures were normal as were pupil size and

reactivity, visual fields, and ophthalmoscopy. He had a left abduction deficit consistent with a left sixth cranial nerve palsy. Head CT suggested a left CCF based on enlargement of the superior ophthalmic veins bilaterally and prominence of the cavernous sinuses, with the left greater than the right.

Over the next 24 hours, he developed severe left proptosis, chemosis, and ophthalmoplegia, and visual acuity in the left eye deteriorated to only light perception (Fig. 1A). He became drowsy and developed left facial numbness, marked dysarthria, and gait ataxia.

Brain MRI revealed findings consistent with a left CCF, as well as diffuse T2 signal hyperintensity in the brainstem (Fig. 2A) and enhancement of the left basis pontis (Fig. 2B).

An urgent cerebral angiogram confirmed a direct high-flow left CCF with robust filling of the cavernous sinuses and ophthalmic veins bilaterally (Fig. 3A). Abnormal early venous contrast filling of posterior fossa veins was visible (Fig. 3B). Significant tortuosity and ectasia of both carotid and vertebral arteries and intracranial vessels was evident, suggesting an underlying connective tissue abnormality.

Near complete obliteration of the fistula was achieved with a single detachable latex balloon placed directly into the fistula, maintaining patency of the internal carotid artery (Fig. 3C). Although a small residual shunt was present at the end of the procedure (Fig. 3C), the proptosis and chemosis had completely resolved at 6 months follow-up (Fig. 1C). Visual acuity had recovered to 20/25 bilaterally, and eye movements were normal. Speech and coordination had normalized. Brain MRI at 1 year follow-up demonstrated complete resolution of the abnormal signal observed in the brainstem (Fig. 2C).

## DISCUSSION

The clinical presentation of CCFs depends on the degree of shunting and the route of venous drainage (1,2). Under normal conditions the cavernous sinus receives drainage anteriorly from the superior and inferior ophthalmic veins and superiorly from the sphenoparietal sinus and cortical veins. Blood flow into the cavernous sinus drains posteriorly into the inferior and superior petrosal sinuses

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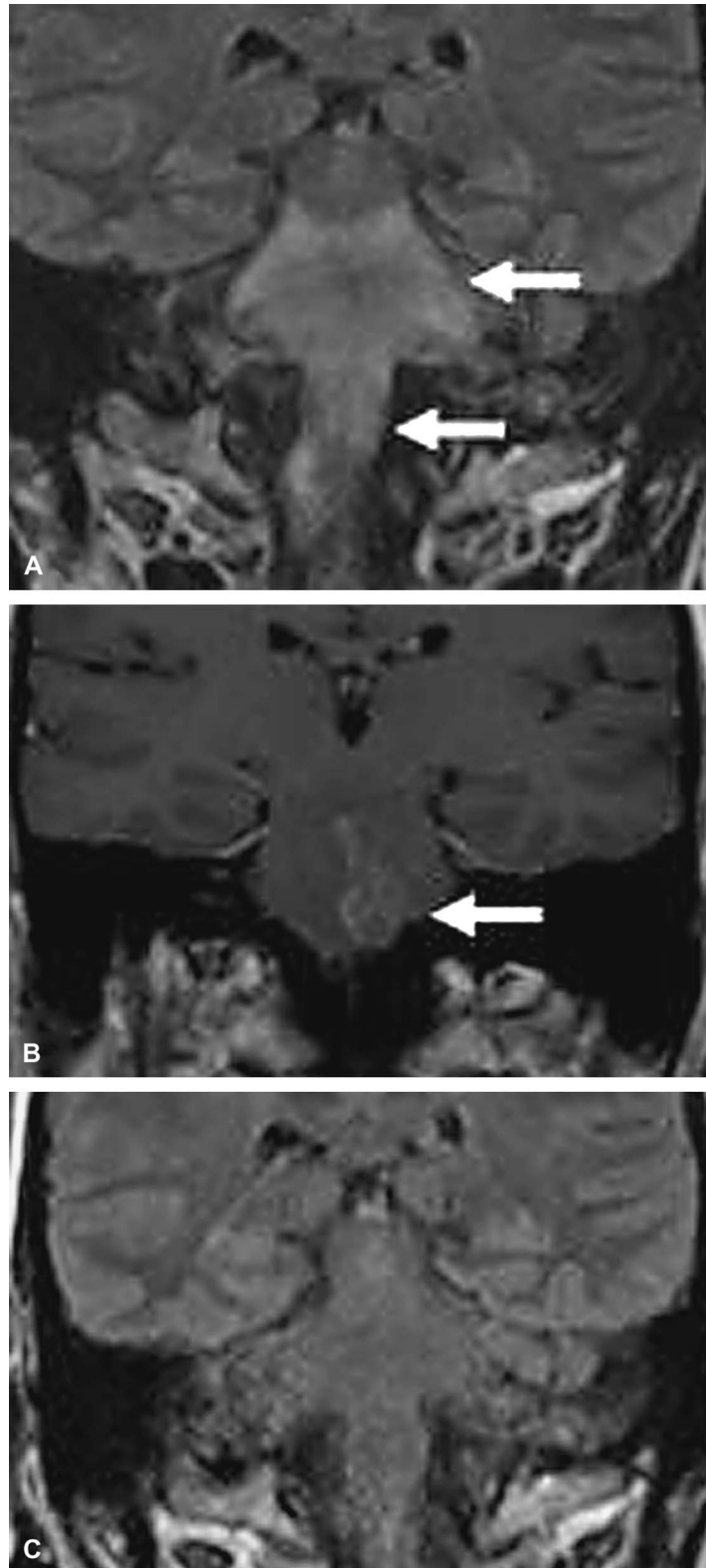
**FIG. 1. A.** Before treatment of the fistula, our patient shows left eye proptosis and chemosis and conjunctival chemosis. **B.** Three days after treatment, the proptosis and chemosis are reduced. **C.** Six months after treatment, all signs have disappeared.

and inferiorly into the pterygoid plexus. The shunt created by CCFs causes increased blood flow through normal drainage pathways and may cause reversal of flow into tributaries such as the ophthalmic veins. Rarely, reflux into cortical veins may occur, which can lead to focal hemispheric signs, seizures, venous infarction, or intracranial hemorrhage (1,2).

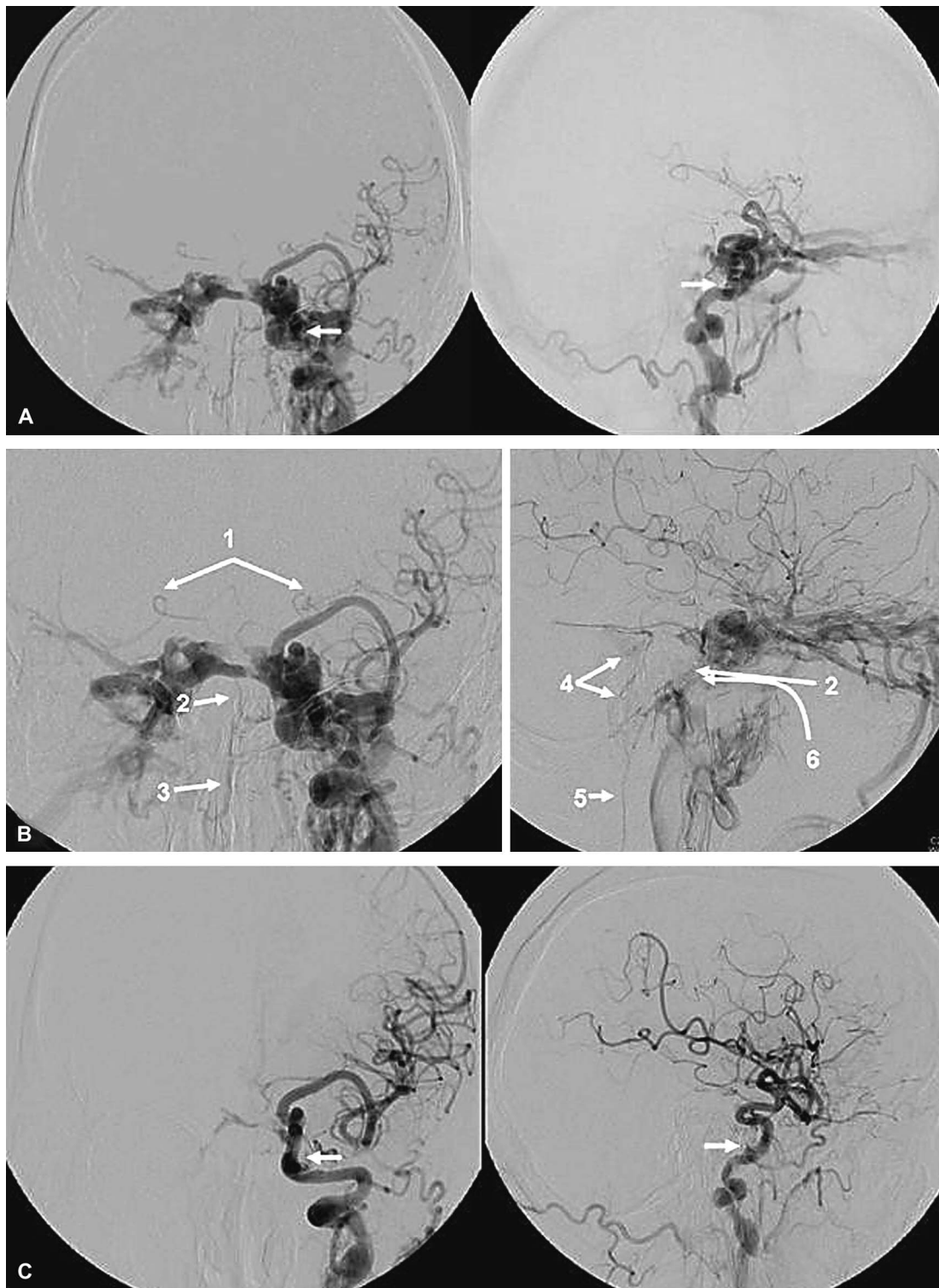
We have described a patient with venous congestion of the brainstem due to a direct CCF. Four such patients have been reported previously (3–5). There have been several additional reports of patients with indirect CCFs presenting with brainstem venous congestion (6–12). Increased shunting of blood into the petrosal system, which also drains the brainstem, is hypothesized to cause

venous hypertension and edema formation in the brainstem. Alternatively, thrombosis of dural sinuses or veins of the posterior fossa may cause increased venous congestion. Kai et al. (7) have speculated that contrast-enhancing brainstem lesions in patients with CCFs may indicate irreversible damage. Clearly, these changes can be reversible, however, as illustrated by our patient and the patient reported by Iwasaki et al. (6).

Brainstem venous congestion is a rare but potentially serious complication of CCFs. Recognition of symptoms, signs, and radiographic findings associated with this complication warrants urgent investigation and definitive treatment to achieve the best possible patient outcome.



**FIG. 2.** Brain MRI. **A.** Coronal FLAIR study demonstrates increased signal diffusely in the brain stem (*arrows*). **B.** Postcontrast T1 coronal study demonstrates enhancement of the left side of the pons (*arrow*). **C.** Coronal FLAIR study demonstrates complete resolution of the abnormal signal at 1 year follow-up.



**FIG. 3.** Left common carotid cerebral arteriogram. **A.** Anterior-posterior (left) and lateral (right) views demonstrate the left-sided direct carotid-cavernous fistula (*arrows*). **B.** Anterior-posterior (left) and lateral (right) views demonstrate abnormal early venous contrast filling of posterior fossa veins. (1. Peduncular veins, 2. Anterior pontomesencephalic vein, 3. Median anterior medullary vein, 4. Vein of cerebello-pontine fissure, 5. Median spinal vein, 6. Cerebral plexus.) **C.** Anterior-posterior (left) and lateral (right) views after balloon detachment demonstrate near complete occlusion of the fistula. The faint outline of the balloon can be discerned at the prior site of the fistula (*arrows*).

## Acknowledgment

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