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Clinical Images

A Quarterly Column

Transorbital Coil Embolization of a Carotid Cavernous Fistula

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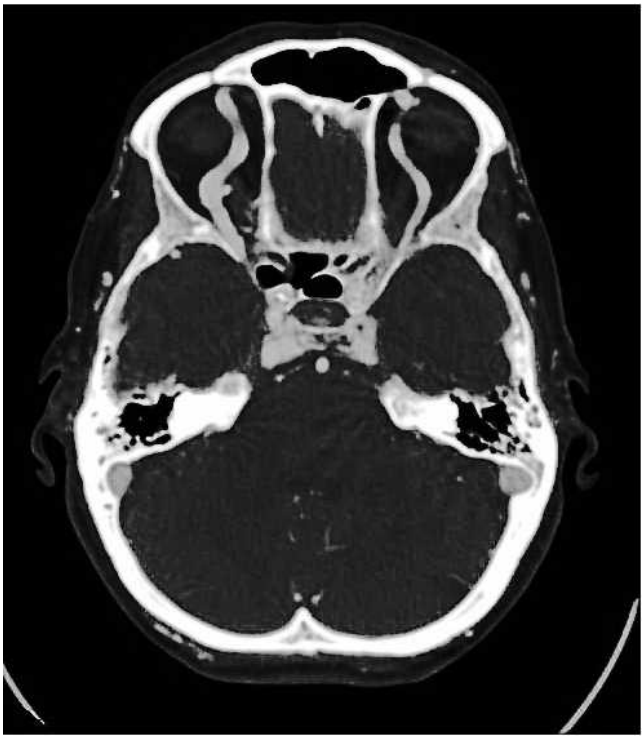

Figure 1. Computed tomography angiogram showing dilated superior ophthalmic veins bilaterally with the right greater than left. They are opacified during the arterial phase of the injection.

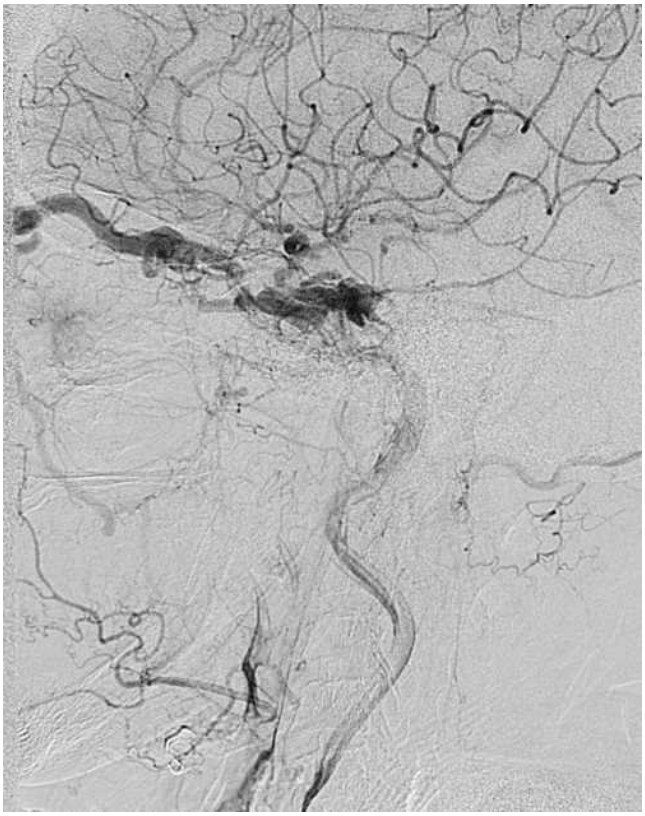

Figure 2. Lateral digital subtraction angiogram of the head shows early filling of the cavernous sinus and the dilated right superior ophthalmic vein in the arterial phase.

INTRODUCTION

A carotid cavernous fistula (CCF) is an abnormal arteriovenous connection between the internal carotid artery (ICA) or the external carotid artery (ECA) and the cavernous sinus. Symptoms range from benign ocular disturbances and cranial nerve palsies to severe or rapid vision loss and subarachnoid hemorrhage. Primary treatment options include transarterial and transvenous embolization.[1,2]

HISTORY

An 84-year-old woman presented to her local emergency room for diplopia and loss of visual acuity. Computed tomography (CT) of the head and CT angiography (CTA) showed no infarction, but the CTA revealed enlarged superior ophthalmic veins, suggesting a CCF (Figure 1). The emergency department

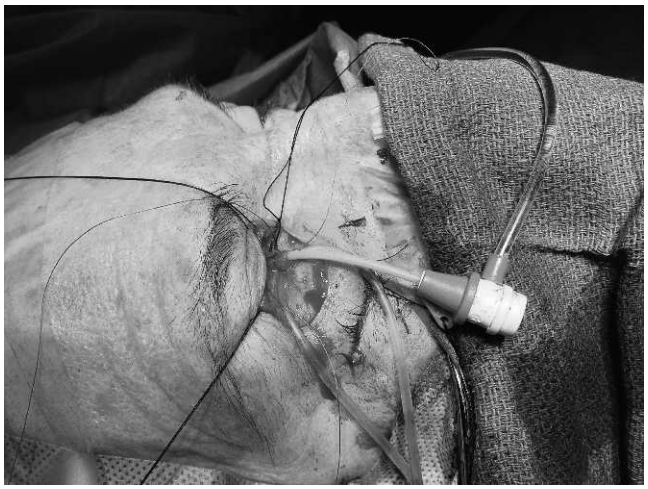

Figure 3. Gross image of the surgical exposure of the superior ophthalmic vein with a 4-French sheath inserted.

referred her to Interventional Neuroradiology for treatment options. Physicians in the oculoplastics service also saw her before the procedure.

RADIOGRAPHIC APPEARANCE AND TREATMENT

Cerebral angiography was performed via the right femoral approach, demonstrating an indirect CCF supplied by small branches of both the right and left ICAs as well as ECA branches with primary venous drainage into the right superior ophthalmic vein (Figure 2). Immediately after the diagnosis of CCF was confirmed on diagnostic transfemoral arterial angiogram, a transfemoral venous approach was used in an attempt to treat the CCF via the jugular veins and the inferior petrosal sinus (IPS). This approach was unsuccessful because the IPS was completely occluded bilaterally.

The patient returned for definitive treatment with the assistance of the ophthalmology department. In the angiography suite, the oculoplastic surgeon performed a cut down to expose the right superior ophthalmic vein.[3] The neurointerventionalist then punctured the exposed vein with a micropuncture needle to advance an 0.018-inch guidewire into the cavernous sinus (Figure 3). A 4-French sheath was placed over the wire into the vein with its tip terminating at the midportion of the dilated superior ophthalmic vein. A Penumbra PX SLIM (Alameda, CA) microcatheter was then advanced over a microwire, and coils were used to occlude the cavernous sinus starting in the posterior cavernous sinus and extending forward into the superior and inferior ophthalmic veins. The neurointerventionalist deployed 150 cm of Penumbra 0.020-inch caliber detachable coils to occlude the CCF (Figure 4).

During follow-up clinic visits, the patient's vision showed continued improvement. Her visual acuity improved on postoperative day 1, and she showed further improvement on later clinic visits. Her diplopia slowly improved, and her proptosis also decreased. She had a cranial nerve VI palsy that improved by 50% at 3 months. Her chemosis resolved with treatment as well.

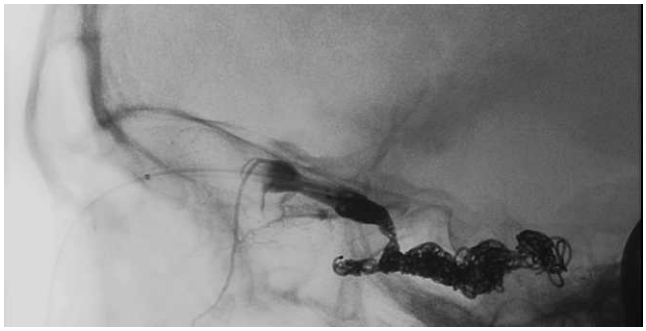

Figure 4. Posttreatment images showing bare metal coils packing the cavernous sinus, the inferior ophthalmic vein, and the superior ophthalmic vein. Contrast injected into the superior ophthalmic vein adjacent to the coils demonstrated stasis. Final common carotid angiography confirmed complete occlusion of the carotid cavernous fistula.

DISCUSSION

CCFs occur in two types: direct (high flow) and indirect (low flow). Direct CCFs are typically sequelae of traumatic ICA tears or ruptured cavernous segment ICA aneurysms. Indirect CCFs, also known as cavernous sinus dural fistulas (CSDFs), are the result of abnormal connections between the dural branches of the ICA or ECA and the cavernous sinus. The exact etiology of CSDFs is unknown but they have been associated with trauma, sinusitis, pregnancy, and cavernous sinus thrombosis.

CSDFs develop slowly over time and present with symptoms when the normal drainage pathways exiting the sinus narrow or occlude. This occlusion redirects the primary venous outflow into the ophthalmic veins, producing orbital symptoms. Venous congestion leads to bruits, pulsating exophthalmos, orbital edema, orbital erythema, headache, increased intraocular pressure, diplopia, loss of visual acuity, or cranial nerve palsies. The arterialized cavernous sinus venous flow may also be directed into the cortical veins via the sphenoparietal sinus, producing cerebral venous congestion and hemorrhage.

The most definitive treatment option is transvenous embolization of the cavernous sinus, usually using transfemoral access via the jugular and inferior

petrosal veins. If this access is not available, direct entry via the superior ophthalmic vein is another option.[4,5]

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