SUPERIOR OPHTHALMIC VEIN PUNCTURE

An alternative approach to treat complex cavernous sinus fistulae

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Complex cavernous sinus fistulae (CCF) are a technical challenge to neuro-vascular specialists. Their clinical symptoms are usually a triad of chemosis, ptosis and exophthalmia. Among the most serious symptoms are visual loss, and consequences of brain hemorrhage. The most commonly performed endovascular treatment consists in embolization through a venous approach by the inferior petrosal sinus and occlusion of the cavernous sinus with coils. Not always a conventional route is feasible, imposing alternative routes. We describe two cases of CCF treated with technical success using the superior ophthalmic vein (SOV) as a route to reach the cavernous sinus.

CASES

Case 1

A 73 year-old male presented with acute-onset right periorcular pain, diplopia, ocular injection, progressive proptosis, and periorcular swelling after an episode of trauma. His past medical history was unremarkable.

The CT scan of the brain demonstrated enlargement of the right SOV posterior to the eye globe. A cerebral angiography demonstrated a direct carotid cavernous dural sinus fistula. The right internal carotid artery supplied the fistula, but also the posterior circulation, due to the presence of a persistent trigeminal artery. The fistula drained only to the anterior compartment of the cavernous sinus, through the superior ophthalmic vein. There was no suitable alternative access to the fistula: the inferior petrosal sinus was occluded and the angular and facial veins were tortuous, such that accessing the fistula through these routes would be difficult (Fig 1). Direct balloon embolization was discarded because of the risk of occluding the persistent trigeminal artery, which was calibrous. An endovascular attempt to recanalize the inferior petrosal sinus was unsuccessful. Conventional surgery with direct occlusion of the cavernous sinus was attempted, but also not successful, as the patient remained symptomatic and control angiograms done in the post-

Fig 1. Patient 1. Left carotid angiography depicting a carotid-cavernous direct fistula, with exclusive venous drainage by a dilated SOV and a prominent persistent trigeminal artery.

Fig 2. Patient 1. Post-therapeutic angiogram demonstrating total occlusion of the fistula, with added coils inside the cavernous sinus.
operative period showed partial occlusion of the cavernous sinus and persistence of the fistula. At this point it was decided to access the cavernous sinus via direct puncture of the SOV.

The patient was placed under general anesthesia. After the arterial anatomy of the lesion was studied through a complete 4 vessels angiography, a 5 French catheter was placed in the carotid artery for observation of the shunt, angiographic control and road mapping. After surgical dissection of the right SOV, the puncture was done with 20G-Teflon intravenous type-catheter, and a microcatheter (Tracker Excel 14- Boston Scientific, Freemont, CA) was placed directly through it. The correct position of the microcatheter was confirmed with both transvenous and transarterial angiography. Embolization was performed using Guglielmi detachable platinum coils (Boston Scientific/Target, Fremont, CA) and Hidrogel 14 coils (MicroVention, Inc., Aliso Viejo, CA). With progressive packing of the fistulous pouch, transarterial angiography was repeated sequentially until no evidence of fistulous drainage was visible. The patient recovered well, remaining with a residual diplopia.

Case 2

A 65 year-old woman presented with a 15 year-history of proptosis, low visual acuity and a pulsatile supraorbital mass. There was no history of trauma. Her past medical history was remarkable for Chagas disease, with cardiomyopathy.

The CT scan of the brain demonstrated a giant SOV and exuberant venous collateral circulation in the supraorbital region and forehead. A cerebral angiography was done, which demonstrated a direct carotid cavernous dural sinus fistula. Both internal carotids were occluded at origin, and recanalization of the distal segments through anastomotic branches of the external carotid arteries was observed. The fistula was fed mainly by the posterior circulation. The direct puncture of the SOV was chosen as the via to access the cavernous sinus.

After the arterial anatomy of the lesion was studied through a complete 4 vessels angiography, a 5 French catheter was placed in the left vertebral artery for observation of the shunt and angiographic control. Road-map images of the venous phase were obtained through transarterial injections from the carotid artery catheter, to guide the venous puncture and microcatheter navigation. The puncture of a collateral enlarged supra-orbital vein was done with 20G-Teflon intravenous type-catheter, and a microcatheter (Tracker Excel 14- Boston Scientific, Freemont, CA) was placed directly through it. There was no surgical exposure of the vein, as in Case 1. The puncture was done under direct road map visualization. The correct position of the microcatheter was confirmed with both transvenous and transarterial angiography. Embolization was achieved using DCS 18 (Cook, Bloomington, IN, USA). With progressive packing of the fistulous pouch, transarterial angiography was repeated sequen-
An artery-vein shunt is a malformation that creates a direct connection between a blood vessel that carries blood away from the heart (artery) and one that returns blood to the heart (vein). This can lead to increased blood flow in the systemic circulation, which can cause symptoms depending on the location of the shunt. The surgical treatment of high-flow traumatic lesions because of its safety and efficacy. However, indirect CCFs are rarely, if ever, curable with balloon occlusion or particulate embolization, because the complex arterial supply from multiple ECA and/or cavernous ICA branches. Trans-arterial treatment may be performed, but only in cases of simple fistulae with a single pedicle, or few dilated pedicles. Injection should be done only if distal catheterization allows good flow control, enabling glue to reach the venous side. In these cases, complete cure of the fistulae may be achieved.

Transvenous embolization has become the treatment of choice for such lesions after the introduction of this method by Uflacker et al. in 1986. Generally, an approach may be performed trough the femoral vein and inferior petrosal sinus. When the inferior petrosal sinus is not opacified, an attempt to catheterize or recanalize it must be made. It may be impossible because of thrombosis or anatomic variation, such as cases of plexiform inferior or petrosal sinus or absence of connection with the jugular vein. An alternative to these cases is catheterization of the facial and angular veins. This may be technically very difficult owing to the tortuosity of the vein. In these cases, direct puncture of the SOV, percutaneously or after surgical exposure, may be a good alternative.

The complications of this approach include risk of damage to the trochlea or other orbital structures, causing retro-ocular hemorrhage and infection. Other potential complications include injury of the supra-orbital nerve or elevator palpebrae muscle. Goldberg et al. concluded that, if the SOV is not dilated, or if it is located deep in the orbit, then transorbital venous approach may not be possible. Miller et al. reported severe visual loss and neurovascular glaucoma complicating the SOV approach. Devoto et al. described acute exophthalmus occurring during treatment of a carotid-cavernous fistula via the SOV. Aihara et al. reported worsening of ocular symptoms occurring after occlusion of the cavernous sinus. Fukami et al. described late central retin vein occlusion after transvenous embolization of a DAVF.

In our cases, we chose the venous approach due to the safety and efficacy of this type of treatment. In Patient 1 there was no route except for the SOV. The angular vein was too tortuous and the inferior petrosal sinus was occluded. An attempt was made to recanalize it, but it was worthless. Even surgical direct embolization failed, and the only choice left was surgical exposure and di-

**DISCUSSION**

Intracranial dural arteriovenous fistulae are acquired arteriovenous shunts located inside the dura-mater. They account for 10–15% of all intracranial arteriovenous lesions. Their arterial supply arises from meningeal branches of the internal or external carotid arteries, and rarely from cortical branches. The clinical presentation of dural fistulae is due to arterialization of the venous system. CCF usually presents with neuro-ophtalmologic symptoms secondary to ophthalmic veins arterialization, such as chemosis (88%), exoftalmos (70%), III cranial nerve palsy (35%), VI nerve palsy (64%) and bruit (47%) in a series presented by Klisch et al. Acute-onset fistulae or high flow fistulae may also be associated with periorcular swelling. Patients with visual deterioration require early intervention to preserve their sight. Once visual loss is complete, even with treatment, optic nerve function is hardly recovered. When cortical drainage is present, there is a major risk factor to intra-parenchymal hemorrhage. The clinical presentation of our patients was periorcular pain, diplopia, ocular injection, proptosis, periorcular swelling, low visual acuity and a pulsatile mass.

The CT scans in patients with dural vascular malformations are often normal. Sometimes enlargement of a dural sinus or a dilated draining vein can be identified. In carotid cavernous fistulae an enlarged cavernous sinus is seen in half of cases, and a dilated superior ophthalmic vein can be identified on most contrast-enhanced scans. On magnetic resonance studies, the finding of dilated cortical veins without an identifiable parenchymal nidus suggests the presence of a dural shunt. Routine T1 and T2, spin echo weighted MR images can demonstrate complications of dural fistula, such as infarction and hemorrhage. Conventional MRI is less successful at direct visualization of the exact fistula site, although dural engorgement can sometimes be seen on post-contrast scans. Enlarged superior ophthalmic vein are readily identifiable. In our cases, the pre-treatment CT scan findings were enlargement of the SOV and collateral circulation.

Manual carotid-jugular or ocular compression was a classically proposed therapeutic method for CCF, not frequently used today. It may precipitate vaso-vagal attack or even ischemic stroke. Hamby and Gardner in 1933 described the first successful intracranial ligation of the carotid artery for treatment of this disease. Endovascular treatment of direct CCFs has evolved since the introduction of detachable balloons and is now the definitive treatment of high-flow traumatic lesions because of
rect puncture of the SOV. In Patient 2 both internal carotids were occluded at origin, and there was an exuberant SOV and collateral circulation, making it a good and easy approach.

There weren’t any technical difficulties or complications in our cases with the direct transvenous approach of the SOV. It can be reached either with surgical exposure or catheterization trough a collateral vein. When other routes are not available, the superior ophthalmic vein puncture is a feasible approach and carries a low risk of complications.

REFERENCES