The superior ophthalmic vein approach for the treatment of carotid-cavernous fistulas: our first experience

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Abstract: Complex cavernous sinus fistulae (CCF) are still a technical challenge to neurovascular team. The most commonly performed treatment consists in endovascular embolization of the lesion through an arterial or venous approach. Not always these conventional routes are feasible, requiring alternative routes. We report a case of a 44-year-old woman with a complex indirect (Barrow D) carotid cavernous sinus fistula treated by two interventional sessions that imposing a retrograde direct transvenous approach via the superior ophthalmic vein.

Key words: cavernous sinus fistulae, superior ophthalmic vein

Introduction

Intracranial dural arteriovenous fistulae are predominantly idiopathic arteriovenous shunts located inside the dura-mater. They account for 10–15% of all intracranial arteriovenous malformations. Carotid-cavernous fistulas (CCF) are abnormal vascular communications between the cavernous sinus and the carotid artery and/or its branches. These vascular lesions are broadly grouped into two: direct and indirect types [1, 3, 8]. The direct CCF are direct shunt between the cavernous sinus and the internal carotid artery whereas the indirect CCF are dural fistulas between the cavernous sinus and extradural branches of the internal carotid artery, the external carotid artery, or both.

Over time and development of new embolization materials, the endovascular therapy has become the treatment of choice for CCFs. The most commonly performed endovascular treatment consists in transarterial or transvenous embolization of cavernous sinus. In special cases with complex CCF a combined microsurgical and endovascular treatment via a superior ophthalmic vein approach can be necessary. A direct operative cannulation of the superior ophthalmic vein ensures a direct, fast and reasonable route to the cavernous sinus.

In this article, we describe the treatment of a type D CCF, using a combined microsurgical and endovascular treatment via a superior ophthalmic vein approach performed in two session [1, 3].
Case Presentation

A female patient of 44 years old was admitted in our clinic in August 2015. The clinical presentation of our patients was left periocular pain, episodes of diplopia, ocular injection, proptosis, periocular swelling, low visual acuity and a pulsatile mass. These symptoms occurred evolving a month. The TOF MRI of the head demonstrated enlargement of the left SOV posterior to the eye globe and vascularization around left cavernous sinus (figure 1).

A cerebral angiography showed a complex Barrow type D dural carotid cavernous sinus fistula. The fistula is fed by multiple meningeal branches of the external carotid artery on both sides and by the internal carotid artery on the left side (figure 2). The CCF drained from the cavernous sinus into the right SOV. The left inferior petrosal sinus could not be angiographically detected. The treatment required two sessions.
Operative Technique

The first session consisted in bilateral meningeal branches of the external carotid artery occlusion with glue injection. The patient was with local anesthesia. Both ECA were cauterized turn with a 6F Launcher guiding catheter (Medtronic, USA) supported by 0.035 Poseidon hydrophilic guidewire (SP Medical, Denmark). 6 meningeal branches were approached and occluded by GluBran 2 injection via 6 Marathon™ flow directed microcatheter (ev3 Neurovascular, USA). Almost complete occlusion of the supply from ECA was obtained (figure 3). External manual carotid compression was performed for a period of 3 weeks.

Because the neurologic deficits of the patient did not completely improve, the patient was treated 3 weeks later in a second session while under general anesthesia. Thus, a microsurgical approach in the catheter laboratory, with exposure of the SOV on the left side, was chosen. We performed a 2-cm skin incision in the upper eyelid and the orbital septum was opened by the craniofacial surgeon. Microsurgical preparation of the orbital fat and exposure of the dilated SOV were done.
The SOV was punctured with a 0.018-gauge cannula (Braun, Melsungen, Germany) and temporarily fixed with a 4.0 suture.

This was used as a sheath for introducing a Excelsior SL-10 (Stryker Neurovascular) with a 0.014-in TransendEx microguidewire (Boston Scientific, Fremont, CA). 14 GDCs were introduced and detached into the left cavernous sinus with completely angiographic occlusion. After the embolization, the cannula was removed and the opening in the SOV was closed by using a 7.0 suture. In the end, the skin was closed with interrupted 5.0 nylon sutures (figure 4).

Figure 4 - Intra-operative aspects of left SOV microsurgical exposure and canulation; Coil occlusion of left cavernous sinus via SOV
Discussion

Complex cavernous sinus fistulae are complex vascular lesions still considered a technical challenge to most neuro-vascular teams. According to Barrow et al [1], carotid cavernous fistulae are divided into 4 categories: - Type A CCF are high flow direct shunts between the cavernous sinus and the internal carotid artery usually caused by traumatic laceration of the internal carotid artery; - Type B CCF are fistulae between meningeal branches of the internal carotid artery and the cavernous sinus; - Type C are dural abnormal communications between meningeal branches of the external carotid artery and the cavernous sinus; - Type D are fistulae between meningeal branches of both the internal carotid artery and external carotid artery and the cavernous sinus.

Etiology

CCFs can be classified based on etiology (traumatic or spontaneous), rate of flow (high versus low flow), or the angiographic architecture (direct or indirect).

Clinical symptoms

The clinical presentations of carotid cavernous fistulae are usually represented by neuro-ophtalmologic symptoms due to indirect arterialization of the ophthalmic veins. Thus, the most common symptoms encountered are the chemosis (88%), exoftalmos (70%), III cranial nerve palsy (35%), VI nerve palsy (64%) and bruit (47%) as the Klisch et al reported in their series [1,3]. Acute-onset fistulae, high flow fistulae or those with fulminant evolution may be associated with pericocular swelling and sever visual deterioration which require emergency intervention to preserve their visual function. Once the loss of vision is complete, even with treatment, optic nerve function is difficult to recover. In case of cortical vein drainage implication the risk of intra-parenchymal hemorrhage must be taken in to consideration [3, 8].

Diagnosis

First steps for diagnostic imaging requires an initial evaluation by cranio-cerebral CT scan and/or MRI. The noncontrasts CT scan in patient with carotid cavernous fistulae are usually normal, in rare cases can highlight a dilated superior ophthalmic vein or edema due to venous congestion. On contrast-enhanced CT scan is more likely to identify a dilated superior ophthalmic vein or an enlarged cavernous sinus. Routine T1 and T2, spin echo
weighted MR images can show dilated vessels or vascular enhancement without an identifiable parenchymal nidus, venous pouches and signs of venous hypertension in high-grade lesions (as white matter hyperintensity, venous infarction or intracranial hemorrhage). Conventional CT and MRI are less successful for arterial pedicle identification and direct visualization of the exact fistula site. For any suspicions mentioned above supplementary evaluation by dynamic CTA, MRA, or DSA are indicated. Even if CTA is specifically useful for a clear diagnostic of carotid cavernous fistulae presence it is characterized by a low sensitivity for endovascular interventional planning. MRA is a reliable imaging technique for carotid cavernous fistulae detection and screening but it is limited by low resolution, restricted FOV and saturation artifacts.

Conventional angiography remains the most accurate method for detection and classification of carotid cavernous fistulae by precise location and delimitation of fistula site and superior visualization of arterial feeders and venous outflow [1, 3, 4, 8].

Treatment

Over time it has been reported different types of approaches for the management of carotid cavernous fistulae such as conservative treatment, surgery, endovascular intervention, radiosurgery or combination of them.

External manual carotid compression is a classically non-invasive method for carotid cavernous fistulae treatment. Its questionable efficiency and potential complications represented by vaso-vagal attack or even isquemic stroke made this technique not frequently used today.

Surgical ligation of the internal carotid artery or packing of the cavernous sinus was the successful surgical techniques described for the treatment of carotid cavernous fistulae [Hamby and Gardner in 1933]. However sacrifice of the ICA is performed carefully due to significant risk of cerebral infarction even after successful balloon test occlusion studies [2, 3, 5].

With the advent and development of embolization techniques and materials, endovascular treatment has become the treatment of choice in case of CCFs. Currently endovascular treatment in CCFs can be done about both arterial and venous route. The first and simple arterial endovascular approach were performed since the introduction of detachable balloons and demonstrate its efficiency for a long period of time especially in type A fistulae. Since the introduction of GDCs embolization, transarterial occlusion of the cavernous sinus through the fistulous site became the recommended treatment.

However, indirect CCFs are more complex vascular lesions requiring more advanced techniques.

Trans-arterial particulate embolization of indirect CCFs may be performed with a success rate of up to 50% of cases. In these cases delayed recanalization or change in drainage pattern were reported. This technique is specially indicated in CCFs fed by only few meningeal branches of the external carotid artery. Also, arterial embolization by glue injection can be performed especially in simple fistulae with clear arterial fed pedicles. A distal catheterization with good flow control especially at the venous side allows a complete
treatment.

With the introduction of transvenous embolization through the femoral vein and inferior petrosal sinus by Uflacker et al. in 1986 these technique become the treatment of choice for such lesions [1, 3, 6, 8]. So, transvenous embolizations by occlusion of the cavernous sinus with coils become treatment of choice for Barrow type B–D indirect CCFs. In highly complex fistulas combinations of endovascular treatment by transarterial and transvenous approach and/or additional stereotactic radiosurgery were reported.

In case of thrombosis or anatomic particularities, such as tortuosity, plexiform inferior petrosal sinus or absence of connection with the jugular vein, the transfemoral venous approach is compromised. An alternative technique in such cases was represented by direct superior ophthalmic vein catheterization after its surgical exposure. This approach offers a reasonable and safe route to cavernous sinus occlusion with coils or onyx.

Complication

The most known potential surgical complications of the SOV approach are hemorrhage, damage to the trochlea or other orbital structures, and infections. The technical complication of this approach is the over-packing of the cavernous sinus resulting in nerve palsies, dural dissections, or penetrations. Retrograde SOV thrombosis was also reported.

Conclusions

Our case shows that a complex CCF requires interdisciplinary treatment. When the classical routes are not available, the direct surgical superior ophthalmic vein catheterization is a feasible approach and carries a low risk of complications.

References