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Endovascular treatment of a cavernous sinus dural arteriovenous fistula by transvenous embolisation through the superior ophthalmic vein via cannulation of a frontal vein

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Abstract We describe a new approach for transvenous embolisation of cavernous sinus dural arteriovenous fistulae through the superior ophthalmic vein (SOV), i.e., via percutaneous cannulation of a frontal vein. Modern neurointerventional angiographic materials make it possible to reach the SOV in this way without puncturing it in the orbit or a surgical exposure. Orbital phlebography should still be in the repertoire of interventional neuroradiology units in large centres.

Keywords Dural arteriovenous fistula · Cavernous sinus · Transvenous embolisation · Frontal vein · Orbital phlebography

Introduction

Transvenous embolisation is the best treatment for patients with cavernous sinus dural arteriovenous fistulae (DAVF) requiring active treatment [1, 2, 3, 4]. Transvenous embolisation is generally performed via puncture of the femoral or jugular vein and catheterisation of the inferior petrosal sinus (IPS), the safest route to the cavernous sinus. When the IPS cannot be catheterised, other routes may be employed: via the contralateral pterygoid plexus [5], superior petrosal sinus [6, 7], superior ophthalmic vein (SOV) by

catheterisation of facial or angular veins [1, 2, 3, 4, 8], or via cortical draining vein [9]. When none of these procedures is possible, the fistula may be reached via direct puncture of the facial vein at the base of the mandible [10], of the SOV in the anterior [11] or posterior [12] orbit, or cavernous sinus [13], or surgical exposure of the angular vein [14], or SOV [15, 16, 17].

We describe treatment of a cavernous sinus DAVF by access to the SOV through the percutaneous puncture of a frontal vein, using the technique of orbital phlebography [18, 19, 20].

Case report

A 67-year-old white woman presented with an 11-year history of mild bilateral Graves' ophthalmopathy, treated for about 10 years with methimazole. Recent radioiodine treatment resulted in progressive improvement of her hyperthyroidism but worsening of the ophthalmopathy. On admission, she had bilateral periorbital oedema, lagophthalmos, conjunctival injection, chemosis and proptosis, and complained of eye discomfort, epiphora and diplopia. There was no history of cranial trauma. Laboratory tests showed subclinical hyperthyroidism. The patient was then given steroids for 3 months, which resulted in improvement in the left eye, but had only mild effects on the right.

Persistent right proptosis, severe conjunctival injection, and chemosis 5 months later prompted further investigation. CT of the orbits showed bilateral thyroid ophthalmopathy, and enlargement of the right SOV. Four-vessel angiography was performed for definitive diagnosis and therapeutic planning. Right common carotid arteriography showed arteriovenous shunts in the dura mater on the right of the clivus, supplied mainly by meningeal branches of the right ascending pharyngeal artery, and draining exclusively into the right SOV (Fig. 1a, b). The shunts were also shown with injection of the left common carotid artery, via subtle clival branches of the ipsilateral external carotid artery. The retrograde flow into the SOV opacified the right facial and both frontal and angular veins: the right facial vein was narrow in its midportion and its lower end could not be seen (Fig. 1b). The petrosal sinuses were not seen. There were no signs of a left cavernous sinus DAVF.

Since the patient's symptoms did not improve after 2 months of conservative treatment, including right medial canthus compression, endovascular treatment was suggested, to which the patient gave informed consent. Treatment was carried out under general anaesthesia, with tracheal intubation. A 5F catheter was left in the right common carotid artery throughout the procedure for assessment of flow in the fistula. A 4F guiding catheter was inserted in the right internal jugular vein, where a 0.010" guidewire with hydrophilic coating and a hydrophilic-coated 2.4–1.8F microcatheter were advanced. However, catheterisation of the right IPS was not possible. Navigation into the right facial vein was attempted, but its narrow portion did not allow the microcatheter to be advanced.

A further period of conservative treatment was then considered, but as the patient did not improve, a last attempt was made, using percutaneous cannulation of the right frontal vein, as in orbital phlebography, given the pattern of retrograde flow. The right frontal vein was distended by maximal posterior flexion of the head and compression of the jugular veins in the neck, then cannulated using an 18G needle-cannula, which was inserted only a few millimetres, allowing introduction of the hydrophilic-coated 0.010" guidewire and microcatheter; these were advanced to the inferior tributary of the right SOV using "road-mapping". Through the SOV, the microcatheter finally reached the fistula (Fig. 1c, d) where Guglielmi detachable coils were deployed, as they were in the posterior cavernous sinus. Dense coil packing resulted in progressively less filling of the right SOV with injection into the right common carotid artery (Fig. 1e, f). There were no complications, and 6 days later angiography confirmed a stable result. Clinical follow-up at 15 months showed clear improvement of the ocular symptoms, with no neurological complications.

Discussion

The issue of the occurrence of a cavernous sinus DAVF in a woman with thyroid ophthalmopathy is beyond the scope of this report, and will be addressed elsewhere. Cavernous sinus DAVFs presenting with

ocular symptoms have a more benign natural history than DAVFs at other sites. Their course is generally indolent, with low morbidity, and a high rate of spontaneous resolution, especially after slight changes in the haemodynamics [1, 2, 3, 16, 21]. Patients are usually managed conservatively by manual carotid-jugular, ocular or medial canthus compression, or antiaggregant therapy [1, 2, 3]. However, conservative treatment may be unsuccessful, and the symptoms and signs may progress to decreasing vision, glaucoma, severe exophthalmos, persistent ophthalmoplegia, intolerable diplopia or bruit, or unacceptable cosmetic disfigurement. These patients require more aggressive therapy, whose aim is complete cure of the fistula, occluding the fistula itself, and part of its venous side. An arterialised cavernous sinus is generally excluded haemodynamically, and its occlusion is assumed not to be dangerous [1]. The fistula may be occluded by surgery, irradiation, endovascular treatment or a combination of these. Endovascular treatment has increasingly become the therapy of choice but it is not devoid of risk, with a reported morbidity of 4%, and possible complications including development of another DAVF, brain abscess, and glaucoma with visual loss [16, 21]. Endovascular treatment may be transarterial and/or transvenous and various materials including particles, glue and detachable metallic coils; the treatment plan varies with the origin of the feeding vessels and type of venous drainage. Transarterial embolisation with polyvinyl alcohol 150–250 μ particles has been proposed as the initial therapy when the fistula is fed only by meningeal branches of external carotid artery: this approach may result in cure of up to half of cases, but delayed recanalisation has been observed [1, 2]. Transarterial embolisation with glue may be performed only for simple fistulae with one or a few dilated feeding vessels, and only if the venous side can be reached by the glue, since proximal embolisation invariably results in recanalisation [1, 21]. Complications include reflux into the carotid siphon, or branches supplying cranial nerves III–VII, and IX–XII palsies, and redirection of flow into remaining vessels pathways causing aggravation of the clinical presentation. The transarterial approach alone is often inadequate because: external carotid artery supply most commonly includes innumerable plexiform branches which cannot be satisfactorily reached; dangerous anastomoses between external and internal carotid or vertebral arteries often exist even if not evident on pretreatment angiograms; and internal carotid artery supply is frequent, and cannot usually be embolised safely [22].

Transvenous embolisation with coils has proved safer and more effective for occlusion of the venous side of the fistula, and transarterial embolisation is now reserved as an adjunct to transvenous embolisation. The routes employed have been discussed above. Advances in guidewire and microcatheter technology have markedly

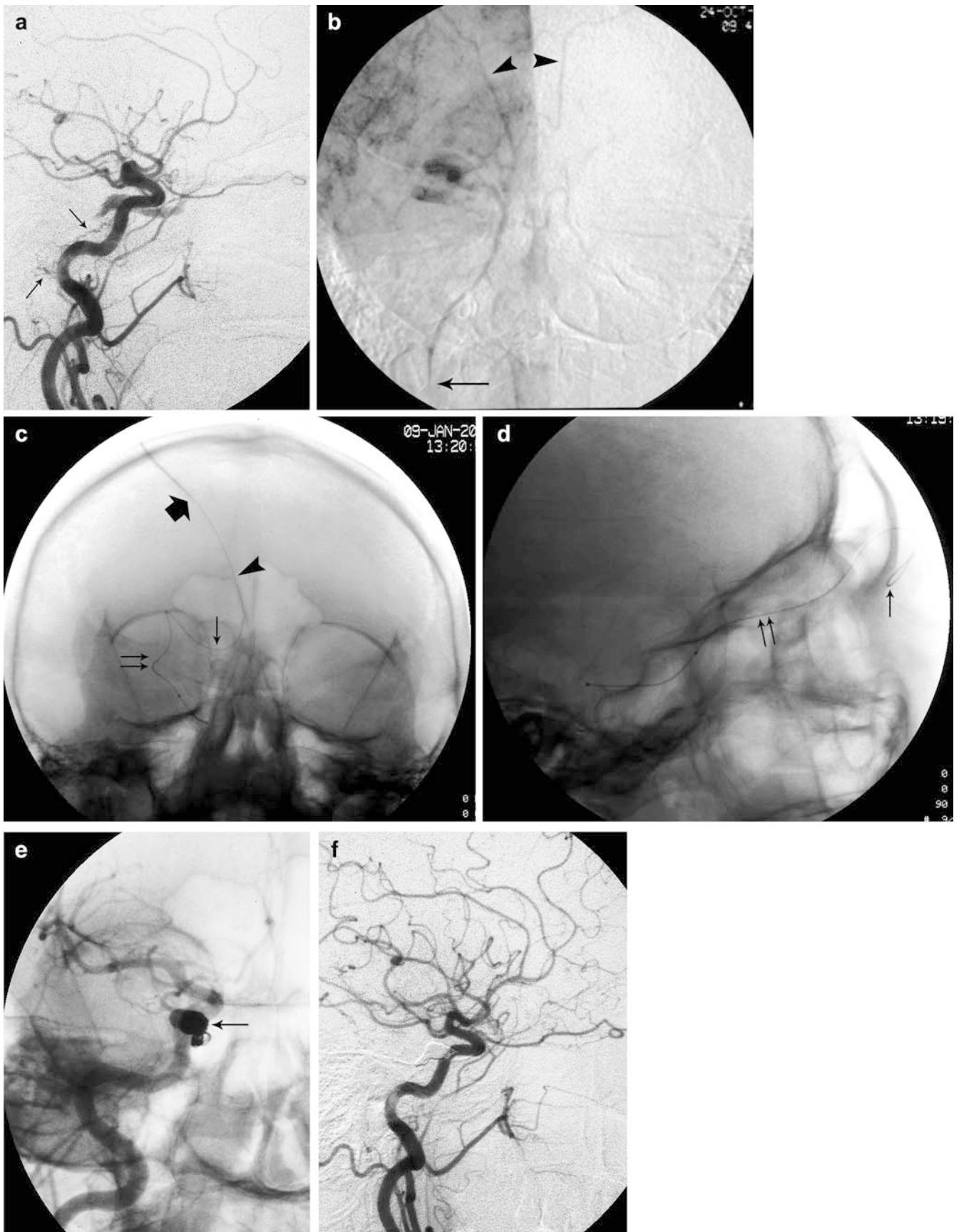




Fig. 1a–f A 67-year-old woman with thyroid ophthalmopathy, presenting with severe congestion of the right orbit. **a** Right common carotid arteriogram, early phase, lateral view, shows clival meningeal branches (*arrows*) of the right ascending pharyngeal artery feeding a dural caroticocavernous fistula, draining to a large right superior ophthalmic vein. **b** Anteroposterior view, late phase, shows retrograde flow via the right superior ophthalmic vein filling both frontal veins (*arrowheads*), and the angular, and right facial veins: the middle portion of the right facial vein is narrow (*arrow*), with no evidence of its lower end. **c, d** Anteroposterior and lateral views during endovascular treatment show successful navigation of the microcatheter via the percutaneous cannula (whose distal end is indicated by the *large arrow*) to the site of the fistula through the right frontal vein (*arrowhead*), inferior root of superior ophthalmic vein (*small arrow*), and superior ophthalmic vein (*double small arrows*). **e, f** Right common carotid artery angiograms, unsubtracted anteroposterior and subtracted lateral views, demonstrate dense packing of coils (*arrow*), and complete occlusion of the fistula

reduced the risk of IPS rupture and subarachnoid haemorrhage, which justifies an attempt to catheterise the sinus even when it is not opacified [1, 4]. However, the IPS may be inaccessible due to its plexiform nature, thrombosis, flow reversal because of shunts or lack of connections with the internal jugular vein: in these cases transvenous navigation to the fistula site may be attempted through the routes listed above. When these other procedures are unsuccessful or impossible due to thrombosis or the anatomical situation, as in our patient, cavernous sinus may be reached in a more invasive way [10, 11, 12, 13, 14, 15, 16, 17]. Direct access to the SOV has been recommended as the route of choice for treatment of fistulae with predominantly anterior venous drainage through the vein [15]. However, direct puncture or surgical exposure of the SOV carry the risk of a number of complications including orbital haematoma or infection and damage to anterior orbital structures such as the trochlea, levator palpebrae superioris and supraorbital nerves. Furthermore, catheterising the SOV may be difficult and risky due to possible abrupt angulation and narrowing as it exits the superior orbital fissure, or if it is thrombosed [5]. Injuries to the SOV may result in orbital haemorrhage, loss of vision and/or redirection of venous drainage to intracranial pathways [12, 16].

In our patient, modern high-quality interventional neuroradiological equipment allowed access to the cavernous sinus via a frontal vein, and the inferior tributary of the SOV, with minimal trauma to frontal and orbital veins. To our knowledge, there is no previous report of a cavernous sinus DAVF treated in this way. Its advantages are the obviation of direct puncture to or surgical exposure of the SOV, and that most of the catheterisation is extracranial; the IPS has a long intracranial portion. We therefore think that, when feasible, this route may be as safe as or safer than the IPS route. Its disadvantages lie in the difficulties of frontal vein

cannulation, and navigation through the SOV and its tributaries.

On both sides of the forehead, a single frontal vein usually forms from a converging frontal venous network which communicates with frontal tributaries of the superficial temporal vein, and descends near the midline, parallel with its fellow. Occasionally, frontal veins unite in a single trunk which divides into two at the root of the nose. The medial canthus is the junction point for frontal and supraorbital vessels from the forehead and the angular vein from below through the internal frontal vein, and marks the origin of the SOV. At the medial canthus the main frontal vein is generally continuous with both the superior tributary of the SOV and the internal frontal vein. The superior tributary of the SOV, accompanied by the supraorbital nerve and artery passes through the supraorbital notch, penetrates the orbital septum above the trochlea, and then courses obliquely posteriorly and slightly superiorly, between the orbital roof above and the tendon of levator palpebrae superioris and the lateral portion of superior oblique muscle below. About 4–5 mm behind the trochlea, it is joined by the inferior tributary of the SOV, one of the two cranial continuations of the angular vein, which penetrates the orbital septum below the trochlea, coursing slightly upwards, laterally, and posteriorly to join the superior tributary. The internal frontal vein is in direct continuation with the angular vein, which has a subcutaneous course down the side of the nose lateral to the angular artery, and crosses the nasal edge of the medial palpebral ligament near the medial canthus. The angular vein has three main tributaries superiorly, including a medial or prenasal arch, the inferior tributary of the SOV and the internal frontal vein, and inferiorly is continuous with the facial vein below its junction with the superior labial vein [19].

A frontal vein is the simplest and safest route for orbital phlebography, a technique almost abandoned over the last two decades due to the development of CT, ultrasonography and MRI [18, 20]. Many methods may be used to produce venous distension, including lowering the head, possibly with compression of the jugular veins [18], or placing one or more elastic bands over the face [20]. A distended frontal vein is then punctured and cannulated, and a slow infusion may be maintained while patient is positioned for radiography. The patient is also instructed to compress the angular veins during injection of contrast medium, to avoid preferential drainage to external jugular vein tributaries and no filling of orbital veins. If the venous flow is preferentially to one orbit, compression of the veins along its medial superior quadrant may be necessary to force contrast medium to the opposite side. Compression of this area is also necessary when filling of only one orbit is desired for the lateral projection.

Although further evaluation of the feasibility, safety and possible indications of the technique we describe, modern neurointerventional angiographic materials enable this approach to be attempted in selected cases. The technique of orbital phlebography via frontal vein

cannulation should still be within the capability of the interventional neuroradiological units of large centres.

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