Treatment of dural carotid-cavernous fistulas via the superior ophthalmic vein.

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Treatment of Dural Carotid-Cavernous Fistulas via the Superior Ophthalmic Vein

Symptomatic patients with dural carotid-cavernous fistulas often require treatment. Traditional therapies, which often are not completely successful, include manual common carotid artery compression and embolization via transarterial routes. This report describes four symptomatic patients with spontaneous dural carotid-cavernous fistulas who were treated unsuccessfully with transarterial embolotherapy and subsequently treated successfully by having a detachable balloon introduced into the cavernous sinus via the superior ophthalmic vein, which was surgically exposed. The fistulas resolved without complications.

Treatment of dural carotid-cavernous fistulas by means of the transvenous approach via the superior ophthalmic vein may be of benefit in selected patients.


Dural carotid-cavernous fistulas (CCFs) are characterized by slow flow, low morbidity, and a high rate of spontaneous cures or disappearance of symptoms [1-7]. Therefore, initial therapy should be as noninvasive as possible. Some investigators recommend manual common carotid artery compression [8, 9]. Infrequently, more aggressive therapy is indicated when the clinical course is progressive or unacceptable. Some use irradiation [10, 11], others have described a transarterial or transvenous approach. The transarterial route usually involves occluding portions of the external carotid arterial system [6, 12, 13]. The transvenous approach involves alteration of flow within the cavernous sinus or its branches [1, 6, 14-20]. Varying surgical and nonsurgical techniques are required for each procedure.

We describe four patients in whom surgical exposure of the superior ophthalmic vein allowed passage of a detachable balloon into the cavernous sinus, resulting in successful treatment of a dural CCF. We are unaware of any previous reports describing the treatment of dural CCFs by placing detachable balloons in the cavernous sinus via a surgically exposed superior ophthalmic vein.

Materials and Methods

Four consecutive patients with a dural CCF were treated via the superior ophthalmic vein (Table 1). Clinical findings were present from 3 weeks to 15 years before this treatment. Our patients with symptomatic dural CCF first undergo attempted ablation via embolization of external carotid artery (ECA) feeders. If this is unsuccessful then transvenous therapy is considered if they have progressive symptoms, decreasing vision, glaucoma, persistent ophthalmoplegia, or an unacceptable physical appearance. An attempt is initially made to reach the cavernous sinus via the inferior petrosal sinus. If this is unsuccessful the superior ophthalmic vein approach is used.

Prior to each surgical procedure an unsuccessful attempt was made to pass a catheter into the cavernous sinus via the inferior petrosal sinus using a transfemoral approach. This was done in an angiographic suite. On a subsequent day the superior ophthalmic vein was
TABLE 1: Summary of Patient Data

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Clinical Presentation</th>
<th>Prior Treatment</th>
<th>Angiographic Findings</th>
<th>Balloon Type</th>
<th>Follow-up Angiography</th>
<th>Follow-up Clinical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>43</td>
<td>Progressive proptosis, chemosis, conjunctival injection, bruised left eye for 15 years; sudden increased proptosis, decreased vision, lagophthalmos, relative afferent pupillary defect prior to venous approach</td>
<td>Embolization of left internal maxillary feeders with Gelfoam at 10 years; embolization of left internal maxillary feeders with polyvinyl alcohol at 15 years prior to transvenous approach</td>
<td>Left internal maxillary feeder, left meningohypophyseal feeder</td>
<td>#9 Debrun</td>
<td>No fistula at 2 days</td>
<td>Complete cure at 3 and 6 months (minimal residual proptosis)</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>Proptosis, chemosis, conjunctival injection, bruised left eye for 6 weeks, increased intraocular pressure, optic disk hyperemia, retinal vein dilated, and peripheral retinal hemorrhages of left eye for 8 months</td>
<td>Embolization of bilateral internal maxillary and ascending pharyngeal with polyvinyl alcohol at 2 years and right internal maxillary artery at 1 year with IBCA glue</td>
<td>Left inferolateral trunk, left internal maxillary and ascending pharyngeal feeders</td>
<td>#9 Debrun</td>
<td>No fistula postoperatively or at 8 months</td>
<td>Complete cure at 8 months</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>Proptosis, conjunctival injection, pain, diplopia of left eye for 8 months, 6th nerve palsy</td>
<td>Embolization of left internal maxillary and ascending pharyngeal with polyvinyl alcohol 8 months before venous approach; embolization of right internal maxillary with IBCA glue 1 month before transvenous approach</td>
<td>Right internal carotid feeder, right internal maxillary feeder, left internal carotid feeder, left external carotid feeder</td>
<td>#16 Debrun</td>
<td>No fistula at 4 days</td>
<td>Complete resolution over several months</td>
</tr>
<tr>
<td>4</td>
<td>67</td>
<td>Bilateral ophthalmoplegia, ptosis, conjunctival injection for 3 weeks</td>
<td>Embolization of clival branch of ascending pharyngeal with NBCA glue 1 week prior to venous approach</td>
<td>Right internal carotid feeder filling right CS and SOV; left internal carotid feeder filling left and right CS and left SOV</td>
<td>2 #16 Debrun</td>
<td>One week after treatment, right internal carotid injection fills right CS and not SOV; left internal carotid injection fills left and right CS but neither SOV</td>
<td>Complete resolution by 2 months</td>
</tr>
</tbody>
</table>

Note.—CS = cavernous sinus, SOV = superior ophthalmic vein.

Discussion

Carotid-cavernous fistulas are best classified as direct (type A) or dural types depending on the character of the arteriovenous shunt that is present, and they may be either spontaneous or posttraumatic [1, 21]. Dural CCFs are further defined by the origin of their multiple small dural arterial connections from the internal carotid artery (ICA) (type B), the ECA (type C), or from both (type D).

Most direct CCFs result from a posttraumatic connection between the ICA and the cavernous sinus through a single tear in the arterial wall resulting in a high-flow state. In a few cases a direct CCF may occur spontaneously, such as after rupture of an aneurysm of the cavernous ICA. Dural CCFs usually occur spontaneously as a result of multiple small dural arterial connections between meningeal branches of the ICA siphon, the ECA, or usually a combination of both (Figs. 1 and 2) and have relatively slow flow.

Dural CCFs usually have an indolent course, low morbidity, and a high rate of spontaneous resolution. Complete cures have been reported spontaneously, after manual carotid compression [8, 9], angiography, or airplane flights. This suggests that in many cases a slight change in the hemodynamics of the fistula is enough to initiate closure. This may...
explain why embolization of only ECA feeders in a patient with a dural CCF may result in a complete cure.

Therapy is indicated in any patient with a dural CCF that causes progressive symptoms, decreasing vision, glaucoma, persistent ophthalmoplegia, or an unacceptable physical appearance. An initial trial period of waiting or manual common carotid artery occlusion is probably warranted when there is no threat to vision. Each of our patients went through an adequate waiting period lasting 3 weeks to 15 years before treatment. Since dural CCFs are chronic disorders, patients will almost always have an adequate trial period and there is usually no urgency for treatment.

Although manual common carotid artery compression has been recommended, we do not believe it is of significant value. The fistulas of patients who appear to benefit from manual common carotid artery compression probably would have resolved spontaneously. Nevertheless, if the carotid bifurcation is free of disease then a trial period of common carotid artery compression (with or without anticoagulants) probably cannot be faulted.

If conservative therapy is unsuccessful or if there is an immediate threat to vision, more aggressive treatment is necessary. A number of approaches have been described for treatment of these patients. Generally, they can be divided into transarterial and transvenous approaches. Transarterial procedures usually involve endovascular occlusion of ECA branches or feeders. This is often time-consuming, has potential complications, and a variable rate of success.

Dural CCFs have also been treated via a transvenous approach. There have been descriptions of successful treatment of dural CCFs via catheterization of the cavernous sinus or its branches and embolization or induction of thrombosis with a variety of materials. Fibrin glue [14], wire [14], coils [15, 16], sclerosing agent [15], needles [17], bone wax [18], oxidized cellulose [16, 18], detachable balloons [18, 19], and cyanoacrylate glue [16] have been used with variable success. We achieved complete clinical cures with detachable balloons in four patients. It is important that the balloons be filled with contrast material rather than a polymerizing substance. Experience with latex balloons shows that they deflate over a period of several weeks. This is time enough, however, for the fistula to close and thrombose. If a polymerizing substance is used it increases the risks of permanent compression of the fifth, sixth, and third cranial nerves.

On the basis of our recent experience and that of others [14–19] we believe that the transvenous approach should
also be considered in the treatment of dural CCFs. If the inferior petrosal sinus is patent, trial embolotherapy through this vessel may be attempted. If this is not successful and there is a dilated superior ophthalmic vein, access to the cavernous sinus may be obtained via surgical exposure of this vessel. Coils may be used when a balloon cannot be introduced.

We have shown that transvenous embolization of the cavernous sinus after surgical exposure of the superior ophthalmic vein can be a safe and successful means of
treatment of dural CCFs. By their nature, dural CCFs are chronic disorders, therefore the venous branches should be arterialized by the time the patient presents for treatment. Nevertheless, a number of potential complications exist, including accidental rupture of the superior ophthalmic vein during surgical exposure with subsequent severe hemorrhage and loss of vision; infection; damage to anterior orbital structures, such as the trochlea; cerebral hemorrhage; and worsening of ocular findings after diversion of venous flow. We believe that when the exposure and embolization is performed by an experienced team made up of a neurosurgeon, ophthalmologist, and neuroradiologist, successful completion of the procedure is maximized and complications are minimized.

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REFERENCES

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