Endovascular treatment of carotid cavernous fistulae (CCF). Direct venous puncture using road mapping in dural CCF

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Abstract  Introduction: Endovascular treatment offers different options to treat CCF by less invasive approach avoiding morbidity and residual fistulas. The choice depends on the anatomy of the fistula and operator/Institutional preferences.
Objective: Describe options in endovascular treatment of Barrow Type A and Type D Carotid Cavernous fistulas (CCF).
Patients and methods: We report 10 cases of carotid cavernous fistula (CCF) that received endovascular treatment using coils and liquid embolic materials. Seven cases Barrow Type A, done through an arterial approach and three cases Barrow Type D, done through venous approach. The cavernous sinus in Type D cases was approached via the inferior petrosal sinus in one case and the other two cases by direct puncture of the facial and supra-orbital veins using road mapping. Using road mapping in venous approach to treat CCF is not reported in the literature before.
Results: Successful obliteration of the fistula was achieved in all cases with transient VI nerve palsy in one case and post procedure ophthalmic vein thrombosis in another.
Conclusion: With the observed favorable outcomes and lack of significant procedural morbidity, direct puncture of the facial and supra-orbital veins using road mapping to reach the cavernous sinus, might be an addition in the armamentarium available for endovascular treatment of CCF.

1. Introduction

CCFs can be classified based on etiology (traumatic or spontaneous), rate of flow (high versus low flow), or the angiographic architecture (direct or indirect). The most commonly used classification scheme divides the CCFs into four types, depending on the arterial supply. Direct fistulas (type A) are direct communications between the internal carotid artery (ICA) and the cavernous sinus, usually associated with high flow rates. Indirect fistulas (types B, C, and D) are dural arteriovenous fistulas (DAVFs) fed by the meningeal arteries of the ICA (Type B).
Early treatment of CCF consisted of trapping of the fistula by ligation of the cervical and intracranial ICA as described in the 1930s. Alternatively, carotid sacrifice was performed via embolization using different materials delivered by a direct carotid exposure. Although surgical trapping with ligation of the ICA is still considered an effective treatment for direct CCFs, sacrifice of the ICA is performed sparingly because of a significant risk of cerebral infarction even after successful balloon test occlusion studies [2].

Endovascular treatment now can offer similar results with a less invasive approach, avoiding the difficult surgical drilling needed for exposure of the clinoideal segment of the ICA, the associated morbidity from cranial nerves and residual fistulas. The exact method chosen in each case depends on the anatomy of the fistula and operator/Institutional preferences [2].

The most common symptoms of cavernous sinus CCF are ocular symptoms like exophthalmos or proptosis caused by anterior venous drainage [1–5]. Aggressive neurological symptoms such as intracranial hemorrhage are extremely rare because of the benign venous drainage pattern but can occur in association with dangerous venous drainage patterns, including (a) cortical venous reflux without other venous drainages (hemorrhagic infarction), (b) dominant deep venous drainage (hemorrhage, edema) and (c) thrombosis of the central retinal vein(blindness) [6,7]. Spontaneous regression of cavernous sinus dural AVFs is well recognized, being observed in 10%–50% of cases [2,8]. In most cases, a “wait and see” strategy may be appropriate [3,9–11]. Complications of CCF can be understood based on the communications of the cavernous sinus shown in (Fig. 1). In cases of rapidly deteriorating ocular symptoms and/or cortical venous drainage, urgent interventional therapy is indicated [9].

2. Patients and methods

We report the successful occlusion of ten CCF (seven cases Barrow type A and three case type D) using coils and liquid embolic materials, six males and four females with a mean average age of 38 years. Using a Siemens Monoplane Artis Zee and Philips Biplane Allura 20/10 machines, all the procedures were done under general anesthesia.

All patients received 5000 IU Heparin after the femoral puncture and were maintained after the 1st hour on 3000 IU Heparin/hour during the procedure. All patients were tested for permanent internal carotid artery sacrifice angiographically by venous phase timing during balloon test occlusion [12]. At the end of the procedure half the amount of Heparin given in the last 2 h was antagonized by IV protamine sulfate and all patients were kept on S.C. low molecular antiagouulant (Clexane 40 mg) /12 h for 4–7 days to prevent thrombosis in the dilated venous drainage of the fistula.

All patients’ data, type of CCF, treatment option used, occlusion test result and post-procedure outcome are listed in Table 1.

In all patients with direct CCF there was a clear history of trauma. The patients on the following days started complaining of gradually progressive proptosis of the affected eye with continuous tinnitus and headache in some cases (depending on the flow across the fistula). CTA done showed the dilated tortuous superior ophthalmic vein SOV consistent with the diagnosis of cavernous sinus fistula. The three cases with dural CCF were two females and one male. All had a long standing history (1 year up to 14 years) of proptosis and injection of the affected eye with no history of trauma.

The arterial route was used in the treatment of traumatic CCF cases (patients 2–7 and 10) by placing a 6F sheath in the femoral artery through which a 6F guiding catheter is advanced till the origin of the internal carotid artery (ICA). In dural CCF cases (patients 1, 8 and 9) the venous approach was used as a route for treatment of these cases and the arterial axis was used to do control angiograms. In patients (1 and 9) a direct percutaneous puncture of the facial and supra-orbital veins respectively using road-mapping was done to access the cavernous sinus. To our knowledge this technique is not reported before in the literature (Fig. 4). In the third case of Dural CCF (patient 8) the cavernous sinus was reached through femoral vein, the contra-lateral inferior petrosal vein through the midline intercavernous sinus to the diseased side.

3. Results

In patients with direct CCF cerebral angiogram revealed direct fistula (Barrow A) between the cavernous carotid and the cavernous sinus with variable degrees of failure to opacify the distal supraclinoid and intracranial carotid due to steel effect of the fistula. The fistula is drained through the SOV mainly, and in some cases also to the ipsilateral pterygoid plexus and to the opposite cavernous sinus.

In patients with dural CCF angiogram of the three cases showed that the fistula is supplied from both ICA and ECA (Barrow D).
The traumatic CCF was crossed to the cavernous sinus using a microcatheter e.g. (Echelon 10 - Microtherapeutics-EV3). In case 1 (Fig. 2) test occlusion was negative and the ICA had to be spared. A $4 \times 10$ mm balloon (HyperGlide - Microtherapeutics- EV3) was placed in the cavernous carotid to be intermittently inflated during embolization to prevent the reflux of the embolizing material in the ICA. The sinus was packed with electrically detachable platinum coils (GDC-10 & GDC-18 Boston/Target, Fremont, CA and Axi- um- Microtherapeutics- EV3) to occlude the sinus (cases 5 and 6). Adding liquid embolic material to complete sinus occlusion was done using Onyx 18 Microtherapeutics- EV3 (in cases 2–4) or $N$-Butyl cyanoacrylate, Braun, Aesculap AG, Tuttingen, Germany (in case 7). Final angiogram

<table>
<thead>
<tr>
<th>Patients</th>
<th>Sex</th>
<th>Age</th>
<th>Type of CCF</th>
<th>Treatment option</th>
<th>Occlusion test</th>
<th>Procedure outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pat. (1)</td>
<td>M</td>
<td>36</td>
<td>Barrow A</td>
<td>Onyx 18 &amp; coils</td>
<td>–</td>
<td>CCF closure</td>
</tr>
<tr>
<td>Pat. (2)</td>
<td>F</td>
<td>44</td>
<td>Barrow D</td>
<td>Onyx 18 &amp; coils</td>
<td>+</td>
<td>Transient 6th palsy</td>
</tr>
<tr>
<td>Pat. (3)</td>
<td>M</td>
<td>36</td>
<td>Barrow A</td>
<td>Onyx 18 &amp; coils</td>
<td>+</td>
<td>ICA occlusion</td>
</tr>
<tr>
<td>Pat. (4)</td>
<td>M</td>
<td>28</td>
<td>Barrow A</td>
<td>Onyx 18 &amp; coils</td>
<td>+</td>
<td>CCF closure</td>
</tr>
<tr>
<td>Pat. (5)</td>
<td>M</td>
<td>14</td>
<td>Barrow A</td>
<td>Coils</td>
<td>+</td>
<td>ICA occlusion</td>
</tr>
<tr>
<td>Pat. (6)</td>
<td>F</td>
<td>40</td>
<td>Barrow A</td>
<td>Coils</td>
<td>+</td>
<td>CCF closure</td>
</tr>
<tr>
<td>Pat. (7)</td>
<td>M</td>
<td>22</td>
<td>Barrow A</td>
<td>Coils and Glue 30%</td>
<td>+</td>
<td>ICA occlusion</td>
</tr>
<tr>
<td>Pat. (8)</td>
<td>M</td>
<td>62</td>
<td>Barrow D</td>
<td>Coils</td>
<td>–</td>
<td>CCF closure</td>
</tr>
<tr>
<td>Pat. (9)</td>
<td>F</td>
<td>18</td>
<td>Barrow D</td>
<td>Onyx 18 &amp; coils</td>
<td>+</td>
<td>CCF closure</td>
</tr>
<tr>
<td>Pat. (10)</td>
<td>F</td>
<td>80</td>
<td>Barrow A</td>
<td>Coils</td>
<td>–</td>
<td>CCF closure</td>
</tr>
</tbody>
</table>

**Fig. 2** (A and B) Right ICA showing Anterior and interstellar drainage of the CCF with non visualized distal course (C and D) Right ICA post embolization with complete closure of the fistula and restored intracranial flow. (E and F) Left ICA post embolization.

The traumatic CCF was crossed to the cavernous sinus using a microcatheter e.g. (Echelon 10 - Microtherapeutics-EV3). In case 1 (Fig. 2) test occlusion was negative and the ICA had to be spared. A $4 \times 10$ mm balloon (HyperGlide - Microtherapeutics- EV3) was placed in the cavernous carotid to be intermittently inflated during embolization to prevent the reflux of the embolizing material in the ICA. The sinus was packed with electrically detachable platinum coils (GDC-10 & GDC-18 Boston/Target, Fremont, CA and Axi- um- Microtherapeutics- EV3) to occlude the sinus (cases 5 and 6). Adding liquid embolic material to complete sinus occlusion was done using Onyx 18 Microtherapeutics- EV3 (in cases 2–4) or $N$-Butyl cyanoacrylate, Braun, Aesculap AG, Tuttingen, Germany (in case 7). Final angiogram
confirmed total occlusion of the sinus and the draining routes. In four patients (cases 3–5 and 7) the ipsilateral ICA was sacrificed with no clinical consequences and the rest showed normal flow in the supraclinoid and intracerebral branches of the ICA.

In dural CCF cases the diseased sinus was closed with coils and Onyx-18 (Figs. 3 and 4) and coils alone (patient 8). In case 7 the glue burst the protective balloon and caused non-intended occlusion of the ICA which had no clinical consequences due to positive test occlusion confirmed at the beginning of the procedure.

There was a dramatic relief of symptoms along the following days of the procedures. The patients were discharged one week later and were symptom free at three months follow up.

Patient 2 had transient 6th palsy post-procedure that resolved spontaneously within two weeks. Patient 5 (previously enucleated eye ball after trauma) had further ipsilateral painful swelling and was diagnosed as superior ophthalmic vein thrombosis and responded to anticoagulant and pain killers over one week.

4. Discussion

Type A fistulas are high flow fistulas that show spontaneous resolution in only 0.05% [3,4], hence, they usually require therapeutic intervention. The decision to treat a dural CCF requires recognition of the patient and physician that the signs and symptoms merit the risks of the intervention. The choice of treatment modalities requires a complete set of diagnostic angiographic sequences. Higher risk fistulas associated with neurological deficits, intradural hemorrhage, venous thrombosis, or altered mental status deserve the most aggressive mea-

![Fig. 3](A and B) Both ICA selectively injected showing dural feeders to the CCF. (C) Direct percutaneous puncture of the facial vein using the road mapping technique. (D) The microcatheter introduced through the facial, angular and superior ophthalmic veins to reach the cavernous sinus. (E and F) Bilateral CCA showing complete obliteration of the CCF post embolization.
sures to eradicate the fistula. Low-risk lesions with mild symptomatology may not require active intervention and can be managed expectantly. Patients with low-risk lesions can be given reassurance, educated regarding potential changes in symptoms, and allowed time for potential spontaneous closure of the fistula [5,6].

The goal of treatment in direct CCFs is to occlude the site of communication between the ICA and the cavernous sinus while preserving the patency of the ICA. This goal can be accomplished through either transarterial obliteration of the fistula with a detachable balloon [7–13] or trans-arterial or transvenous obliteration of the ipsilateral cavernous sinus with coils or other embolic materials [9–14] or deployment of a covered stent across the fistula [15]. Transvenous may be an option in direct CCFs that cannot be treated by a transarterial route because of inaccessibility of the proximal ICA secondary to traumatic injury, severe tortuosity, and/or inability to catheterize the ICA tear [10].

Transarterial embolization with detachable balloons has been widely accepted as the preferred method for treating direct CCF. However, this technique results in a rate of pseudo-aneurysm formation as high as 30-44% because of the remnants of the wall defect of the ICA at the site of the previous fistula that forms [16] after the detachable balloon deflates.

Archondakis et al. reported eight patients with post-traumatic CCF treated by positioning a coronary covered stent...
in the intracranial internal carotid artery (ICA) to occlude the fistula. One year follow up showed that asymptomatic occlusion occurred [17] in one case, persistent fistula in one case and complete occlusion in six patients.

A recent report of three patients with direct CCF treated by transarterial graft-stent placement stated that detachable coils have proven to be an excellent therapeutic option for traumatic fistula occlusion under circumstances when detachable balloons cannot be used. Nevertheless, detachable coils have their limitations of parent artery occlusion or thrombo-embolism by herniation or jumping [18] of coils into the parent vessel.

N-Butyle-Cyanoacrylate (n-BCA) (Histoacryl; Braun, Aesculap AG, Tuttlingen, Germany) was the commonly used liquid embolic material to occlude the CCF direct or dural [11]. However, it provides limited time of injection, and may polymerize and glue the microcatheter inside the sinus. In contrast to n-BCA Ethylene–vinyl alcohol copolymer (Onyx, Microtherapeutics- Irvine, Calif.) is a non-adhesive liquid that allows a longer injection time and rates and can be used safely and showed excellent penetration in the different compartments of the cavernous sinus allowing longer duration and controlled injection [9–14].

In indirect (dural) CCF the goal of treatment is to interrupt the fistulous communications and decrease the pressure in the cavernous sinus. This goal can be accomplished by occluding the arterial branches supplying the fistula (transarterial embolization) or, more effectively, by occluding the cavernous sinus that harbors the fistulous communication (transvenous embolization) [2].

The most commonly used venous pathway for cannulation of the cavernous sinuses is via the inferior petrosal sinus (IPS), because of easy accessibility and excellent results. However, in many patients, the inferior petrosal sinus does not fill angiographically, perhaps owing to an acquired thrombosis or flow reversal related to the shunt. The superior ophthalmic vein provides direct access to the cavernous sinus and it may be an equally-if not more-accessible approach to the indirect fistulas than the inferior petrosal sinus [19]. Other transvenous approaches include the pterygoid venous plexus, superior petrosal sinus, cortical veins, or the contralateral IPS or SOV with access into the ipsilateral cavernous sinus through the circular sinus [20,21].

In extremely difficult cases of venous occlusion, stenosis, or marked tortuosity, combined surgical and endovascular approaches may be needed to access the cavernous sinus. Direct transorbital puncture or indirect puncture through the superior or inferior ophthalmic vein (SOV/IOV) allows straightforward access to the cavernous sinus [22]. Seven cases with CCF treated by retrograde catheterization of the facial vein were reported [23] with one case report where sonographically guided percutaneous facial vein puncture was used to access the cavernous sinus [24].

Although in most patients the trans-SOV embolization is a quick and efficient therapeutic route, many factors may make catheterization difficult. Anatomic variations in the location and size of the orbital veins may be confusing. The SOV could be small and non-dilated, be located more medially or inferiorly in relation to the trochlea, and have small bridging veins. Careful preoperative and intraoperative review of angiographic anatomy can help identify anatomic variations [25].

Previous reports were published using coils and Onyx18 to embolize dural CCF [25] using the transvenous approach.

In case 2 and 4 road mapping enabled direct percutaneous puncture of the facial vein. Road mapping is a soft ware that allows getting a contrast map of the opacified vessels to be overlapped on the next fluoroscopy. It is done by injecting the ICA and waiting the passage of contrast through the CCF to opacify the draining veins and using it subsequently to puncture the vein. This technique is simple and safe and obviates the need for surgical manipulations of the orbit. To our knowledge this was not described before in the literature.

Dense packing of the cavernous sinus with coils should be avoided because of the risk of cranial nerve deficits due to compression of the cranial nerves by the mass effect [26]. In case 5 dense packing created a mass effect that caused non-intended occlusion of the ICA which had no clinical consequences due to positive test occlusion confirmed at the beginning of the procedure, and as a result of previous enucleation of the eyeball in this case there was no antegrade flow after closure of the fistula to maintain the patency of the dilated superior ophthalmic vein in spite of the prophylactic heparin given, the condition responded to therapeutic anticoagulant and pain killers over one week.

In case 2 transient 6th nerve palsy was encountered in spite of loose packing (only three coils) that resolved spontaneously within 2 weeks. We think that this transient nerve palsy occurred from the mass effect of the Onyx cast combined with the swollen fresh thrombus formed in the sinus cavity. This effect resolved spontaneously within 2 weeks after clot retraction.

The dural fistulae frequently have a posterior location within the cavernous sinus [27]. Posterior fistulae may drain into the inferior petrosal sinus, causing distension of the sinus and compression of the VI nerve. Compression of the nerve against the petroclinoid ligament or interruption of the fragile arterial blood supply would cause VI nerve palsy [28].

5. Conclusion

Direct puncture of the draining veins using road mapping can be safely used as a venous approach in the treatment of dural CCF when other venous routes are difficult to access. Carotid occlusion test is an important pretreatment step that helps to decide the strategy of endovascular treatment weather including the possibility of carotid sacrifice or the necessity to preserve patency.

References

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