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Treatment of Carotid Cavernous Fistulas

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Opinion statement

The treatment of a carotid cavernous fistula (CCF) depends on the severity of the clinical symptoms, its angiographic characteristics, and the risk it presents for intracranial hemorrhage. In most instances, endovascular treatment is preferred.

High-flow direct CCFs usually are traumatic or are caused by rupture of a cavernous aneurysm into the sinus, but a small percentage can be spontaneous. They usually present with sudden development of a clinical triad: exophthalmos, bruit, and conjunctival chemosis. All direct CCFs should receive treatment, because they carry a high probability of intracranial hemorrhage or neurologic deterioration.

Low-flow indirect or dural CCFs, either incidental or with minimal symptoms, are not associated with significant risk of intracranial hemorrhage. The accepted practice is to treat ocular symptoms conservatively with medical management or manual carotid compression. If the patient cannot tolerate the symptoms, or if signs of ocular morbidity occur, endovascular treatment is offered.

The first treatment option should be endovascular embolization with a combination of detachable balloons, coils, stents, or liquid embolic agents. The procedure can be performed from either an arterial or venous approach. Use of these materials and techniques can yield a high cure rate with minimal complications.

If the patient is not amenable to embolization or if the embolization fails, then surgery (surgical ligation of the internal carotid artery or packing of the cavernous sinus) should be offered.

Stereotactic radiosurgery may be an elective treatment for low-flow CCFs, but it has no role in the treatment of high-flow CCFs.

Introduction

Carotid cavernous fistulas (CCFs) are abnormal arteriovenous communications between the carotid artery or its branches and the cavernous sinus (CS). Their manifestations and course can be benign, intermediate, or severe, depending on their angioarchitectural and hemodynamic characteristics. Patients classically present with development of a triad of exophthalmos, cephalic bruit, and conjunctival congestion. Patients may also experience intractable headache, visual deterioration, elevation of intraocular pressure refractory to medication, diplopia, or an intolerable cosmetic deformity.

CLASSIFICATION

CCFs are classified by 1) etiology (posttraumatic or spontaneous), 2) flow rate (high flow or low flow), and 3) angioarchitecture (direct fistulas with a connection between the internal carotid artery (ICA) itself and the CS, or indirect or dural arteriovenous fistulas (DAVFs) between meningeal arteries and the CS. The term CCF generally is applied to both direct and indirect types, based on the older angiographic literature. The most commonly used classification scheme, established by Barrow and colleagues [1], is shown in Table 1. It divides CCFs into four types depending on the arterial supply. Direct (type A) fistulas are usually associated with high flow rates. The most common indirect CCF is type D; type B is relatively uncommon.

ETIOLOGY

Direct (type A) fistulas usually are traumatic, caused by motor vehicle accidents or penetrating injuries [2]; these generally affect young males. About 20% of type A CCFs may be spontaneous [3, Class IV]; these are more common in older women, although a few have been reported in children [4,5]. They usually are caused by the rupture of a cavernous aneurysm or by the spontaneous rupture of a congenitally weakened, atherosclerotic, or diseased artery. Predisposition to spontaneous CCFs has been shown in fibromuscular dysplasia, Ehlers-Danlos syndrome, and pseudoxanthoma elasticum [6,7].

Indirect CCFs (types B, C, or D) have a predilection for spontaneous occurrence in postmenopausal women. Sinus thrombosis, hypertension, and diabetes have been suggested as predisposing factors [8,9; Class IV]. Trauma is less commonly associated with symptomatic indirect CCFs [10]. Congenital indirect CCFs have been reported in children, including infants as young as 5 weeks of age [11].

CLINICAL PRESENTATION

The classic presentation for a direct, high-flow CCF is the sudden development of a clinical triad: exophthalmos, bruit, and conjunctival chemosis (Fig. 1). Direct CCFs can develop following a traumatic tear of the cavernous segment of the ICA, rupture of a cavernous ICA aneurysm, or both [12–14]. Complete disruption of the ICA wall allows highly pressurized arterial blood to be transmitted directly into the cavernous sinus and the ophthalmic veins, leading to venous hypertension. The manifestations of venous hypertension include ocular signs and symptoms (proptosis, chemosis, conjunctival injection, cranial nerve pareses, and visual deficits), bleeding (from mouth, nose, or ears), and cerebral complications (intracranial hemorrhage, increased intracranial pressure, and steal phenomena) [15,16]. Intracranial hemorrhage develops in 5% of patients, and 1–2% manifest life-threatening epistaxis. Epistaxis can be acute or can be remote from the initial trauma, following rupture of a pseudoaneurysmal cavernous sinus varix.

Table 1. Barrow classification of carotid-cavernous fistulas

Type	Description
Type A (direct)	Direct communication between the ICA and the cavernous sinus
Type B (indirect)	Supplied only by dural branches of the ICA
Type C (indirect)	Supplied only by dural branches of the ECA
Type D (indirect)	Supplied by dural branches of the ICA and ECA

ECA—external carotid artery; ICA—internal carotid artery.
(*Adapted from* Barrow et al. [1].)

Figure 1. Photograph of a patient with a direct carotid-cavernous fistula demonstrates arterialization of conjunctival vessels.

Compared with direct CCFs, indirect fistulas have a gradual onset, generally with a milder clinical presentation. Indirect fistulas usually are low-flow, acquired lesions that result from sinus thrombosis leading to venous congestion. Subsequently, abnormal arteriovenous shunting develops through the recanalized dural veins [17]. DAVFs of the CS often do not demonstrate the classic triad of symptoms. Patients with these fistulas have chronically red eyes because of tortuous arterialization of the conjunctival veins. An ocular bruit may or may not be present. Unlike type A fistulas, most spontaneous indirect CCFs improve with time, and many will heal with medical management [18,19; Class IV]. About 20–50% of indirect or dural CCFs heal spontaneously within days to months after symptomatic presentation.

DIAGNOSTIC IMAGING

CT and MR imaging often are used in the initial workup of a possible CCF. CT findings in CCFs include proptosis, enlargement of the extraocular muscles, enlargement and tortuosity of the superior ophthalmic vein, and enlargement of the ipsilateral cavernous sinus. MRI findings are similar to those seen on CT, with the addition of orbital edema and abnormal flow voids in the affected cavernous sinus [20]. In the setting of a high-flow fistula and retrograde cortical venous reflux, MR or CT studies may reveal dilatation of leptomeningeal and cortical veins. In patients who have cerebral venous congestion and elevated intracranial pressures, cerebral edema, hemorrhage, or both may be encountered.

Digital subtraction angiography is essential in confirming the diagnosis, classifying the fistula, and delineating the venous drainage pathways. Conventional angiography best characterizes the flow rate of the fistula and clearly distinguishes between direct and indirect fistulas, showing the exact anatomic location of the ICA tear versus dural feeders of the ICA or external carotid artery (ECA). Moreover, it helps to assess the draining venous pathways (anterior vs posterior), cortical venous reflux, venous stenosis, or occlusions that could limit transvenous access into the cavernous sinus.

A complete and detailed diagnostic angiogram is recommended for planning either endovascular or surgical treatment. Selective ICA and ECA injections allow accurate classification of the fistula. In addition, vertebral artery injections are helpful in fully appreciating the intracranial collateral circulation and circle of Willis, in case ICA sacrifice must be considered as an option.

Treatment

- The treatment of a CCF varies according to the symptoms and the risk that it may represent for the patient.
- For indirect CCFs with low risk of intracranial hemorrhage or minimal clinical symptoms, no treatment or conservative treatment may be an acceptable option. Radiosurgery may have a limited role in treatment of indirect, low-flow CCFs.
- CCFs that present with severe clinical symptoms or significant risk of intracranial hemorrhage are usually first treated with endovascular embolization. Surgery has a very limited role in treatment.

Conservative treatment

- There is no role for conservative treatment of high-flow (type A) CCFs.
- Unlike high-flow CCFs, low-flow indirect or dural CCFs are not associated with increased mortality or significant risk for intracranial hemorrhage. An accepted practice is to treat the patient's ocular symptoms medically with prism therapy or patching for diplopia, topical agents for elevated intraocular pressure, lubrication for proptosis-related keratopathy, and/or systemic corticosteroids if needed [21, Class IV].

Manual carotid-jugular compression

Intermittent, self-administered manual carotid-jugular compression alone can result in a cure in 30% of patients with indirect CCFs [18, Class IV]. Compression of the common carotid and jugular veins causes flow obstruction, which theoretically leads to stasis and thrombosis of the fistula.

The patient compresses the affected common carotid at the neck for 30 seconds many times a day for 4–6 weeks. The compression must be performed with the contralateral hand, so that if hemispheric ischemia occurs, the hand will lose strength and perfusion to that side of the brain will be restored.

Endovascular embolization: Direct CCFs

- A number of different endovascular treatment options for CCFs are currently available. The method chosen in a given patient depends on the anatomy of the fistula and the preference of the operator or institution.
- The goal in treating a direct CCF is to occlude the site of communication between the ICA and the cavernous sinus while preserving the patency of the ICA.

Transarterial embolization

- Transarterial embolization of direct (high-flow) CCFs in countries other than the United States most commonly consists of placing a detachable balloon through the rent in the ICA [22]. In the United States, transarterial embolization is commonly performed with coils or other embolic material, given the unavailability of a detachable balloon. This technique consists of placing a microcatheter across the tear into the cavernous sinus and then filling the sinus with detachable coils, n-butyl cyanoacrylate (n-BCA), or ethylene vinyl alcohol copolymer (EVOH) to occlude the direct communication [23–25]. During transarterial embolization, a temporary balloon may be placed in the cavernous segment of the ICA (across the site of the tear) to protect the parent vessel and to prevent migration of the embolic material into the distal intracranial circulation.

- If there is a large arterial tear in the ICA, coils or balloons may migrate through the defect into the parent vessel, potentially causing cerebral ischemia.
- Recently, dedicated self-expanding intracranial stents have become available. These stents can provide a valuable scaffolding to reconstruct a severely injured ICA [26].
- Placement of a covered stent is an additional treatment option. It may immediately obliterate a direct CCF by placing an impermeable barrier across the site of the fistula [27].

Transvenous embolization

- The transvenous route may be an option in treating direct CCFs that cannot be treated by a transarterial route because of inaccessibility of the proximal ICA secondary to traumatic injury, severe tortuosity, or inability to catheterize the ICA tear.
- Occlusion of a direct CCF from a transvenous route usually involves a posterior approach through the internal jugular vein and the inferior petrosal sinus (IPS) up into the shunt involving the CS [28]. If the IPS is occluded or absent, access into the cavernous sinus can be obtained from an anterior approach through the superior ophthalmic vein via the facial vein [29]. Other venous approaches include the contralateral pterygoid plexus, superior petrosal sinus, and cortical veins [30,31]. Direct transorbital puncture of the cavernous sinus or access via the inferior ophthalmic vein has also been reported [32]. Once cavernous sinus access is obtained, disconnection of the venous outflow from the feeding arteries at the level of the fistula can be completed with detachable coils or liquid embolic agents.
- Rarely, if the defect is large and cannot be repaired, the ICA may need to be sacrificed or trapped from an endovascular approach, using coils or a vascular plug [33]. A temporary balloon test occlusion of the ICA is carried out before permanent occlusion of the artery.
- In most direct CCFs, a combination of techniques and materials must be used. Endovascular embolization can lead to high occlusion rates (55–99%) [12,34–36; Class IV]. The percentage of patients experiencing morbidity associated with embolizations of direct CCFs, such as ICA occlusion or worsening of ocular palsy, has ranged from 10% to 40% [12,37–39; Class IV].

Endovascular embolization: Indirect CCFs

Transarterial embolization

- Transarterial embolization of indirect low-flow CCFs generally is cumbersome because of the small size, tortuous anatomy, and multiplicity of arterial feeders (Fig. 2A, B).
- The procedure often involves distal catheterization of the small meningeal branches supplying the fistula. Ideally, superselective

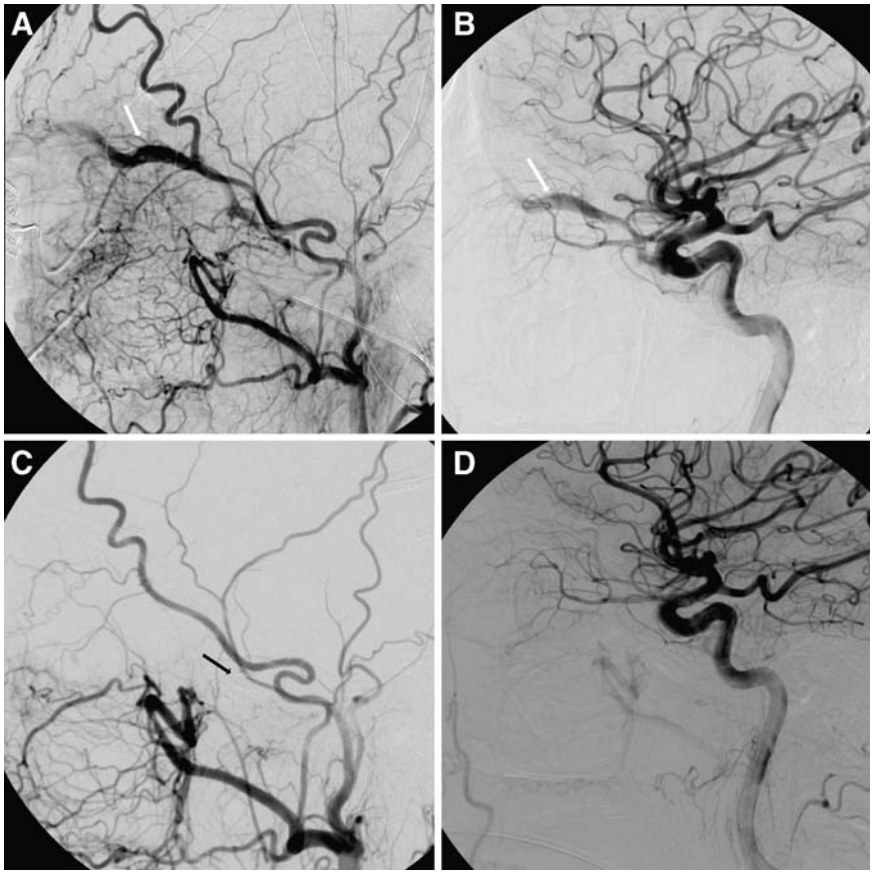

Figure 2. A, Lateral external carotid angiogram in a patient with a type D carotid cavernous fistula shows early filling of the superior ophthalmic vein (*white arrow*). **B,** Lateral internal carotid angiogram in the same patient, demonstrating early filling of the superior ophthalmic vein (*white arrow*) from dural branches off the cavernous segment of the internal carotid artery. **C,** Lateral external carotid angiogram after intraarterial injection of ethylene vinyl alcohol copolymer (EVOH) shows no filling of the fistula (*black arrow*). **D,** Lateral internal carotid angiogram after EVOH injection shows no filling of the fistula.

microcatheter placement is performed with the microcatheter tip placed as close as possible to the point of fistulous communication. Once a satisfactory position of the microcatheter is achieved, an embolic agent is injected under fluoroscopic control with the goal of occluding the fistulous connections and penetrating the cavernous sinus.

- Although coils and particulate agents can be used, these agents cannot cause permanent occlusion of the fistula by themselves. The most commonly used agents for transarterial embolization of indirect CCFs are n-BCA and EVOH [40–42•].
- Although transarterial embolization is a good treatment option, selective distal access into multiple tiny feeder vessels is often difficult or impossible and may require multiple sessions in a staged approach.

n-Butyl cyanoacrylate (n-BCA) glue

The most often used agent is n-BCA glue, a monomeric liquid adhesive approved in the United States for use in presurgical embolization of cerebral arteriovenous malformations. The viscosity and polymerization time of n-BCA is controlled by the addition of Lipiodol.

The advantages of n-BCA include its thrombogenic nature and permanent occlusion of the injected feeders.

Drawbacks include its rapid polymerization time (a few seconds), adhesive nature (risk of catheter retention), and the relatively long learning curve for its optimal use.

Ethylene vinyl alcohol copolymer (EVOH)

EVOH (Onyx; ev3, Inc., Irvine, CA) is another useful liquid embolic agent that may be used for transarterial embolization of indirect CCFs. It was recently approved by the US Food and Drug Administration for the preoperative embolization of brain arteriovenous malformations. EVOH is a nonadhesive liquid embolic agent with a lavalike flow pattern. It is supplied in ready-to-use vials with a mixture of EVOH, dimethyl sulfoxide (DMSO) solvent, and tantalum. Currently 6% and 8% EVOH concentrations (dissolved in DMSO) are available in the United States. When the mixture contacts aqueous media such as blood, DMSO rapidly diffuses away from the mixture, causing in situ precipitation, solidification of the polymer, and the formation of a spongy embolus. Polymerization occurs more slowly than with n-BCA; because EVOH does not adhere to the walls of the vessel or the microcatheter, it allows prolonged injection times with less chance of permanent microcatheter retention.

Because of the slower polymerization times, the ability to make controlled injections over minutes, and the ability to direct and push the embolic agent into the desired location, the use of EVOH may allow better and more distal penetration of the nidus or fistula than is possible with n-BCA.

EVOH offers the possibility of venous sinus packing/occlusion using a transarterial approach, an advantage that may be quite helpful in cases in which venous access to the cavernous sinus is limited because of stenosis or occlusion of the major draining venous pathways (Fig. 2C, D).

Transvenous embolization

- Transvenous embolization has become the preferred method of treating indirect CCFs. The advantages of this technique are its simplicity compared with transarterial methods, the ability to cure the fistula (often in a simple session), and a high long-term success rate.
- The most commonly used pathway for cannulation of the cavernous sinus is the IPS. If the IPS is inaccessible, the pterygoid venous plexus, superior petrosal sinus, facial vein, and superior ophthalmic vein can be used [28–31].
- Popular choices for embolic materials from a transvenous approach include coils, n-BCA, and EVOH. These may be used in isolation or in combination.
- The advantages of coils are their radioopacity, ease of use, and ability to be redeployed or removed if the initial placement is not optimal.
- Liquid embolic agents are increasingly being used, either alone or in combination with coils [37,43], because they can permeate different compartments, allowing complete occlusion of the fistula.

- The reported complete cure rate for embolization of indirect CCFs is 70–90%, with a complication rate of 2.3–5% [19,44,45; Class IV].

Surgical treatment

- There is no indication for any type of nonendovascular (open surgical) approach for CCFs as the primary form of treatment. Surgical techniques have a cure rate between 31% and 79%, but the operative mortality and cerebral morbidity in the surgical approach to CCFs has been distressingly high. Given the higher morbidity and mortality of surgical treatment, it is primarily reserved for cases in which endovascular treatment is unsuccessful.
- Trapping of the fistula by ligation of the cervical and intracranial ICA is still considered an effective treatment for direct CCFs.
- Carotid sacrifice has also been performed via embolization using different materials delivered by direct carotid exposure [46, 47]. However, sacrifice of the ICA is performed sparingly because of a significant risk of cerebral infarction even after a successful balloon test occlusion.
- Successful surgical exposure and packing of the cavernous sinus with preservation of the ICA has also been reported [48].
- Rarely, orbital surgery or decompression can be offered in cases in that do not respond to treatment or those in which elevated intraocular pressures persist despite closure of the fistula [21].

Radiosurgery

- Stereotactic radiosurgery has no primary role in the treatment of high-flow direct CCFs (type A). It has been used to treat indirect CCFs. In selected small series of patients with indirect CCFs, the treatment results have been good, with cure rates of 75–100% [49,50]. There are no reports in the literature of cure for direct CCFs.
- Stereotactic radiosurgery requires stereotactic angiography and fusion with CT or MR images to delineate the treatment target. The treatment target is irradiated to 30–40 Gy. The effect of the treatment on an indirect CCF may appear several months later. Vessel thrombosis following radiation is a slow and progressive process, often taking 2 years to occur (latency period). Therefore, the treatment is usually an option given to older patients with minimal symptoms.
- Stereotactic radiosurgery is commonly used following incomplete transarterial or venous embolization of an indirect CCF. Embolization has also been used to decrease the number of arterial feeders and minimize the risk of bleeding or ocular symptoms during the latency period of radiosurgical treatment [51].
- Because the natural history of some indirect CCFs includes spontaneous healing and because radiosurgery has a latency period, treatment should be reserved for low-risk fistulas or those with

minimal ocular symptoms. Radiosurgery may have a role in treating direct CCFs only if the results of embolization, surgery, or both are incomplete.

Disclosure

No potential conflicts of interest relevant to this article were reported.

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