CASE REPORT

Carotid-Cavernous Fistulas: Pathogenesis and Routes of Approach to Endovascular Treatment

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ABSTRACT

The etiology, classification, clinical presentation, complications, and intravascular routes to image and treat carotid-cavernous fistulas percutaneously are described. Endoarterial and transvenous approaches (through the jugular, inferior petrosal, or cavernous veins) are discussed in relation to the etiology, size, and characteristics of the fistulas, as well as in relation to the planned therapeutic approach and its possible complications. Possible outcomes, with particular attention to the internal carotid circulation, side effects, and complications also are discussed in relation to etiology and type of fistula. Four exemplary cases are presented.

KEYWORDS: Carotid-cavernous sinus fistula, cerebral angiography, therapeutic embolization, balloon occlusion

Carotid-cavernous fistulas (CCF) are abnormal communications between the carotid arterial system and the cavernous sinus. They can be spontaneous or acquired and are classified as direct or indirect. According to the Barrow classification,[1] there are four angiographically identifiable types of CCF: type A, a direct shunt between the intracavernous internal carotid artery (ICA) and the cavernous sinus; type B, a dural arteriovenous (AV) fistula supplied by the ICA; type C, a dural AV fistula supplied by the external carotid artery (ECA); and type D, a dural AV fistula supplied by both the ICA and ECA. Types B, D, and C are often grouped under the common definition of dural or indirect carotid-cavernous fistulas.[2] Direct CCF often demonstrate high flow, whereas indirect fistulas are often low-flow lesions.[3]

A different approach to the classification of CCF considers the etiopathogenetic origin of these lesions. Four mechanisms have been described for

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the genesis of fistulas.[4] In this approach, type 1 is a traumatically acquired direct AV fistula. The rupture of the ICA wall at its emergence from the petrous bone with direct flow into the cavernous sinus may be the result of a fracture or of a spontaneous rupture caused by collagenopathy. In the latter condition, endovascular treatment is still indicated and is technically identical, but the risk of mortality with simple cerebral angiography in patients with Ehlers-Danlos syndrome is high (20 to 30%). Type 2 is caused by rupture of a pre-existing intracavernous carotid aneurysm. Two abnormal communications form, one at the neck of the aneurysm and the other along the wall of the aneurysm. Type 3 is a dural-type AV fistula involving the cavernous sinus, supplied indirectly by dural branches of the ECA or by meningeal perforators originating from the ICA. The difficulty of treatment varies with the path of the originating vessels. Type 4 is a combination of direct- and indirect-type fistulas.

ETIOLOGY

The most frequent cause of CCF is trauma, which accounts for 70 to 90% of cases.[5] The carotid artery enters the cavernous sinus at the foramen lacerum, turns anteriorly about 2 cm, ascends along the medial aspect of the anterior clinoid process, and exits the cavernous sinus perforating the dura, to which it is attached. Motor vehicle accidents, falls, and penetrating injuries to the head and orbit are the most frequent causative events. They can cause shearing of the dural attachments of the carotid artery or intra-arterial penetration of bony spicules originating from skull base fractures. Typically the laceration is 2 to 5 mm in diameter. The fistulas tend to be unilateral, probably because bilaterality portends to earlier and more frequent death. Iatrogenic injuries may also occur during carotid angioplasty and endoarterectomy, trans-sphenoidal hypophisectomy, trigeminal rhizotomies, and nasopharingeal biopsies.

Spontaneous direct CCFs are often associated with underlying collagen deficiencies, such as Ehlers-Danlos syndrome.[6] Such diseases are thought to weaken the media of the ICA wall with a relatively high frequency. Affected patients have an abnormally short life expectancy caused by similar alterations elsewhere in the vascular system, and they often die from aortic dissection, myocardial or aneurysmal ruptures, or bowel perforations.[7]

The indirect types (Barrow types B, C, and D) are AV fistulas located within the dura surrounding the cavernous sinus and result from spontaneous rupture of small dural arteries. These low-pressure, low-flow CCFs have only rarely been associated with trauma. They tend to occur spontaneously, most frequently in middle-aged women, probably secondary to hypertension or hormonal factors associated with pregnancy and menopause. These circumstances could exert a weakening action upon pre-existing congenital or acquired AV malformations of the dura.[6] The collagen deficiency syndromes are also thought to represent underlying predisposing abnormalities.[7]

CLINICAL PRESENTATION

The cavernous sinus drains into the jugular bulb through the inferior and superior petrosal sinuses. The superficial middle cerebral veins drain through the sphenoparietal sinus into the cavernous sinus; the superior and inferior ophthalmic veins drain the orbit into the cavernous sinus. The fistula provides an abnormal low-resistance pathway between the high-pressure carotid artery and the low-pressure venous system. If increased blood flow cannot be accommodated through the veins of the basilar and petrous systems, the increased pressure in the cavernous sinus is relieved through the inferior and superior ophthalmic veins.[1,6] This pattern of flow

may therefore result in orbital venous congestion, variable proptosis, eyelid swelling, corneal ulcerations, pulsating exophthalmos, chemosis, secondary glaucoma (9%),[8] and compromised retinal perfusion (~30%), with visual impairment in as many as 85% and complete loss of vision in as many as 25% of cases. In contrast, diplopia tends to be related to mass effect within the cavernous sinus, causing venous hypertension and muscular edema. Neuropathic diplopia can also be related to ischemia in the vasa nervorum, particularly of cranial nerve VI.[1,6] Occasionally, the presence of a circular sinus may cause bilateral orbital symptoms from a unilateral CCF. Symptoms and signs of CCFs are related to the size, duration, location, and route of venous drainage of the fistula[9] and only rarely occur in the immediate post-traumatic period. More typically, they evolve and become apparent over days or weeks.

The onset of venous hypertension in the cerebral cortex is less frequent and raises the risk for cerebral and subarachnoid hemorrhage, which may occur in as many as 6% of patients.

Large-caliber fistulas may have a dramatic and acute clinical presentation. Arterial steals are responsible for retinal hypoperfusion and optic and cranial nerves III, IV, V, and VI ischemic neuropathy. They also can generate ischemia of large cerebrovascular territories in patients with poor collateral circulation due to atherosclerotic compromise or congenitally incomplete circle of Willis.

Indirect fistulas appear to have more benign clinical course and evolution than direct fistulas, probably because of the small size of the cavernous sinus in relation to the transverse sinus. Dural fistulas are most probably acquired by alteration of the physiological dural AV shunts that occur in relation to venous thrombosis. Their course is capricious and they usually heal spontaneously. The onset of symptoms is often subtle and protracted in relation to the slow progression of the underlying pathophysiologic anomaly. The rates of orbital and cerebral complications and death are characteristically lower than the complication rate associated with post-traumatic lesions. Their benign course most likely reflects the combination of a low-flow fistula and their much longer course, which undoubtedly allows physiologic compensation of the hemodynamic abnormality.

DIAGNOSIS AND EVALUATION

Computed tomography (CT), performed with thin-section axial and coronal planes, helps identify and characterize skull fractures, frequently the cause of direct CCF. Both CT and magnetic resonance imaging (MRI) are useful in assessing the degree of associated cerebral parenchymal injury, edema, and orbital changes such as the proptosis, dilated ophthalmic veins, bulging of the cavernous sinus, and increased thickness of the extraocular muscles. MRI may reveal extraneous causes of injury to the cranial nerves and is highly accurate in identifying intracerebral hemorrhage and ischemia.

Digitally subtracted angiography is pivotal in characterizing the vascular abnormality as well as in guiding endovascular treatment. The initial angiographic evaluation is performed to assess the size and location of the fistula; to detect any associated carotid aneurysm; to classify the fistula as direct or indirect; to identify the outflow pathway from the cavernous sinus; to detect high-risk hemodynamic abnormalities such as cortical venous drainage, pseudo-aneurysms, or cavernous sinus varix; and to diagnose associated vascular injuries in trauma cases.[10]

High-flow traumatic lesions may require implementation of the Mehringer-Hieshima maneuver (gentle selective injection of the carotid artery with manual compression of the ipsilateral carotid) to slowly opacify the fistula and therefore detail its morphology by eliminating the high-pressure inflow from the ipsilateral carotid artery. The Heuber maneuver, a similar technique, opaci-

fies the fistula through a patent posterior communicating artery during injection of the vertebrobasilar system and compression of the ipsilateral carotid artery. Both maneuvers also may unveil the unusual case of multiple lesions or complete transection of the ICA.

The delayed phase of the arteriogram depicts the outflow pathway of the fistula, the direction of venous flow, and the presence of a cavernous sinus varix. In particular, patency of the petrosal sinuses and ophthalmic veins provides mapping for transvenous access to embolization.

TREATMENT

Direct CCFs rarely undergo spontaneous resolution and, without treatment, eventually cause loss of vision in 80 to 90% of cases from central retinal vein occlusion or glaucoma. The higher risk for other complications such as epistaxis, intracerebral hemorrhage, and death mandate prompt treatment. Indirect CCFs may resolve spontaneously in 20 to 50% of cases.[1,11] In the past these lesions have been treated by cervical arterial ligation, carotid artery banding, placement of muscle plugs into the carotid artery, and direct repair of the fistula under cardioplegia. All of these techniques have been abandoned for what today is considered the treatment of choice: transarterial detachable balloon occlusion.[12] Embolization can also be performed with a combination of coils and balloons.[13]

The advantages of endovascular treatment are well known and are represented by its minimally invasive nature, the possibility of performance without general anesthesia, and the faster postprocedural recovery time. With contemporary catheters and balloon material, direct CCF obliteration has been obtained in 80 to 90% of cases with the rate of carotid artery patency as high as 80%.[14] Although the morbidity rate associated with endovascular treatment is minimal compared to older therapies, there are potential complications. There is a reported 3% rate of transient ischemic attacks and permanent neurologic deficits. Reversible oculomotor palsies have also been reported in as many as 20% of patients. Balloon deflation can lead to pseudoaneurysm formation, some of which may become symptomatic requiring sacrifice of the carotid artery, as may traumatic or iatrogenic carotid dissection.[14]

In some instances the transarterial approach fails or cannot be performed because of high-grade carotid stenosis, extreme tortuosity of vessels, or traumatic occlusion of the proximal ICA. In these cases endovascular embolization can also be performed through venous access, either retrogradely through the cavernous sinus or after surgical exposure of the superior ophthalmic vein.[15–17]

Indirect fistulas can be cured successfully by transarterial embolization if the blood supply is solely from the ECA territory. Otherwise, the transvenous approach appears to have a higher rate of success with these low-flow indirect lesions, probably due to the decreased resistance imparted upon the small-bore catheters while navigating through tortuous anatomy.[17] Dural fistulas also may be more challenging technically when multiple arterial feeders are involved, especially when the latter originate from both the ECA and ICA. A role for external beam radiation therapy has been advocated in the treatment of low-flow fistulas, as an adjuvant to endovascular treatment or as the sole therapy. Cure rates of as high as 75% have been reported.[2]

Balloon Occlusion

Most CCFs can be occluded by deploying detachable inflatable balloons, delivered through the arterial route, thereby maintaining patency of the ICA. The balloon can be inflated to a diameter exceeding that of the abnormal communication in order to prevent future dislodgment. Potential causes of failure for this approach are the presence of an aperture small enough to prevent entry of the bal-

loon, a venous compartment too small to allow appropriate inflation of the balloon, or the presence of bony spicules that can puncture the balloon itself. When the balloon alone only partially occludes the fistula, the remaining flow can be halted by additional balloons or platinum microcoils. Sacrificing the ICA is rarely necessary for proper treatment. In such cases, a temporary test occlusion must be performed to assess for the presence of residual feeding to the fistula and for adequacy of the cerebral perfusion after occlusion.

The device of choice is a detachable silicon balloon filled with polymerizing agent and a mixture of saline solution and hydrosoluble iodinated contrast medium. The balloon can also be delivered by a flow-control guide catheter to increase the safety of the procedure. After the balloon is placed in the desired location, an angiogram is performed to confirm occlusion of the fistula before deployment.[13,14]

Coil Embolization

This technique represents a valid alternative in patients in whom balloon occlusion is unsuccessful. In long-standing fistulas, redistribution of blood flow to and from the orbital, petrosal, and sphenoparietal veins occasionally aggravates or causes the denovo onset of ocular and/or cerebral symptoms.

This procedure is performed electively through the transvenous route after venous access has been gained through a common femoral vein. The cavernous sinus can be occluded through selective catheterization of the inferior petrosal sinus or facial vein. As a last resort, the superior ophthalmic vein can be exposed surgically.

After access has been gained to the cavernous sinus, a mass of coils can be delivered to obtain venous occlusion. The more dangerous visualized vein (the superior ophthalmic vein or the inferior petrosal sinus) should be addressed first. Care must be exercised to prevent errant coils in the carotid lumen. This problem can be prevented by temporary balloon occlusion of the fistulous communication during the coiling procedure.[18,19]

Potential complications of endovascular treatment are thromboembolic and ischemic events related to the balloon and catheter manipulation; formation of pseudoaneurysms secondary to balloon injury; and changes in arterial hemodynamics that could cause hemorrhage, edema, and progression of ocular damage.

CASE DESCRIPTIONS

Case 1

An 18-year-old male sustained a penetrating injury to the skull base that extended from the left maxillary sinus to the posterior fossa, creating a left carotid cavernous fistula. After undergoing debridement of the skull base through a combined ENT and neurosurgical approach, the patient was referred for embolization of the fistula.

At angiography, the left ICA was dissected in the region of the carotid canal, with very slow flow of contrast at the level of the skull base.

The anterior and posterior communicating arteries appeared patent, with retrograde filling of the left ICA and opacification of the left CCF during selective angiogram of both the right ICA and left vertebral artery. A sump effect with secondary slow flow in the left anterior and middle cerebral arteries was also demonstrated. No contribution was detected from the branches of the left ECA.

A retrograde approach to the fistula was selected (Fig. 1). A 6-F guiding catheter was introduced into the left proximal vertebral artery. A microcatheter and microwire were coaxially (Fig. 1B) introduced through the left vertebral and basilar artery, proximal posterior cerebral artery into the posterior communicating artery, and retrogradely into the ICA. The tip of the catheter was placed within the CCF through the laceration in the ICA.

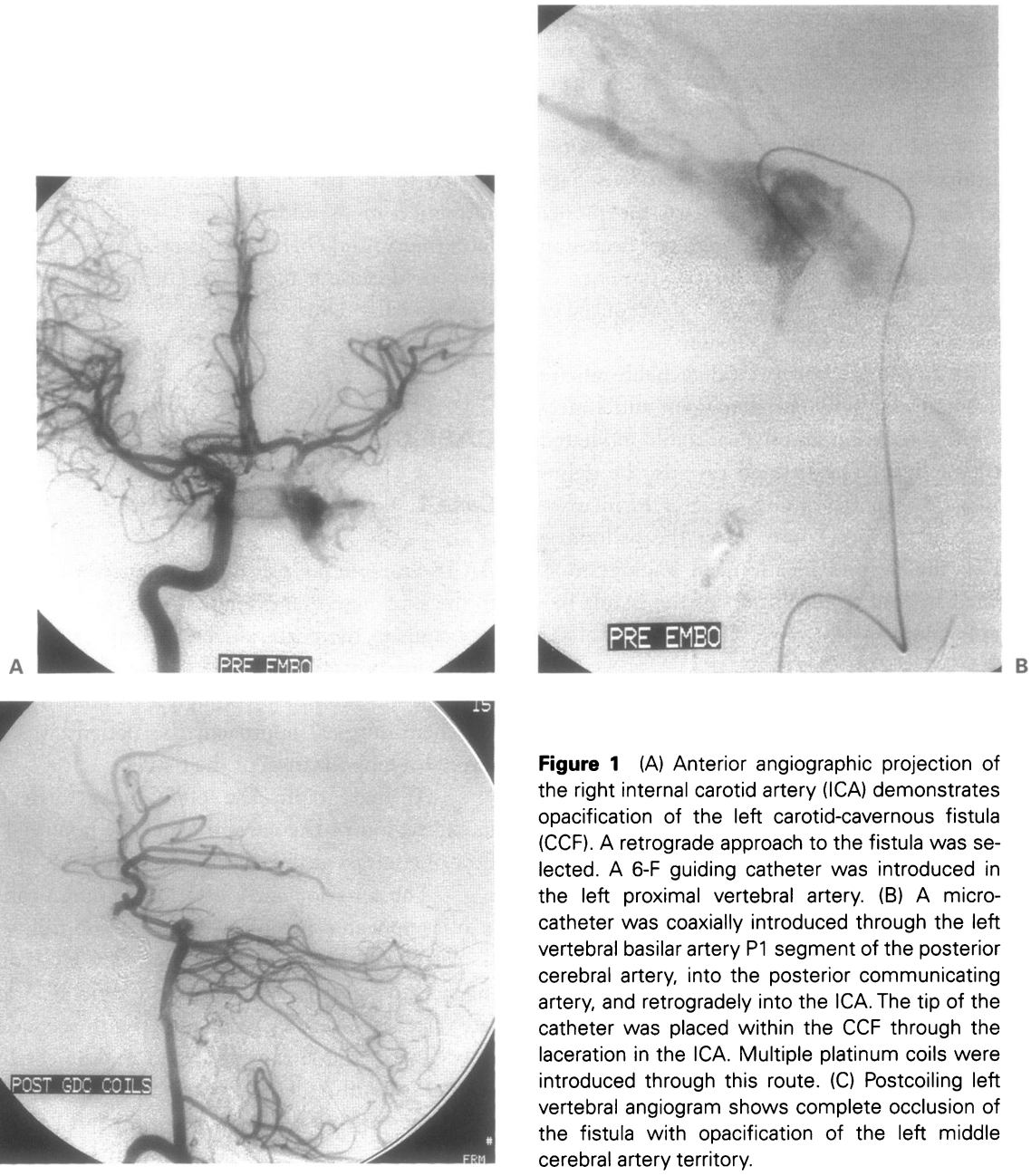

Figure 1 (A) Anterior angiographic projection of the right internal carotid artery (ICA) demonstrates opacification of the left carotid-cavernous fistula (CCF). A retrograde approach to the fistula was selected. A 6-F guiding catheter was introduced in the left proximal vertebral artery. (B) A microcatheter was coaxially introduced through the left vertebral basilar artery P1 segment of the posterior cerebral artery, into the posterior communicating artery, and retrogradely into the ICA. The tip of the catheter was placed within the CCF through the laceration in the ICA. Multiple platinum coils were introduced through this route. (C) Postcoiling left vertebral angiogram shows complete occlusion of the fistula with opacification of the left middle cerebral artery territory.

Multiple platinum coils of various sizes were introduced. Postcoiling angiography through the left vertebral artery demonstrated complete occlusion of the fistula with opacification of the territory of the left middle cerebral artery. A 9-F guiding catheter was introduced into the left common carotid artery (CCA) and proximal left ICA. A silicon detachable balloon was coaxially introduced and detached in the left carotid bulb. Postballoon detachment angiography (Fig. 1C) of the left CCA

demonstrated a complete cutoff of the blood supply to the left ICA and normal filling of the territory of the ECA.

Case 2

A 27-year-old female was hospitalized unconscious after a high-speed motor vehicle accident. The patient presented with a CCF with left proptosis and also underwent intravascular stenting of the internal meningeal artery for a dural leak.

Angiography of the right CCA demonstrated a pseudoaneurysm of the petrous portion of the ICA. The anterior communicating and posterior communicating arteries were patent. Angiography of the left ICA demonstrated a CCF with opacification of the superior ophthalmic vein (Fig. 2). There was slow antegrade flow in the left ICA distal to the fistula, as well as in branches of the middle cerebral artery, likely due to sump effect. Angiography of the left ECA showed a small dural fistula communicating with the superior ophthalmic vein. Selective left internal maxillary angiography demonstrated that the dural fistula originated from a branch of the middle meningeal artery.

The left CCF was treated by catheterizing the left ICA, left cavernous sinus, right ICA, and left ECA while the patient was under general anesthesia. A detachable balloon was deployed through an 8-F guiding catheter, through the left ICA, into the cavernous sinus and was finally inflated. Before and after balloon detachment, angiography demonstrated occlusion of the fistulous tract and normal antegrade flow in the distal ICA, with opacification of the anterior and middle cerebral arteries (Fig. 2C).

Case 3

A 57-year-old female presented after a fall with a fracture of her sphenoid and occipital bones and a three-quarter loss of vision in her left eye due to a persistent CCF. She complained of persistent throbbing of her right temporal area. At angiography a 6-F Weinberg catheter was advanced into the proximal left ECA and into the maxillary artery. There the arterial phase demonstrated a fistula of the middle meningeal artery, immediately before the origin of the anterior and posterior divisions, with early venous opacification. A left ICA angiogram demonstrated a slow-flowing CCF communicating with both cavernous sinuses and with preferential venous drainage into the contralateral inferior petrosal sinus, sphenoparietal vein, and jugular vein (Fig. 3). There was only faint opacification of the middle cerebral artery territory. Multiple attempts to profile the lesion angiographically in the carotid artery were unsuccessful.

At a later date, a microcatheter was advanced coaxially through an 8-F banding catheter positioned in the left ICA (Fig. 3B). The cavernous sinus was selectively catheterized and a 12 mm x 3.0 cm 18-F coil was introduced into the cavernous sinus. Angiographically, however, the coil could not be separated from the ICA and was therefore removed. The following day a combined retrograde venous and arterial approach was selected for selective embolization of the cavernous sinus. A 4-mm balloon was coaxially introduced through the ICA and positioned across the tear to prevent intra-arterial dislodgement of the coils deployed through the venous route. A Berenstein catheter was introduced into the right internal jugular vein. A microcatheter was coaxially introduced into the right inferior petrosal sinus, right cavernous sinus, and left cavernous sinus, where opacification of the fistula was demonstrated. Two detachable microcoils were introduced and deployed within the left cavernous sinus, with the carotid balloon inflated. Postcoiling angiography demonstrated some residual slow flow in the CCF.

Six months later the patient returned for further treatment. Left ICA angiography demonstrated residual flow in the fistula (Fig. 3C) as well as prominent cortical venous flow through the sphenoparietal plexus. The sump effect caused

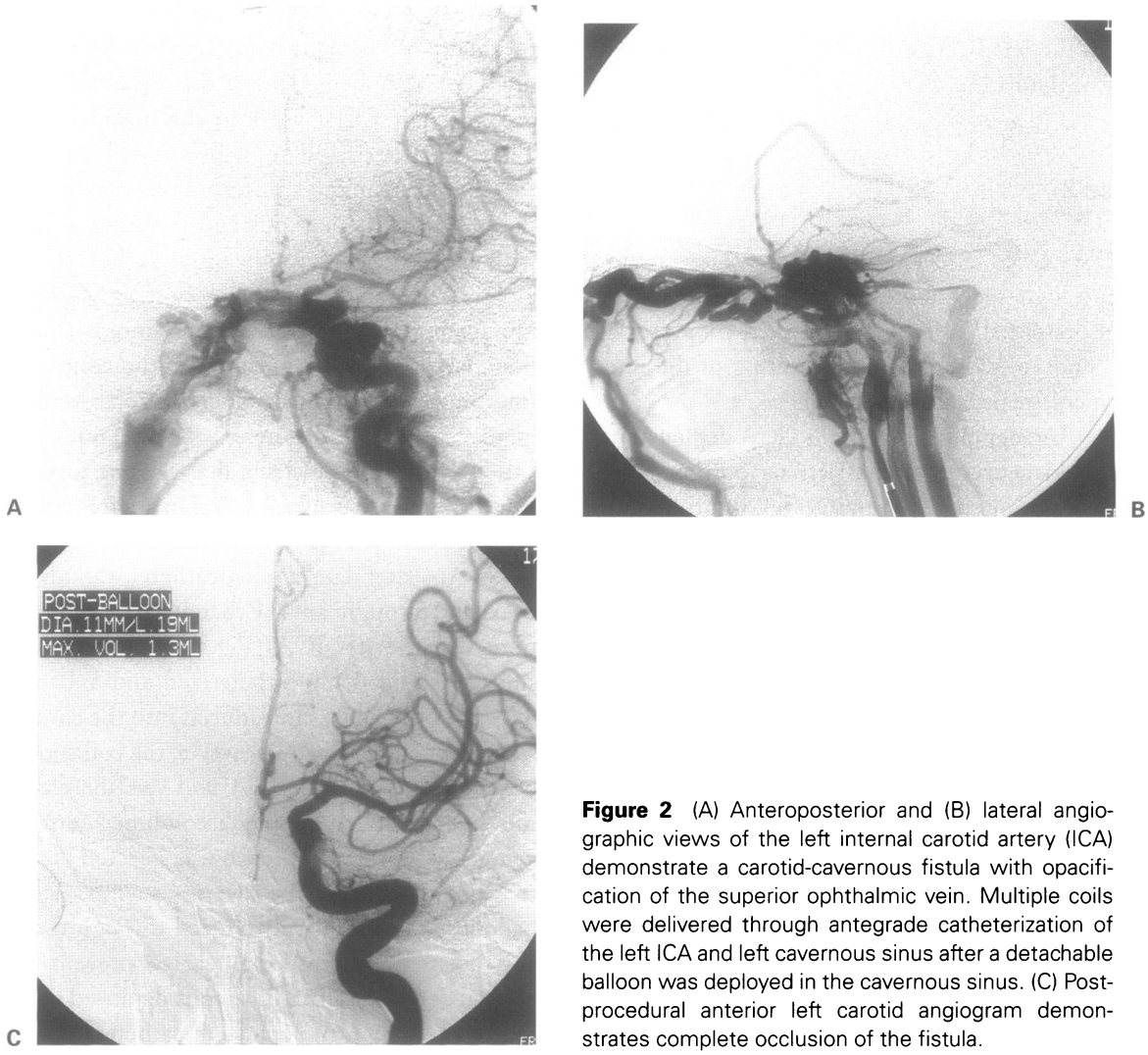

Figure 2 (A) Anteroposterior and (B) lateral angiographic views of the left internal carotid artery (ICA) demonstrate a carotid-cavernous fistula with opacification of the superior ophthalmic vein. Multiple coils were delivered through antegrade catheterization of the left ICA and left cavernous sinus after a detachable balloon was deployed in the cavernous sinus. (C) Post-procedural anterior left carotid angiogram demonstrates complete occlusion of the fistula.

markedly diminished antegrade flow in the ICA and collateral opacification of the middle cerebral artery territory during vertebrobasilar angiography. Through a left ICA guiding catheter, a detachable balloon was positioned across the fistula and inflated. Before detachment, an angiography demonstrated obliteration of the fistula, normal antegrade flow in the ICA territory, and normally delayed venous phase opacification. The balloon was detached and its therapeutic effect confirmed by an additional (three-dimensional) angiography. A month later the balloon deflated, and the procedure was repeated successfully.

Case 4

A middle-aged woman presented with right proptosis secondary to a CCF. At angiography bilateral dural indirect CCFs were demonstrated. The lesion on the left was treated with coil embolization through a left ICA access.

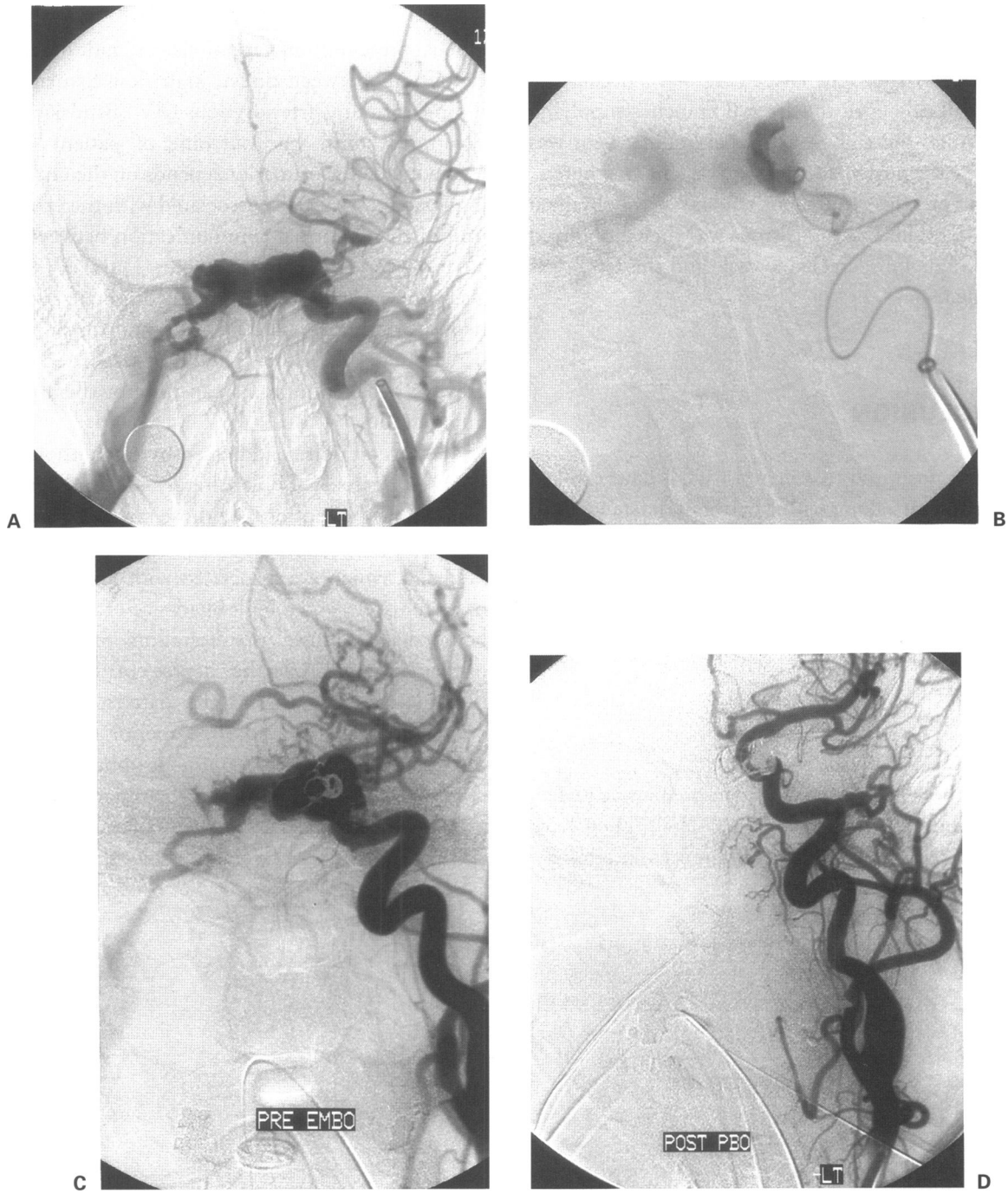

Figure 3 (A) Anterior angiogram of a left internal carotid artery (ICA) demonstrates a slow-flowing carotid-cavernous fistula (CCF) communicating with both cavernous sinuses and predominant venous drainage into the right inferior petrosal sinus. (B) At another setting, a turbo 18 microcatheter was advanced coaxially through an 8-F banding catheter positioned in the left ICA. The cavernous sinus was selectively catheterized, and coils were introduced after a balloon was positioned across the tear to prevent intra-arterial dislodgement of the coils. (C) Postcoiling angiogram demonstrates some residual slow flow in the CCF. Six months later the patient returned for further treatment. Through a left ICA guiding catheter, a detachable balloon was positioned across the fistula, inflated, and detached. (D) Antegrade flow in the ICA territory is normal on a postprocedural angiogram.

The right fistula was supplied by very small branches of the right ECA (Fig. 4A). Because of the small caliber of the arterial branches supplying the fistula, the right superior ophtalmic vein was exposed surgically, and retrograde microcatheterization of the cavernous sinus was obtained (Fig. 4B). The cavernous sinus was then embolized with Guglielmi detachable coils and the CCF was occluded.

CONCLUSION

The diagnosis and treatment of CCF have evolved in recent years along with a better understanding of their etiology and biological history. Using different endovascular routes to treat these potentially lethal lesions relies upon accurate anatomic mapping of the lesions and the ability to implement multiple strategies and, when necessary, varied expertise.

COMMENTS FOR PUBLICATION

The authors have provided a comprehensive review of carotid-cavernous fistulas (CCFs). They have emphasized the significant differences in etiology, clinical presentation, natural history, and therapeutic options between direct, high-flow fistulas and indirect or dural arteriovenous (AV) fistulas of the cavernous sinus. The outcome of patients with traumatic CCFs primarily depends on the underlying head injury usually associated with the production of the abnormal communication between the carotid artery and cavernous sinus. Dural AV fistulas of the cavernous sinus usually originate spontaneously and are associated with a much more benign natural history although associated progressive ocular symptoms can prompt therapeutic intervention.

Several points addressed by the authors deserve emphasis. CCFs are rarely life-threatening. The primary exceptions are those rare fistulas associated with retrograde leptomeningeal venous drainage, which are associated with an aggressive natural history and a high incidence of intracranial hemorrhage. Clinical manifestations of CCFs are primarily related to the degree of blood flow through the fistulas and the pattern of venous drainage. Drainage into the superior ophthalmic vein is associated with prominent ophthalmologic manifestations whereas drainage primarily into the petrosal sinus may be associated with minimal ocular findings and an intolerable, subjective bruit.

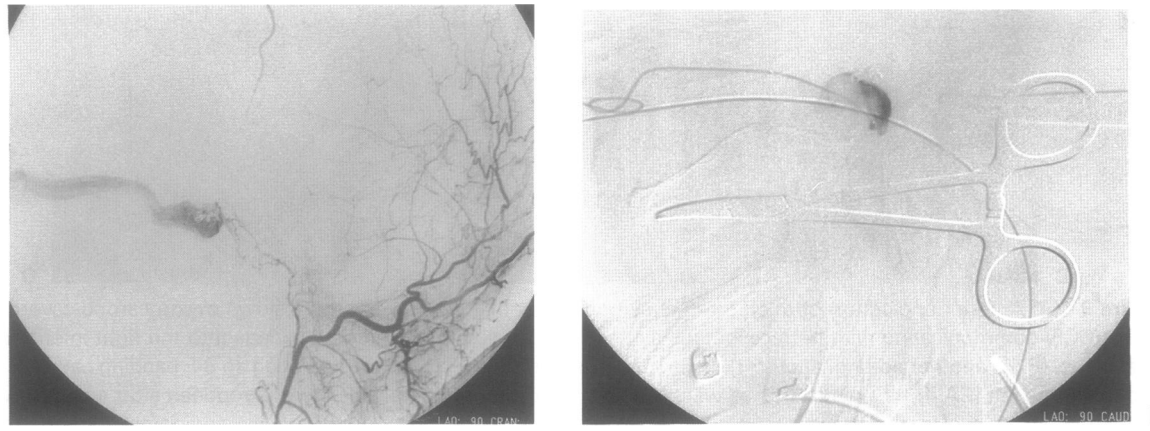

Figure 4 (A) Lateral view from angiographic right external carotid artery demonstrates a dural carotid-cavernous fistula. The right superior ophthalmic vein was exposed surgically and (B) retrograde microcatheterization of the cavernous sinus was used to deliver microcoils to occlude the fistula.

Because CCFs are rarely life-threatening, therapeutic options must be associated with low risk. Currently, most CCFs, direct or indirect, are treated by endovascular techniques; indications for surgical intervention are rare. Both transarterial and transvenous endovascular routes may be used to treat direct (type A) CCFs while indirect fistulas (types B, C, and D) are more often managed with transvenous techniques.

In 1985, when we proposed a classification system for CCFs, transarterial embolization was the primary therapeutic option. The precise arterial supply to the fistulas was much more important before transvenous endovascular techniques were available. Now, this supply to indirect fistulas is of less importance than the access to venous drainage. In the rare event that transvenous access by a catheter cannot be obtained, alternatives include surgical exposure of the superior ophthalmic vein or open craniotomy with direct puncture of the cavernous sinus to introduce embolic material using intraoperative angiography. More recently, we have abandoned these techniques in favor of percutaneous transocular puncture of the cavernous sinus through the superior orbital fissure for direct introduction of embolic material into the cavernous sinus.[1,2] The development of this technique has almost eliminated the need for surgical intervention for CCFs.

As discussed by the authors, in the rare case that requires carotid sacrifice, it is essential to demonstrate that the endovascular balloon occludes the ICA as well as the entire fistula. If the balloon is proximal to the fistula, the fistula remains open resulting in a "steal" and a high risk of cerebral ischemia. Therefore, when carotid sacrifice is the only therapeutic option, we catheterize both femoral arteries to perform contralateral carotid angiography before detaching the balloon to be absolutely certain there is no retrograde filling of the fistula.

The case examples in this report outline the various endovascular therapeutic options in the management of CCFs with differing angioarchitecture. I would point out that the 6-month delay between therapeutic interventions placed the patient in Case 3 at high risk of intracranial hemorrhage. The first therapeutic intervention left the patient with cortical venous drainage, which is associated with a very aggressive natural history. In the event that partial treatment of a CCF results in retrograde leptomeningeal venous drainage, the fistula should be excluded completely as soon as possible. In Case 4, the surgical exposure of the right superior ophthalmic vein was a perfectly appropriate therapeutic option for management of this fistula. We would now treat this type of fistula by direct puncture of the cavernous sinus through the superior orbital fissure, as described by Teng et al.[1,2]

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