Endovascular strategies for carotid cavernous and intracerebral dural arteriovenous fistulas

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Interventional neuroradiology is a less invasive modality than open neurosurgery for the treatment of a wide range of neurovascular disorders. Refinements in technique and improvements in the design of microcatheters, guide wires, and embolic materials have yielded superior clinical outcomes and provided an impetus to treat more of these lesions endovascularly rather than surgically. Endovascular therapy is the standard of care for direct and indirect carotid artery cavernous sinus fistulas and may also be curative for dural arteriovenous fistulas (AVFs) in other locations.

The authors provide review of the clinical features, pathophysiology, and management of intracranial AVFs focusing on contemporary endovascular treatment options.

KEY WORDS • embolization • carotid artery • cavernous sinus • intracranial • arteriovenous fistula • transvenous approach

Carotid artery cavernous fistulas can be classified in two ways: as “direct,” consisting of a tear in the wall of the cavernous ICA causing an arteriovenous communication with the cavernous sinus; or as “indirect,” which is synonymous with a DAVF in communication with the cavernous sinus.

Transarterial–transfistula balloon embolization for direct-type fistulas and transvenous coil embolization of indirect fistulas are often curative. Intracranial DAVFs in other locations may also be embolized transarterially or transvenously. Those that are incurable by endovascular embolization alone are usually amenable to preoperative embolization for minimization of intraoperative bleeding.

A thorough review of these lesions and the current endovascular strategies for their treatment is presented along with a range of clinical results.

DIRECT CACFS

Direct CACFs may be spontaneous or acquired lesions.

Abbreviations used in this paper: CA = carotid artery; CACF = CA cavernous fistula; CCA = common CA; DAVF = dural arteriovenous fistula; ECA = external CA; ICA = internal CA; VA = vertebral artery.

Closed head injury in which the base of the skull has fractured and the ICA sheared from its dural attachment to the foramen lacerum and anterior clinoid process are the most common cause of this lesion. Alternative origins include rupture of an intracavernous ICA aneurysm, iatrogenic injury during transsphenoidal pituitary resection, fibromuscular dysplasia, or collagen vascular diseases such as Marfan syndrome and Ehlers–Danlos syndrome.^{6,8,27}

Clinical Presentation

Direct irritation or associated trauma to the cranial nerves traversing the cavernous sinus can result in third, fourth, fifth, and sixth nerve palsies. Retrograde flow of arterialized blood through the superior and inferior ophthalmic veins into the orbit may cause proptosis, chemosis, pain, and reduced visual acuity. Retinal perfusion pressure can be compromised to the extent of inducing permanent blindness.⁹ These ocular symptoms may be exacerbated by hypoplasia or thrombosis/occlusion of the inferior petrosal sinuses that can occur as a result of prolonged arteriovenous artery–vein communication.

Sumping of ICA blood flow into the cavernous sinus may compromise antegrade intracranial flow and precipitate hemispherical hypoperfusion if the circle of Willis collateral structures are inadequate.

Treatment Options

Intraocular pressures exceeding 40 mm Hg or rapidly progressive visual deterioration are indications for emergency therapy to avoid permanent loss of vision. Topical acetazolamide and β -agonists can reduce intraocular pressure, and lateral canthotomy may be a temporary measure for decompression of orbital contents prior to definitive closure of the fistula.

Diagnostic angiography can demonstrate the extent of intracerebral collateral pathways, the severity of arteriovenous communication, and residual antegrade flow in the ICA beyond the fistula. Various maneuvers can be useful in identifying the exact location of the hole in the ICA. The Mehringer maneuver (Fig. 1 *left*) consists of inducing CCA compression during contrast injection into the ICA.²⁸ Compression of the CA during VA injection (Huber maneuver) may delineate the fistula site through retrograde opacification via the posterior communicating artery (Fig. 1 *right*).²⁰

The preferred endovascular procedure is transarterial-transfistula balloon embolization to preserve the ICA while the fistula is occluded using detachable silicone balloons passed from it, through the fistula, and into the cavernous sinus. A detachable silicone balloon is tested for leakage and mounted on a suitable microcatheter. A 0.010-in guide wire can be used to facilitate passage of the catheter-mounted detachable silicone balloon in the presence of CA tortuosity. The balloon is gently inflated as it approaches the fistula site to facilitate its passage through the arterial tear into the cavernous sinus. Once there, it is inflated incrementally during small test injections of contrast through the guiding catheter to determine any change in the degree of shunting and/or compromise of antegrade flow in the ICA. After the correct volume for occluding the fistula while preserving ICA patency has been determined, the balloon is detached by gently pulling back on the catheter (Fig. 2). Filling the balloon with hydroxyethylmethacrylate, which polymerizes within an hour at body temperature, makes the balloon less likely to deflate or migrate than iodinated contrast alone.

If transarterial-transfistula balloon occlusion is not feasible, transvenous coil embolization may be performed us-

ing a microcatheter positioned in the cavernous sinus via the inferior petrosal sinus. Even when the inferior petrosal sinus cannot be visualized angiographically, a channel can usually be found by probing with a steerable guide wire in up to 75% of cases.^{13–15,48} Direct percutaneous access to the superior ophthalmic vein in the orbit or, less commonly, the angular facial vein has been reported.^{25,39} Pushable fiber coils are then positioned within the cavernous sinus while avoiding coil prolapse into the ICA through the fistula.

Sacrifice of the ICA may be considered if adequate intracranial collateral pathways exist. Balloon test occlusion can be useful in this regard. Care must be taken to avoid occluding the ICA proximally without complete fistula closure because retrograde shunting could further compromise cerebral perfusion. Using three detachable silicone balloons—one above, one at, and the last one proximal to the site of the fistula—minimizes the risk of this occurrence.

Anecdotal reports of placing a covered stent across the fistula and stent-assisted coil embolization have been described.^{22,23,36,47}

INTRACRANIAL DAVFS

Dural arteriovenous fistulas account for 10 to 15% of all intracranial arteriovenous shunts.³² They are acquired lesions, thought to originate from an insult to a dural venous sinus that stimulates an inflammatory response with subsequent neovascularization, angiogenesis, and development of pathological shunts at the arteriolar level.³¹ The initial insult may be thrombophlebitis, trauma, intracranial surgery, or dural venous thrombosis. Alternatively, the fistula could be the initial event with turbulent arterialized blood flow in the dural venous sinus inciting a process of thrombosis, stenosis, and occlusion.^{31,40}

The most common presentation is in perimenopausal women and is least common in men (27%), suggesting an underlying hormonal influence.^{32,33,38,42} The transverse and sigmoid sinuses are most commonly involved and followed by the cavernous sinus, the latter also referred to as indirect CACFs.

Spontaneous resolution of angiographically proven DAVFs has been reported to occur in 5 to 10% of cases.

Classification of DAVFs

Of the various classification systems for DAVFs, perhaps the most widely accepted is that proposed by Merland, et al.,²⁹ which indicated that the risk of intracranial hemorrhage is related to the venous drainage pattern. Low-risk fistulas exhibit antegrade venous drainage into a venous sinus whereas high-risk fistulas exhibit reflux into cortical vein(s) from the dural venous sinus, or direct drainage into a cortical vein when the adjacent sinus is occluded (Fig. 3 *upper* and *center*). The cortical veins develop tortuosity, stenoses, and venous aneurysms that are potential sites of hemorrhage.

Ongoing pathophysiological changes may occur with persistent arteriovenous fistulas and result in venous stenoses and sinus occlusions that develop in cases of low-risk fistulas, which are then transformed into higher-risk lesions. Any change in the patient's symptoms such as

Fig. 1. Techniques for enhanced localization of fistula site. *Left*: The Mehringer maneuver. Compression of the ipsilateral CCA during slow ICA injection of contrast defines the proximal extent of the fistula site. *Right*: The Huber maneuver. Compression of the ipsilateral CA during VA injection demonstrates the distal extent of the fistula site.

Fig. 2. Angiogram obtained in a 20-year-old man who sustained a gunshot wound to the head. *Left and Center:* Anteroposterior (*left*) and lateral right (*center*) ICA angiograms revealing a direct CACF. Note the dilated superior ophthalmic veins bilaterally (*arrows*) and outline of the bullet (BT) lodged in the left temporal lobe. *Right:* Following transarterial–transfistula detachable balloon embolization (*arrowheads*), there is no residual arteriovenous fistula.

worsening headaches or change in the quality of pulsatile tinnitus may indicate alteration in the venous drainage pattern and risk of hemorrhage.⁴⁴

Both computerized tomography and magnetic resonance imaging may be useful in diagnosis, but conventional catheter arteriography is necessary for definitive diagnosis, risk assessment, and treatment planning.^{5,17,43,45,46}

Presentation of DAVFs

In patients with low-risk fistulas, the lesions may be discovered incidentally during angiography or present with pulsatile tinnitus or headache. High-risk fistulas may present with hemorrhage into the subarachnoid space or brain parenchyma resulting in focal neurological deficits or death. Cortical venous drainage with associated venous hypertension is a well-known cause of neurological deficit and severe headache.²¹ High-risk DAVFs therefore warrant aggressive therapy, either endovascular or surgical, whereas low-risk fistulas can undergo palliative treatment if symptoms such as pulsatile tinnitus are troublesome.⁴

Carotid artery cavernous fistulas may present with ophthalmoplegia, proptosis, chemosis, pain, and reduced visual acuity. Pulse-synchronous bruit is an inconstant finding, occurring in 40 to 50% of patients.¹⁰

Treatment Options

A complete diagnostic cerebral angiography examination is performed to identify the arterial feeding structures in addition to the site of arteriovenous communication within the diseased venous sinus. Typically the ECA is supplied by the ascending pharyngeal, middle and accessory meningeal, occipital, and distal internal maxillary arteries.

The plethora of tortuous feeding arteries often limits the success of transarterial embolization as a definitive treatment. Cure may be achieved if the number of feeding arteries is limited and permanent embolic agents such as n-BCA glue or ethanol can be used safely. Transarterial preoperative embolization involving particulate emboli and coils is of low risk and can significantly reduce intraoperative bleeding.

Arterial feeding structures from branches of the ICA or VAs are usually not amenable to embolization unless

microcatheter-based stable superselective catheterization can be performed because reflux of embolic material cannot be afforded. Ethanol may be more suitable in such cases because reflux into the parent artery would be rapidly diluted by antegrade blood flow. Successful embolization of cavernous CA branches has been reported;¹¹

Fig. 3. Angiograms obtained in a 37-year-old woman who experienced headaches and confusion. *Upper and Center:* Anteroposterior (*upper left*) and lateral (*upper right*) ECA angiograms revealing a DAVF at the junction of the right transverse and sigmoid sinuses supplied by numerous feeding arteries. Note the grossly abnormal venous drainage with occlusion of the right sigmoid sinus downstream from the fistula as well as retrograde opacification of the superior sagittal sinus, straight sinus, and reflux into dilated, tortuous cortical veins. *Lower:* Lateral CCA angiogram obtained after transvenous placement of 30 pushable fiberoptic coils into the fistula and adjacent venous sinus, demonstrating no residual arteriovenous shunting. Note the outline of the coil mass.

Fig. 4. Angiograms obtained in a 41-year-old man who presented with left-sided hemiparesis due to a right thalamic hemorrhage. *Upper Left and Right:* Lateral ICA (*upper left*) and ECA (*upper right*) angiograms revealing a DAVF of the superior petrosal sinus supplied predominantly by branches of the middle meningeal, occipital, and ophthalmic artery (deep recurrent meningeal branch). The superior petrosal sinus is occluded with consequent cortical venous drainage via the anastomotic lateral mesencephalic vein, which harbors a varix (*arrow*). *Lower Left:* Unsubtracted anteroposterior angiogram demonstrating a microcatheter with its tip at the fistula site after having been navigated through the cortical vein and varix via the right sigmoid sinus. *Lower Right:* After embolization with 15 electrolytically detachable coils, a lateral CCA angiogram demonstrated no residual fistulas.

however, ethanol-based embolization of the meningohypophyseal trunk has resulted in posterior pituitary dysfunction and diabetes insipidus.³⁴

Conservative treatment for indirect CACFs consists of self-applied CA compression by using the contralateral hand every 30 minutes for 1-minute intervals during waking hours. This can be successful in as many as 30% of cases.^{9,14,16} By compressing the CA sheath contents, antegrade flow is diminished and venous pressure elevated, promoting thrombosis of the fistula. Should hemiparesis result from CA compression, the contralateral arm will fall away, thus reestablishing cerebral perfusion.

In transvenous embolization of DAVFs, coils are deposited at the site of arteriovenous shunting. When possible, it can result in cure without having to involve the myriad of feeding arteries (Fig. 3 *lower*). When transvenous access is prevented by occlusion of the recipient venous sinus, the site of shunting can be occasionally accessed transarterially or via a cortical draining vein (Fig. 4). Transcranial embolization after limited craniotomy and direct venous sinus catheterization has been described.¹⁸ Embolization of indirect CACFs by direct puncture through the superior orbital fissure⁴⁰ and transvenous access to the cavernous sinus via the facial and superficial temporal veins has been reported.³

Although detachable coils have the advantage of being able to be repositioned or removed if placement is unsatisfactory, they are significantly more expensive and less thrombogenic than pushable fibered coils. A combination of the two types may be placed as a compromise.

RESULTS OF ENDOVASCULAR TREATMENT

Chemosis and proptosis typically improve rapidly after successful obliteration of CACFs. Visual acuity and ophthalmoplegia may take longer to resolve and can be exacerbated in the short term by thrombosis and associated swelling within the cavernous sinus.³⁷ In a retrospective review of 135 patients with indirect endovascularly treated CACFs, 121 patients (90%) were clinically cured after a mean follow-up period of 56 months.³⁰ In a study of 133 patients undergoing transvenous embolizations for indirect CACFs, good clinical recovery (modified Rankin Scale score of 1–2) was demonstrated in more than 97% of the population at long-term follow-up examination.^{30,35} Although uncommon, reversal of blindness after successful embolization of direct and indirect CACFs has been described.²

In nine patients in whom transvenous coil embolization was performed for indirect CACFs, two patients suffered

temporary deterioration of ocular motor function.¹ There is some evidence that this effect may be limited by the use of corticosteroid agents or heparin in the postoperative period.²⁴

High success rates and durable results in direct CACFs can also be achieved when using transarterial-transfistula balloon embolization, and the ICA can also be preserved.⁷ In a review of 87 patients with direct CACFs, detachable balloon embolization allowed ICA preservation; during the mean follow-up period of 10 years, diplopia improved in all patients and there was no recurrence of bruit, chemosis, proptosis or conjunctival edema.²⁶

In a recent study of 135 patients in whom endovascular treatment was conducted for intracranial DAVFs, cure was achieved in 74 patients and significant improvement in 54.¹⁹ In another series of 11 patients with DAVFs of the transverse and sigmoid sinuses, preoperative transarterial embolization was undertaken in seven patients. Endovascular cure was achieved in one of four patients who underwent transvenous embolization, and arteriovenous communication was significantly reduced in the remaining three patients.¹⁵ In our experience of treating 18 patients with DAVFs of the superior petrosal sinus, four patients (22%) were cured by transvenous embolization alone. The remaining 14 patients (78%) underwent preoperative transarterial embolization followed by successful resection of the fistula.

CONCLUSIONS

Neurovascular embolization therapies can have a role in the treatment of all intracranial arteriovenous fistulas. It is the preferred treatment for both direct and indirect types of CACFs and may be curative for intracranial DAVFs in other locations, particularly when transvenous access to the fistula site can be achieved. In cases in which endovascular cure is not possible, embolization alone can be palliative, or it can be used preoperatively for minimization of intraoperative hemorrhage. Future advances in endovascular equipment in conjunction with technical refinements will expand on the current applications and further minimize procedure-related complications.

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