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Review Article

Transvenous Approaches to Embolization of Dural Arteriovenous Fistulae of the Cavernous Sinus

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Dural arteriovenous fistulae of the cavernous sinus (CS) (previously often referred to indirect carotid cavernous fistulas) are rare vascular shunts involving meningeal branches and osseous branches of the external or internal carotid arteries and the CS. They typically present with ocular symptoms including pain, conjunctival injection, and proptosis. Left untreated there may be a risk of vision loss, and fistulas with cortical venous reflux through either the deep or superficial venous system may cause intracranial venous congestion or hemorrhage. Endovascular embolization is the standard treatment, and while transarterial routes may appear possible, transarterial embolization has considerable risks of ischemic complications. Conversely, transvenous routes achieve a high rate of fistula occlusion with a low risk of peri-procedural morbidity. Procedural success depends on identification of the venous outflows from the fistula and localization of the fistulous point, to select the best route of access to the CS, including the inferior petrosal sinus (IPS), intercavernous sinus, or superior ophthalmic vein, among others. Even if the IPS is not visualized, it may be possible to recanalize it to gain access to the CS. Embolization can be performed with a combination of coils, fibered coils, and liquid embolic agents, focusing on occlusion of the fistulous point or blocking high-risk venous outflow pathways. In this review we will highlight procedural pearls and potential pitfalls and our typical approach to these lesions based on illustrative examples.

Keywords ▶ dural arteriovenous fistula, cavernous sinus, carotid cavernous fistula, endovascular, embolization

Introduction

A carotid cavernous fistula (CCF) is an abnormal connection between the carotid artery or its branches and the cavernous sinus (CS). This connection may arise from a defect in the wall of the cavernous segment of the internal carotid artery (ICA), creating a direct CCF, or from meningeal branches of the ICA or external carotid artery (ECA), creating an indirect CCF. Indirect CCFs thus are true dural arteriovenous fistulas (DAVFs) and are the principal focus of this review.

Classification

Although the Barrow system[1)] has conventionally been used for classifying CCFs, it has limited utility in describing DAVFs of the CS. Direct and indirect CCFs are distinct disease processes, with different etiologies and treatment strategies. Moreover, classification of CS DAVFs by arterial supply may be less useful when transvenous approaches are often preferred for treatment, so other systems have developed focusing on the venous drainage of the fistula. Yu et al.[2)] described fistulas based on whether the anterior and posterior compartments of the CS are opacified, and then subtyped them by drainage according to involvement of the facial vein (FV) and inferior petrosal sinus (IPS). The Thomas system[3)] divides CS DAVFs based on the direction of their drainage, which is shown to correlate with symptomatology and treatment approach[4)] (**Table 1**). The Borden and Cognard

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Table 1 Thomas system for classification of CCFs

Characteristics	Type	Venous drainage
Indirect (dural)	Type 1	Posterior/inferior drainage only
	Type 2	Posterior/inferior and anterior drainage
	Type 3	Anterior drainage only
	Type 4	Retrograde drainage into cortical veins ± other routes
Direct	Type 5	High flow direct shunt between cavernous ICA and CS (Barrow Type A) ± multiple route of venous drainage

Thomas classification for carotid cavernous fistulas.[3] Anterior drainage: superior and inferior ophthalmic veins; Cortical drainage: superficial middle cerebral veins, perimesencephalic, and cerebellar; posterior and inferior drainage: inferior petrosal sinus, superior petrosal sinus, pterygoid, and parapharyngeal plexus. CCF: carotid cavernous fistula; CS: cavernous sinus; ICA: internal carotid artery

classifications used elsewhere for DAVFs are also applicable but not specifically suited to the anatomy of the CS. The Barrow classification system is of limited value in the assessment of dural AVF of the CS.[5–7]

Etiology

It is theorized that spontaneous venous thrombosis of the CS may be a contributing event to the formation of a CS DAVF, with the fistula forming from collateralization induced by the thrombus.[6] There is some evidence that a hypercoagulable state, such as Factor V Leiden[8] and other hereditary thrombophilias,[9,10] is associated with DAVF formation, but these studies are not specific to fistulae of the CS. Pregnancy and childbirth are known predisposing factors, which may represent a combination of hormonal factors and the physiologic stress of delivery. The higher incidence in menopausal and postmenopausal women may also be hormonally mediated, but this is superimposed on the increasing incidence with age of CS DAVFs in both men and women that may result from atherosclerotic changes in the vessel wall.[6]

Epidemiology

There is relatively little data on the population prevalence of CS DAVFs. An estimate in the Japanese population placed the overall incidence of DAVFs at 0.29 per 100000 adults per year.[11] CS DAVFs represent only a fraction of these cases but were found to be proportionately more numerous in Japan than in European or North American studies. Benndorf[6] has summarized several case series of CCFs and reports considerable variation in the distribution of the Barrow fistula types between studies. Among spontaneous CCFs, DAVFs account for approximately two thirds of cases.[12]

Presentation

Patients with a CCF typically present with ocular symptoms, including chronic eye redness, periorbital swelling, pain, and diplopia. Physical examination may show corkscrew episcleral vessels, chemosis, asymmetrically elevated intraocular pressure, and proptosis.[13] In some cases, an orbital bruit may be auscultated. Retinal findings include retinal vein dilation, intra-retinal or pre-retinal hemorrhage, and disc hyperemia.[14] Left untreated, symptoms can progress to permanent loss of vision through retinal vein occlusion, ischemic optic neuropathy, or glaucomatous optic nerve damage. As with other DAVFs pulsatile tinnitus is occasionally reported (e.g., the case shown in **Fig. 1**). CS DAVFs with cortical venous reflux present a risk for venous congestion and intracranial hemorrhage, which can occur without associated ocular findings.[15]

Imaging diagnosis

Clinical findings suspicious for a CS DAVF should prompt imaging investigation, which would generally start with CTA or MRA. On CTA, depending on contrast timing the degree of intracranial venous enhancement can be variable, but other sinuses can be used as a control to assess the CS for relative hyperenhancement (**Fig. 1A**). On time-of-flight MRA, any high signal in the CS is abnormal and should raise suspicion for a fistula (**Fig. 1B**). The CS itself may be expanded, and for anteriorly draining fistulae, the superior ophthalmic vein (SOV) may also be dilated (**Fig. 2B**). A recent study on the role of CTA in the diagnosis of CCFs found that SOV early enhancement and dilation had a 100% sensitivity and 77.8% specificity, while arterial phase enhancement in the CS had 88.9% sensitivity and 66.7% specificity.[16] On ocular ultrasound the SOV may show flow reversal with blood traveling away from the orbital apex. Supporting nonvascular features include edema and stranding in the retrobulbar fat, proptosis, and extra-ocular muscle enhancement. Dynamic contrast-enhanced MRA will show relative early enhancement of the affected CS and can be performed to assess the arterial supply and venous drainage of the fistula, although due to its relatively low spatial and temporal resolution it usually does not obviate the need for DSA, which remains the definitive modality for diagnostic evaluation.

Once identified on cross-sectional imaging, a CS DAVF should be further investigated with DSA for treatment planning. The DSA needs to include angiograms of the bilateral ECAs and ICAs, with selective catheterization of ECA branches as required with evaluation of both arterial

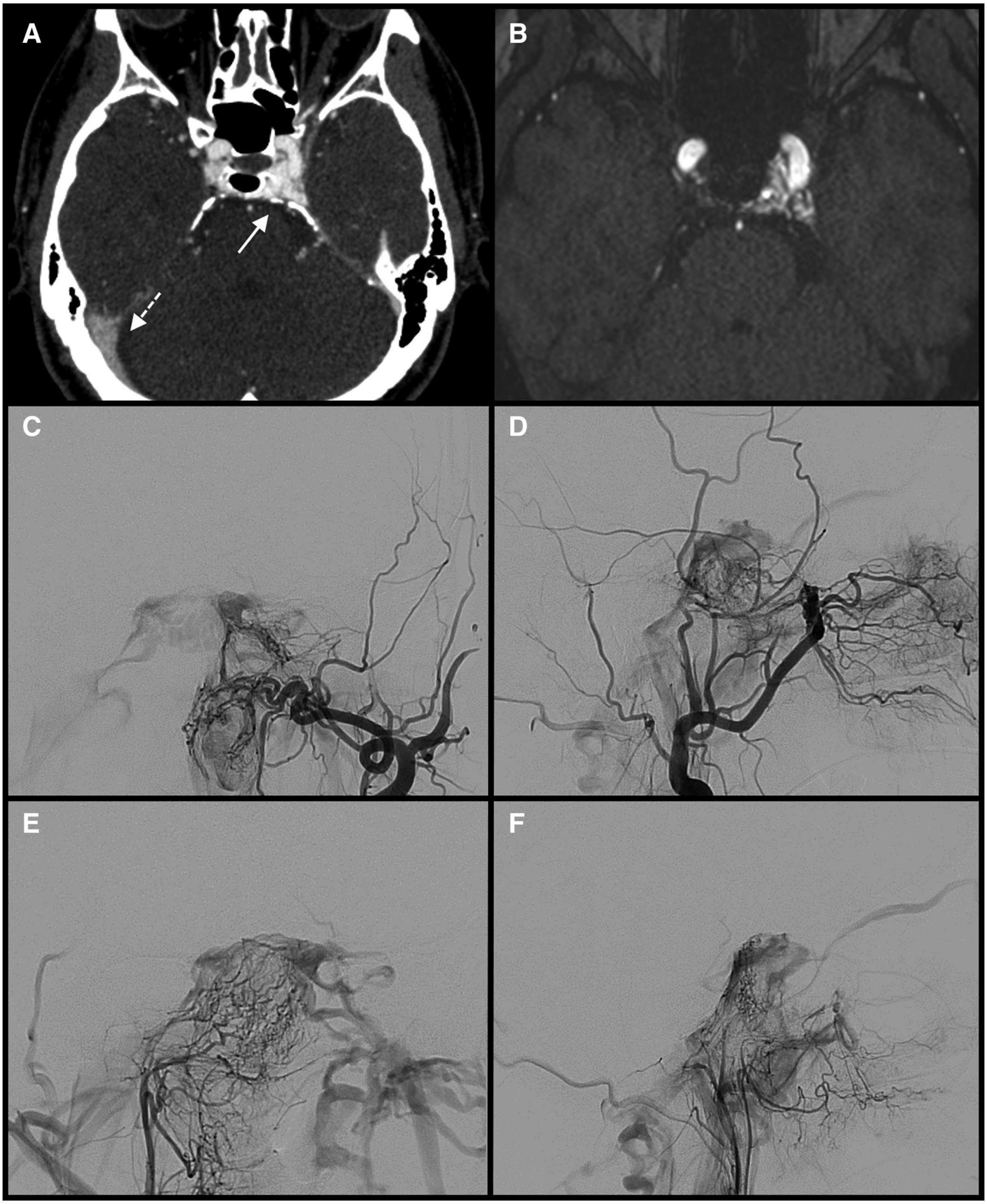

Fig. 1 A 46-year-old man presented with pulsatile tinnitus. (**A**) CTA showed early enhancement of the left CS, consistent with a CCF (compare the attenuation of the left CS marked with a solid arrow to the right transverse sinus marked with a dashed arrow) (**B**) Time-of-flight MRA showed abnormal high signal in the left CS. DSA of the (**C**) left ECA AP view, (**D**) left ECA lateral view, (**E**) right ECA AP view, and (**F**) right ECA lateral view demonstrates a left CS DAVF supplied by bilateral ascending pharyngeal and internal maxillary artery branches. There was minor contribution from meningeal branches of the bilateral ICAs (not shown). Drainage is via the intercavernous sinus, inferior petrosal sinus, and pterygoid plexus, with a small amount of relatively delayed drainage through the superior ophthalmic vein. AP: anterior-posterior; CCF: carotid cavernous fistula; CS: cavernous sinus; DAVF: dural arteriovenous fistula; ECA: external carotid artery; ICAs: internal carotid arteries

and late venous phases. Angiograms of the vertebral arteries should also be obtained: while vertebral artery contribution to a CS DAVF is extremely rare, it is important to exclude DAVFs at other intracranial locations, and vertebral-venous fistulas have been described as a cause for ocular symptoms mimicking a CCF.[17] Features to be

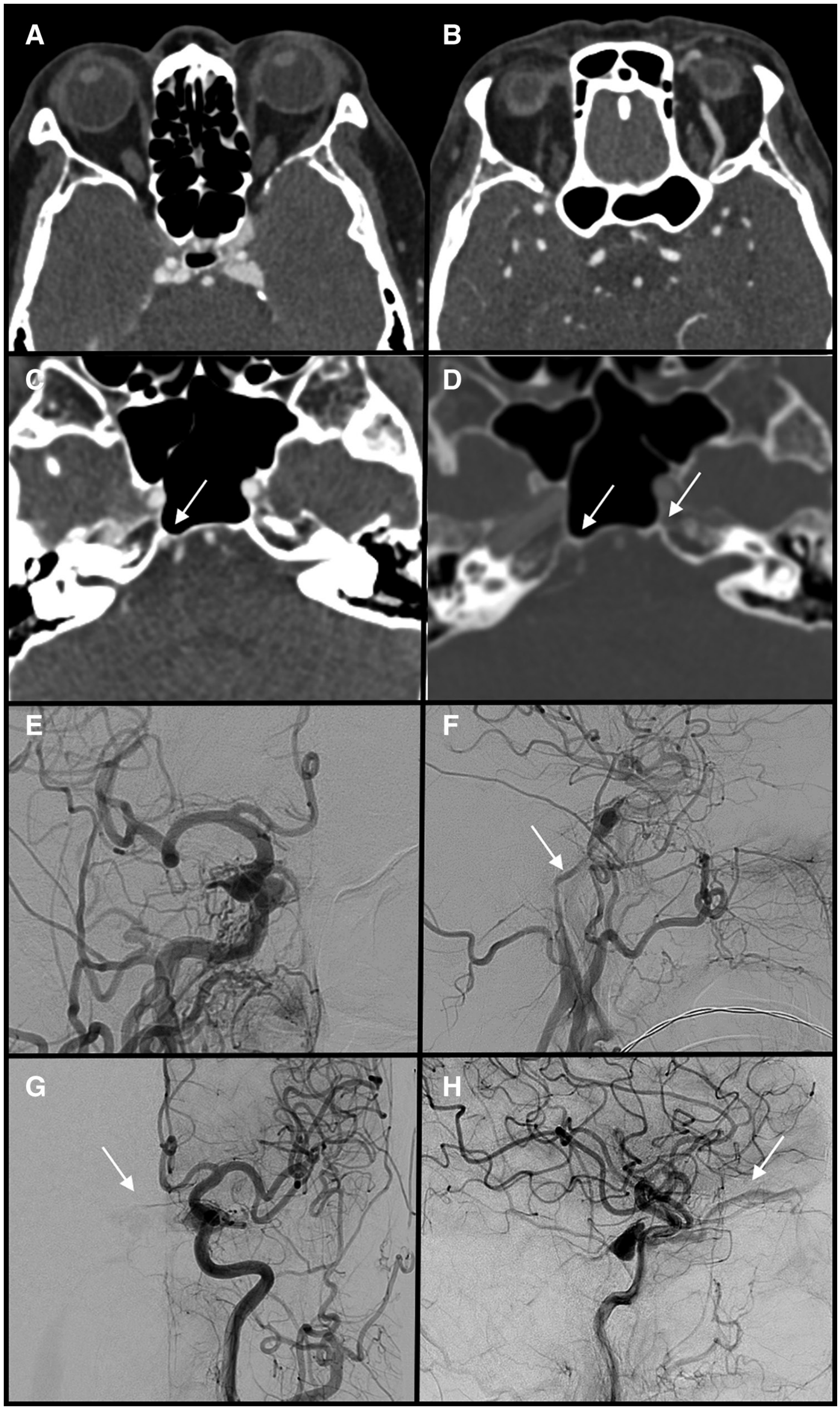

Fig. 2 A 71-year-old woman presented with several months of left orbital swelling and pain. CTA showed early enhancement of the bilateral cavernous sinuses (**A**) and dilation of the left superior ophthalmic vein (**B**). (**C**) Contrast is seen in the right IPS (arrow) but not in the left. (**D**) However, the presence of bony notches in the clivus bilaterally (arrows) indicates that the left IPS is developmentally present but may have thrombosed. DSA of the right CCA in the AP (**E**) and lateral (**F**) projections shows supply to the right-sided fistula from the meningohypopheal trunk and internal maxillary artery, with drainage via the right IPS (arrow in **F**). DSA of the left CCA in the AP (**G**) and lateral (**H**) projections shows supply to the right-sided fistula from a dural branch of the left ICA (arrow in **G**), while venous drainage of the left-sided fistula is through the superior ophthalmic vein (arrow in **H**). AP: anterior-posterior; CCA: xxx xxx xxx: ICA: internal carotid artery; IPS: inferior petrosal sinus

assessed on DSA include the routes of arterial supply (which often include the inferolateral trunk and meningohypophyseal trunks of the ICA, the middle meningeal artery, the accessory meningeal artery, the internal maxillary artery via the artery of foramen rotundum, and the ascending pharyngeal artery via the neuromeningeal trunk), venous outflow (which may include the IPS, superior petrosal sinus [SPS], SOV, intercavernous sinus [ICS], and sphenoparietal sinus), and the presence or absence of cortical venous reflux (which can be seen into the uncal veins, superficial middle cerebral veins [SMCVs], petrosal vein, prepontine bridging veins, or inferior cerebral veins). The routes of venous outflow provide possible endovascular approaches for transvenous embolization, and the sites of cortical venous reflux are priorities for occlusion during treatment.

Goals of Treatment

The primary goal in treatment of a CS DAVF is to occlude the fistulous point, thus curing the disease, but if this is not possible, the venous reflux to the brain should be obliterated to remove the risks of intracranial hemorrhage and venous congestion.

Angiographic and clinical cure of a CS DAVF is best achieved by localized embolization of the fistulous point within the CS. Identification of the exact fistulous point is therefore of utmost importance but may be challenging. Features to assess include:

1) The site of earliest opacification in the CS (facilitated by performing the DSA at high frame rate).
2) The confluence of enlarged arterial feeders to the fistula, and convergence on a single venous drainage.
3) Dilution of contrast from dark gray to light gray at the site of admixture with non-opacified venous blood.

Pinpointing the site of the fistula enables the embolization microcatheter to be navigated as close as possible to the fistulous point, and thus the use of a minimum volume of embolic material. Smaller volumes of embolic material are associated with a lower risk of procedural complications such as cranial nerve palsies from mass effect in the CS.[18]

Access Routes to the CS

As with other DAVFs, those of the CS can be treated via a transarterial or transvenous route, but there is a strong preference for transvenous embolization whenever possible. A rich network of arterial collaterals exists near the CS, including the dangerous anastomoses between the ECA and the ICA.[19] Embolization through the ECA branches that supply the CS DAVF therefore risks migration of embolic material into the ICA with the potential for stroke from further migration into the anterior cerebral artery or middle cerebral artery.[20] Even with detailed mapping of the arterial anatomy before treatment, these ECA–ICA anastomoses may not be evident and only reveal themselves during embolization in response to alterations in flow dynamics.

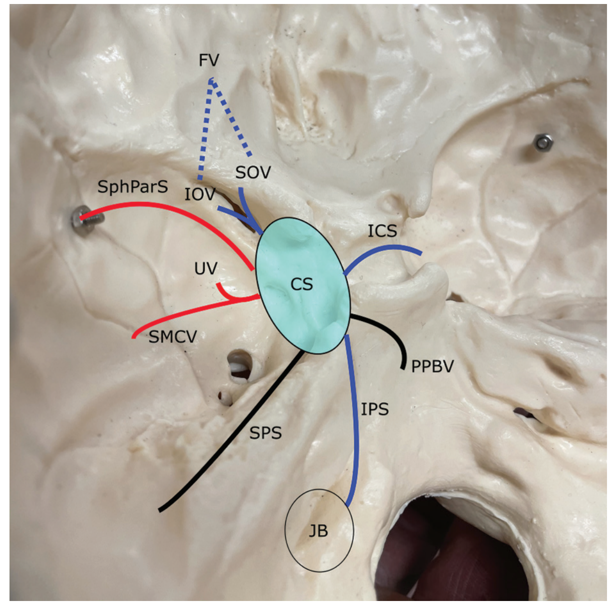

Fig. 3 Venous connections to the CS. Paths shown in blue are commonly used access routes to the CS for endovascular embolization of CS DAVFs. Paths shown in red are potentially dangerous routes for venous reflux, with elevated risks for intracranial hemorrhage or venous congestion if involved in draining the fistula. CS: cavernous sinus; DAVFs: dural arteriovenous fistulas; FV: facial vein; ICS: intercavernous sinus; IOV: inferior ophthalmic vein; IPS: inferior petrosal sinus; JB: jugular bulb; PPBV: prepontine bridging veins; SMCV: superficial middle cerebral vein; SOV: superior ophthalmic vein; SphParS: sphenoparietal sinus; SPS: superior petrosal sinus; UV: uncal vein

Transvenous approaches do not present this risk of ICA embolism to the same degree, but it remains essential to monitor for arterial migration of embolic material from the venous side past the fistulous point. A variety of transvenous approaches are possible (**Fig. 3**) and depend on the outflow from the fistula and the ease of catheter navigation, as discussed below.

IPS access

For most posteriorly draining CS DAVFs (Thomas Types 1, 2, and some Type 4), access via the ipsilateral IPS is the most facile approach and provides relatively direct access to the CS. The IPS exits the CS posteriorly and inferiorly, and then travels in the inferior petrosal sulcus, a bony notch lateral to the clivus at the junction of the petrous part of the temporal bone and the basilar part of the occipital bone, before draining into the jugular bulb (JB). The IPS occasionally terminates in the internal jugular vein (IJV) well below the JB; so if the IPS is not identified connecting with the JB, the IJV more inferiorly should also be explored.

On CTA, the IPS is often visualized in the inferior petrosal sulcus (**Fig. 2C**), confirming patency for access to the IPS. However, if the IPS is not visible directly, the presence of the inferior petrosal sulcus (**Fig. 2D**) provides evidence that the IPS was present during development. Even if the IPS subsequently thrombosed, it may be possible to recanalize it with a wire and catheter to gain access to the CS, as discussed further below.

Intercavernous access

In some cases, the IPS may be hypoplastic or thrombosed on the side of the fistula but accessible on the other side. It may be possible to then reach the affected CS from the contralateral CS, either through the clival venous plexus posterior to the sella, or through the ICSs, variable small venous channels located anterior, inferior, and posterior to the pituitary. They are not generally resolvable on cross-sectional imaging; although if hyperenhancement is seen in both the left and right CS, this suggests either bilateral fistulae or venous drainage into the contralateral CS through the ICSs or clival venous plexus. On DSA the ICSs are sometimes faintly resolvable, best seen on the anterior-posterior (AP) view crossing midline near the floor of the sella turcica.

SOV access

For anteriorly draining CS DAVFs (Thomas Type 3) without achievable access via the IPS, the CS can be reached via the SOV (or less commonly the inferior ophthalmic vein [IOV]). The SOV and IOV connect to the angular vein, a branch of the FV that itself arises off the IJV near the angle of the mandible or the external jugular vein (in 5% of people).[21]

Other routes

The three routes described above are generally the safest and most straightforward means of accessing the CS and should be considered first in any CS DAVF embolization, but if none of them are technically feasible several other approaches have been described. In a series of 44 patients, Yoshida et al. found that transvenous embolization of CS DAVFs was feasible via the IPS in 90% of cases, although one patient required access through the SPS and one through the FV.[22]

Navigation of the facial and angular veins via a traditional transfemoral venous approach may be limited by the small caliber of the vein, so direct puncture of the SOV is an alternative. This can be performed by surgical cut down or percutaneous micropuncture under fluoroscopic guidance.[23] Similarly, if transfemoral venous access to the FV is limited by tortuosity at its origin, surgical cutdown[24] or ultrasound-guided direct puncture[25] of the FV are options. Access to the SOV may also be achieved by direct cannulation of the superficial temporal vein.[26] Access to the CS by cannulation of an emissary vein of the foramen ovale[27] has been described in a case report, when no other venous route to the CS was accessible. In some cases, the contralateral CS may be accessed by catheterizing the IPS and navigating the catheter across midline in the basilar plexus.[28]

In patients with Thomas Type 4 fistulas and predominantly cortical venous drainage, transfemoral venous access to the CS has been described through cortical veins such as the vein of Labbe and SMCV.[29] Hybrid open surgical and endovascular strategies include image-guided burr hole placement for direct catheterization of the sylvian vein.[30]

Choice of Embolic Materials

Embolization of CS DAVFs may be achieved through deployment of coils, n-butylcyanoacrylate (NBCA) glue, ethylene vinyl copolymer such as Onyx (Medtronic, Minneapolis, MN, USA) or Squid (Balt, Irvine, CA, USA), or a combination of materials. Coils are typically the initial embolic material of choice and can be used to occlude or partially occlude the venous outflow from the CS to reduce the rate of blood flow. Dense packing of conventional platinum coils physically occludes the venous outflow, and this can be supplemented with fibered coils to promote thrombosis.

Sufficiently dense packing of coils at the site of venous outflow and the fistulous point may suffice to cure the fistula without the need for liquid embolic agents, although if needed these agents can be used to consolidate the coil mass and reflux into the fistulous point. Glue has the advantage of being inherently thrombogenic, although only a single injection is possible before the catheter must be withdrawn. The ethylene vinyl copolymer agents are not thrombogenic but do enable repeated injections through the same catheter, allowing the agent to migrate toward the fistulous point once venous outflow is blocked.

Technical Details for Transvenous Embolization

Vascular access

Both common femoral arterial and venous access are required: catheterization of the CS and embolization occurs through the venous access, but arterial access is required for control angiograms to monitor the progress in occlusion of the fistula, and to provide roadmaps for catheter navigation. The catheter for angiography is typically a 4Fr or 5Fr berenstein catheter, positioned in the distal common carotid artery (CCA), proximal ICA, or proximal ECA according to the principal site of arterial supply to the fistula. Once vascular access is obtained, the patient is systemically heparinized for the duration of the procedure. Direct retrograde catheterization of the IJV under ultrasound guidance is an alternative to femoral venous access and provides a shorter route to the CS,[31] which may also be advantageous in patients with tortuous venous anatomy or occlusion/stenosis of the central veins.

Cannulation of the CS

For IPS access an angled tip guide catheter, such as a Benchmark (Penumbra, Alameda, CA, USA) or Envoy (Codman, Raynham, MA, USA), is brought up to the JB. Angiography through the arterial catheter can provide a roadmap for navigation of a microcatheter and microwire into the IPS and then the CS. The choice of microcatheter depends on the planned embolic material, and in particular the catheter should be Dimethyl Sulfoxide (DMSO) compatible with a detachable tip if Onyx will be injected. For additional catheter stability, it is often advantageous to have a triple coaxial catheter system, comprising a guide catheter and long sheath, intermediate catheter, and microcatheter; this is particularly true when taking a more tortuous route to the CS, such as via the FV.[32]

If the IPS on the side of interest is occluded but review of the pre-procedure CT shows a well-defined inferior petrosal sulcus, it is often possible to recanalize the IPS to gain access to the CS. A 5Fr berenstein catheter is navigated through the guide catheter over a 0.035″ hydrophilic guide wire. The tip of the catheter is directed medially at the JB and moved gently along the wall of the bulb to engage the ostium of the IPS. The ostium is then explored with the guide wire, incrementally advancing the wire along the expected course of the IPS, superomedially on the AP view and anteriorly along the clivus. Consistent forward tension on the wire with intermittent short movements may help it advance. As the wire advances, the angled tip of the catheter can be brought along it into the IPS. On the lateral view, it is important to ensure that the wire moves anteriorly toward the CS and not posterior to the clivus, in which case it may be in the basilar plexus and further wire advancement could lead to venous subarachnoid hemorrhage. Once the guidewire reaches the CS, it can be removed and a microcatheter brought up over a microwire through the channel it created. In a variation of this technique,[33] the 0.035″ hydrophilic guidewire is used to probe the orifice of the IPS and then advanced into the occluded IPS. A jugular venographic roadmap is obtained with the wire in place, and then the wire is removed leaving a bright line on the roadmap from the wire tract through the IPS. A microcatheter and microwire can then be advanced through the guide catheter along the wire tract defined on the roadmap to reach the IPS.

An alternate microguidewire looping technique[34] for accessing the CS through an occluded IPS involves bringing a guide catheter up to the insertion of the occluded IPS on the IJV or JB, and then advancing a microwire into the occluded IPS which forms a loop due to the resistance encountered from the occlusion. The guide catheter is then brought into the orifice of the IPS to enhance support, and the looped microwire is used to tunnel through the IPS until the CS is reached, then bringing a microcatheter up over the microwire into the IPS.

Catheter positioning in the CS

Once the microcatheter is in the CS, it should be positioned at the site of the venous outflow furthest from where the catheter entered the CS, since once embolization begins this site may no longer be accessible. This may involve navigating the catheter around multiple septations in the CS to reach the SOV at the orbital apex. Dural septations may also be encountered at the lateral CS, which can present a challenge on navigating the microcatheter to the origin of the SMCV if there is cortical venous reflux that needs to be occluded. An angiogram through the microcatheter can be used to confirm the position of the catheter tip and can also be used to help identify the fistulous point. Additionally, a cone beam CT with injection of 20% dilute contrast can localize the microcatheter within the compartments of the CS (**Fig. 4C** and **4D**), to ensure it is at the site of venous outflow.

Embolization

Embolization will typically begin with deployment of coils matching the vessel diameter at the site of venous outflow, seeking to form a compact, occlusive plug of coils

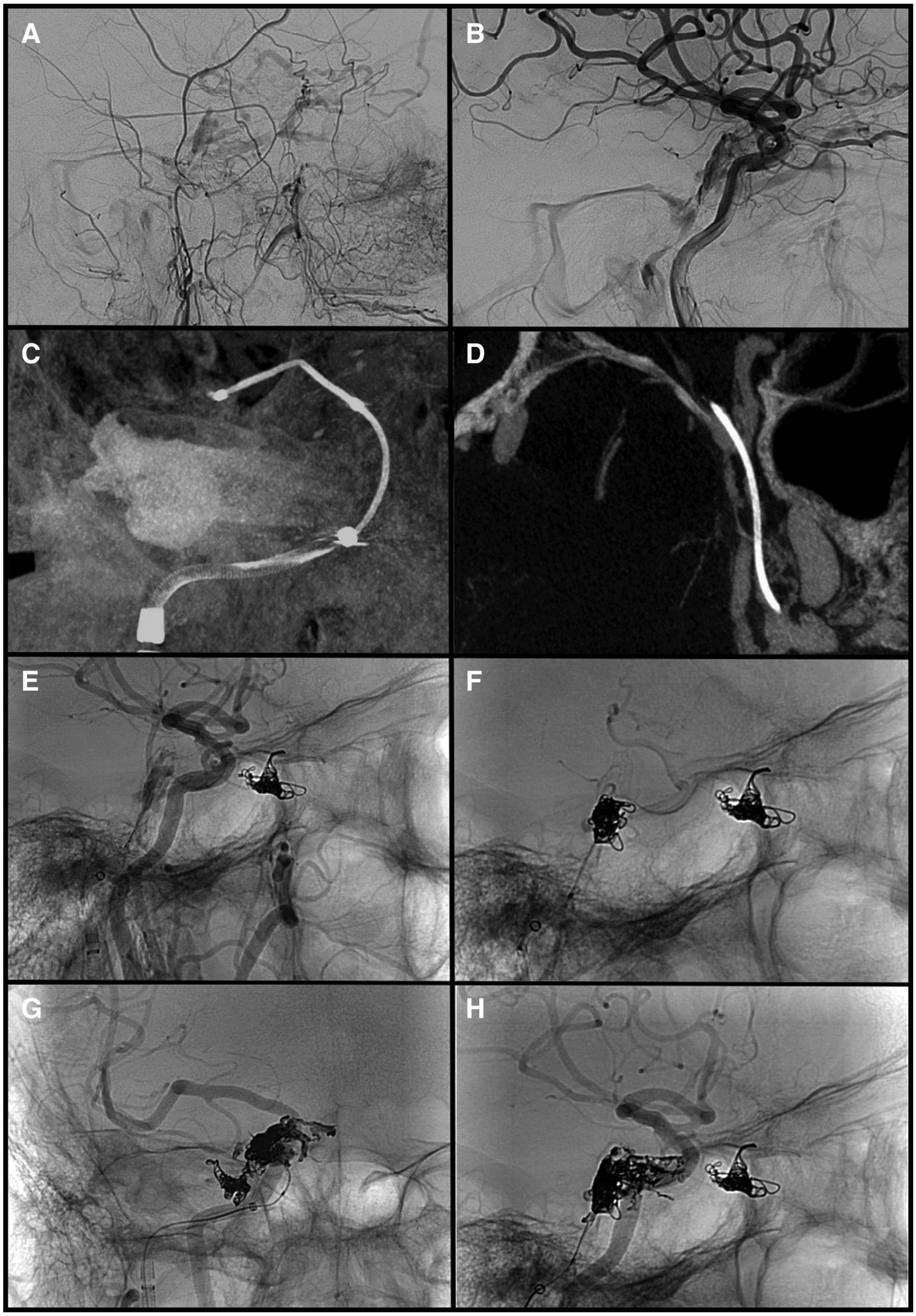

Fig. 4 Treatment via the IPS approach. A 61-year-old man presented with a 2 weeks history of right eye proptosis, chemosis, and diplopia. DSA in the lateral projection of the (**A**) ECA and (**B**) ICA showed a CS DAVF with venous drainage via the SOV, anterior cerebral vein, superficial middle cerebral vein, IPS, and superior petrosal sinus into the sigmoid sinus. There is cortical venous reflux into the anterior cerebral vein. (**C**) A microcatheter was navigated via the right IPS to reach the confluence of the ophthalmic veins at the orbital apex, shown on 3D MIP. (**D**) A cone beam CT with injection of dilute contrast shows the position of the microcatheter in relation to the compartments of the CS. (**E**) Coiling through the microcatheter occluded the drainage via the SOV, then the catheter was withdrawn and more coils deposited posteriorly in the CS (**F**) to reduce drainage through the sphenoparietal sinus, though there is still a small amount of venous reflux into the anterior cerebral vein. The embolization was completed with Onyx injection through the microcatheter into the posterior coil mass, and final control AP (**G**) and lateral (**H**) DSA of the right CCA show resolution of the fistula. AP: anterior-posterior; CCA: xxx xxx xxx; CS: cavernous sinus; DAVF: dural arteriovenous fistula; ECA: external carotid artery; ICA: internal carotid artery; IPS: inferior petrosal sinus; MIP: xxx xxx xxx; SOV: superior ophthalmic vein

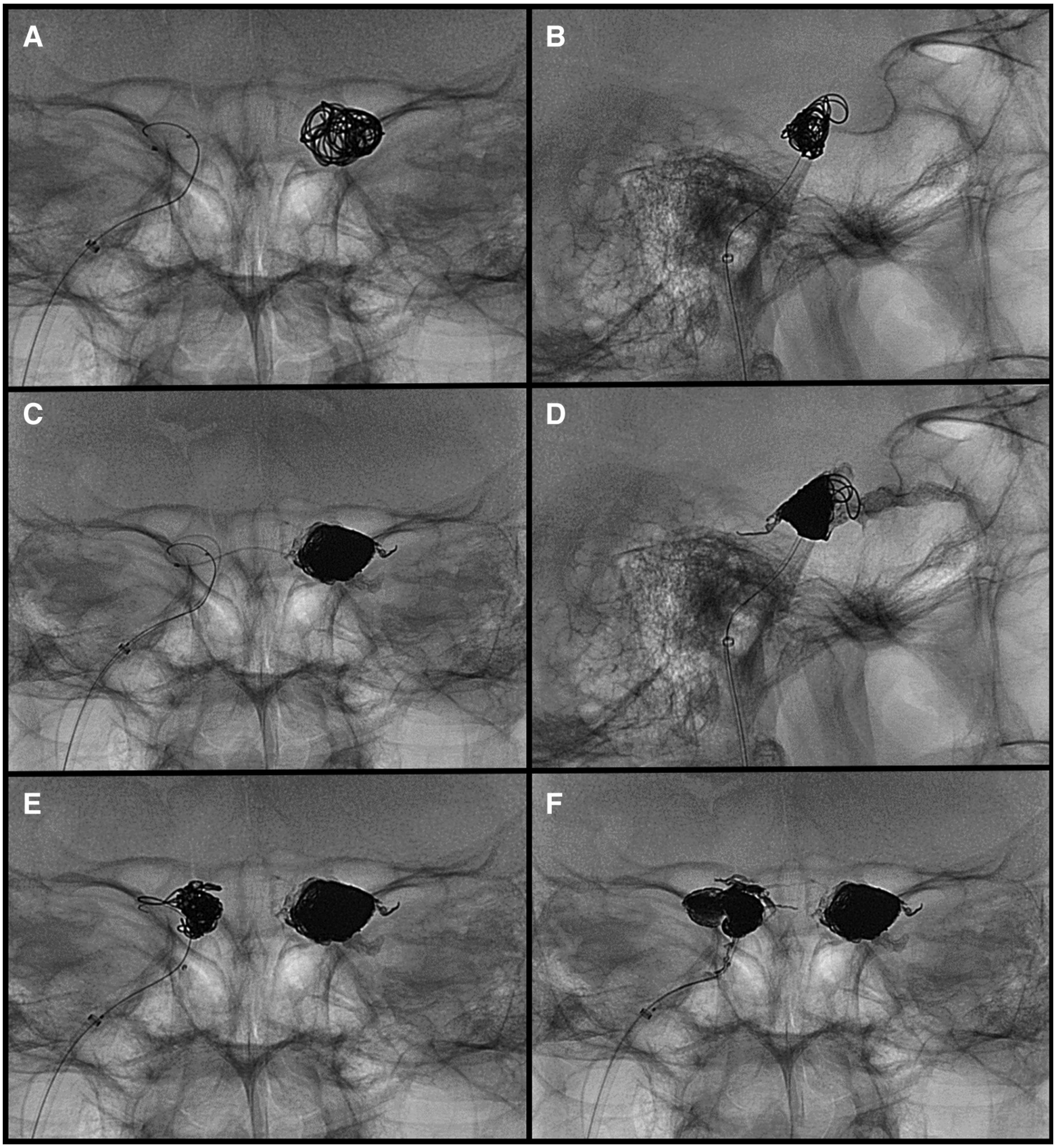

Fig. 5 Embolization of bilateral CS DAVFs (shown in **Fig. 2**) via an intercavernous approach. A 6Fr destination sheath was positioned at the origin of the right IPS, and an Echelon 10 microcatheter was navigated through intercavernous venous channels to reach the left CS, while an Apollo microcatheter was positioned in the right CS. Coils were deployed in the left CS to reduce the flow rate, shown in AP (**A**) and lateral (**B**) projections. Onyx was then injected through the Echelon microcatheter (**C** and **D**) to consolidate the coils and embolize the left-sided CS DAVF. The Echelon microcatheter was removed and the same procedure performed on the right through the Apollo microcatheter, with coiling (**E**) followed by Onyx injection (**F**). Post-embolization DSA showed complete occlusion of the fistula. AP: anterior-posterior; CS: cavernous sinus; DAVFs: dural arteriovenous fistulas; IPS: inferior petrosal sinus

(**Fig. 4E**). The microcatheter can then be repositioned at another site of venous outflow for further coiling (**Fig. 4F**), or coiling can continue back into the CS to occlude the fistulous point.

Coiling alone may be adequate to occlude the fistula; at a minimum it should substantially reduce flow through the fistula, enabling liquid embolic agents to then complete the occlusion. If glue is to be used, the catheter should be positioned near the fistulous point, and then flushed with dextrose solution. A mixture of 33%–50% glue in lipiodol is injected until the fistulous point is reached, and then the catheter is immediately withdrawn under aspiration.

With Onyx or Squid, the DMSO-compatible catheter is flushed with DMSO, and then the embolic agent is injected slowly, monitoring for penetration into the fistulous point while ensuring there is no intra-arterial extension, which creates a risk of cranial nerve ischemia or migration of liquid embolic material into the ICA. Angiograms are performed intermittently through the arterial catheter, and injection stops once the fistula is occluded. The microcatheter is then removed.

Flushing of the catheter with DMSO or injection of Onyx or Squid can precipitate a trigeminocardiac reflex,[35] caused by stimulation of the trigeminal nerve and characterized by abrupt severe brachycardia or asystole. It is therefore prudent to advise the anesthesia team before injecting these agents, so that the patient's hemodynamic status can be carefully monitored.

It is particularly important to ensure that at the conclusion of embolization no cortical venous reflux is evident on angiography, since it could present an ongoing risk for intracranial hemorrhage or cerebral venous congestion if left untreated. If there is residual reflux not treatable from the catheter position in the CS, either the catheter may need to be repositioned or the CS accessed from a different route.

Two catheter techniques

In the case of bilateral or complex CS DAVFs, it may be beneficial to have two microcatheters for embolization. In **Fig. 5**, bilateral fistulae are treated via an IPS approach with one microcatheter navigated through intercavernous venous channels into the contralateral CS, and the other microcatheter in the ipsilateral CS. Coil and Onyx embolization of the contralateral fistula is performed first, followed by the ipsilateral fistula. Unilateral CS DAVFs may also benefit from a two-catheter technique if there are multiple venous outflows to be occluded.

Post-procedure care

Typically, heparinization does not need to continue after the procedure, although a patient with a fistula showing extensive cortical venous reflux and stasis in cortical veins after embolization may remain on a heparin infusion overnight, with a CT or MRI the next day 6 hours after cessation of the heparin to ensure there is no cortical venous thrombosis. Mass effect from embolic material or coils in the CS may lead to cranial nerve palsies (particularly affecting the sixth cranial nerve due to its central location within the CS near the ICA), which can be mitigated by intravenous (IV) dexamethasone during the procedure and a short course of oral dexamethasone afterwards.

Procedural success rates

Transvenous occlusion of CS DAVFs generally has a high success rate with low morbidity. In a single center case series of 38 patients, 23 of whom were treated transvenously, the angiographic complete occlusion rate was 74% and the clinical symptom resolution or improvement rate was 94%.[36] Another case series of 27 patients showed angiographic occlusion in 89% and clinical cure in 96%, with no permanent procedure-related morbidity.[37]

Conclusion

Transvenous embolization of dural and indirect CCFs is a safe and effective technique, but it depends on a thorough understanding of the angioarchitecture of the fistula, including the arterial supply, venous outflows, and fistulous point, to determine the optimal catheter approach and choice of embolic agent.

Disclosure Statement

The authors declare that they have no conflicts of interest.

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