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Treatment of Dural Arteriovenous Malformations and Fistulae

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Dural arteriovenous malformations or dural arteriovenous fistulae (DAVFs) are acquired lesions consisting of one or more fistulous connections within the leaflets of the dura mater. They account for 10% to 15% of intracranial arteriovenous malformations [1,2]. We believe that the term *malformation* is a misnomer for two reasons: the term *malformation* implies a congenital etiology when, in fact, most if not all of these lesions are acquired; and the term *malformation* also implies that there is a true nidus, and although these lesions often have a complex angiographic appearance because of the recruitment of numerous arterial pedicles, they can frequently be isolated to a single or a few discrete fistulous sites of arteriovenous shunting.

Etiology and pathogenesis

Given the variable locations and complexity of dural fistulae, there are multiple etiologies responsible for fistula formation. Specific factors are known to predispose to fistula formation, however, including sinus thrombosis, trauma, and surgery. There are several cases of documented sinus thrombosis with subsequent fistula formation associated with the involved sinus [3–6]. In such cases, the primary cause of sinus thrombosis may be a generalized hypercoagulable state or an infection of the mastoid or sphenoid sinus. It is thought that the fistula occurs during the phase of attempted recanalization and neovascularization within the sinus.

Conversely, not all DAVFs are associated with thrombosis or stenosis of a major dural sinus. Subsequent sinus occlusion may then occur because of turbulence and venous hypertension within the sinus, ultimately leading to occlusion. Experimentally, rats in a carotid-jugular fistula venous hypertension model have been shown to develop DAVFs, suggesting that thrombosis in and of itself may not be the primary event [7–9]. A recent histopathologic study of DAVFs describes 30-μm "crack-like" vessels within the dural sinus wall and postulates that steno-occlusive disease of the venous sinuses triggers the development of these vessels. Subsequent sinus thrombosis is then an epiphenomenon that occurs because of turbulent flow and sinus wall thickening [10].

Finally, conditions associated with vascular fragility, such as fibromuscular dysplasia, neurofibromatosis type I, and Ehlers-Danlos syndrome, have been associated with DAVFs [11–15]. As a whole, despite the variety of potential factors that may be responsible for DAVF formation, it seems that pathologic findings on the venous side are probably the major contributor. Furthermore, most of the management and treatment decisions are primarily determined by the pathologic findings within the venous system.

Clinical presentation and angiographic considerations

The clinical presentation of DAVFS is highly varied and is primarily determined by the location of the fistula and the subsequent pattern of venous

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drainage. Other factors include the degree of arteriovenous shunting, venous hypertension, and presence of venous stenoses or ectasias. The two most common locations are the transverse or sigmoid sinus and the cavernous sinus [16,17], followed by the following sites: deep venous, superior sagittal sinus, superior petrosal sinus, ethmoidal, marginal sinus, and inferior petrosal sinus. The reason for this discordant distribution has not been clearly established. A delay in the development of the external carotid territory and numerous emissary veins near the skull base have been proposed as two possible mechanisms [18]. Theoretically, any site along the dura is a potential source for fistula formation. The primary factor in determining the aggressive behavior of DAVFs, however, is the presence of leptomeningeal venous drainage, which can engender venous hypertension, progressive neurologic deficit, infarction, and hemorrhage [19].

Lesions involving the transverse sinus are the most common (38%) (Fig. 2) [17]. The clinical presentation can range from asymptomatic to overt hemorrhage. Common symptoms may include a simple pulsatile bruit or headache. If there has been long-standing venous hypertension and swelling, the presentation can mimic transient ischemic attacks or ischemic infarcts of the involved temporal lobe [20]. The arterial supply typically occurs through transmastoid branches of the occipital artery; branches of the middle meningeal artery; neuromeningeal branches of the ascending pharyngeal artery; branches of the vertebral artery, including the posterior meningeal and artery of the falx cerebelli; and tentorial branches of the meningohypophyseal trunk. A complete evaluation of the venous drainage should include identifying downstream stenosis or occlusion of the ipsilateral sinus, the presence of flow across the torcula, and the presence of cortical venous drainage. Particular attention should paid to the direction of flow in the vein of Labbé and its point of insertion, because this information has significant implications for the endovascular options for treatment. If the vein of Labbé flows in a retrograde fashion, its origin in the sinus can be occluded from a transvenous approach. If the flow is antegrade, occlusion of its origin can exacerbate the venous hypertension, leading to a worsening of symptoms and possible hemorrhage.

Lesions involving the cavernous sinus frequently manifest with ocular pathologic changes. The classic signs of orbital venous hypertension include pulsatile exophthalmos, chemosis, and conjunctival injection. A progressive cavernous sinus syndrome can also include increased ocular pressure, extraocular muscle paresis (especially of the third and sixth cranial nerves), decline in visual acuity, optic neuropathy, and proptosis. These are all indications for treatment. Tinnitus and ocular bruits are also relative indications. The arterial supply can include branches from the inferolateral or meningohypophyseal trunk, branches of the middle or accessory meningeal artery, the artery of foramen rotundum, and the ascending pharyngeal artery, among others. A large draining superior ophthalmic vein can easily be identified on MRI, and surgical access into this vein provides a route for potential therapy. Access into the cavernous sinus via the inferior petrosal sinus provides yet another route for endovascular therapy. Again, particular attention should be paid to possible intracranial cortical venous drainage from the cavernous sinus into the superficial and deep sylvian systems.

Ethmoidal dural fistulae typically derive supply from the anterior and posterior ethmoidal branches of the ophthalmic artery and may recruit supply from the distal branches of the internal maxillary artery. The drainage is almost always into a pial vein along the floor of the anterior cranial fossa, which ultimately drains into the superior sagittal sinus. As a result, the most common presentation is a frontal lobe hemorrhage. Occasionally, drainage can occur into the cavernous sinus, resulting in chemosis, proptosis, and elevated intraocular pressures. Such DAVFs have a male preponderance, and surgical coagulation of the vein is the preferred method of treatment because of its low morbidity and high cure rate [21].

Superior sagittal sinus DAVFs are rare and are varied in their presentation (Fig. 1). Because of the distant location between the superior sagittal sinus and the auditory apparatus, early detection secondary to pulsatile tinnitus is rare. The presentation is thus predominantly secondary to hemorrhage, either subarachnoid, subdural or intraparenchymal; headache; or symptoms from venous hypertension. The arterial supply is generally derived from branches of the middle meningeal artery, the anterior falcine artery from the ophthalmic artery, or the posterior meningeal artery. Frequently, the arterial supply is bilateral. Should endovascular therapy fail to achieve complete obliteration, surgical excision can be contemplated, with care noted to identify the presence of collateral venous drainage to prevent exacerbation of the venous hypertension.

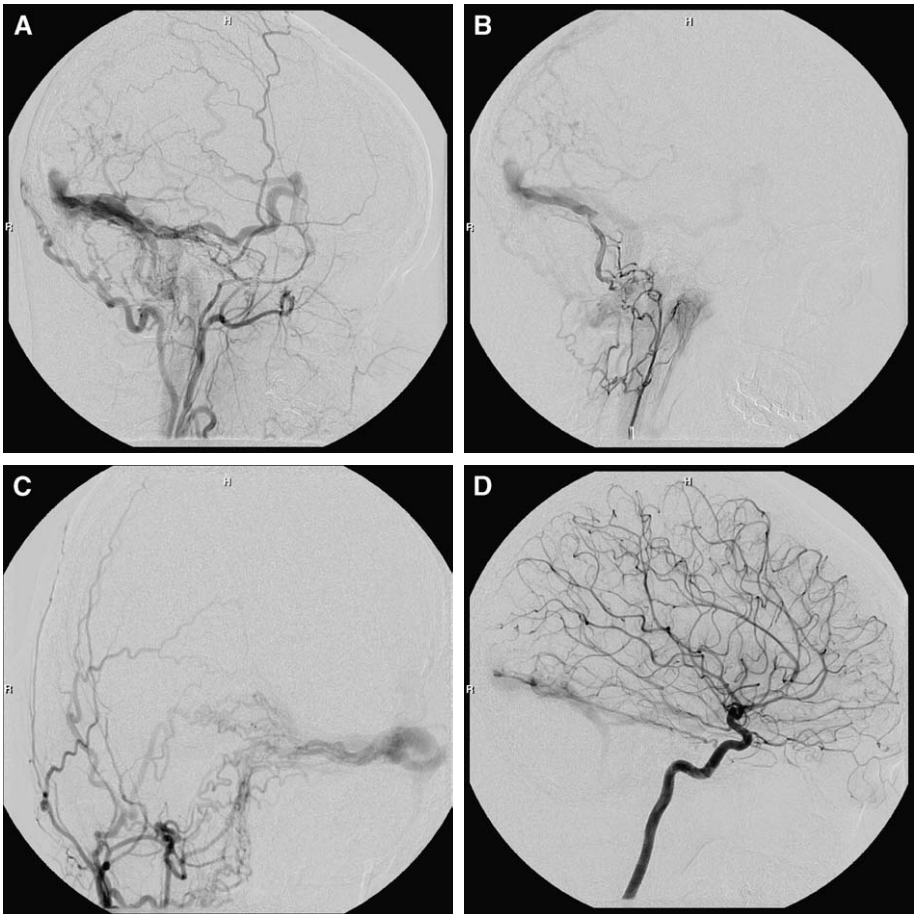


Fig. 1. A 29-year-old woman who presented with headaches and pulsatile tinnitus. On examination, she was noted to have a vigorous mastoid bruit. Radiographic evaluation documented this large transverse-sigmoid dural arteriovenous fistula. (*A*) Preprocedural lateral left external carotid angiogram demonstrates a vigorous left transverse sinus fistula with supply from the occipital, ascending pharyngeal, and middle meningeal arteries. Note the retrograde flow through the vein of Labbé and occlusion of the ipsilateral left jugular vein. (*B*) Preprocedural lateral ascending pharyngeal angiogram demonstrating the contribution from the neuromeningeal trunk. (*C*) Preprocedural anteroposterior (AP) right external carotid angiogram demonstrating the contribution from the right occipital and posterior auricular arteries. (*D*) Preprocedural lateral left internal carotid angiogram demonstrating the contribution from the meningohypophyseal trunk artery of Bernasconi and Cassinari. (*E*) Preprocedural AP left vertebral angiogram demonstrating extensive supply from the artery of the falx cerebelli and recruitment of pial supply from the posterior cerebral artery. (*F*) AP roadmap image demonstrating the position of the microcatheter traversing the contralateral jugular vein across the torcula and into the left transverse-sigmoid junction. (*G*, *H*) AP and lateral projections of a middle meningeal injection after coil occlusion of the transverse sinus. This is the position from which N-butyl-cyanoacrylate was injected. (*I*) AP roadmap image of the glue cast demonstrating liquid embolic not only in the sinus but in the proximal portion of the vein of Labbé and other arterial collaterals. (*J-M*) Postprocedural lateral angiograms of the left external carotid, occipital, ascending pharyngeal, and internal carotid arteries demonstrating complete angiographic obliteration of the fistula. (*N*, *O*) Postprocedural AP angiograms of the left vertebral and right external carotid arteries confirming an angiographic cure. The patient was discharged home on the following day neurologically intact. (*P*, *Q*) Three month follow-up lateral angiograms of the left common carotid artery and left pharyngo-occipital trunk demonstrating regression in the caliber of all the previously enlarged vessels. The complete angiogram demonstrated no evidence of recurrence.

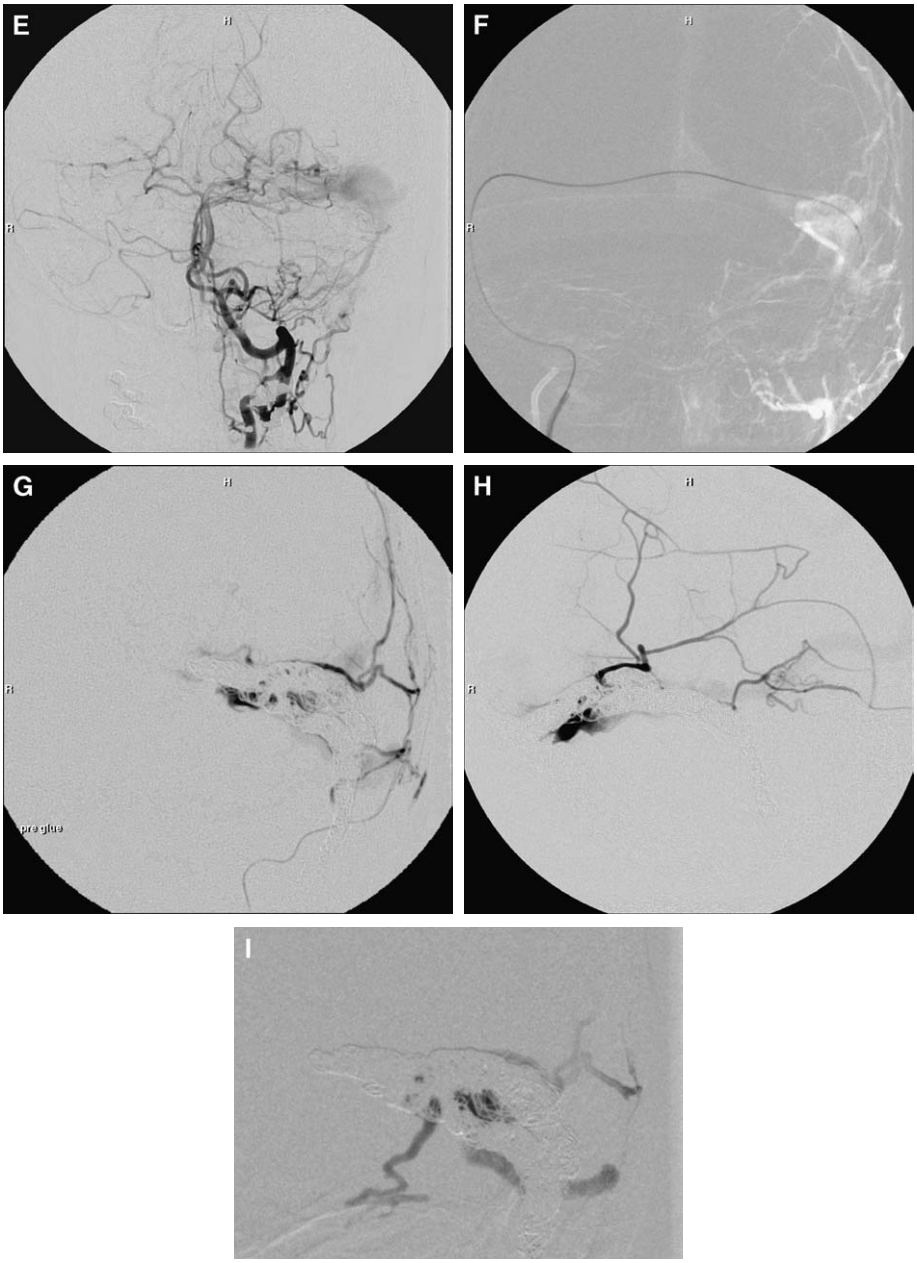
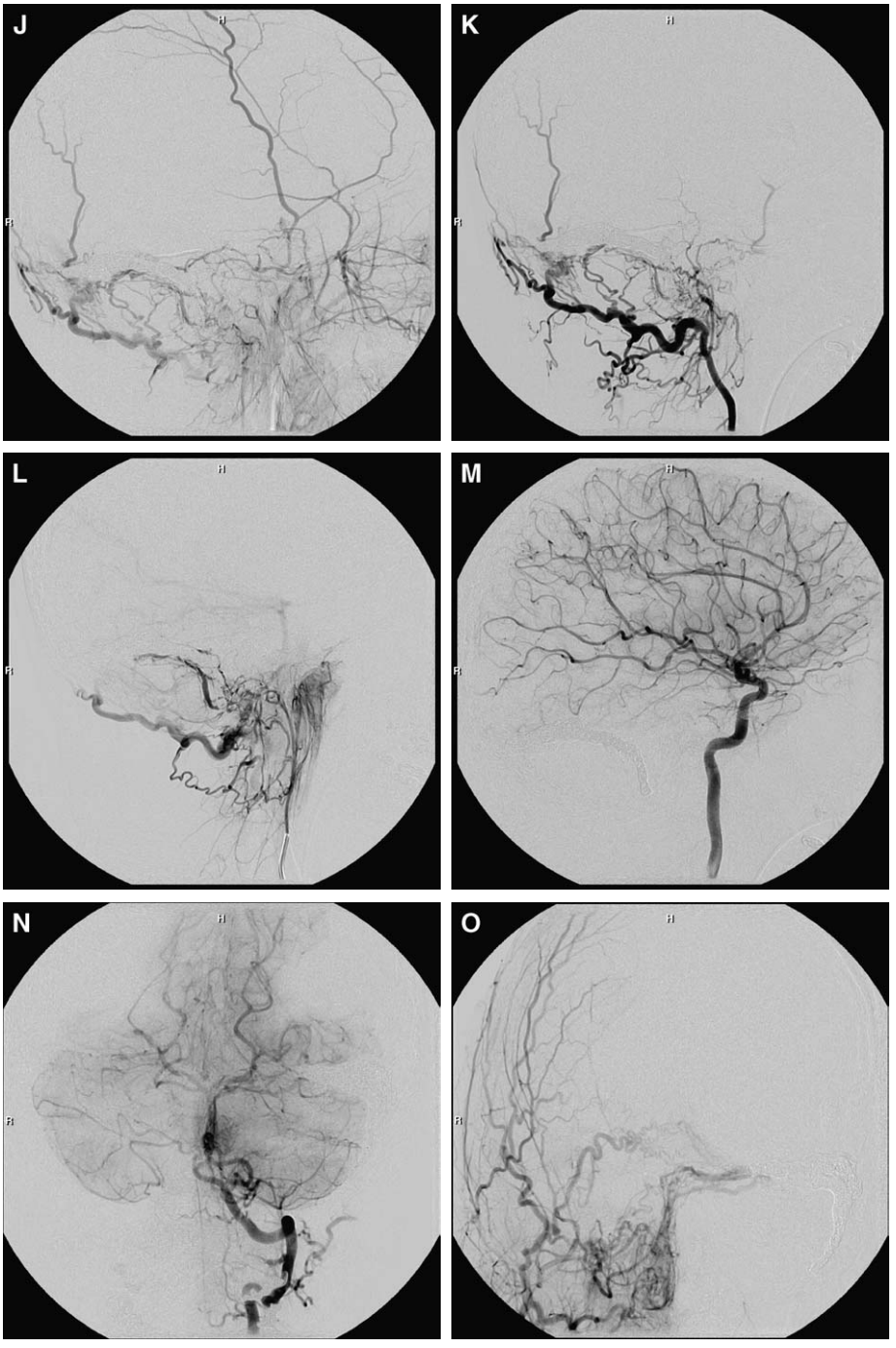


Fig. 1 (*continued*)

Lesions involving the superior petrosal sinus, also referred to as tentorial DAVFs, generally present with hemorrhage or mass effect from dilated veins (Fig. 3). The arterial supply typically arises from the artery of Bernasconi and Cassinari off of the meningohypophyseal trunk as well as from the petrosal and petrosquamosal branches of the middle meningeal artery. The venous drainage usually involves the superior petrosal sinus and the pontine and perimesencephalic veins. Even if endovascular treatment fails to obliterate the fistula, it can aid in localization of the fistula on postprocedure axial imaging and can minimize the blood loss during surgical resection [22].



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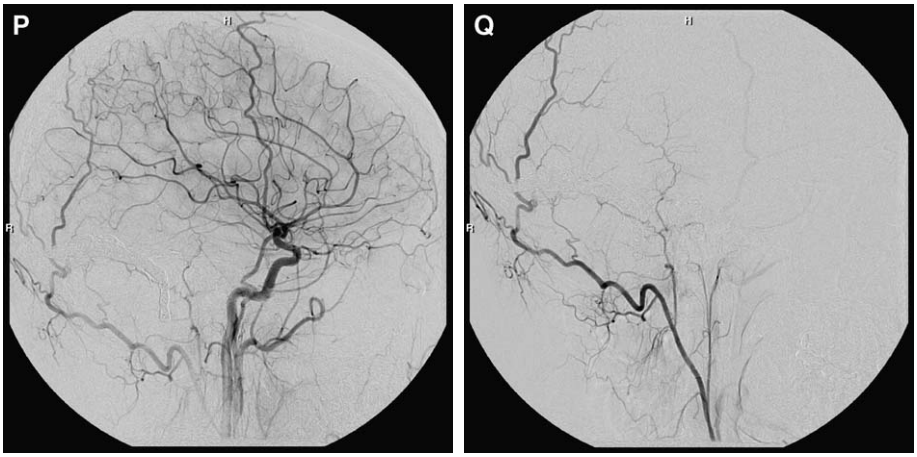

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In summary, the location and pattern of venous drainage are the key components in determining the clinical presentation. The arterial supply is largely determined by the location of the fistula, and the venous drainage and degree of venous hypertension indicate the potential for a malignant clinical course. The overall angiographic anatomy aids in determining whether endovascular therapy by transarterial, transvenous, or a combined treatment can obliterate the fistula, and an endovascular approach is generally the first line of therapy for most of these lesions. Should endovascular therapy fail to cure the lesion, it can aid in localization of the fistula on axial CT imaging and minimize the blood loss during surgical resection.

Radiographic diagnosis

The preliminary diagnosis of a DAVF is based on clinical presentation. CT, CT angiography, MRI, and magnetic resonance angiography often support the clinical diagnosis by revealing engorged cortical veins, sinus stenosis or occlusion, hemorrhage, osseous changes from hypertrophied and ectatic vessels, or parenchymal abnormalities from venous hypertension [23]. All patients with clinical and radiographic evidence suggesting a DAVF should undergo cerebral angiography. If a DAVF is revealed, a thorough angiographic evaluation should delineate the fistula's location, arterial feeders, sinus drainage, cortical venous drainage, occlusions, stenoses, ectasias, and blood flow dynamics. A complete angiogram may require evaluation of internal and external carotid arteries, vertebral arteries, and possibly the ascending and deep cervical systems.

Classification

There are numerous classification schemes for DAVFs dating back to the initial scheme proposed by Djindjian and Merland in 1978 [24]. The most useful and modern are the revised Djindjian classification proposed by Cognard et al [25] and the classification proposed by Borden et al [26], both of which are based on that initial scheme. No matter the classification system, they all focus on the patterns of venous drainage and the clinical implications of presentation, treatment, and prognosis associated with them (Tables 1 and 2).

Treatment decision making

The decision to treat DAVFs depends primarily on the clinical presentation of the patient and the angiographic characteristics of the fistula, especially on the venous side. A simple fistula draining into a sinus in a patient with a mild bruit or who is asymptomatic is best served with conservative or compression therapy. It is important to continue to follow these patients, because DAVFs can progress to a more malignant state. If a pulsatile bruit resolves, it is an indication for repeat angiography, because the sinus may have thrombosed and the venous drainage may be redirected into the leptomeningeal or deep venous system, portending a more aggressive course.

There is a subset of patients who are symptomatic (most commonly a bruit) and whose activities of daily living are affected but do not harbor aggressive angiographic features. In these cases, subtotal obliteration can palliate the

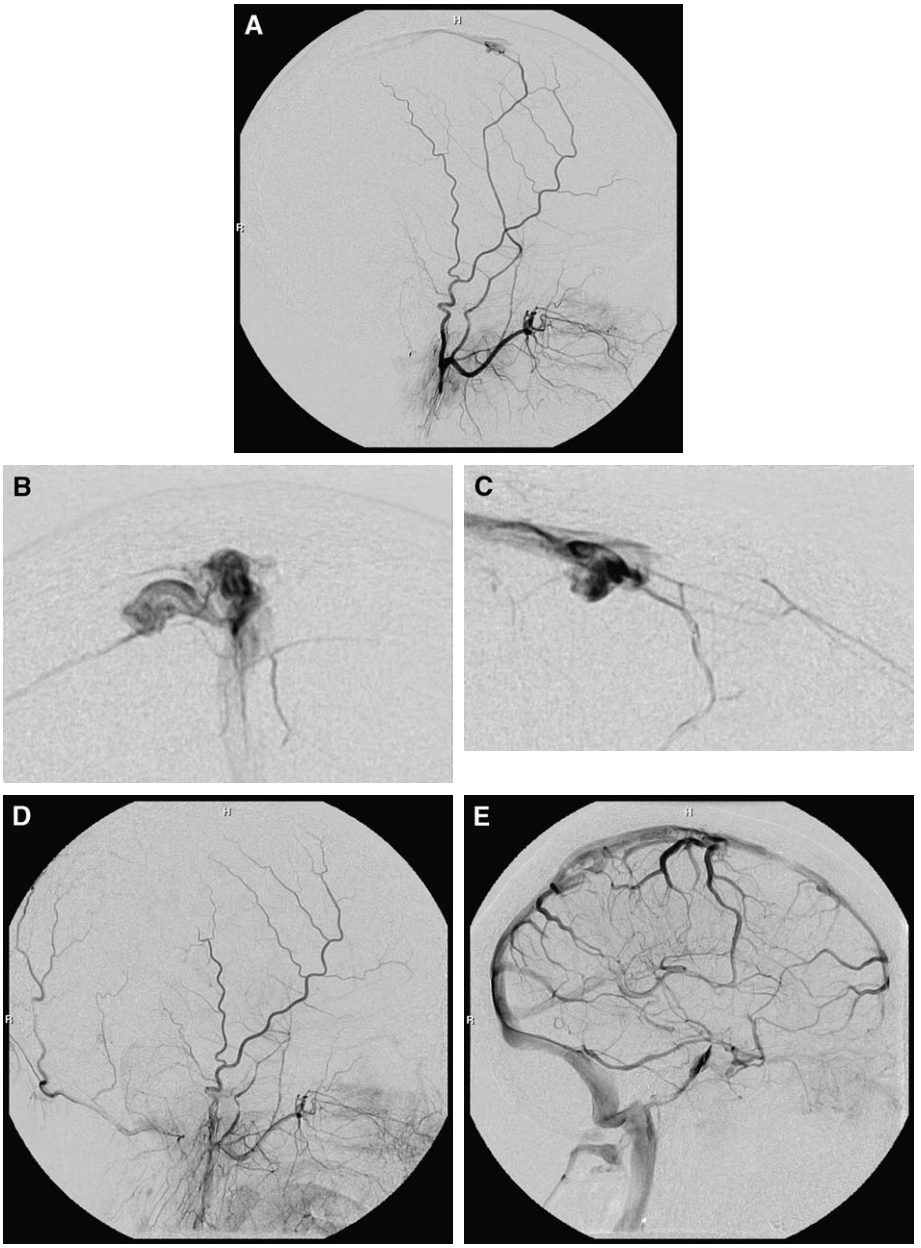

Fig. 2. A 49-year-old woman who, during evaluation of a left facial droop, was noted to harbor this sagittal sinus dural arteriovenous fistula. (*A*) Preprocedural right external carotid angiogram demonstrates supply from the middle meningeal artery. (*B*, *C*) Anteroposterior and lateral projections of a superselective middle meningeal injection demonstrate drainage into a cortical vein and, ultimately, the superior sagittal sinus. N-butyl-cyanoacrylate was injected from this position. (*D*) Postprocedural right external carotid angiogram demonstrates angiographic obliteration of the fistula. (*E*) Postprocedural venous phase of a right internal carotid injection demonstrates patency of the superior sagittal sinus. The patient was discharged home the following day neurologically intact.

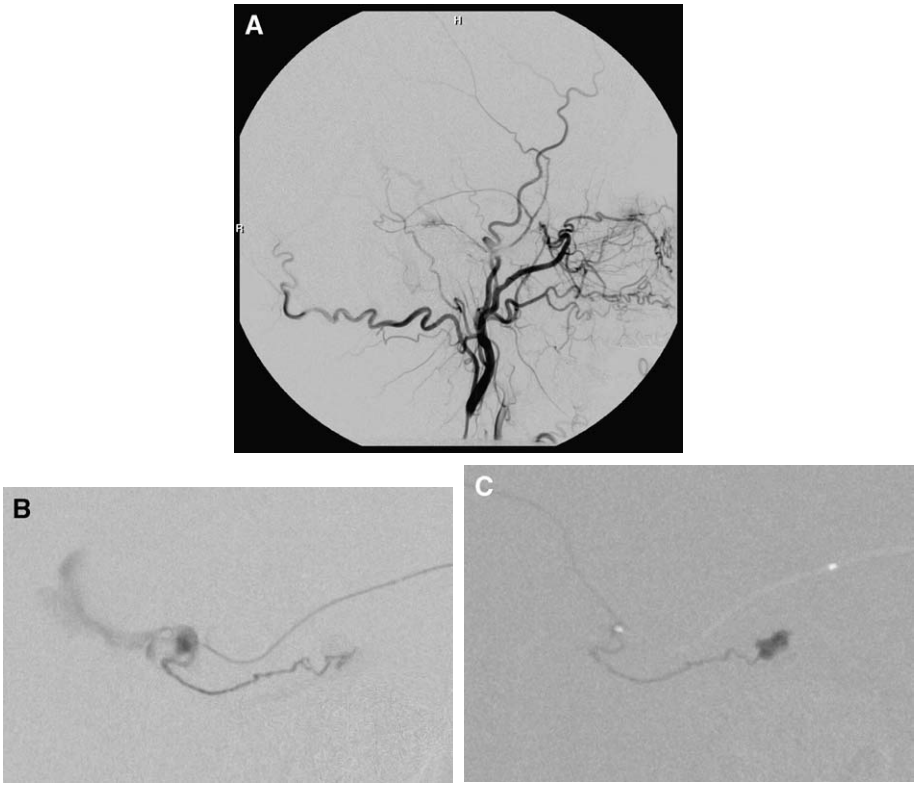

Fig. 3. A 60-year-old man with an incidentally discovered superior petrosal dural arteriovenous fistula (DAVF) during an evaluation for seizures. (*A*) Preprocedural lateral left external carotid angiogram demonstrating a small fistula supplied by the petrosquamosal and petrosal branches of the middle meningeal artery. (*B*) Superselective lateral angiogram of the petrosquamosal branch of the middle meningeal artery. Note the small amount of extravasation caused by attempts to navigate the severe tortuosity. Fortunately, a flow arrest position was obtained, and N-butyl-cyanoacrylate (NBCA) was injected from this position, sealing the defect and preventing an epidural hematoma. (*C*) Glue cast from the middle meningeal embolization. (*D*) Lateral left internal carotid angiogram demonstrating the remainder of the fistula supply from the meningohypophyseal trunk draining into the superior petrosal sinus and the petrosal vein, and, ultimately, into the vein of Galen via the lateral mesencephalic vein. (*E*) To catheterize the meningohypophyseal trunk, a balloon was inflated in the cavernous segment of the internal carotid so that the microcatheter would deflect off of the balloon and into the meningohypophyseal trunk. (*F*) Microcatheter injection of the meningohypophyseal trunk. Note the presence of reflux into the carotid artery. The balloon was then positioned over the origin of the meningohypophyseal trunk and inflated during the injection of NBCA. (*G*) Glue cast from meningohypophyseal trunk embolization. (*H*, *I*) Postprocedural lateral angiograms of the left internal and external carotid arteries demonstrating complete occlusion of the DAVF and angiographic cure without evidence of glue emboli in the intracranial circulation.

symptoms effectively. An aggressive angiographic cure may not be required and may even impose unnecessary risks [27,28].

The treatment goal for any patient presenting with hemorrhage, symptoms of cortical venous hypertension, or significant ocular pathologic findings should be complete obliteration. Even the asymptomatic patient whose fistula demonstrates significant cortical venous pathologic findings should be considered for aggressive treatment. In this high-risk population, there is little evidence to support the possibility that incomplete obliteration reduces the risk of hemorrhage, venous infarction, or visual loss.

Treatment methods

Compression therapy

A small percentage of DAVFs involving the transverse or sigmoid sinus or cavernous sinus can

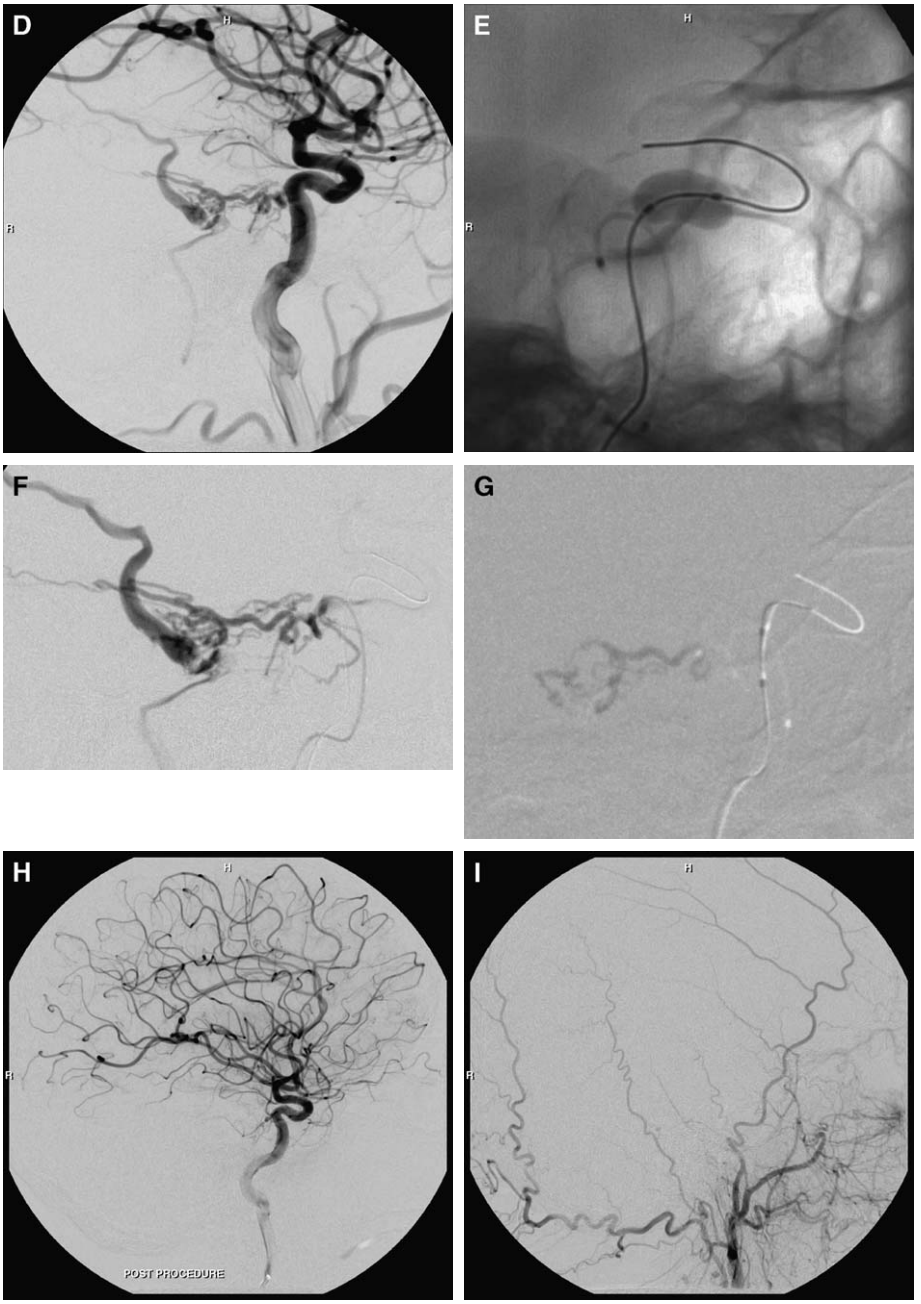

Fig. 3 (*continued*)

be treated with compression therapy. This involves compression of the involved occipital artery or the carotid artery (carotid atherosclerosis must be excluded) with the contralateral hand for 30 minutes several times a day. For small fistulae, this may promote thrombosis in up to 30% of the cases [20,29,30].

Endovascular treatment

Transvenous embolization of fistulae has historically been used with good success and is especially effective in the treatment of transverse sinus DAVFs. Venous access into the transverse sinus is generally not an issue, and even if the

Table 1
Classification schemes of venous drainage and the clinical application: revised Djindjian classification

Type I	Antegrade drainage into a sinus
Type IIa	Reflux into the sinus (retrograde flow)
Type IIb	Reflux into cortical veins
Type IIa+b	Reflux into both sinus and cortical veins
Type III	Direct cortical venous drainage without venous ectasia
Type IV	Direct cortical venous drainage with venous ectasias
Type V	Spinal venous drainage

Data from Djindjian R, Merland J. Meningeal arteriovenous fistula. Superselective arteriography of the external carotid artery. New York: Springer-Verlag; 1978. p. 405–6.

Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, et al. Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. Radiology 1995;194(3):671–80.

sinus is occluded at the level of the sigmoid sinus or jugular bulb, access into the involved sinus can be obtained by crossing the torcula. Consideration of the venous drainage of the fistula itself as well as the drainage of normal cerebral tissue is paramount in minimizing risk. Care must be taken not to reroute the pattern of venous drainage into the cortical veins, which can exacerbate venous hypertension. For transverse sinus fistulae, particular attention must be paid to the vein of Labbé. If the flow in the vein of Labbé is antegrade, embolization cannot proceed across the origin of the vein of Labbé without compromising normal venous drainage. If it is retrograde, however, occlusion across the vein of Labbé is well tolerated and may be required to provide a definitive cure.

There are many methods of occluding the sinus with balloons or coils. Advocates of balloon occlusion [31] espouse the advantage of possible balloon test occlusion of the sinus. Unlike arterial infarction, however, venous infarction generally does not occur for hours, and occasionally for days, after the permanent occlusion. A short temporary period of balloon occlusion therefore does not suggest that the patient will tolerate long-term occlusion. Furthermore, placement of a detachable balloon against the blood flow into a sinus may not be technically feasible in every case and certainly is not possible for Borden type III DAVFs, where the drainage occurs directly into subarachnoid veins. The other option to balloons is coil occlusion. Unfortunately, even dense packing with coils may not permanently occlude the fistula and may require further thrombosis within the coil mass itself. Currently, we favor coil embolization of the sinus, which decreases the degree of shunting, followed by transarterial N-butyl-cyanoacrylate (NBCA) embolization through a preselected arterial pedicle.

Table 2
Classification schemes of venous drainage and the clinical application: Borden classification

Type I	Drainage into the dural venous sinus
Type II	Drainage into the dural venous sinus with retrograde drainage into subarachnoid veins
Type III	Drainage into subarachnoid veins
Subtype a	Simple fistula
Subtype b	Multiple fistulas

Data from Borden JA, Wu JK, Shucart WA. A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment. J Neurosurg 1995;82(2):166–79.

The initial results of transarterial treatment of DAVFs were suboptimal because of the low rates of cure and the subsequent recruitment of collaterals [20,29,32]. The difficulty in curing DAVFs transarterially probably resulted from the use of polyvinyl alcohol (PVA) particles and proximal occlusion of feeding pedicles during NBCA injections [33,34]. It is well known that embolization with PVA ultimately results in recanalization. In addition, NBCA embolization that does not traverse the fistulous connection into the venous side will, over time, recruit collaterals that are certain to be more difficult to catheterize selectively. With the advances in microcatheter and microguidewire technology, greater distal access can be established; however, the potential for dangerous overt or occult external carotid–to–internal carotid or vertebral anastomoses or ischemic cranial nerve palsies could preclude safe and effective embolization. A recent series of 21 patients treated transarterially under flow arrest conditions demonstrated cures in all fistulae without complications [35]. Although the definite curative embolization occurred under flow arrest conditions, a significant portion of these patients underwent adjunctive embolization with PVA or NBCA or prior transvenous coiling of the recipient venous structure. This served to devascularize the collateral inflow to minimize NBCA fragmentation, prevent systemic venous embolization, and increase the probability of polymerization within the pathologic shunt itself. This illustrates the complex angioarchitectural

spectrum of DAVFs and the expertise in multimodality treatments required to engender safe and effective outcomes.

Surgical treatment

Unlike pial arteriovenous malformations of the brain or spinal cord, the venous drainage of a DAVF can frequently be safely ligated, excised, or occluded before occlusion of all the arterial pedicles [36]. Profuse bleeding can occur during the exposure and bone flap elevation, however, because of the arterialized dura, pedicles, and drainage into the intradiploic vascular channels [37]. Sinus skeletonization or excision of DAVFs should be reserved for those cases in which endovascular therapy has failed to effect a cure but has at least decreased the flow to reduce blood loss. Surgical access of a recipient venous structure, such as the superior ophthalmic vein for cavernous DAVFs, to deliver endovascular materials continues to play a significant role. As previously mentioned, for ethmoidal fistulae [21] and some petrosal sinus fistulae, surgical excision of the draining vein is the primary treatment modality.

In a recent series, 34 patients with primarily transverse or sigmoid, superior sagittal, or superior petrosal sinus DAVFs were cured by surgical treatment [36]. The authors separated their patient population into two groups depending on whether the fistulous drainage occurred purely through leptomeningeal veins (nonsinus fistulae) or whether the fistula drained into a sinus with retrograde flow into the leptomeningeal circulation (sinus fistulae). In the former scenario, the surgical treatment required disconnection of the draining veins at the point where they exited the dural wall of the sinus. In the latter, surgical excision of the involved sinus segment after preoperative embolization represented a safe and definitive treatment, because this segment did not serve to drain the normal cerebrovasculature. Their cure rate was 100%, and there were no instances of mortality or permanent morbidity. Again, the treatment goals were determined by a careful evaluation of the venous anatomy, and the importance of preoperative embolization was emphasized.

Radiosurgery

The role of stereotactic radiosurgery in the treatment of DAVFs is continuing to develop, and the experience with this modality is growing [38–44]. Because of the complex nature of DAVFs and the relatively small number of patients in each series, the rate of angiographic obliteration is still uncertain. It does appear, however, that the complication rates of the initial treatment are relatively low. There is the possibility of hemorrhage or symptomatic clinical events that could occur from the time of treatment until obliteration is obtained, however. This may be untenable for patients who present with hemorrhage, significant venous hypertension, or high-risk angiographic profiles.

Because of the relative efficacy of endovascular and surgical treatment of these lesions, we view stereotactic radiosurgery as the third-line treatment modality. There is, however, a relatively small patient population in which endovascular or surgical treatment may be extremely difficult or risky, as is the case for the elderly population with significant comorbidities, where stereotactic radiosurgery may play a role.

Summary

DAVFs comprise a highly complex series of lesions clinically and angiographically. The clinical presentation can range from asymptomatic to devastating intracranial hemorrhage. The location of the fistula is a primary factor in determining the method of presentation. Angiographically, the pattern of venous drainage is the main factor in determining the ultimate prognosis. The goal of treatment of any DAVF that exhibits cortical venous drainage should be angiographic obliteration and cure. The method of treatment should be highly individualized to the angiographic architecture of each DAVF and can comprise endovascular, surgical, or a combination of methods to achieve the appropriate treatment goal and to minimize risk. In general, we think that the endovascular approach is the primary mode of therapy for transverse sinus, cavernous, superior sagittal, or petrosal sinus fistulae and that surgery is the primary mode of therapy for ethmoidal fistulae. Stereotactic radiosurgery should be reserved for lesions in which endovascular or surgical options have failed or would subject the patient to inordinate risk. The importance of a multidisciplinary approach to these highly complex lesions cannot be overemphasized and engenders the safest and most effective outcomes.

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