Chapter 21
Dural Arteriovenous Fistulae: Endovascular Embolization Indications and Techniques

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Description and Classification

Cranial dural arteriovenous fistulas (DAVFs) are abnormal dural-based connections between meningeal arteries and venous sinuses, meningeal veins, or cortical veins, without an intermediate capillary network or nidus. They account for 10–15% of aberrant intracranial arteriovenous connections [1, 2]. DAVFs are mostly acquired conditions that occur in middle-aged adults. Lesions in the pediatric population, although rare, are often much more complex and beyond the scope of this chapter.

Exact etiology of adult DAVFs remains unknown. An association with venous sinus thrombosis has been found, and hypercoagulable states have therefore been implicated. Thrombosis has also been reported as a consequence of dural arteriovenous shunting. Other provocative factors have been reported, such as trauma, intracranial surgery, and radiation exposure. However, many DAVFs occur without an identifiable cause.

Two pathophysiological mechanisms for the development of DAVFs have been proposed. One theory holds that these lesions develop in normally occurring vascular channels [3], which open due to venous hypertension secondary to outflow obstruction or venous sinus thrombosis [4]. The second theory proposes that the lesions form due to neovascularization in the dura occurs due to release of angiogenic factors secondary to hypoxia or increased venous pressure in the setting of thrombosis or occlusion. Studies of lesion histology as well as animal models are used to support this hypothesis [5, 6, 7] [1, 8].

Symptoms and clinical course of dural arteriovenous fistulas depend predominantly on the drainage pattern of the lesion, as well as the location, arterial supply, and degree of shunting [1, 9]. A review of 191 reported cases found that lesions

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without cortical venous drainage (CVD) typically presented with more benign symptoms related to shunting, such as headache, proptosis, pulsatile tinnitus, and cranial nerve deficits secondary to arterial steal. These lesions can also be asymptomatic and incidental in nature. The natural history of a vast majority of benign DAVFs suggests that they remain stable over the course of many years and thus do not require surgical intervention. Benign DAVFs are a dynamic disease with potential risk of progression of the venous thrombosis, which may reroute the venous drainage. Two scenarios are potentially important: the possible significance of retrograde flow in the sinus and the small, but concerning risk of conversion into an aggressive DAVF. The change of flow in the sinus to retrograde, as outlined by Cognard type IIa, has been associated with papilledema and raised intracranial pressure. Theoretically, retrograde flow in the sinus can preclude cortical veins from draining into the implicated sinus, thereby resulting in increased intracranial pressure. In a Toronto series of 112 conservatively managed patients, 14 (12.5%) patients underwent spontaneous occlusion of their fistulas, and 4 (4.0%) patients converted from having a benign fistula to an aggressive angioarchitecture with new corticovenous reflux [10].

Lesions with CVD often presented with more substantial or aggressive features including neurological sequelae such as hemorrhage, nonhemorrhagic focal neurological deficit (NHND), dementia, or even death [11]. Studies have shown significant variation in the rates of hemorrhage, focal nonhemorrhagic neurological deficit, and mortality in patients with CVD [12, 13, 14]. One study with a mean follow-up of 4.3 years found a 35% rate of hemorrhage, 30% rate of nonhemorrhagic neurological deficit, and 45% rate of mortality in lesions associated with CVD [15]. A meta-analysis of outcomes after intracranial hemorrhage secondary to DAVFs in 326 patients found the rate of mortality after a median follow-up of 12 months to be 4.7% and an overall rate of poor clinical outcome (mRS $\geq$ 3) of 8.3% [16].

Various classification schemes for dural arteriovenous fistulas have been proposed. The most widely utilized classification schemes are those proposed by Borden and Cognard. Both schemata recognize the presence of CVD as a factor predictive of the severity of the lesion. The Borden classification simply stratifies fistulae using their venous drainage and remains the most widely used clinical classification system (Table 21.1) [17]. The more complex Cognard classification accounts for the direction of venous flow, as well as the degree of venous distortion associated with the lesion (Table 21.2). The degree of venous ectasia was used to further stratify the risk of hemorrhage in lesions with CVD. Cognard type V lesions are used to designate lesions with spinal venous drainage [18]. More recently, Zipfel et al. have proposed to modify the Borden and Cognard classification schemes to include the presence of absence of symptoms in aggressive DAVFs [8, 19], as stud-

Table 21.1 Borden classification of dural arteriovenous fistulae

Type 1	Venous drainage into a dural sinus; no CVD
Type 2	Venous drainage into a dural sinus with associated CVD
Type 3	Drainage into cortical veins (CVD)

Table 21.2 Cognard classification of dural arteriovenous fistulae

Type I	Venous drainage into dural sinus with antegrade flow
Type II a	Venous drainage into dural sinus with retrograde flow
Type II b	Venous drainage into dural sinus with antegrade flow and CVD
Type II a + b	Venous drainage into dural sinus with retrograde flow and CVD
Type III	Venous drainage into cortical veins (CVD)
Type IV	CVD with associated venous ectasia(s)
Type V	Venous drainage into spinal perimedullary veins

Table 21.3 Barrow classification of carotid-cavernous fistulas

Type A	Direct shunt from internal carotid artery
Type B	Shunt from meningeal branches of internal carotid artery
Type C	Shunt from meningeal branches of external carotid artery
Type D	Shunt from both internal and external carotid artery branches

ies have indicated a higher annual rate of ICH in symptomatic high-grade DAVFs than in asymptomatic lesions (7.5% vs 1.5%, respectively) [20, 21].

A second category of cranial DAVFs involve the cavernous sinus. Lesions of the cavernous sinus have also been classified using the Barrow classification of carotid-cavernous fistulas (Table 21.3). This anatomical classification divides cavernous sinus shunts into direct and indirect types, with the indirect types of lesions (type B, C, and D) being DAVFs [22]. The indirect fistulas may be from meningeal branches of the ICA (Type B), ECA (Type C), or both (Type D). They present with symptoms that are more insidious and less severe than Type A lesions [23]. Symptoms include red eye, proptosis, increased intraocular pressure, diplopia, pain, and bruit [24]. These fistulas may resolve spontaneously in many cases. Interestingly, high rates of spontaneous resolution following diagnostic angiography have been noted. Symptoms are classically waxing and waning in nature, with worsening symptoms possibly attributable to changing venous drainage patterns.

Common Sites and Feeding Vessels

Dural arteriovenous fistulas are predominantly located around the venous sinuses. These include the cavernous sinus, sigmoid and transverse sinuses, and the superior sagittal sinus. Anterior cranial fossa, occipital sinus, and tentorial lesions have also been described. The sigmoid and transverse sinuses are the most common location, accounting for 30–50% of DAVFs [25, 26]. Multiple DAVFs may occur in 7–10% of cases [25].

Multiple arteries from the extra- and intracranial circulation may supply DAVFs in a given location. All branches of the external carotid artery (ECA) may be implicated, as well as dural arteries of the internal carotid artery (ICA). The vertebral artery (VA) may also supply a lesion through meningeal branches. As well, terminal cerebral branches of the ICA and the VA may supply the lesion directly through pial branches. The diverse routes of arterial supply to the lesion make it imperative that diagnostic angiography of all potential feeders be carried out as part of the diagnostic evaluation of the DAVF. After partial treatment through arterial embolization, the DAVF may recruit new feeding vessels from the contralateral circulation. Possible arterial feeding vessels for common DAVF locations, including carotid-cavernous fistulas, are summarized in Table 21.4 [27].

Treatment of Dural AVFS

Successful treatment of DAVFs requires a complete knowledge and understanding of the complex cerebral anatomy. Surgical ligation of fistulas remains a standard therapy. A multidisciplinary team of cerebrovascular surgeons and interventional

Table 21.4 Major supply to intracranial DAVFs

Location	ECA	ICA	Vertebral
Sigmoid-transverse sinus	Occipital Ascending pharyngeal Posterior auricular Middle meningeal Superficial temporal	Bernasconi-Cassinari meningohypophyseal artery Inferolateral trunk	Posterior meningeal Cerebellar falcine
Cavernous sinus	Middle meningeal Artery of foramen rotundum Ascending pharyngeal Posterior auricular	Meningohypophyseal trunk Inferolateral trunk McConnell's capsular branches	
Tentorial-incisural	Ascending pharyngeal Occipital Middle meningeal Accessory meningeal	Meningohypophyseal trunk	Posterior meningeal Cerebellar falcine Posterior cerebral Superior cerebellar
Superior sagittal sinus	Middle meningeal Superficial temporal Occipital	Anterior falcine Meningohypophyseal trunk	Posterior meningeal
Anterior cranial fossa	Internal maxillary Middle meningeal	ICA Anterior/posterior ethmoidal	

neuroradiologists is required to successfully treat the patients. The goals of treatment vary depending on the presentation. For patients with significant tinnitus but no cortical venous drainage, a less aggressive approach can be undertaken, and in some situations, partial occlusion with reductions of symptoms is adequate. Care must be taken in these patients to ensure that future corticovenous drainage does not develop. This is done with serial imaging follow-up, usually MRI. If a patient presents with a change in their symptomology, such as a reduction or discontinuation of their pulsatile tinnitus, cerebral angiography is suggested, as this may represent a change in the venous outflow.

Lesions presenting with CVD must be angiographically cured. This includes patients presenting with or without hemorrhage. Often a combination of endovascular embolization followed by open surgical ligation is required. Surgery should be considered when (1) a simple fistula exists which is surgically accessible, (2) partial embolization has created a more acceptable lesion for surgery, and (3) the arterial supply to the fistula is by important branches that cannot be embolized without risking cranial nerves or other arterial anastomoses such as the ophthalmic artery. This is the case for most anterior cranial fossa fistulas, as often the feeders arise from ethmoidal branches that cannot be safely embolized with a liquid embolic material due to the proximity of the central retinal artery (Fig. 21.1).

Transarterial Embolization

Transarterial embolization necessitates penetration of the embolic material into the fistulous connection and venous pouch of the fistula. This is required to ensure that the often-multiple feeders to the fistula are interrupted. Standard aneurysm coils are not used but because of their inability to be placed distally are usually reserved for transvenous embolization.

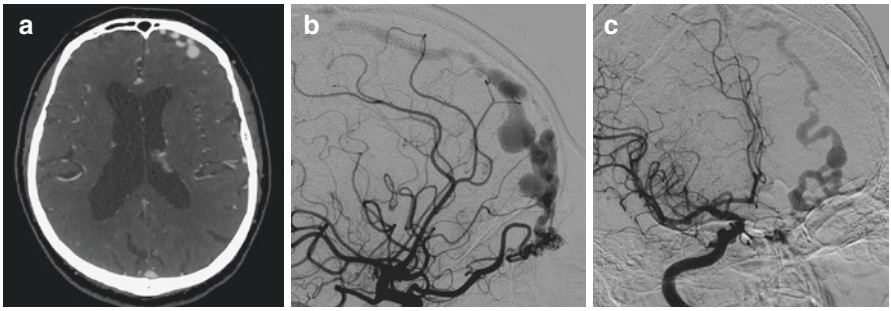

Fig. 21.1 A 79-year-old man presented with an acute left fourth cranial nerve palsy. (**a**) CTA showed the presence of dilated frontal vessels, raising the suspicion of anterior cranial fossa DAVF. (**b**, **c**) Right ICA injections in lateral and AP orientation show supply from the right anterior ethmoidal artery to the left-sided DAVF, with retrograde cortical venous drainage (Borden type 3, Cognard III). Surgical ligation was recommended and successfully performed, leading to total cure of the patient's cranial nerve palsy

Generally, there are two approved embolic materials for embolization of DAVFs: N-butyl cyanoacrylate (NBCA), marketed in the United States as TRUFIL (Cordis Neurovascular, Inc., Miami Lakes, FL, USA), and ethylene vinyl alcohol copolymer or Onyx (Medtronic Neurovascular Inc., Irvine, CA, USA). Onyx is an ethylene vinyl alcohol copolymer that is dissolved in dimethyl sulfoxide. Tantalum powder is dissolved in the material to provide visualization under fluoroscopy. A third material that has become available is PHIL from Microvention. This is available in Europe and is reported to have superior visualization of the liquid embolic, an ability to see the microcatheter tip during injections, minimal streak artifact on imaging, and no tattoo effect from the tantalum powder when treating superficial lesions. It is similar to Onyx in that it is a DMSO-dissolved product, requiring DMSO-compatible delivery catheters, and it is available in three different concentrations/viscosities [28].

The overall principle of using an embolic material is to penetrate and occlude the fistulous connection of the lesion (Fig. 21.2). If this is not successful, then either surgical ligation or transvenous embolization is required. There are multiple techniques available for transarterial embolization. Newer distal access catheters allow for excellent access to the feeding vessel. This reduces the requirement to re-navi-

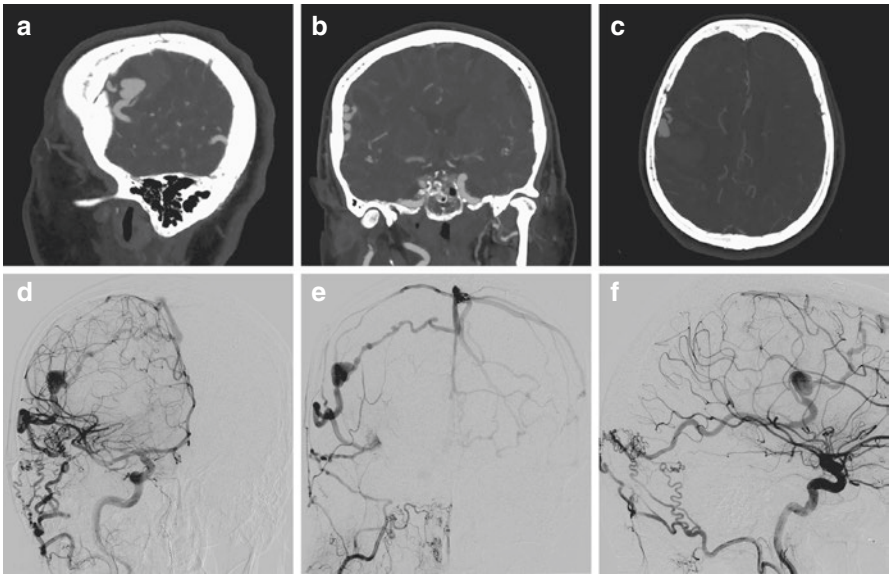

Fig. 21.2 This 69-year-old woman presented with a sudden-onset headache and a right-sided frontoparietal intracerebral hemorrhage; she was diagnosed with a Borden type 2/3 and Cognard IV dural AVF, with occlusion of the superior sagittal sinus. (**a**, **b**, **c**) CTA demonstrated multiple dilated vessels in the region of hemorrhage, raising the suspicion of DAVF. (**d**) Right ICA injection in the AP projection showed takeoff of the occipital artery from the R ICA and multiple occipital artery branches feeding the fistula, with evidence of venous ectasia and occlusion of the superior sagittal sinus. (**e**) Right ECA lateral injection in the AP projection showing supply to the fistula from the posterior division of the middle meningeal artery and cortical venous reflux. (**f**) Lateral view of the right ICA injection showing occipital artery supply to the fistula

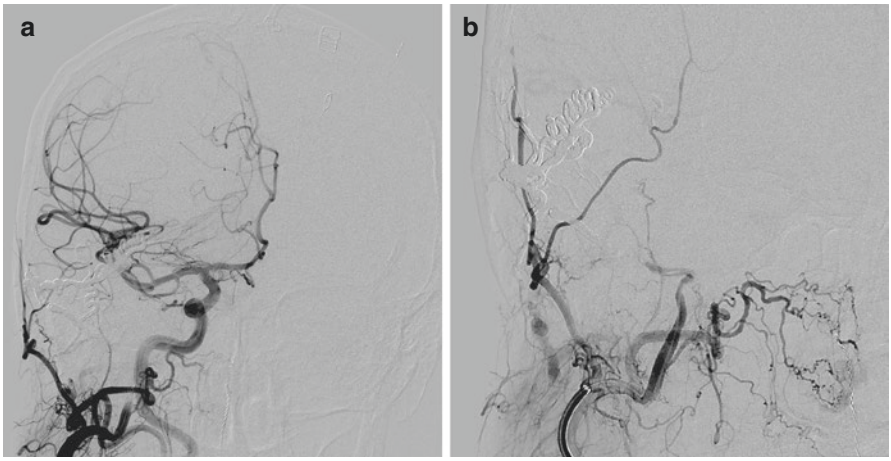

Fig. 21.3 (**a**, **b**) Posttreatment angiogram from the patient in Fig. 21.2, following transarterial Onyx embolization form the middle meningeal artery. AP and lateral views of right CCA injection show complete occlusion of the fistula and venous connection and absence of early venous filling

gate the vessels to get the microcatheters out the various feeding vessels. The distal access catheters also allow for better distal placement of microcatheters to achieve penetration into the fistula (Fig. 21.3). There are multiple microcatheters that are available for embolization with either NBCA or Onyx. Onyx requires the use of catheters that are resistant to the DMSO and will not degrade. A newer technique has been developed called the balloon augmented technique. A Scepter balloon (Microvention Inc. Tustin, CA, USA) can be advanced into the arterial feeder of the fistula. The traditional technique of Onyx embolization required the creation of a plug around the microcatheter. This can require a great deal of time and reflux, and gluing of the microcatheter can result in permanent catheter retention. By inflating the Scepter balloon, this plug is immediately created, and the Onyx can be pushed into the fistula, and reflux is limited. The limitation of the technique is that the balloon catheter is much less navigable into the distal vessel than a flow-directed microcatheter.

Transvenous Embolization

Transvenous embolization should be considered in all cases. The transvenous route allows access to the fistulous connection and venous pouch in many cases. This allows for disconnection of the fistula and angiographic cure. Also, in cases where the sinus is either occluded or there is proximal occlusion, embolization of the residual sinus connection can achieve angiographic cure. Often traditional aneurysm coils can be utilized to occlude the venous pouch via the transvenous approach.

Treatment of Carotid-Cavernous Fistulas

Carotid-cavernous fistulas (CCFs) have variable arterial supply, and this is outlined in Table 21.3. The key to successful management is identification of the supply. For high-flow lesions that occur secondary to a ruptured aneurysm or trauma, transarterial approach can be considered. This usually involved entry into the CCF via the internal carotid artery rent with subsequent coil embolization. Use of liquid embolic material is not recommended because of reflux into the intracranial circulation. Other options include placement of covered stents and vessel sacrifice. However, most lesions can be managed with either a transarterial or transvenous approach to the CCF.

Transvenous approaches remain the mainstay of treatment for indirect CCFs. Access is usually obtained via the inferior petrosal sinus (IPSS) (Fig. 21.4). Even in

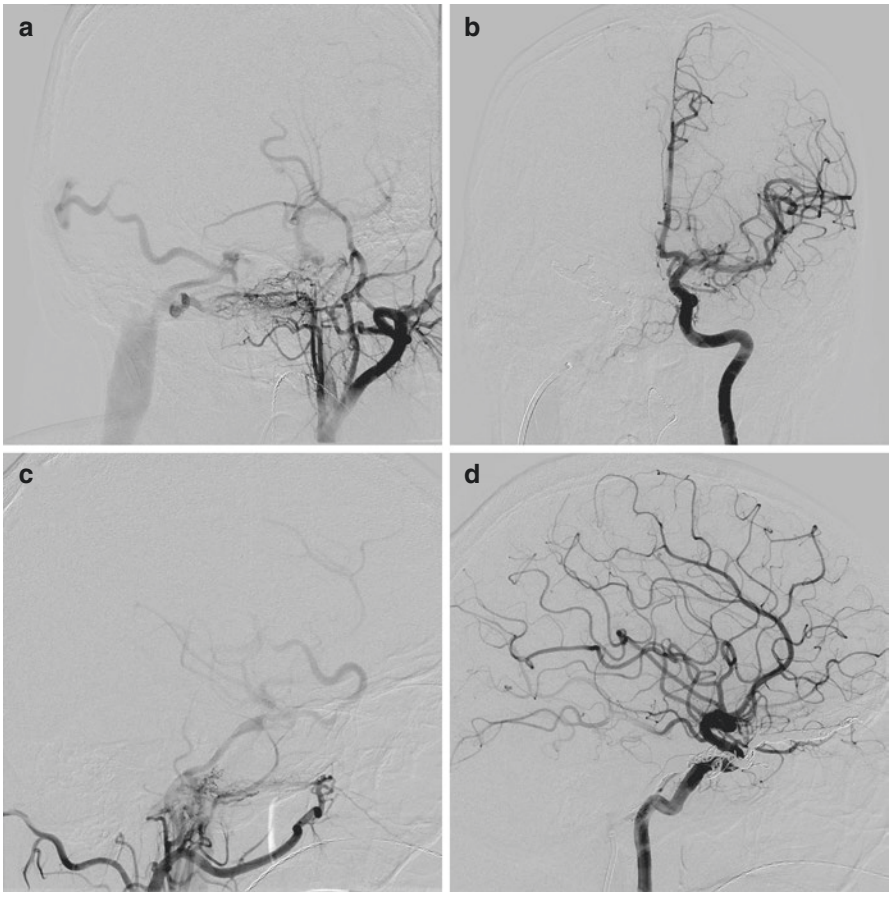

Fig. 21.4 This 42-year-old man presented with right-eye proptosis, chemosis, and diplopia and was found to have a right-sided, indirect CCF with supply from bilateral ICA and ECA branches (Barrow class D). (**a**, **c**) AP and lateral ECA injections showing early filling of the right inferior petrosal sinus and internal jugular vein (IJV). (**b**, **d**) AP and lateral arterial runs show near-complete occlusion of the CCF after transvenous embolization (see Fig. 21.5)

cases where the IPSS is not angiographic visible, we have had success probing the opening and successfully navigating into the cavernous sinus. In cases where access via the IPSS is not viable, other routes must be entertained. This includes direct cutdown or percutaneous access to the superior ophthalmic vein or access via the transfacial vein [29]. In our experience, this is infrequently required. Once the CCF has been entered with the microcatheter, progressive coil embolization is performed. We prefer to get as far into the cavernous sinus as possible and even place coils in the superior ophthalmic vein if allowed (Fig. 21.5). Progressive coiling with serial angiography will show progressive occlusion. Care must be taken during the initial diagnostic angiogram to target the correct side so that unilateral cavernous sinus occlusion will result in angiographic cure. In the occasional cases, bilateral embolization is required to cure the CCF.

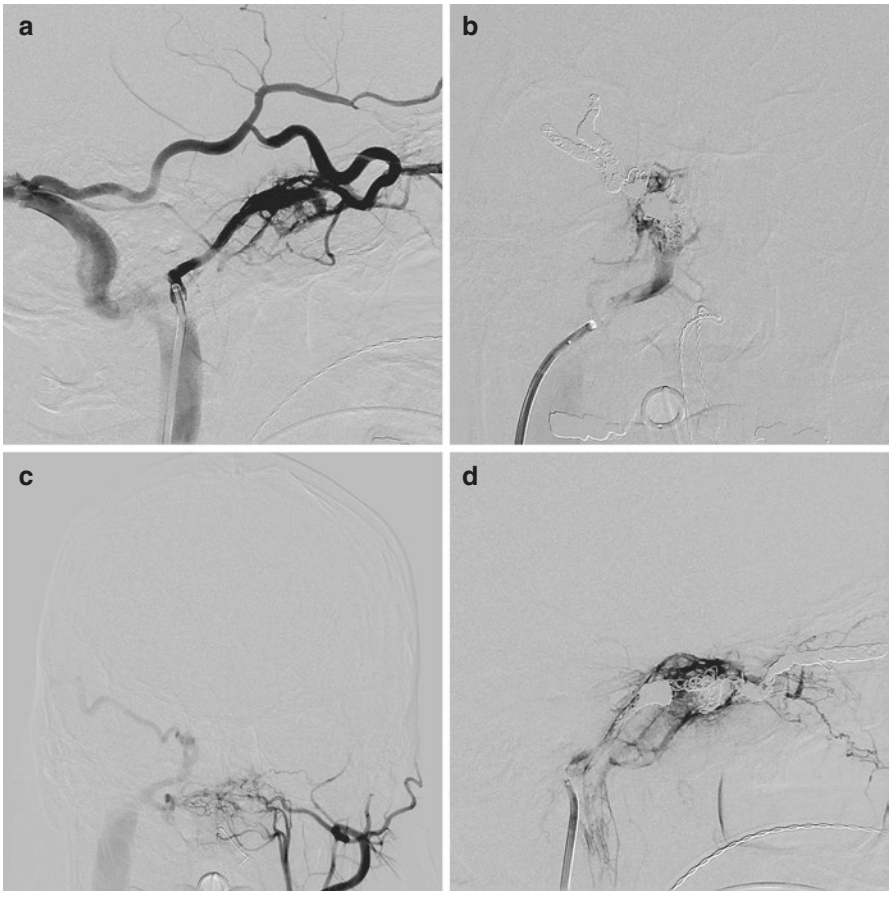

Fig. 21.5 Transvenous embolization of the right-sided CCF with supply from bilateral ICA and ECA meningeal branches: (**a**) Pretreatment venous injection from the inferior petrosal sinus, showing dilated venous anatomy of the cavernous sinus. (**c**) Pretreatment AP view of left ECA injection showing contribution to the right-sided CCF and early filling of the right IJV. (**b**, **d**) Posttreatment AP and lateral views of venous injection showing coils in the right superior ophthalmic vein, cavernous sinus, proximal cortical draining vein, and the inferior petrosal sinus

Complications

Both transarterial and transvenous endovascular treatment of DAVFs have associated complications. The primary concern with endovascular treatment of these lesions revolves around under- and overtreatment of the shunts. Incomplete treatment of DAVFs can lead to recruitment of more arterial feeders and a more difficult-to-treat lesion, while leakage of embolic material into the sinus can lead to pulmonary embolism or venous sinus thrombosis and infarction. Persistent neurological deficits and cranial nerve palsies have also been reported.

Due to the small numbers of cases included in most series, as well as the evolving nature of endovascular therapy for DAVFs, a consistent rate of complications is hard to define. A recent review of 260 cases over 30 years, carried out by Gross et al., places the overall complication rate at 8%. The rate of permanent neurological complications was 3% and included venous infarction, MCA territory infarction, facial nerve palsy, anesthesia dolorosa, and worsened ophthalmoparesis following cavernous DAVFs. The authors found no difference in the rate of complications between DAVFs treated in the pre-Onyx era and after the advent of Onyx [30]. Another review of fewer patients treated solely in the Onyx era found the overall rate of complications to be in the same range, at 10% [31]. A prior study of 40 patients, also treated solely using Onyx, found a slightly higher overall rate of complications, at 23% [32]. Complication avoidance is of paramount importance. This is achieved by a thorough understanding of the anatomy of the lesion. For example, lesions supplied by the neuromeningeal branch of the ascending pharyngeal artery can have higher complication rates during embolization because of the arterial supply to the cranial nerves in the jugular foramen. As well, anastomotic channels between the external carotid artery and intracranial ICA and vertebral artery must be identified and considered to prevent embolic material from entering the intracranial circulation.

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