Cranial Dural AV Fistulas: Making Sense of Who to Treat and How

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Introduction

Cranial dural arteriovenous fistulas (DAVFs) are abnormal vascular malformations of the cranial dura that involve direct connections between meningeal arteries draining into veins adjacent to dural venous sinuses. Their etiology remains unknown, though dural fistulas are known to occur after venous sinus thrombosis and trauma. Microsurgical, radiosurgical, and interventional approaches all play critical roles in dural fistula management. These modalities can be used in isolation or in tandem depending on a number of factors including location, anatomy of the fistula, and the feasibility of both arterial and venous access to the nidus and draining vein(s). Observation may be appropriate for select dural fistula when they are low grade and not affecting quality of life. Careful assessment of clinical symptoms, the physical exam, noninvasive imaging, and a thorough cranial angiogram are the bedrock of safe and effective patient management. This chapter is augmented with a compendium of interesting illustrative case examples of DAVFs that have been managed by the senior author and clearly illustrates the concept of a multimodal management strategy.

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Table 19.1 Location distribution of DAVFs in the brain [3]

DAVF location	Frequency (%)
Transverse and sigmoid sinus	61
Cavernous sinus	10
Middle fossa	7.7
Convexity	6.5
Confluence of sinuses	3.1
Frontobasal	3.1
Tentorium	2.7
Superior sagittal sinus	2.3
Foramen magnum	1.9
Other	1.9

Epidemiology

The rate of detection of dural arteriovenous fistulas has been increasing in parallel to the evolution of imaging modalities [1–4]. Yearly population incidence rates for DAVFs are 0.15 per 100,000 individuals, but have been reported as high as 0.29 per 100,000 (Japan) to 0.51 per 100,000 (Finland) [3, 5, 6]. These lesions have a 1:1 male to female predilection and typically present during the fifth and sixth decades [1, 3]. The anatomic distribution of cranial dural fistulas varies widely as outlined in Table 19.1.

Clinical Presentation

The range and severity of symptoms depends on the location, hemodynamics, and venous drainage patterns of the dural AVFs [7]. Patients may present with a wide variety of symptoms ranging from mild to severe and even fatal [8–11]. Symptoms are known to correlate to location. DAVFs related to the cavernous sinus typically manifest with ocular symptoms such as exophthalmos, visual disturbance, orbital pain or swelling, and ophthalmoplegia (Case 19.5) [1, 12, 13]. DAVFs related to the transverse-sigmoid junction can present with pulsatile tinnitus as they are contiguous to the auditory apparatus [12, 14]. DAVFs draining into the superior sagittal sinus or deep veins may manifest with symptoms of prolonged intracranial hypertension and venous congestion, such as hydrocephalus, seizures, and papilledema [1, 10, 14]. Fistulas involving the brainstem may present with cranial nerve involvement, motor weakness, or paralysis [15]. Rarely, fistulas may present with cognitive dysfunction and memory loss (Cases 19.1 and 19.4).

Diagnostic Approach and Imaging

The history, physical exam, and noninvasive imaging are all important for proper selection of patients for further workup with a diagnostic cerebral angiogram. MRA and CTA in patients who present with brain hemorrhage can show clues that point toward the diagnosis of dural fistula (Case 19.3). Fundamentally a cerebral angiogram remains the gold standard of diagnosis and the most helpful

modality for treatment decision-making [3, 16–18]. It is crucial to include both external carotid systems in the angiographic injection, in addition to the internal carotid and vertebral systems.

Grading

The first classification system for dural arteriovenous fistulas was proposed by Djindjian and Merland who analyzed angiographic findings in relation to hemorrhagic risk. Accordingly, they classified dural fistulas into grades I–IV (Table 19.2). Contemporary classification systems currently followed in clinical practice, however, are the Cognard and Borden classification systems [10, 19]. Borden classified DAVFs into three grades

Table 19.2 Merland and Djindjian classification of dural AVFs [19]

Merland and Djindjian type	
I	Dural sinus or meningeal vein
II	Dural sinus with cortical venous reflux
III	Purely into cortical vein
IV	Cortical vein with supra- or infratentorial venous lake

Table 19.3 Borden classification of dural AVFs [19]

Borden classification	Description
1	Venous drainage directly into venous sinus or meningeal veins
2	Venous drainage into dural venous sinus with CVR
3	Venous drainage directly into subarachnoid veins (CVR) only

Table 19.4 Cognard classification of dural AVFs [10]

Grade	Venous drainage	Venous sinus flow	Cortical venous flow	Hemorrhage risk (%)	Description
I	Venous sinus	Antegrade	Antegrade	~0	Venous drainage into dural venous sinus with antegrade flow
IIa	Venous sinus	Retrograde	Antegrade	~0	Venous drainage into dural venous sinus with retrograde flow, no cortical vein involvement
IIb	Venous sinus	Antegrade	Retrograde	20	Venous drainage into dural venous sinus with antegrade flow and cortical vein reflux
IIa+b	Venous sinus	Retrograde	Retrograde	6	Venous drainage into dural venous sinus with retrograde flow and cortical vein reflux
III	Cortical veins	N/A	Retrograde	45	Drains directly into cortical veins
IV	Cortical veins	N/A	Retrograde	60	Drains directly into cortical veins with venous ectasia
V	Spinal medullary veins	N/A	N/A	N/A	Spinal perimedullary venous drainage, associated with progressive myelopathy

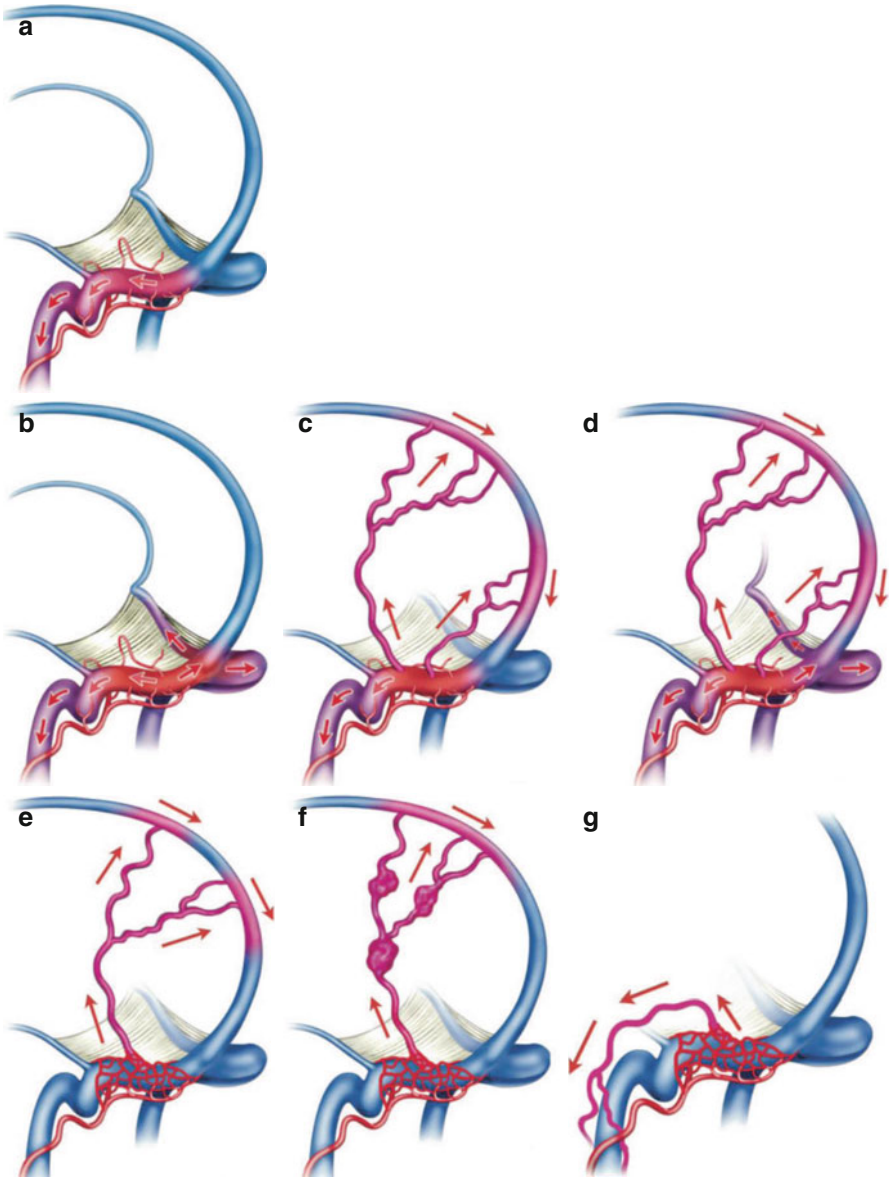

Fig. 19.1 Cognard (C) and Borden (B) DAVF classification. The fistula is located in the wall of the transverse sinus in each example: (**a**) C1, B1, antegrade flow in sinus. (**b**) C2a, B1, retrograde flow in the sinus. (**c**) C2b, B2, retrograde reflux into cortical veins with antegrade flow in sinus. (**d**) C2a+b, B2, retrograde flow in the sinus and reflux into cortical veins. (**e**) C3, B3, direct fistula drainage into cortical veins. (**f**) C4, B3, similar to E but associated with venous ectasia. (**g**) C5, drainage directly into perimedullary spinal veins [20]

Table 19.5 Barrow classification of carotid cavernous fistulas [21]

Grade	Characteristics
A	Direct internal carotid artery (ICA)-cavernous sinus fistula
B	Dural ICA branch-cavernous sinus fistula
C	Dural external carotid artery branch-cavernous sinus fistula
D	ICA/ECA dural branches-cavernous sinus fistula

(I–III) (Table 19.3), while Cognard classified the lesions into five grades (grade I–V) with three subtypes for class II DAVFs (IIa, IIb, and IIa+b) (Table 19.4) (Fig. 19.1). Based on the Cognard classification, the annual risk of hemorrhage from a type I or type IIA arteriovenous fistula is zero. Type II B is associated with an overall risk of 20%, whereas types III and IV are associated with an overall hemorrhage risk of 40% and 65%, respectively. In both classifications, type I and II fistulas drain into the venous sinuses, and type III into cortical veins. Additionally, in Cognard's classification, type IV fistulas drain into cortical veins and are associated with venous ectasia, and type V drain into perimedullary spinal veins (Fig. 19.1).

Carotid cavernous sinus fistulas (CCFs) are classified separately using the Barrow classification system (Table 19.5). The typical "dural" CCFs are types B–D. Type A is a direct carotid to cavernous sinus fistula that typically presents acutely with aggressive symptomatology [21].

Natural History and Clinical Course

In the absence of cortical venous reflux (CVR), DAVFs typically present as incidental findings or with signs and symptoms of increased dural venous drainage (bruit, tinnitus) [1, 12, 13, 22]. In a study of 68 patients with dural arteriovenous fistulas and no cortical venous drainage [23], none of the patients had neurological deficits and only 1 (1%) developed intracranial hemorrhage during a mean follow-up period of 27.9 months. Furthermore, among 50 patients who underwent angiography at follow-up, only 2 (4%) patients developed cortical venous drainage [23]. Studies tend to indicate that dural fistulas without cortical venous drainage typically follow a benign natural history [24, 25].

In the presence of cortical venous reflux, however, patients with DAVFs are at an increased risk for intracranial hemorrhage or nonhemorrhagic neurological deficits. A study by van Dijk et al. [23] included 20 patients with DAVFs and cortical venous drainage who were treated partially or followed conservatively for a mean period of 4.3 years. 16 (80%) patients developed intracranial hemorrhage or neurological deficits. The calculated annual risks for intracranial hemorrhage and neurological deficits were 8.1% and 6.9%, respectively [23]. In a meta-analysis conducted by Awad et al. on 360 tentorial DAVFs, 31 of 32 (96%) patients with cortical venous drainage developed hemorrhagic or nonhemorrhagic neurological sequelae. Other studies have also shown an increased risk of intracranial hemorrhage and neurological deficits for patients who have venous varices and anomalies involving the deep venous system [1, 24, 26, 27].

Treatment

Patient Selection for Treatment

The key decision in DAVF management is to identify patients who need treatment. The presence of cortical venous reflux (Borden types II, III; Cognard types II b, II a+b, III, IV, and V) is a potentially concerning feature and should lead to strong consideration of treatment [28–34]. Careful consideration should be given not to attribute an unrelated hemorrhage or progressive neurological symptoms to a low-grade fistula (Borden and Cognard types I). High-grade fistulas that present with hemorrhage are invariably selected for treatment to obliterate the fistula [30]. The importance of a thorough, unhurried clinical encounter with a patient cannot be overemphasized; unilateral pulsatile tinnitus with a low-grade fistula that is interfering with quality of life is a potential indication for treatment. Observation is a valid strategy for DAVFs that are low grade, but conversion of a low-grade fistula to a high-grade one while on observation can occur at an annual rate of about 1 % [35]. Hence periodic surveillance with MR imaging is warranted in patients who are being observed [36–39].

Formulating a Treatment Strategy

Once a decision is made to treat a DAVF, the goals and methods of treatment should be formulated by careful analysis of all imaging with special emphasis on the angiogram. However, visualizing the fistula is only one component of this analysis. Important questions that should be considered are:

1. The location of the fistula: certain locations favor endovascular therapy while others favor microsurgical treatment.
2. The anatomy of arterial and venous access from both microsurgical and interventional perspectives.
3. The relationship of feeding arteries to cranial nerves and potential extracranial to intracranial collateral.
4. The relationship of normal venous drainage to the arterialized sinus or arterialized cortical veins (drainage can be mixed).
5. What is the safest and easiest way to eliminate CVR?
6. Can the benign anatomic features of the fistula be treated safely and easily?

Endovascular Management

Advances in endovascular surgery and approaches over the past two decades have allowed for an increasing proportion of dural fistulas to be effectively treated with this approach (Case 19.2). Several embolic materials have been used for successful DAVF occlusion: NBCA (n-butyl cyanoacrylate glue) (Codman Neuro, Raynham, MA), Onyx (ethylene vinyl alcohol copolymer) (ev3 Endovascular Inc., Plymouth, MN), and

coils. Our preferred agent for most embolizations is Onyx for a number of reasons: First, Onyx possesses cohesive properties, as opposed to the adhesive character of NBCA, which allows for more controlled and prolonged injections [40, 41]. Second, Onyx allows for the possibility of precise, controlled injections, including stopping intermittently for short durations. Careful analysis of the angiogram can define the potential safety and success rate of both transvenous and transarterial approaches. Transarterial approaches can be quite straightforward if access can be achieved close to the nidus via a meningeal artery branch that is not in close proximity to cranial nerves. When cranial nerve proximity or potential extracranial to intracranial collaterals are at stake, then transvenous approaches may be preferable. When considering occlusion of venous structures, however, careful attention should be given to assure lack of normal venous drainage to those structures (mixed drainage). Adjuncts to transarterial Onyx may be required in technically challenging circumstances such as high-flow fistulas, in which the liquid material may quickly gain access and occlude the venous side or cause pulmonary embolism. This may take the form of balloon or coil assistance (flow control techniques) [41, 42]. Balloon- or coil-assisted Onyx embolization can potentially utilize a transarterial or transvenous route for either the balloon or coil and then employ Onyx through either route in various combinations [42, 43]. Novel dural lumen catheters have been used successfully [44, 45].

In some instances, an occluded sinus may be navigated using a microcatheter [41]. The transvenous route is by far most reliably used in management of type B–D CCFs. For non-CCFs, it has been primarily employed for TS-SS DAVFs (because anatomically they tend to have various routes for a transvenous approach) [46]. On the other hand, some DAVFs located at the tentorial incisura or anterior cranial fossa may not have accessible venous routes. Problems with transvenous occlusion include propagating venous thrombosis [47].

Obliteration rates with Onyx embolization are in the 68–92 % range [40, 48–50]. However, DAVFs that show complete obliteration on immediate postprocedure angiography have been demonstrated to occasionally recanalize or regrow on follow-up [50, 51].

Recanalization rates of around 10 % have been reported [52]. This reinforces the necessity of continued follow-up. It has been suggested that a short-term angiographic follow-up may be more predictive of long-term occlusion than relying on the immediate posttreatment angiogram [51].

Complications of cranial nerve paresis include those in the cavernous sinus (3–6) and the posterior fossa cranial nerves 7 and 9–12 with tentorial and TS-SS DAVFs and are described to occur in about 8 % of cases in most contemporary series [41, 53]. Mechanical complications such as catheter adherence and breakage are known to occur but do not necessarily translate into clinically significant problems.

Microsurgical Management

Despite the vast majority of DAVFs being treated with endovascular means, select circumstances in Borden types 2 and 3 (with CVR) lesions mandate open surgical

approaches. Such cases include instances where embolization was not performed due to difficult access, critical anastomoses, or the presence of arterial feeders with critical normal supply. An example of the latter is ethmoidal DAVFs with ophthalmic artery supply, which present an increased risk of vision impairment with transarterial embolization (Case 19.4). Microsurgery is also considered when embolization is not possible and angiography shows persistent filling [54]. Based on location, tentorial DAVFs are noted to have multiple tortuous arterial feeders that supply several cranial nerves along with less accessible transvenous routes. These factors make microsurgical management for these treacherous lesions an important option [55].

Current surgical techniques are vastly different from the traditional extensive resections in the pre-embolization/early embolization era [56, 57]. Contemporary surgical strategies may take the form of interruption of the draining vein close to the fistula using image guidance, surgical excision of the involved sinus, or direct sinus packing of a nonfunctional sinus. Hybrid approaches may involve burr hole placement to access an arterialized sinus with catheter-based occlusion then delivered endovascularly [58–60]. As a "hybrid" procedure, direct surgical access of the superior ophthalmic vein, a cortical vein, the vein of Galen, or the middle meningeal artery to deliver embolic material can be employed (Case 19.3) [61–63]. Some authors make a distinction between DAVFs with CVR that drain directly into the leptomeningeal vein (non-sinus type) and ones that occur via drainage into a venous sinus (sinus type) [64]. Non-sinus-type lesions are approached with interruption of the vein close to the dura, by clipping or by coagulation, and then sectioning without tackling the arterial feeders. The sinus-type lesions can be dealt with by sinus occlusion. An important caveat for a surgical approach is recognizing that the initial scalp, bony, and dural openings may be excessively bloody due to extensive external carotid arterial feeders. Preoperative embolization can ameliorate this issue [31]. Various technical adjuncts for surgery include frameless stereotaxy, intraoperative angiography or ICG videoangiography, and intraoperative Doppler ultrasound [39].

Stereotactic Radiosurgery

Stereotactic radiosurgery (SRS) has a delayed obliteration effect on dural fistulas. While this latency period may be tolerated for benign fistulas, the high annual hemorrhage rate for fistulas with CVR makes radiosurgery problematic as a first-line therapy [30, 33, 34]. SRS is, hence, chosen as a modality in situations where the lesion is not amenable to safe endovascular/microsurgical methods of obliteration or in a patient with severe medical comorbidities. Benign residual fistula after treatment can also be targeted with SRS to potentially reduce recurrence risk or eliminate residual pulsatile tinnitus. In case of low-flow/low-risk DAVFs such as CCF types B–D or Borden type 1, SRS may have more applicability, especially to treat a postembolization residual lesion [60]. The most common radiosurgery platform used is the Gamma Knife; however, any platform may be used for treatment delivery. The radiosurgical target is the nidus which typically is located in the wall of the

dural venous sinus. The dose depends on the lesion size and location and ranges from 14 to 25 Gy [65]. A recent meta-analysis of SRS for DAVFs reported a complete obliteration rate of 68.2 % over an overall mean follow-up period of 28.9 months [66]. Following SRS, there is an overall risk of hemorrhage that is in the range of 1.2–1.6 % [66, 67] over follow-up periods ranging from 2 to 11.4 years. This risk persists till complete obliteration and is higher in patients with CVR.

Cases

Case 19.1 High-Grade Dural Arteriovenous Fistula Simulating a Bilateral Thalamic Neoplasm

Summary: A 51-year-old male presented with bilateral thalamic lesions causing cognitive dysfunction. MRI demonstrated bilateral enhancing thalamic lesions with minimal mass effect (Fig. 19.2). Angiography revealed a thalamic DAVF supplied by bilateral middle meningeal arteries, marginal tentorial arteries from both ICAs, and a posterior meningeal artery from the left vertebral artery (Fig. 19.3). Transarterial endovascular embolization of the fistula was performed via both middle meningeal arteries using Onyx 18. Post-op angiography revealed complete resolution of the fistula (Fig. 19.4). Patient returned to his cognitive base-line 3 weeks after treatment.

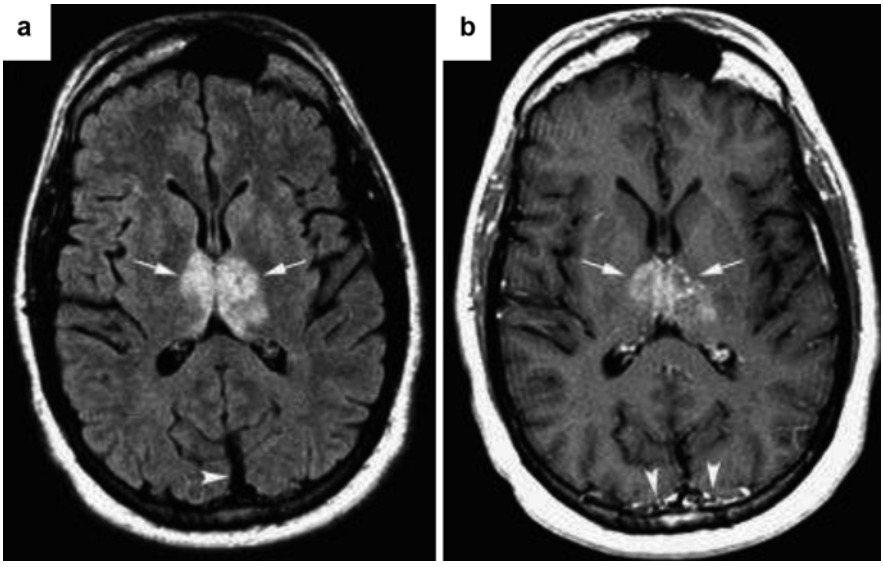

Fig. 19.2 (**a**) Axial FLAIR MRI sequence demonstrating hyperintense bilateral thalamic signal, represented by *arrows*. (**b**) T1 post-gadolinium showing bilateral thalamic enhancement, represented by *arrows*, and increased vascularity near the falcine sinus, represented by *arrowheads*

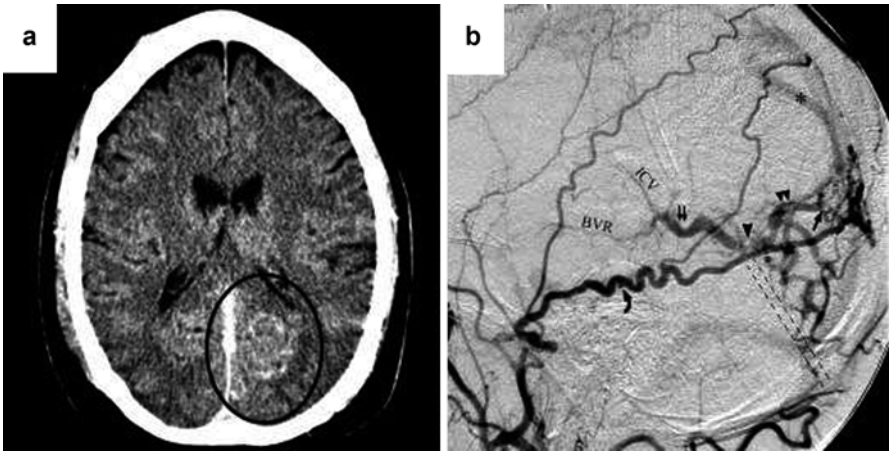

Fig. 19.3 (**a**) "Non-contrast" head axial CT (performed after an abdominal CT study for hematuria of unrelated etiology) shows enhancement of the left occipital cortical veins. (**b**) Right external carotid artery preoperative lateral cranial angiography shows an enlarged middle meningeal artery with a small flow-related aneurysm, represented by curved arrow, and supplying a leash of vessels which converge on a fistula, represented by *arrow*, draining into the posterior falcine sinus, represented by *double arrowheads*

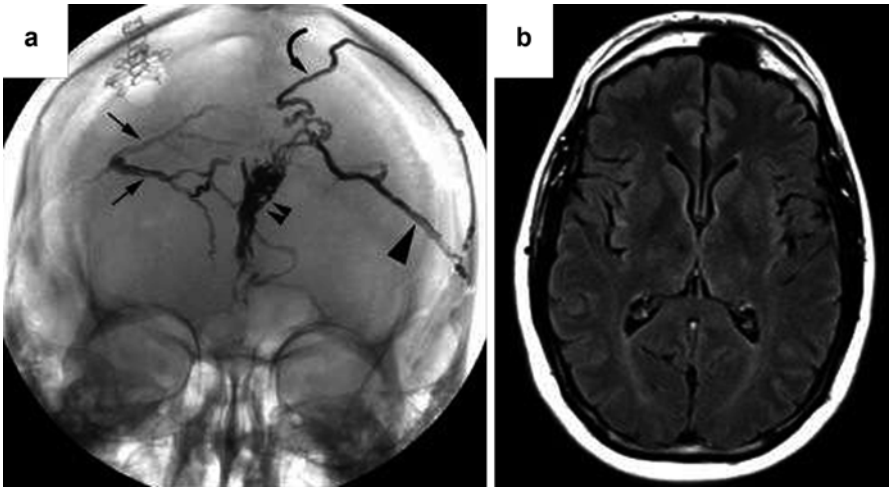

Fig. 19.4 (**a**) Angiogram following embolization of both the right middle meningeal feeders, represented by *arrows*, and left meningeal feeders, represented by *double arrowheads*. These structures were opacified by the injected Onyx. The *curved arrow* pinpoints reflux along a separate dural feeder which occurred during embolization on the *left*. (**b**) Follow-up magnetic resonance imaging (FLAIR) at 3 months interval exhibited resolution of the bithalamic high-signal aberrancy (Source: Sugrue et al. [68])

Case 19.2 Reversal of Diffusion Restriction After Embolization of Dural Arteriovenous Fistula

Summary: A 54-year-old male patient presents with a 5-day history of confusion and mental status changes. Computed tomography (CT) demonstrated gyral swelling and sulcal effacement associated with a small subcortical parenchymal hemorrhage in the left parietal region. MRI that was then performed demonstrated a broad zone of T2/FLAIR hyperintensity and restricted diffusion (Fig. 19.5). Cerebral angiogram revealed a Cognard type III left lateral tentorial DAVF with cortical venous drainage, resulting in significant left parieto-temporo-occipital venous hypertension (Fig. 19.6). The DAVF was treated by occluding the fistulous nidus endovascularly using Onyx 18. Postoperative cerebral angiography demonstrated complete obliteration with normalization of the venous drainage (Fig. 19.7). Follow-up MRI examination performed 4 weeks after the embolization revealed resolution of the previously seen area of restricted diffusion (Fig. 19.8). The patient was neurologically intact and seizure-free upon follow-up.

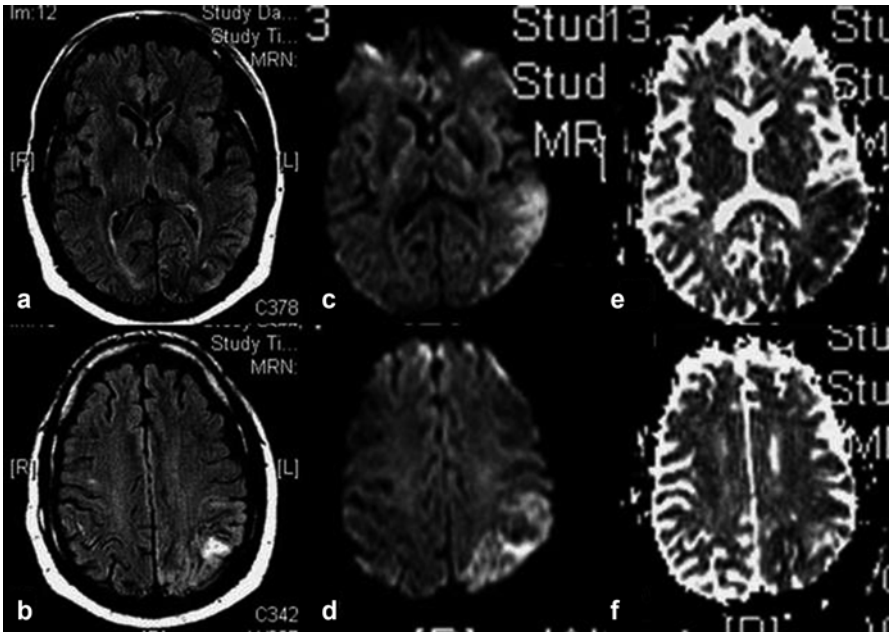

Fig. 19.5 (**a**, **b**) Magnetic resonance imaging (FLAIR sequence) showing a signal increase patient's left parietotemporal region. (**c**, **d**) Diffusion restriction is observed within the same area. (**e**, **f**) ADC map signal reduction is recognized as well

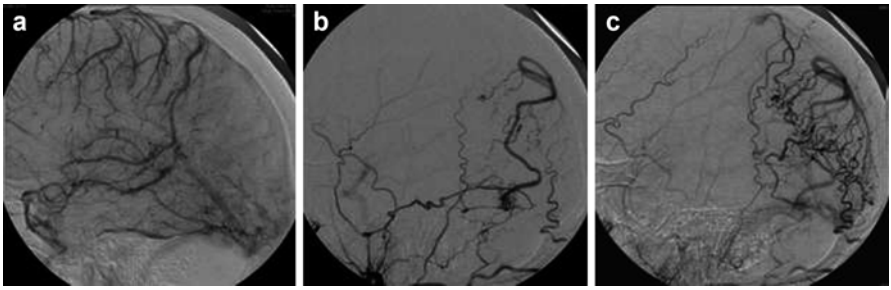

Fig. 19.6 Digital subtraction angiography (DSA) of left parieto-temporo-occipital region showing lack of cortical venous opacification. (**a**) Lateral venous phase of left internal carotid artery injection. (**b**, early phase, and **c**, late phase) Left external carotid artery injection demonstrating a Cognard type III left lateral tentorial DAVF causing considerable left parieto-temporo-occipital venous hypertension

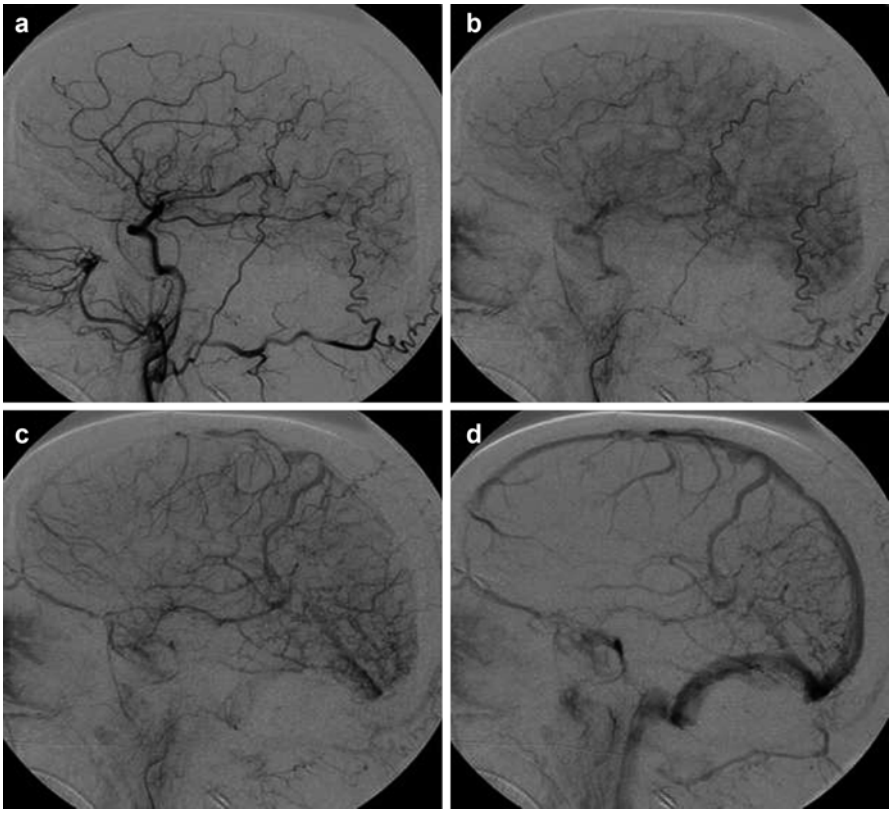

Fig. 19.7 (**a**) Posttreatment cerebral angiography revealing angiographic resolution of the aberrancy. (**b**–**d**) Restoration of the normal cortical venous drainage in the left parieto-temporo-occipital region

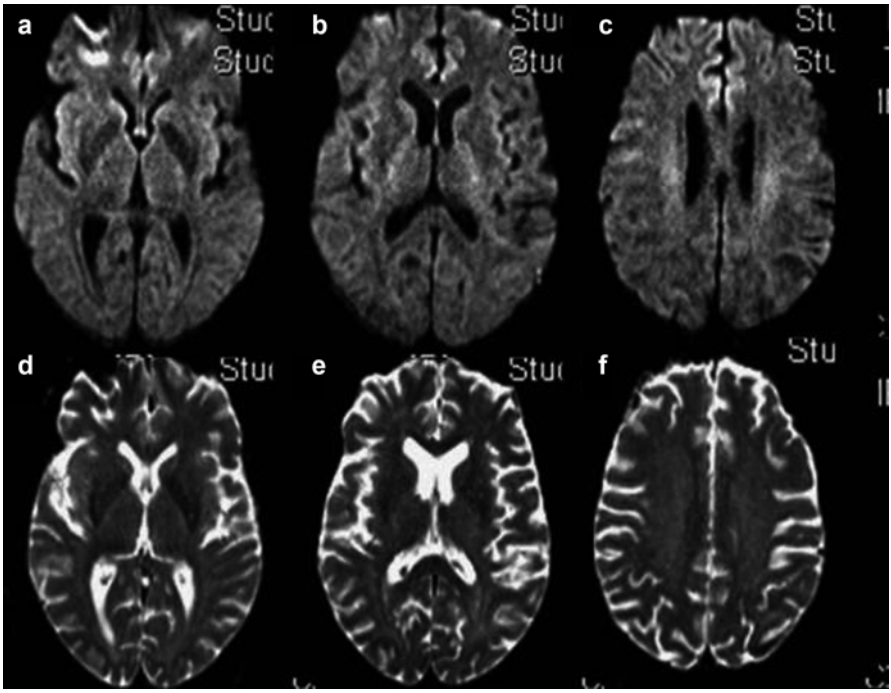

Fig. 19.8 (**a**–**f**) 4-week posttreatment follow-up magnetic resolution imaging (MRI) scans confirms the complete reversal of the diffusion restriction previously seen (Source: Dabus et al. [69])

Case 19.3 Combined Surgical and Endovascular Access of the Superficial Middle Cerebral Vein to Occlude a High-Grade Cavernous Dural Arteriovenous Fistula

Link: http://links.lww.com/NEU/A393

Summary: A 75-year-old female patient presented with 3-month history of left retro-orbital headaches, 1 week of intermittent vertical and horizontal diplopia, and a few days of worsening slurred speech and RUE weakness. Computed tomography head scan without contrast demonstrated an acute left temporal lobe hemorrhage (Fig. 19.9a). Subsequent, magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) studies of the head revealed dilated cortical veins in the left sylvian fissure (Fig. 19.9b). Cerebral digital subtraction angiography revealed the presence of a high-grade left CS-DAVF supplied by the bilateral external carotid branches as well as the left ICA feeders (Fig. 19.10a). Drainage from the left CS was solely through the left SMCV into engorged perisylvian cortical veins without involvement of the right CS (Fig. 19.10b). Venous access to the lesion was determined to be challenging. Subsequently, transarterial embolization was attempted and was unsuccessful due to migration of Onyx into the intraorbital left lacrimal artery. Following this, we elected to perform a left orbitozygomatic craniotomy to provide exposure to the anterior left middle cranial fossa for direct access to the SMCV (Fig. 19.11a). The left

SMCV was then punctured with a 21-gauge micropuncture needle (Fig. 19.11b). The microcatheter was navigated over a microguidewire into the CS under fluoroscopic guidance (Figs. 19.12 and 19.13a). 19 detachable coils were then deployed resulting in the complete occlusion of the fistula (Fig. 19.13). Postoperative angiogram 1 week later confirmed complete obliteration of the fistula. At 6 months follow-up, patient had no neurological deficits and no cognitive dysfunction.

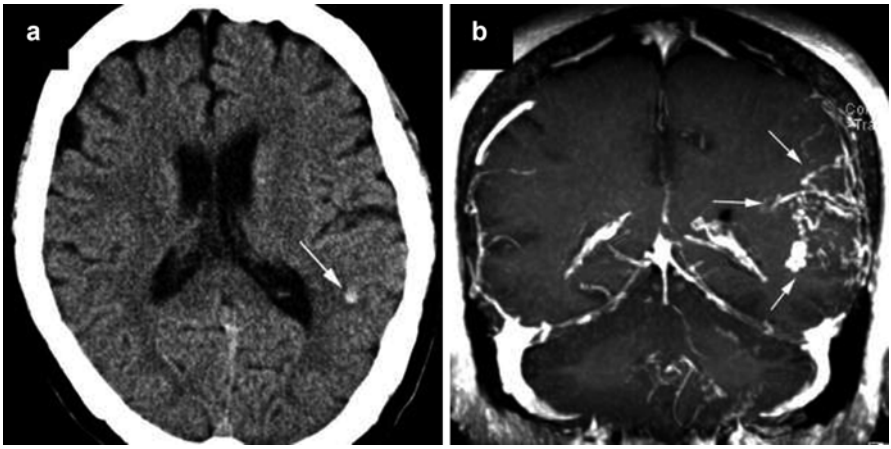

Fig. 19.9 (**a**) Axial head computed tomography without contrast revealing a small left temporoparietal hemorrhage. (**b**) Coronal magnetic resonance imaging (MRI) demonstrating dilated cortical veins in the left perisylvian region

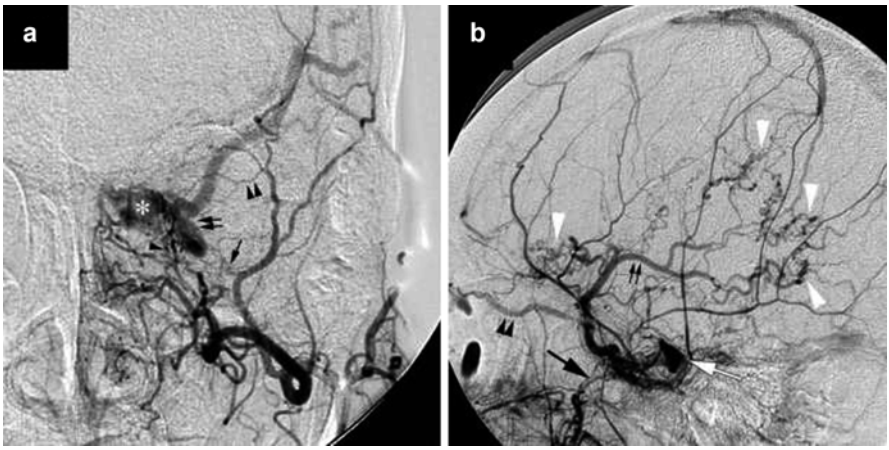

Fig. 19.10 (**a**) AP view of left external carotid digital subtraction angiogram revealing arterial feeders from the middle meningeal cavernous, represented by *arrows*, and recurrent branches, represented by *double arrowheads*, and the artery of the foramen rotundum (FAO), represented by an *arrowhead*, to a fistula centered on the left cavernous sinus (CS), represented by an *asterisk*, draining into the superficial middle cerebral vein (*SMCV*), represented by *double arrows*. (**b**) A lateral view revealing the posterior compartmentalization of the CS fistula, represented by *white arrow*, with FAO feeder, represented by a *black arrow*, and drainage through both the superior ophthalmic vein, represented by *double arrowheads*, and the SMCV, represented by *double arrows*. The *white arrowheads* represent the dilated cortical veins in the perisylvian region

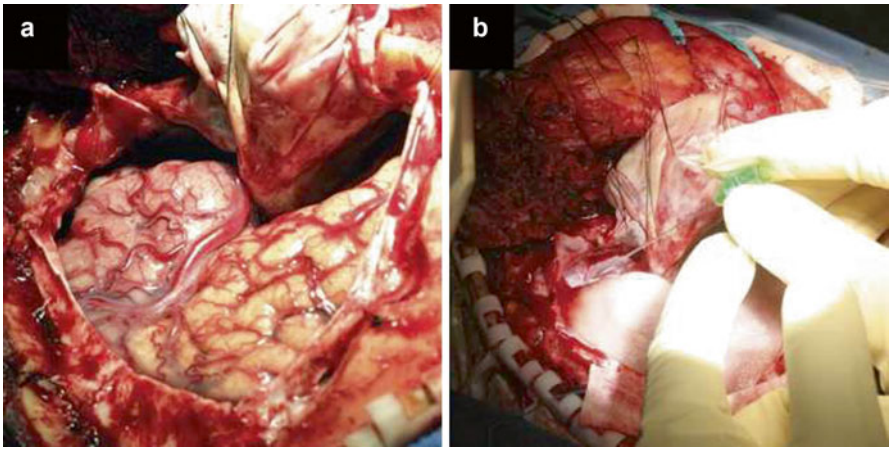

Fig. 19.11 (**a**) Exposure of the arterialized superficial middle cerebral vein (SMCV) and cortical venous tributaries after orbitozygomatic craniotomy. (**b**) Micropuncture of the SMCV

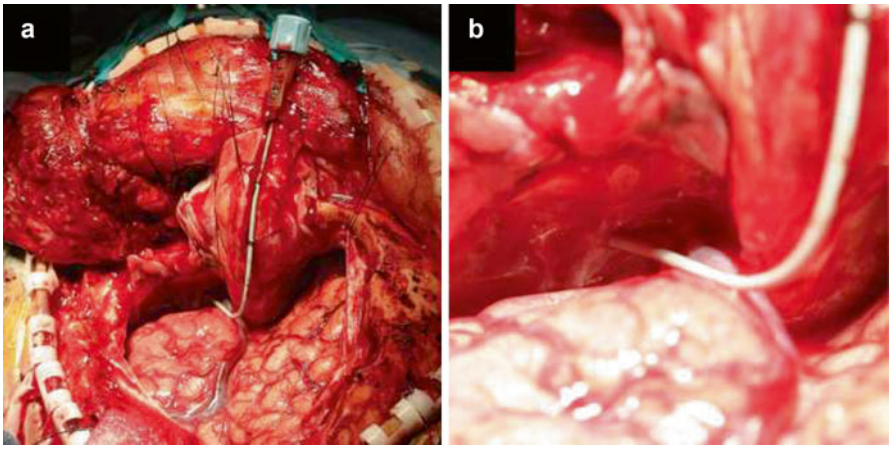

Fig. 19.12 (**a**) 4-French (outer diameter) micropuncture sheath positioned in superficial middle cerebral vein (SMCV). (**b**) Puncture site of sheath into the SMCV

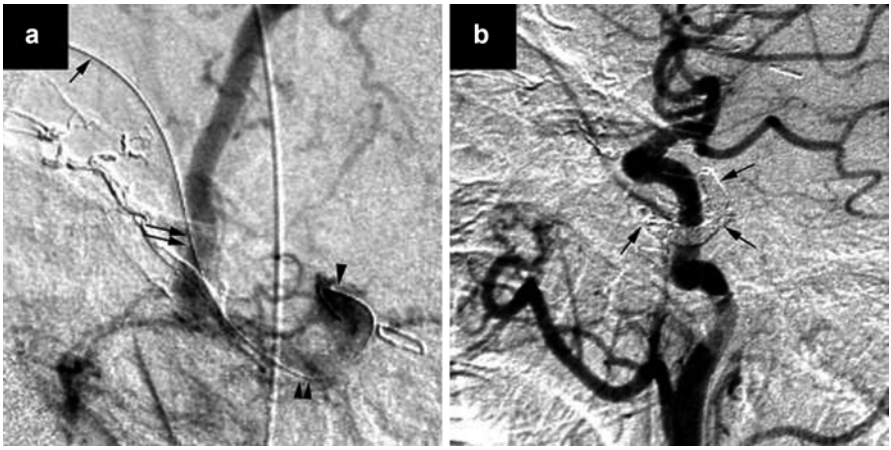

Fig. 19.13 (**a**) Lateral view of intraoperative left external carotid artery DSA showing the microcatheter, represented by an *arrow*, going into the superficial middle cerebral vein (SMCV), represented by *double arrow,* and treading across the floor of the middle cranial fossa, represented by *double arrowhead,* with its tip located in the cavernous compartment, represented by *arrowhead*. (**b**) *Left* carotid angiogram showing the endovascular coils occluding the cavernous sinus (Source: Hurley et al. [70])

Case 19.4 Microsurgical Treatment of an Ethmoidal Dural Fistula: Three-Dimensional Illustration

A 74-year-old male patient presents with memory loss. Magnetic resonance imaging revealed a left high-grade ethmoidal fistula. CTA demonstrated a large vessel emanating from the anterior left skull base with its vein connecting to the superior sagittal vein anteriorly (Fig. 19.14a). Cerebral angiogram revealed the fistula's supply to be from the branches of the ethmoidal artery and meningeal branches of the internal maxillary artery (Fig. 19.14b). The draining on the other hand is through the superior sagittal vein anteriorly. An open procedure was elected due to the risk of ophthalmic artery occlusion via endovascular treatment of ethmoidal fistulas. Subsequently, a bifrontal craniotomy was performed and occlusion of fistula was successfully executed (Fig. 19.15, Video Link 1). Postoperative angiogram revealed complete resolution of the fistula. On 1 month follow-up, patient had no neurological deficits and was able to fully resume his daily activities

Video Link 1: https://www.youtube.com/watch?v=CU5nQfv1BM4

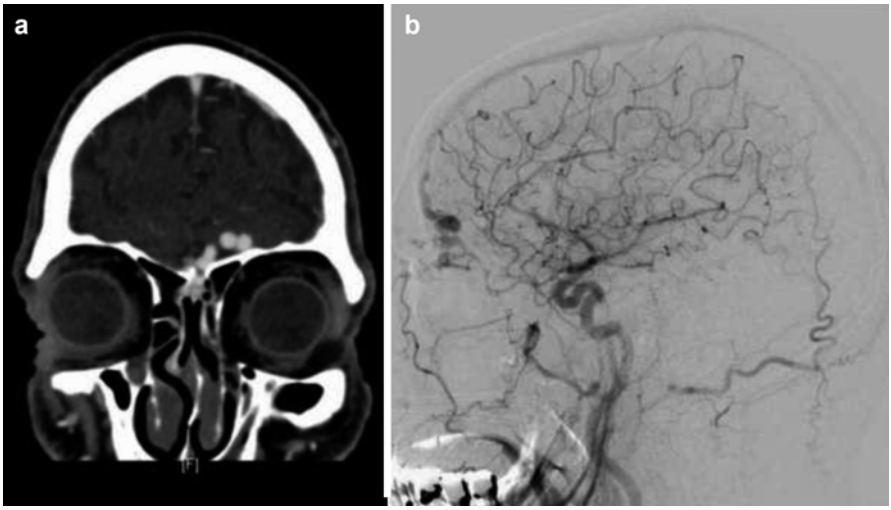

Fig. 19.14 (**a**) Computed tomography angiogram (CTA) and (**b**) digital subtraction angiogram of ethmoidal DAVF showing the large dilated draining vein

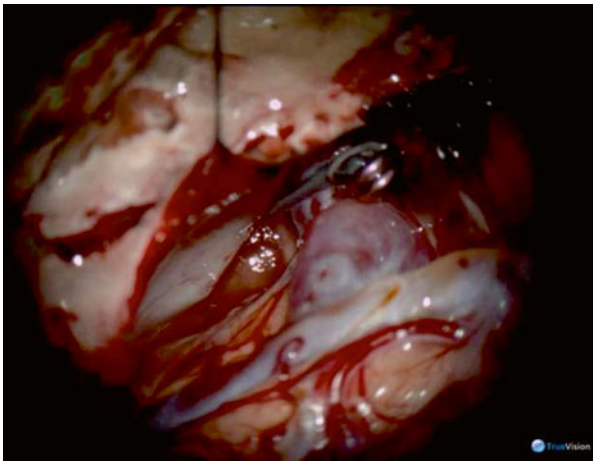

Fig. 19.15 Operative photograph following microsurgical disconnection of DAVF with an aneurysm clip at a site just distal to the exit of the large dilated draining vein just before ablation (Source: Aoun et al. [71])

Case 19.5 *Summary*: A 52-year-old female patient presents with bilateral proptosis and orbital chemosis. Angiography demonstrates bilateral mirror image Barrow type B cavernous sinus fistulas. The fistulas were draining through an ectatic superior ophthalmic vein with focal venous stenosis (Fig. 19.16). A transvenous approach was elected due to the presence of single draining veins from each side accessible through a transvenous approach. Coil-assisted Onyx embolization was carried out on both sides over two procedures with successful obliteration of the fistulas (Figs. 19.17, 19.18, and 19.19). The patient had complete symptom resolution.

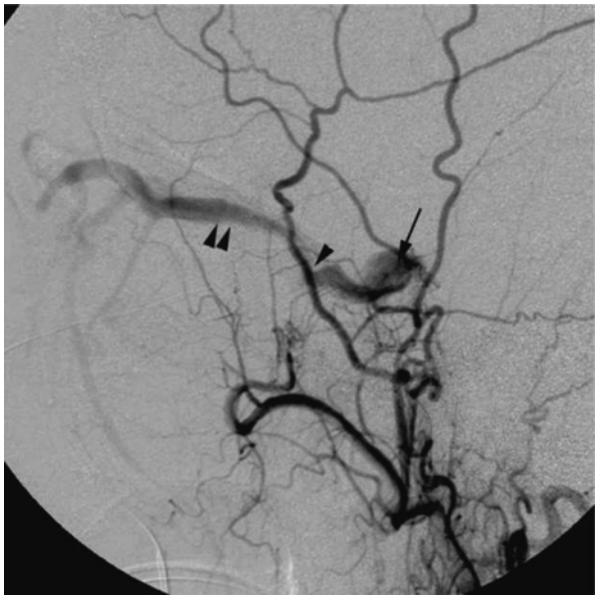

Fig. 19.16 DSA reveals a Barrow type D cavernous sinus fistula, represented by *arrow*, draining through an ectatic superior ophthalmic vein, represented by *double arrowhead*, with a focal venous stenosis, represented by *arrowhead*. Similarly on the contralateral side there was a mirror image fistula of the cavernous sinus (not shown). A transfemoral approach was utilized to navigate the external jugular, angular, and superior ophthalmic veins into the cavernous sinus

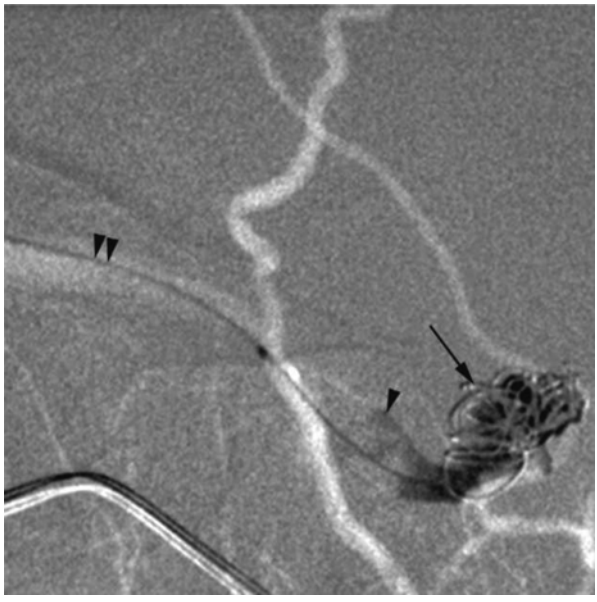

Fig. 19.17 After multiple coils were placed to slow down the flow, 3 cc of Onyx 34 was injected to fill the CS back into the posterior ophthalmic vein

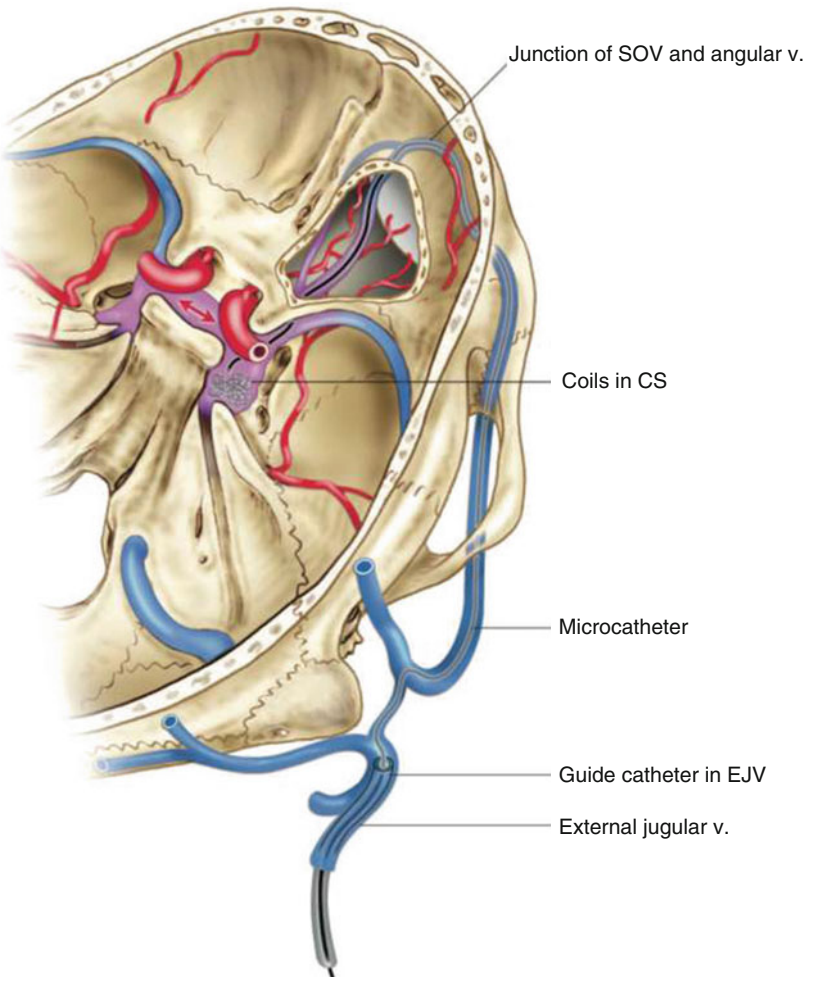

Fig. 19.18 The endovascular technique is rendered above. *CS* cavernous sinus, *EJV* external jugular vein, *SOV* superior ophthalmic vein. Both sides were managed via two elective procedures

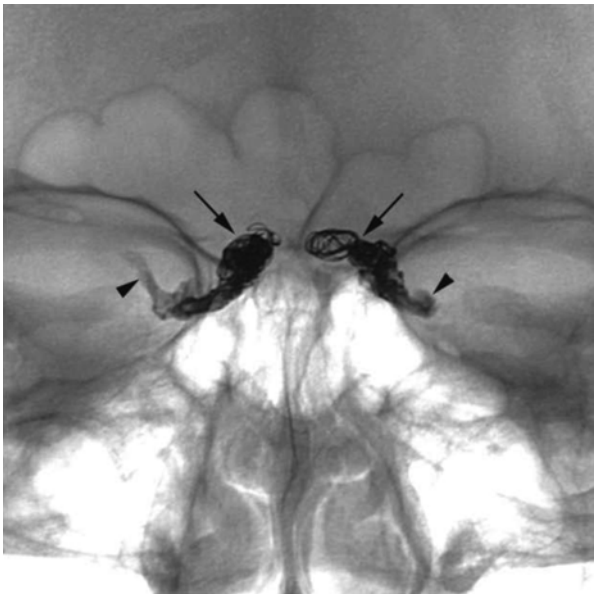

Fig. 19.19 The final appearance of the coils and Onyx is shown above. The *arrowheads* represent the Onyx and the *arrows* represent the coils (Source: Bendok et al. [20])

Conclusions

A comprehensive multidisciplinary evaluation of patients with DAVF is essential to guide best management practices. The decision of whether to observe or treat a fistula should be based on a detailed analysis of clinical and angiographic parameters of the DAVF. These include the presentation, location, and grade of the fistula. Accordingly, if it is decided to pursue treatment, the physician must create and implement an individualized plan based on three types of treatment modalities: endovascular embolization, microsurgery, and stereotactic radiosurgery. Each modality has certain specific strengths and limitations as described in the chapter. It is however fallacious to view the above three options in isolation or mutual exclusion. The ideal approach is an integrative multimodal management strategy that ensures the safety and efficiency of permanent occlusion of cranial dural fistulas.

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