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Intracranial Dural Arteriovenous Fistulas: A Systematic Approach—Diagnosis, Classification, and Endovascular Treatment

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Abstract

Intracranial dural arteriovenous fistulas (DAVFs) are rare lesions accounting for 10 to 15% of intracranial arteriovenous malformations. They involve an anomalous shunt between meningeal arteries, intracranial (venous sinuses and/or cortical veins), or medullary veins. The DAVFs are usually acquired, implying previous aggression of the dura mater. Thus, they are associated with dural sinus thrombosis, previous craniotomy, and trauma. However, they are idiopathic in most instances and have no evident cause. Their clinical presentation is variable, with symptoms depending on their location and venous drainage pattern. However, lesions with cortical venous drainage have the highest risk of causing the most significant morbidity and mortality. High clinical suspicion alongside noninvasive cross-sectional imaging techniques such as computed tomography and magnetic resonance imaging help establish the diagnosis. Digital subtraction angiography is the gold standard for diagnosis and accurate classification, permitting the evaluation of the feeding vessels, cortical venous drainage, and venous ectasia. Accordingly, a prompt diagnosis and precise classification of these lesions are essential. Endovascular treatment is nowadays the primary therapeutic modality for DAVFs. The access route can be divided into transarterial, transvenous, and combined approaches based on angioarchitecture, venous drainage model, and location. Surgical resection and stereotactic radiosurgery may be considered in some cases. A personalized case-by-case approach accomplishes a high complete treatment grade with a low complication rate. This review highlights the epidemiology, pathogenesis, clinical presentation, classification, diagnosis, and endovascular treatment of patients with intracranial DAVFs.

Keywords
- angiogram
- arteriovenous fistulas
- dural
- embolic agent
- embolization

Introduction

Intracranial dural arteriovenous fistulas (DAVFs) are abnormal shunts within the dural leaflets between meningeal arteries, dural venous sinuses, and/or cortical veins, or medullary veins. They account for approximately 10 to 15% of all intracranial vascular malformations.[1] Evidence suggests that DAVFs are rather acquired lesions and present later in life compared to cerebral arteriovenous malformations (AVMs).[2] However, their etiology remains unclear, and they are mainly considered idiopathic. Intracranial venous sinus thrombosis with subsequent venous hypertension seems to be the leading

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mechanism for fistulous arteriovenous connection in the dura mater. Thus, some patients have a history of craniotomy, head trauma, infection, tumor, or dural sinus thrombosis.[3,4] Their clinical course varies from harmless with spontaneous remission to lethal due to cerebral hemorrhage, with symptoms depending on their location and venous drainage pattern. However, lesions with cortical venous drainage have the highest risk of causing the most significant morbidity and mortality.[5] Although DAVFs frequently occur near dural venous sinuses, they can develop anywhere within the intracranial dura mater. The typical locations include the cavernous sinus, the ethmoidal cribriform plate, the transverse sigmoid sinus, and the tentorium.[2] This article will summarize the epidemiology, pathogenesis, clinical presentation, diagnosis, classification, and Treatment of DAVFs.

Epidemiology

DAVFs represent 10 to 15% of all intracranial vascular malformations.[1] The mean age at presentation is between 50 and 60 years, though DAVFs can occur at any age. There is no gender predominance, but some studies reported an increased incidence of hemorrhagic complications in men compared to women.[1,6] The detection rate varies from 0.15 to 0.29 per 100 000 per year[2]. The individual presentation is highly variable, and the detection rate is increased due to the development of cross-sectional imaging techniques.[6] DAVFs seen in adults are thought to be acquired.[2] Nevertheless, those occurring in children are believed to be congenital.[1,2] The most common locations involve the cavernous sinus, the ethmoidal cribriform plate (anterior fossa), the transverse sigmoid sinus, and the tentorium.[2,4,7] DAVFs are usually single, but multiple lesions have been reported.[6]

Pathogenesis

DAVFs are mainly idiopathic, though some studies report patients with prior craniotomy, head trauma, infection, tumor, or dural sinus thrombosis. The leading mechanism proposed is sinus thrombosis with subsequent venous hypertension resulting essentially from two hypotheses; the former is the enlargement of physiologic arteriovenous shunts between meningeal arteries and dural venous sinuses resulting from venous hypertension; the latter is the neoangiogenesis resulting from decreased cerebral perfusion led by venous hypertension due to outflow obstruction.[2,3] Pediatric DAVFs are thought to be congenital due to birth trauma, infection, in-utero venous thrombosis, or maternal hormones.[3]

Clinical Presentation

Incidentally diagnosed DAVFs are increasing due to the wide availability and frequent use of high-resolution computed tomography (CT) and magnetic resonance imaging (MRI).[7] Nevertheless, the majority of DAVFs are diagnosed after the development of symptoms. The clinical presentation of DAVFs highly depends on the venous drainage pattern and the fistula's location and results from either increased dural sinus drainage or the development of cortical venous hypertension.[2,7–9] Antegrade-flow DAVFs (i.e., antegrade drainage into the dural sinus and the ipsilateral jugular vein without cortical vein drainage [CVD]) usually exhibit nonaggressive presentations such as headaches, pulsatile tinnitus, and bruits (►**Fig. 1**). On the other hand, retrograde-flow DAVFs (i.e., retrograde drainage into the dural sinus crossing the torcular down the contralateral jugular vein) result in increased intracranial venous pressure (►**Fig. 2**). Thus, patients present with severe headaches, papilledema, and progressive dementia. DAVFs that exhibit CVD (►**Fig. 3**) usually present with the most aggressive clinical manifestations, inclusive of intracranial hemorrhage (ICH), and nonhemorrhagic neurologic deficits (NHNDs) such as progressive dementia, seizures, parkinsonism, or ataxia due to parenchymal edema or ischemia.[2,7,8] DAVFs draining into perimedullary spinal veins may cause myelopathy and progressive tetraplegia (►**Fig. 4**). The symptoms are, as a rule, benign unless-retrograde dural sinus, cortical, or perimedullary venous drainage occurs.[2] The clinical presentation of DAVFs is closely related to the location. Transverse sigmoid junction fistulas typically demonstrate pulsatile tinnitus due to auditory apparatus closeness. Likewise, middle fossa fistulas are more likely to present with pulsatile tinnitus due to increased drainage through the sigmoid and transverse sinuses.

Cavernous sinus DAVFs (indirect carotid cavernous fistulas) arterialize the ophthalmic vein, thus leading to exophthalmoplegia, proptosis, chemosis, retroorbital pain, decreased visual acuity, and visual loss due to increased intraocular pressure. Anterior cranial fossa DAVFs are fed by ethmoidal arteries and frequently drain into the cavernous sinus. Therefore, these lesions often present with ophthalmological manifestations. Besides, they have a proneness for hemorrhagic presentation, given the frequency of retrograde CVD with these fistulas. Likewise, tentorial DAVFs also have a high propensity for ICH, given their frequent retrograde CVD. DAVFs involving the superior sagittal sinus or those that drain into the deep venous system frequently present with NHNDs. Symptoms of brain stem DAVFs include quadriparesis or lower cranial nerve palsies originating from venous congestion and ischemia within the brain stem.[2,7]

Classification

Current classification systems are based on the venous drainage pattern, which is reliable in predicting the clinical behavior of DAVFs. The Cognard et al[10] and the Borden et al[9] classifications are the most commonly used grading systems.

The Borden classification system consists of three types (►**Table 1**). Type I DAVFs drain directly into a dural sinus without cortical venous reflux. Type II DAVFs drain directly into a dural venous sinus with a retrograde flow into cortical veins. Finally, type III DAVFs drain entirely into a cortical vein. Besides, patients with a single fistula are classified as type a, while patients with multiple fistulas are classified as type b. In this scheme, patients with type II and III DAVFs are at the highest risk of ICH and/or NHNDs.[9]

The Cognard classification system (type I–V) (►**Fig. 5**) is based on the direction of dural sinus drainage (antegrade or

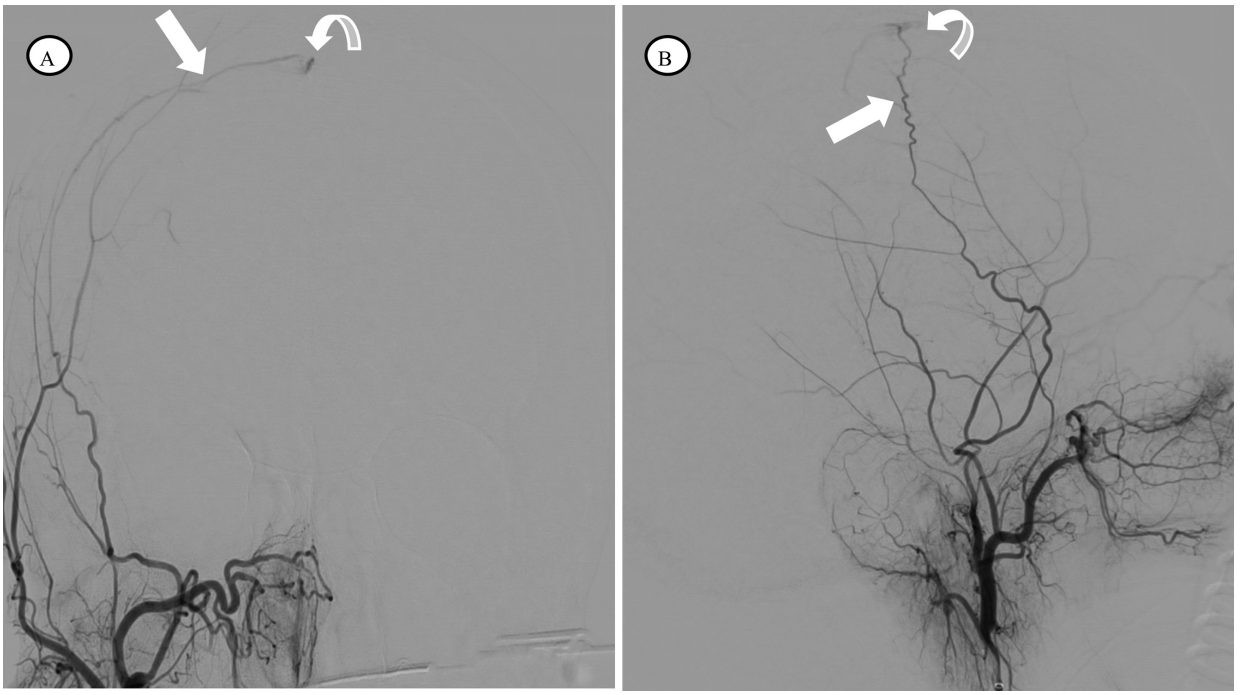

Fig. 1 A 50-year-old man presented with persistent headaches following head trauma. **(A** and **B)** Right external carotid injection, frontal and lateral views, respectively, showing a superior sagittal sinus dural arteriovenous fistula (Cognard I) with arterial supply from the posterior branch of the middle meningeal artery straight arrow. Note the antegrade drainage into the superior sagittal sinus curved arrow.

retrograde), cortical venous drainage (present or absent), and cortical venous angioarchitecture (nonectatic cortical vein, ectatic cortical vein, and perimedullary vein) (►**Table 2**). Type I lesions drain antegrade into a dural sinus without cortical venous drainage. Type IIa lesions drain retrograde into a dural sinus without cortical venous drainage, type IIb lesions drain antegrade into a dural sinus with cortical venous drainage, and type IIa + b drain retrograde into a dural sinus with cortical venous drainage. Type III lesions drain entirely into cortical veins without venous ectasia. Type IV lesions drain into cortical veins with venous ectasia. Finally, type V lesions drain into spinal perimedullary veins.[10]

Diagnosis

The initial radiologic evaluation comprises CT and MRI. The role of noncontrast CT is to point out ICH and edema caused by venous congestion. MRI is comprehensive as it evaluates the

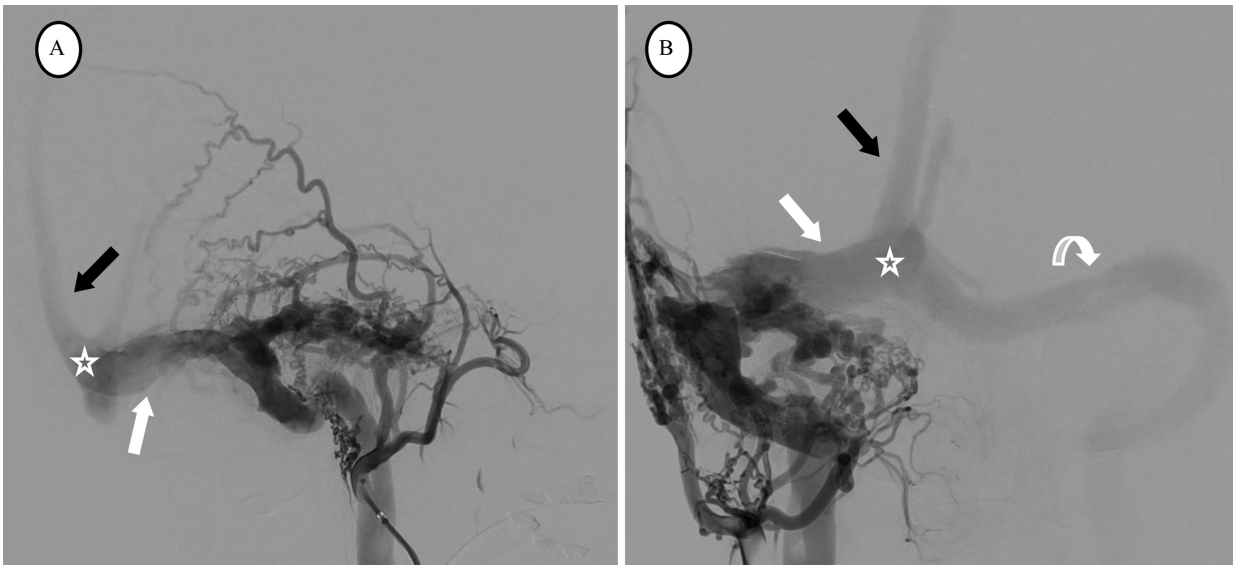

Fig. 2 A 29-year-old man with a history of treated right parotid malignancy presented with severe headache alongside papilledema and pulsatile tinnitus. (**A** and **B**) Right external carotid artery injections, frontal and lateral views, respectively, confirm the presence of right lateral sinus dural arteriovenous fistula (Cognard IIa) with arterial supply from the middle meningeal and superficial temporal arteries. Note the retrograde drainage into the homolateral lateral sinus, the torcular (star), the superior sagittal (black arrow), and the left sigmoid sinuses (curved arrow).

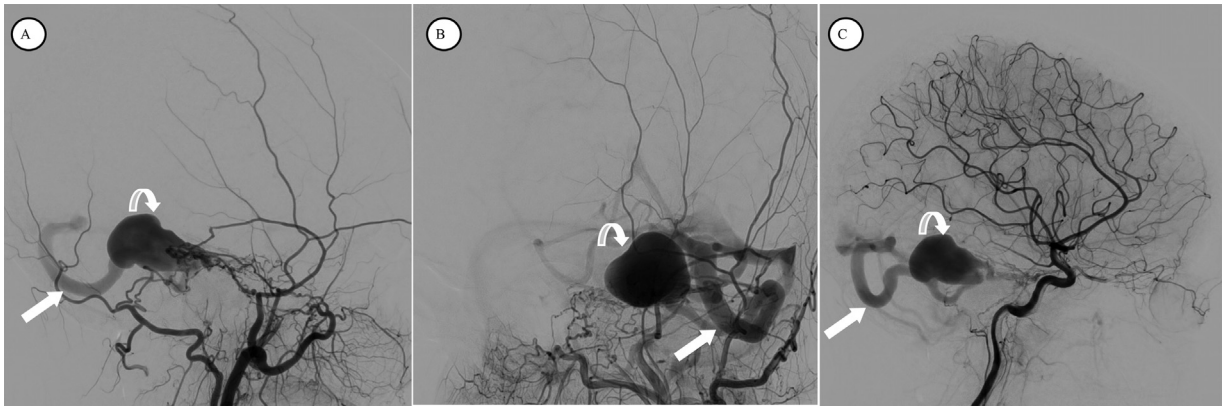

Fig. 3 A 59-year-old woman with a subarachnoid hemorrhage presented severe headaches accompanied by seizures. (**A** and **B**) Left external carotid injections, lateral and frontal views, respectively, and (**C**) left internal carotid injection, lateral view, showing a tentorial dural arteriovenous fistula (Cognard IV) with arterial supply from middle meningeal, occipital arteries, and branches from meningohypophyseal trunk. Note the direct drainage into a cortical vein (arrow) joining the left lateral sinus and presenting venous ectasia (curved arrow) at its origin.

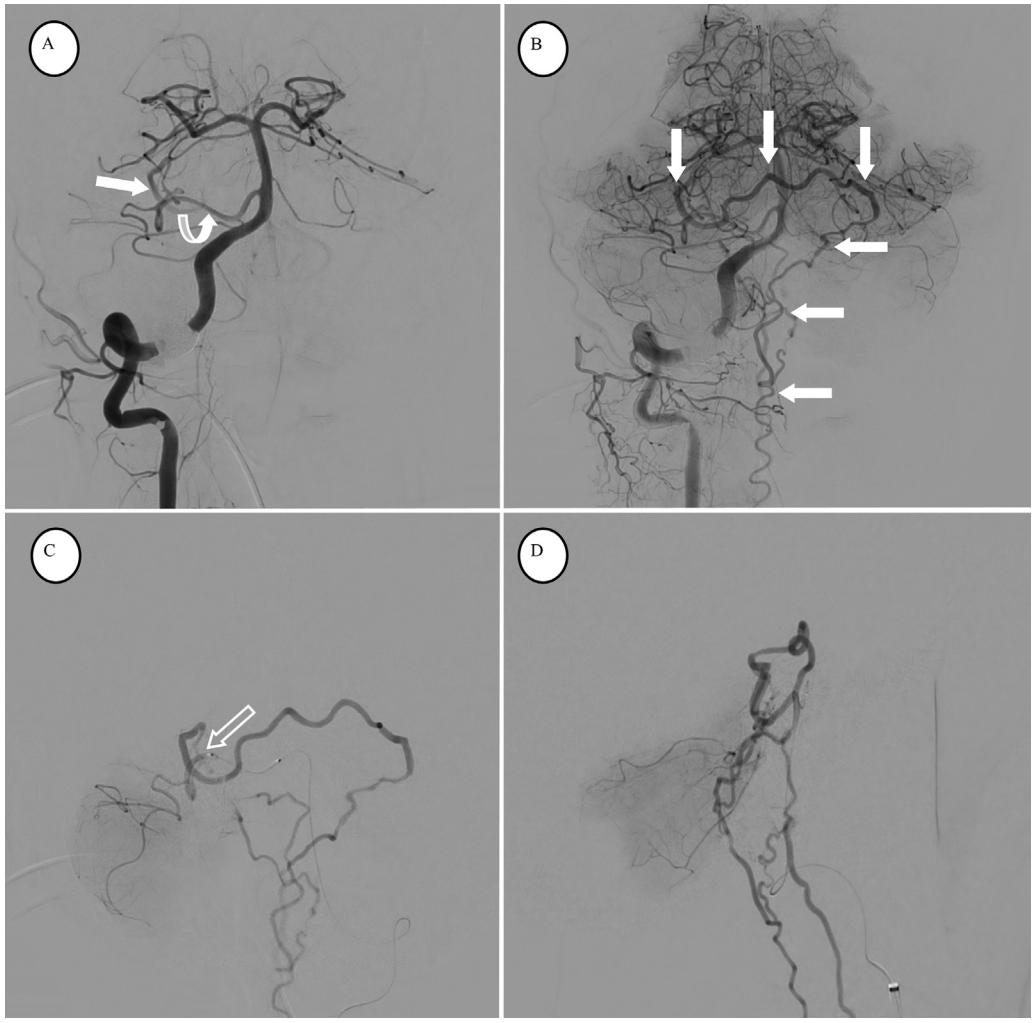

Fig. 4 A 60-year-old man presented with neck pain, progressive upper and lower extremities weakness, and increased reflexes. Medullary magnetic resonance imaging was suspicious for spinal vascular malformation. (**A** and **B**) Right vertebral artery injection, early and late arterial phases, respectively, showing a posterior fossa dural arteriovenous fistula (Cognard V) with arterial supply from the right anterior–inferior cerebellar artery (curved arrow) and draining perimedulllary veins (arrows). (**C** and **D**) Superselective right anterior–inferior cerebellar artery injections, frontal and lateral views, better identify the fistulous site (hollow arrow) and the anterior and posterior draining veins.

Table 1 Borden classification of intracranial dural arteriovenous

Type	Description
Type I	Direct drainage into a dural sinus without cortical venous reflux
Type II	Direct drainage into a dural venous sinus with a retrograde flow into cortical veins
Type III	Drainage into a cortical vein in its entirety

Table 2 Cognard classification of intracranial dural arteriovenous

Type	Description
Type I	Antegrade drainage into a dural sinus
Type II • IIa • IIb • IIa + b	Retrograde drainage into a dural sinus without cortical venous drainage Antegrade drainage into a dural sinus with cortical venous drainage Retrograde drainage into a dural sinus with cortical venous drainage
Type III	Drainage into a cortical vein in entirety without venous ectasia
Type IV	Drainage into a cortical vein in entirety with venous ectasia
Type V	Drainage into a spinal perimedullary vein

anatomy of involved vessels, cortical vein outpouching, and signs of venous hypertension in high-grade lesions (e.g., white matter high signal on T2-weighted images, ICH, or venous infarction). The findings differ depending on the type of DAVF. Type I and II fistulas may display flow-void clustering and ophthalmic manifestations. More aggressive type II and III fistulas are more likely to show dilated vessels, prominent vascular enhancement, and hemorrhage. A further angiographic study using CT angiography (CTA) or MR angiography (MRA) should be ordered for a detected flow void cluster adjacent to a dural venous sinus. With its high spatial resolution, CTA is useful in highly suspecting the diagnosis. However, many studies have reported higher sensitivity for MRA in detecting DAVFs compared to CTA (50 vs. 15.4%). Therefore, time-resolved MRA techniques are increasingly used for DAVF screening and post-treatment surveillance. Nevertheless, due to limited spatial resolution and field of view alongside saturation artifacts, MRA's negative predictive value is insufficient to exclude DAVFs formally.

Digital subtraction angiography (DSA) remains the gold standard for diagnosing and accurately classifying DAVFs, permitting the evaluation of the feeding vessels, cortical venous drainage, and venous ectasia. A six-vessel angiography is mandatory, including both internal carotid arteries, external

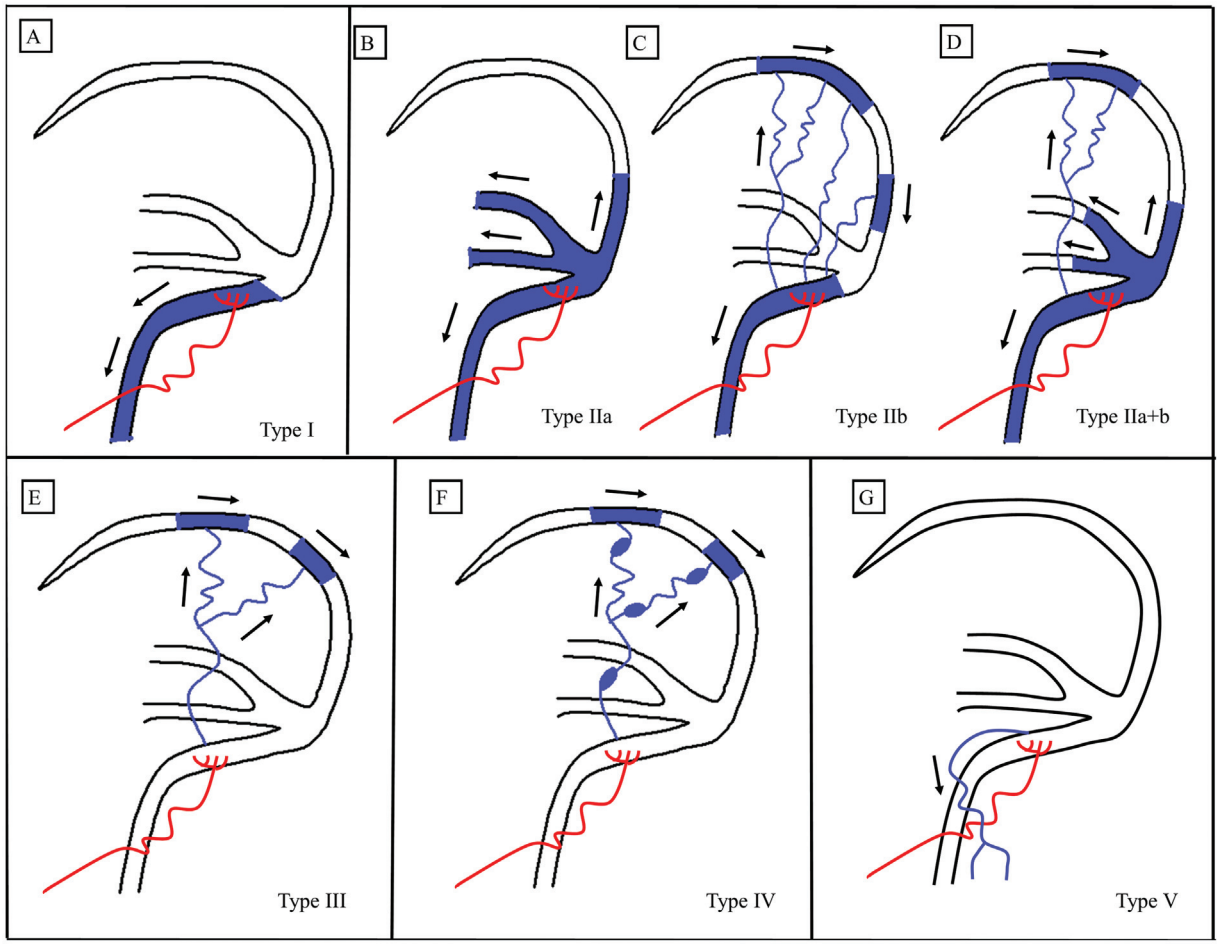

Fig. 5 (A–G) Cognard classification.

carotid arteries, and vertebral arteries. Early venous drainage is the authentication mark of DAVFs due to an anomalous shunt within the dura mater between a meningeal artery and a venous sinus and/or cortical vein. When multiple arterial feeders from different vascular territories supply a complex DAVF, it may not be easy to confirm the fistulous point. The trick is to superimpose the angiographic runs from different arterial injections; since the venous drainage is identical regardless of the arteries injected, the point after which the venous drainage is identical marks the fistulous point. Therefore, a cerebral angiogram establishes the fistula location, arterial supply, venous sinuses' patency, and venous drainage pattern. Thus, allowing for therapeutic strategy establishment.[2,3]

Treatment

Treating DAVFs generally includes conservative treatment, radiation therapy, endovascular intervention, and surgery. The endovascular treatment represents a centerpiece in DAVFs Treatment. However, a multidisciplinary approach is essential, including interventional neuroradiologists, neurosurgeons, neurologists, and radiation therapists. The decisive factors are the patient's medical background, clinical symptoms, and type of lesion (location and classification). The risk–benefit ratio should always be taken into account. High-grade lesions (Borden II and III; Cognard $\geq$ IIb) should be treated promptly to prevent complications. Conversely, low-grade fistulas (Borden I; Cognard I, IIa) are managed with conservative treatment with close follow-up to spot the symptoms' worsening or de novo occurrence. Low-grade lesions with severely debilitating symptoms (e.g., severe tinnitus or ophthalmological symptoms) are candidates for rapid endovascular repair.[3,5]

Endovascular Treatment

The embolization comprises transarterial, transvenous, or occasionally combined approaches. The treatment's primary purpose is the complete closure of the arteriovenous shunt. Nevertheless, an incomplete shunt shutting results in a novel development of arterial feeders and a persistent risk of ICH and NHNDs. The endovascular treatment's optimal approach remains subject to controversy. A reflection on the benefits and disbenefits of transarterial, transvenous, and combined approaches should be given on an individual case-by-case approach.

Transarterial Embolization

Transarterial embolization (TAE) is the first-line therapeutic approach for DAVFs.[3,11,12] TAE consists of superselective distal catheterization of arterial feeders, while the microcatheter tip should be "wedged" in the artery, and the embolic agent should penetrate the fistulous shunt and proximal aspect of the draining vein.[5] Available embolic agents include particles, coils, *n*-butyl cyanoacrylate (*n*-BCA) glue, and Onyx.[5,11,13] Particles' disadvantage is a nondurable obliteration, allowing for recurrent recanalization from collateral recruitment.[5] Coils can be used as an additional treatment to liquid embolic agents in high-flow lesions.

n-BCA is commonly used for TAE; it is injected in liquid form and solidifies once in contact with blood; thus, it closes the target blood vessel. Ethiodol, tantalum, or tungsten powder is often added to *n*-BCA, making the melange radiopaque on fluoroscopy. Before embolization, the microcatheter must be placed the closest possible to the fistulous point. Preferably, the microcatheter tip should be wedged to halt the blood flow, helping the glue progress to the fistulous site and diffusion to the fistulous collaterals. Before *n*-BCA injection, the microcatheter is prepared with a nonionic solution flush, such as 5% dextrose, to avoid glue polymerization within the microcatheter lumen. The glue is injected under direct DSA or negative road map.[14] The injection duration should be short, and an experienced operator is necessary. The thrombogenic properties of *n*-BCA can lead to progressive occlusion of residual shunt flow seen on immediate post-treatment angiography. Several studies have shown that using *n*-BCA for TAE is an effective treatment.[5,13,15] However, multiple procedures are often required for complex lesions using more than one therapeutic approach (e.g., transvenous, transarterial therapy, and/or surgical excision).[13,15] Nontarget embolization of the distal venous system may occur, leading to venous infarction, progressive venous occlusion, or worsening venous hypertension.

More and more studies report the use of Onyx to treat DAVFs.[11,15,16] This nonadhesive embolic agent comprises micronized tantalum powder for radiopacity and ethylene-vinyl alcohol copolymer dissolved in varying dimethyl sulfoxide (DMSO) doses. When the combination comes into contact with blood, DMSO quickly diffuses from it, precipitating the polymer in place without adhering to the vascular wall. The polymer initially forms precipitates in the blood vessel's periphery, occluding the center vessel later, thus, allowing a more prolonged and controlled injection with better diffusion in the vascular bed than *n*-BCA.[17] The operator can also stop the injection of Onyx if it flows toward another nontarget arterial pedicle, venous outflow vessel, or suspicious dangerous anastomosis. Afterward, the injection may be resumed as the Onyx will track toward the low-pressure zones of the residual fistula. Another technical advantage of Onyx is obtaining follow-up angiograms during embolization, assessing residual fistulous shunts, and modifications in the hemodynamics of complex lesions. Furthermore, Onyx may heal complex fistulas with multiple arterial afferents through a sole pedicle.[13] Studies have reported excellent results with this embolic agent, with a high single-session treatment rate.[11,18,19] Using Onyx also has some drawbacks, as fluoroscopy time can be lengthened, and care must be taken to avoid radiation damage.[18,19] Catheter entrapment, DMSO-induced vascular toxicity, and cranial nerve injury have also been reported.[12,18] The knowledge of the potential inducing mechanisms plays a key role in the prevention. DMSO-vascular toxicity can be impeded by slow infusion of Onyx.[18] Catheter entrapment can be prevented by keeping the reflux away from the catheter markers. Cranial nerve injury is circumvented by forbearing injection in blood vessels that supply the lower cranial nerves (petrosal branch of the middle meningeal artery,

stylomastoid branch of the posterior auricular and occipital arteries, and jugular branch of the ascending pharyngeal artery).[3]

Transvenous Embolization

Transvenous embolization (TVE) is a second-line endovascular therapeutic approach. It is indicated when TAE approaches are associated with a moderate-to-high risk of (1)/ischemic cranial neuropathy secondary to vasa nervorum occlusion, (2) parenchymal infarction resulting from inadvertent embolization of external carotid to internal carotid anastomoses, and (3) impossibility to achieve a distal embolization due to small-vessel caliber or high tortuosity.[20] It consists of retrograde catheterization of the dural sinus or cortical vein followed by embolic agent injection and/or coil deployment. This treatment closes the fistulous shunt and/or the CVD while preserving typical venous drainage. The optimal indication for TVE is when the involved sinus has minimal contribution to normal venous drainage and thus can be occluded safely. The situation may be tricky when the sinus substantially contributes to the normal venous outflow, fearing infarction or hemorrhage. Partial embolization should be avoided since the redirection of the flow toward normal venous drainage can aggravate CVD. The ability to repair the fistula in a single session and the relative ease of retrograde venous access to the fistulous region are the main advantages of TVE. DAVFs with multiple arterial afferents that present small caliber and tortuosities are best suited for TVE.[11,21] Cavernous and transverse/sigmoid sinuses DAVFs are more opportune for TVE than other sinuses.[21] Studies have reported high closure rates ranging from 71 to 87.5%.[21,22] Complications of TVE include vessel perforation, ICH, infarction, and temporary or permanent neurologic impairments secondary to venous drainage alterations.[22] Transient cranial nerve impairment causing ophthalmoplegia has been reported in cavernous sinus treatment.[22] Using liquid embolic agents reduces the danger of cranial nerve damage from coil mass-effect or direct coil injury. In 4 to 7% of cases, permanent problems have been documented.[21–23] Despite these dangers, TVE can be a safe and efficient therapy for various DAVFs and can be used with TAE to achieve full recovery.

Surgery

The surgery is a secondary intention treatment when the endovascular approach has failed or is not feasible. Various surgical techniques are applicable, including direct intraoperative embolization of meningeal arteries or veins, resection of abnormal dura, packing of the diseased sinus, disconnection of the retrograde leptomeningeal venous drainage, and skeletonization of the dural sinus with disconnection of the dural arterial supply. Particular anatomic locations are more prone to surgery, including the anterior cranial fossa and the superior sagittal sinus, as vascular access may be laborious and/or sinus exclusion is often undesired.[24,25] However, surgery is usually carried out when the endovascular approaches have wholly failed to cure the lesion. Surgery complications include infection, hydrocephalus, cerebrospinal fluid leak, infarction, cranial nerve palsy, and severe operative bleeding. Preoperative embolization may be performed; it reduces perioperative blood loss.[14] The combined approach proved to be highly effective, nearing 100%, but the risk of morbidity and mortality remains considerable at more than 10%.[3,24,26,27]

Stereotactic Radiosurgery

Stereotactic radiosurgery (SRS) for DAVFs is a developing option. Studies have involved DAVFs with low-risk or not prone to endovascular or surgical approaches. Vessel radiation causes late thrombosis leading to fistula closure after several months. The hemorrhage risk persists up to vessel thrombosis. Thus, SRS cannot be considered the primary treatment in DAVFs with CVD.[28] Better outcomes have been achieved when SRS is combined with endovascular treatment than with sole SRS, with closure rates approximating 93 and 50%, respectively. [28,29] Sole SRS may be an efficient treatment for selected patients with a small-volume, low-risk DAVF.[30] SRS is under evaluation with limited expertise worldwide and should be reserved for carefully selected patients with failing endovascular and surgical treatments.

Conclusions

Intracranial DAVFs are relatively rare lesions involving an abnormal connection, within the dural leaflets, between meningeal arteries and dural venous sinuses and/or subarachnoid veins. The natural history is influenced by the venous drainage pattern and the presentation mode. DSA is the gold standard for diagnosing and evaluating DAVFs. Cognard and Borden established angiographic classifications to distinguish between benign and aggressive lesions, but they do not consider the natural history. The treatment is indicated in the presence of clinical symptoms and/or angiographically aggressive lesions. An experienced multidisciplinary team should evaluate the patient's clinical status and decide on the appropriate treatment modality.

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Conflict of Interest
None declared.

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