Literature Review

UPDATE ON MANAGEMENT OF DURAL ARTERIOVENOUS FISTULAS

Mohammed A Azab[1], Emma R Dioso[1], Matthew C Findlay[1], Jayson Nelson[1], Cameron A Rawanduzy[1], Philip Johansen[1], Brandon Lucke-Wold[2]

Author information: 1 Department of Neurosurgery, University of Utah, Salt Lake City
2 Department of Neurosurgery, University of Florida, Gainesville

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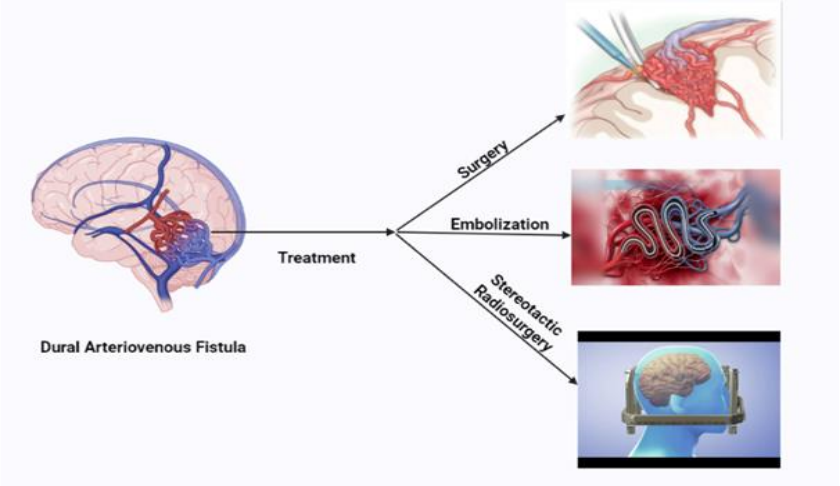
Abstract: Dural Arteriovenous Fistulas (AVF) represent about 10% of all intracranial vascular lesions. Although they seem benign in nature, the presence of retrograde venous makes them aggressive, with a high risk of complications. Patients may be clinically asymptomatic or experience symptoms ranging from mild to severe hemorrhage, depending on their location. Different treatments are available, but recently, the development of catheter intervention allows most patients to be cured with transcatheter embolization. Stereotactic radiosurgery achieves excellent rates of obliteration for low-grade lesions. In this review, we try to highlight the recent advances in the management of dural AVF.



Keywords: Dural Arteriovenous Fistulas, Embolization, Surgical Ligation, Pathophysiology

INTRODUCTION **Dural Arteriovenous Fistulae** Dural arteriovenous fistulas (dAVF) are pathologic shunts between arteries and dural veins. They are the most common spinal vascular malformation, representing approximately 10-15% of all intracranial vascular malformations[1-5]. The location of these vascular anomalies is important for risk stratification and prognostication. Spinal dAVF tend to develop in the thoracic spine, while cranial dAVF are most prevalent in the regions of the transverse, sigmoid, and cavernous sinuses [6-9]. Petroclival dAVF are a rarer but important archetype due to their aggressive nature [6]. Tentorial, galenic, and foramen magnum dAVF are among the most complex and highest-risk lesions [4, 10-12].

There is no clear sex predilection, and there is heterogeneity within the age of presentation, but patients commonly present in the 5th and 6th decades of life [13-18]. There is debate surrounding the formation mechanism; however, it is universally accepted that most

***Corresponding author:** Brandon Lucke-Wold MD, PhD, MCTS
Department of Neurosurgery, University of Florida, Gainesville
Brandon.Lucke-Wold@neurosurgery.ufl.edu

of these lesions develop idiopathically [19-21]. Herein lies the challenge of detecting these lesions as they are without a true, primary, identifiable cause. Often, they are incidental findings or present symptomatically and further along their clinical course.

Dural venous sinus thrombosis or progressive stenosis is the proposed most likely cause of dAVF. Therefore, it is well documented that hypercoagulable states may predispose individuals to dAVF formation. This is supported by the correlation between prothrombotic conditions such as factor V Leiden, protein C, and protein S deficiency and greater frequency of dAVF [19, 22]. Additional causes of hypercoagulation, including hormonal alterations, pregnancy, and menopause, have also been implicated [3]. Other risk factors for developing dAVF include a history of traumatic head injury, previous craniotomy, concomitant tumor, or infection [3, 5, 13, 19, 22].

The underlying pathophysiology of dAVF is occlusion from inflammation, thromboses, and stenosis causing congestion and venous hypertension. The subsequent elevation in venous pressure results in the formation of fistulous connections between meningeal arteries, dural sinuses, and cortical veins as a compensatory mechanism [19, 21]. Over time, abnormal flow can pivot and become retrograde, leading to cortical venous reflux (CVR); this is the hallmark sign of an aggressive dAVF and is associated with increased morbidity [3, 12, 19]. Due to their progressive nature, dAVF can present symptomatically unless found incidentally. Symptoms are non-specific and depend on the fistula location and the venous drainage pattern. They are classified as high-grade or low-grade, which will be discussed in further detail.

Briefly, low-grade fistulas are generally benign but can present with headache, pulsatile tinnitus, vertigo, bruits, and ophthalmologic abnormalities and visual changes [21, 23-25]. Symptom severity is variable and can be minor or severely affect the quality of life. Clinical symptoms can also vary by location; for instance, dAVF at the cavernous sinus frequently presents with ocular symptoms, including ophthalmoplegia, proptosis, chemosis, and orbital pain [22, 26]. Ocular manifestations may become so apparent late in the course of carotid dAVF that the clinical examination alone can suggest a possible diagnosis [27].
High-grade dAVF are defined by the presence of cortical venous drainage (CVD) and are associated with significant mortality and morbidity. They are aggressive lesions with elevated risks of intracranial hemorrhage (ICH) and non-hemorrhagic neurological deficits (NHND) [13, 20, 23, 28-30]. In high-grade dAVF, the risk of ICH is reportedly as high as 20%[13]. Furthermore, if left untreated, the risk of rebleeding after ICH in a ruptured dAVF is 35% within the first two weeks [13, 20, 31, 32]. NHND includes seizures, focal nerve deficits, mental status changes, and progressive myelopathy; symptoms will vary with location [3, 19, 24, 27]. Overall, the combined risk of a severe adverse neurological event is approximately 15% annually, with a mortality rate greater than 10% [22, 27, 28, 31]. For this reason, a full work-up and evaluation are warranted if there is clinical suspicion of a dural fistulous lesion. Prompt treatment is recommended for high-grade dAVF to achieve complete obliteration of the fistula.
Several grading scales have been developed to classify dAVF as low- or high-grade. They are based on the venous outflow and architecture and are used to guide treatment recommendations. Historically, the Borden and Cognard classifications have offered the greatest utility and are the most universally referenced [27, 33]. Borden and colleagues [34] designed a three-category classification scheme to describe dAVF: Type I drains directly into a dural venous sinus or meningeal vein, Type II drains into the venous sinus with retrograde drainage into subarachnoid veins, and type III drains directly into subarachnoid veins. In this system, Type II and Type III dAVF are considered high-grade due to retrograde flow and/or cortical venous drainage, which confer a worse natural history.

Similarly, the Cognard classification delineates dAVF by the presence or absence of retrograde flow and CVR, however, it is an eight-category scale. Types I – Type V, with three subsets of Type II make up this classification scheme [35] (Table 1), (Figure 1). Type I and Type IIa are considered low-grade and equivalent to Borden Type I dAVF with drainage directly into the dural sinus. Type IIa differs by the presence of retrograde flow. Type IIb-Type V are high-grade dAVF exhibiting direct cortical venous drainage, retrograde flow, venous ectasia, and drainage into the perimedullary veins [5, 19, 35].

The Zipfel classification, developed by Zipfel and colleagues [36], registers the presence or absence of aggressive symptoms to subcategorize high-grade dAVF further. The rationale for assessing the lesions based on clinical presentation is that mortality and morbidity are significantly higher when CVD is present with associated ICH or NHND. Zipfel Type I dAVF solely drains into dural sinuses, analogous to Borden Type I and Cognard Types I and IIa lesions. Types II and III lesions display CVR and direct cortical venous drainage, respectively.

Borden			Cognard			Zipfel		
Type	Venous Drainage	CVR	Type	Venous Drainage	CVR	Type	Venous Drainage	CVR
Type I	Dural sinus antegrade	-	Type I	Dural sinus antegrade	-	Type I	Dural sinus	-
Type 2	Dural sinus retrograde	+	Type 2a	Dural sinus retrograde	-	Type 2a	Dural sinus	+/-, asymptomatic
Type 3	Cortical vein	+	Type 2b	Dural sinus antegrade	+	Type 2s	Dural sinus	+/-, symptomatic
			Type 2a+b	Dural sinus retrograde	+	Type 3a	Cortical vein	+, asymptomatic
			Type 3	Cortical vein	+	Type 3s	Cortical vein	+, symptomatic
			Type 4	Cortical vein	+ w/ venous ectasia			
			Type 5	Perimedullary veins	+			

Lasjaunias-Geibprasert		Lawton				
Type	Venous Drainage	Type	Venous Drainage	Location	Dural Base	Venous Sinus
Ventral	Ventral venous plexuses	Type 1, Galenic	Supra/infratentorial	Midline	Anterior faclotentorial junction	Vein of Galen
Dorsal	Dural venous sinuses	Type 2, Straight Sinus	Infratentorial	Midline	Middle falcotentorial junction	Straight Sinus
Lateral	Cortical veins	Type 3, Torcular	Supratentorial	Midline	Posterior falcotentorial junction	Torcula
		Type 4, Tentorial Sinus	Infratentorial	Paramedian	Tentorium	Tentorial Sinus
		Type 5, Superior Petrosal Sinus	Supratentorial	Lateral	Petrotentorial junction	Superior Petrosal Sinus
		Type 6, Incisural	Infratentorial	Paramedian	Tentorial incisura	None

Table 1. Dural arteriovenous fistula classification**.**

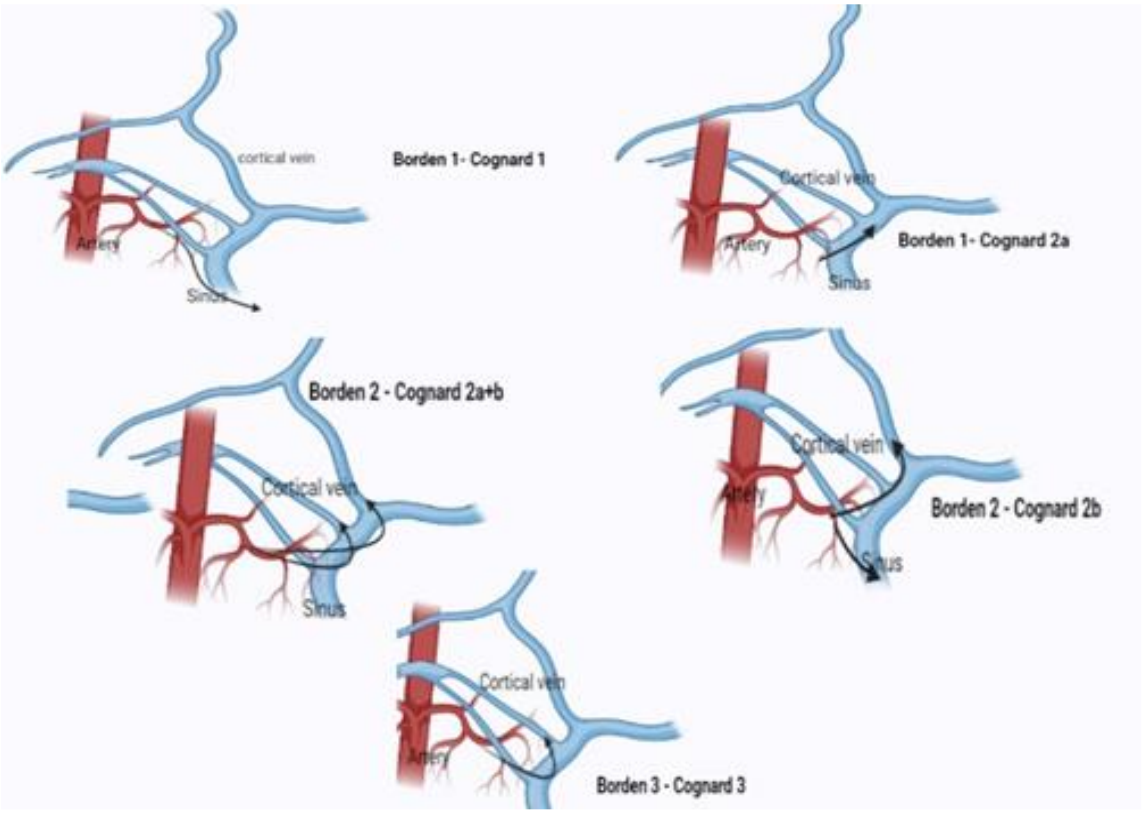


Figure 1. Dural arteriovenous fistula classification.

They are further subdivided into Type II/III A and S, corresponding to asymptomatic and symptomatic presentation [5, 19]. This modification improves accuracy for stratifying which high-grade lesions are of urgent concern versus emergent.

Lasjaunias-Geibprasert and colleagues determined that cranial and spinal dAVFs could be categorized into lesions of ventral, dorsal, and lateral origin [2, 13]. The notion that the embryological origin of the fistula could further illustrate risk is the impetus for developing their system. Accordingly, women are predisposed to ventral origin lesions that drain towards venous plexuses and are typically benign. Dorsal origin fistulas are present at the transverse and sigmoid sinus, draining into dural venous sinuses. The lateral lesions constitute those at the petrous, ethmoid, and tentorial regions and are more common in men. Lateral lesions are also the most frequently discovered SDAVF, and they have the greatest likelihood of being aggressive due to direct CVD and drainage to spinal perimedullary veins, which can cause debilitating myelopathy [8, 13, 37]. Lawton et al. created a new classification scheme for tentorial dAVFs based on fistula location, dural base, the associated venous sinus, and the direction of venous drainage. This classification system, made up of 6 different subtypes, produces an algorithmic approach to the surgical management of tentorial dAVF to achieve better treatment outcomes. Tentorial dAVFs have a high risk of hemorrhage and require microsurgical interruption of the draining veins because they often cannot be obliterated endovascularly [38].

Additional, more nuanced classifications to label specific types of dAVF have also been produced. The existence of a unique dAVF that has features of Cognard Type IV and Type V lesions, aptly named Type IV + V, has been reported [20]. Though rare, the presence of CVD and venous ectasia with pial medullary drainage is concerning and is an indicator for urgent treatment, even in an asymptomatic presentation.

The DES scheme was developed to examine the inherent heterogeneity among high-grade dAVF. It aims to precisely describe the anatomical localization of the shunt and the characteristics of leptomeningeal venous reflux [39]. Shunts can be composed of bridging veins, dural sinuses,

emissary veins, or exist as isolated sinus shunts. If present, leptomeningeal reflux is subdivided into direct, exclusive, or strained. Using the DES scheme, Baltsavias et al. [39] discovered that lesions with the most aggressive presentation had a bridging vein shunt with strained leptomeningeal reflux. These dAVFs were more amenable to microsurgery than endovascular therapy. Their results illustrate the pertinence of accurately ascribing the true risk of dAVF. It can influence when and what kind of intervention is necessary.

CVR is the hallmark sign of high-grade dAVF and is significantly associated with worse mortality and morbidity. The most consequential symptoms are NHND and ICH due to rupture [40, 41]; however, these are not the most common symptoms upon presentation. Patients with both low- and high-grade dAVF can present asymptomatically or most frequently with headache, followed by pulsatile tinnitus [23]. The low-grade dAVF are often considered benign, and treatment is conservative or reserved for symptom management to improve quality of life. There are instances of conversion from low-grade to high-grade, so patients with a known history should be followed for changes in symptoms and monitored with serial imaging [13, 19, 28, 42].

Because dAVFs do not present with a pathognomonic clinical picture, the initial evaluation is with CT and MRI. CT has limited utility and poor sensitivity and specificity for detecting dAVFs. It can detect bleeding, vasogenic edema, and the presence of cortical venous hypertension [19]. MRI is superior to CT, given its better resolution, and some MRI series, such as post-contrast T2, may show leptomeningeal and medullary vessel dilatation, venous ectasia, or venous sinus thrombosis [19, 20]. Hyperintensity on T2/FLAIR sequencing is an important indicator for aggressive dAVFs. Robust FLAIR signaling was identified in patients with CVD presenting with ICH and NHND [43]. Additionally, arterial spin labeling (ASL) reportedly has a high specificity and sensitivity to identify dAVF [44, 45]. The utility of CT and MRI is limited, and they are best used as initial imaging modalities to support clinical suspicions. The gold standard for diagnosis remains diagnostic subtraction angiography (DSA) [20, 44, 46, 47]. Though it is an invasive procedure, DSA is necessary for visualizing arterial feeders, defining the venous architecture, and planning treatment strategies and approaches [3, 13, 19, 20, 31]. The current classification scores used to guide treatment and identify the presence of CVD, venous ectasia, etc., rely on DSA.

The presence of CVD and aggressive symptoms is an absolute indication of treatment. The treatment should be aimed at complete obliteration of the fistula to protect against future hemorrhage and NHND [10, 13, 20]. The presence of asymptomatic CVD requires a more nuanced approach, as these lesions have the potential to progress. A watch-and-wait approach with serial imaging or obliteration on an elective basis may be warranted; this tends to be case-dependent [27]. Currently, the first-line therapy for dAVF is endovascular embolization [16, 48]; however, there are instances where microsurgery, or combined therapy, is more suitable [49]. Surgical obliteration is often more favorable for complex lesions along the tentorium, in the anterior cranial fossa, and involving the transverse and sigmoid sinuses [50]. Endovascular therapy has evolved with advancements in techniques and embolic agents resulting in satisfactory treatment outcomes, but the fistula's architecture limits its efficacy. A proper approach via transarterial or transvenous access is lesion-dependent [30]. The benefit of surgery, though more invasive, is the near 100% obliteration rate and angiographic cure that is routinely achieved [51].

Stereotactic radiosurgery (SRS) is an alternative option usually reserved as a follow-up treatment or for lesions not amenable to surgical or endovascular therapy [31]. SRS is minimally invasive and effective, but dAVFs obliteration rates are reportedly suboptimal [24]. Moreover, there is a latency period between the date of SRS treatment and when the therapy takes effect. It is a poor treatment modality for high-risk lesions that have caused ICH or are at risk of bleeding [28], but it may be a suitable option for appropriately selected low-grade dAVF [31].

Management strategies in cranial dAVF Cranial dural arteriovenous fistulae (dAVF) are vascular malformations consisting of a nidus of arteriovenous shunting within the dura mater. The pathogenesis of this condition involves numerous etiologies, though trauma, previous surgery, venous stenosis, or sinus thrombosis are common causes [31]. While most often benign, retrograde venous drainage and cortical venous reflux cause these structures to carry a high risk of catastrophic intracerebral hemorrhage [31, 52, 53]. Thus, managing cranial dAVF requires careful evaluation to optimize patient outcomes (Figure 2).

Treatment strategies involve the assessment of individual patient characteristics, symptoms, and the risk of adverse events [19]. Of special consideration during dAVF workup is the malformation's anatomic location and venous

drainage pattern, as these factors carry the greatest import on patient symptoms and hemorrhage risk [54]. A conservative approach with regular imaging is preferred if a dAVF is determined to be sufficiently benign (Grades I and II) [54]. Another non-invasive, low-cost treatment that has been successful in a minority of patients, especially those without retrograde venous drainage or severely declined visual acuity, is carotid artery manual compression [55-58]. When the clinical picture demonstrates a more aggressive dAVF, treatment is generally necessary via either microsurgery, transvenous embolization, or stereotactic radiosurgery [59-61].

insufficient or have previously failed, microsurgical disconnection may be used in isolation or tandem with endovascular treatments [68, 69]. While microsurgery is generally safe and highly effective, open surgery brings increased complication risk; thus, this modality is commonly reserved for dAVF, which cannot be treated otherwise or in acute dAVF hemorrhage [34, 70-73].

Lastly, stereotactic radiosurgery is an avenue whereby less aggressive dAVF might be addressed [74]. Radiosurgery gradually obliterates the lesion, which can help prevent venous hypertension or infarction [75]. However, this

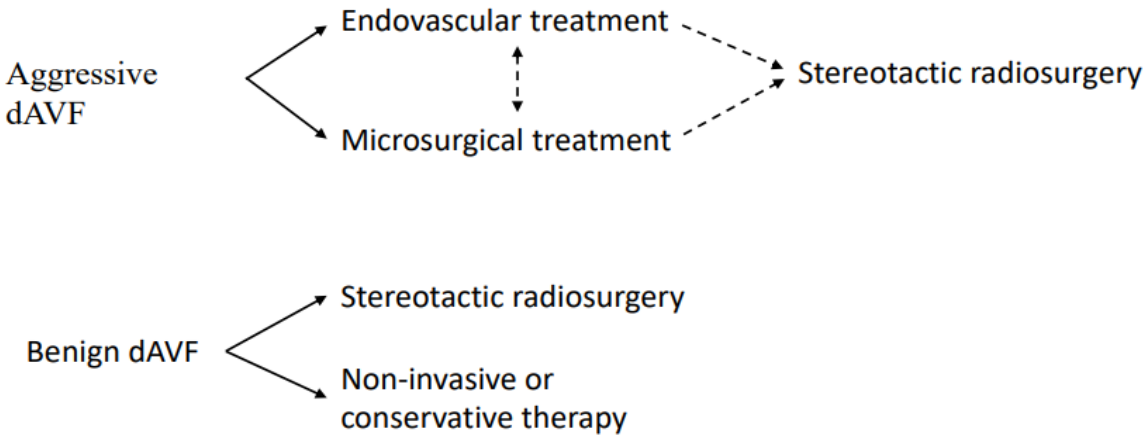


Figure 2. Flowchart outlining the treatment modalities for either benign or aggressive cranial dAVFs. Aggressive dAVF are standardly addressed endovascularly, though microsurgery may be indicated in rare instances. Endovascular and microsurgical approaches may be combined in select dAVF circumstances. If total lesion obliteration is not achieved via endovascular or microsurgical approaches, stereotactic radiosurgery may be employed. For benign lesions, conservative therapy is almost always preferred, though radiosurgery may help reduce patient symptoms.

During invasive treatment of dAVF, complete obliteration of the fistula is ideal, as any residual lesion could lead to dAVF recurrence [31]. In the modern era, endovascular embolization has become the preferred cranial dAVF treatment modality with either an arterial or venous approach [19, 22, 62-65]. Using specialized coils, embolic agents, or particulates, the dAVF may be partially or fully occluded [6, 66, 67]. Careful attention must be given before embolization to fully appreciate the anatomical relationships of the dAVF, including the neurological structures supplied by the dAVF feeder arteries, the presence of anastomoses, and dAVF contributions to nervous tissues [58]. When endovascular approaches are

delayed fistula closure can allow time for adverse events in the presence of aggressive dAVF [76]. For this reason, aggressive dAVF are recommended for endovascular or microsurgical treatments first, with radiosurgery being utilized as secondary therapy to manage residual components [75]. Due to radiosurgery's comparatively less-invasive nature, complications related to this procedure are rare, and rates of dAVF obliteration via radiosurgery are high. Thus, the radiosurgical approach has a firm place in the management of cranial dAVF.

Embolization of dAVF (Figure 3); dAVF can be treated in several ways, the most common of which is endovascular

embolization. Though endovascular embolization is the primary treatment method for most dAVF, the correct technique should be chosen according to the overall presentation, patient age, comorbidities, and prior medical history of hemorrhage [31, 77, 78]. Other important considerations for endovascular management include hemodynamics, angiographic characteristics, and location [76-78]. Alternatively, in cases without cortical venous reflux (CVR) and venous ectasia, dAVF can be managed conservatively [76, 77, 79, 80]. The choice of embolization can also be influenced by hemorrhagic risks, which may, in part, be ascertained from the Borden or Cognard type [35, 81]. The literature suggests that dAVF of Borden Type 2 and 3 (dAVF with CVR) should be treated rapidly due to a higher incidence of intracranial hemorrhage and non-hemorrhagic neurological deficits [79, 82, 83]. Jung Tae Oh et al. report hemorrhage and aggressive symptom rates according to a Borden classification based on a retrospective analysis of 95 patients [81]. In this study, Type I dAVF was associated with a 3% risk of hemorrhage, Type II was associated with a 17% risk of hemorrhage, and Type III had an associated risk of hemorrhage of 46%. Interestingly, in this same study, the authors report success rates (complete obliteration) based on intervention type. The treatment choice of "embolization alone" led to complete obliteration in 80% of cases [81]. In a different study, Strom et al. found that asymptomatic cases of dAVF with CVR resulted in lower intracranial hemorrhage rates (5.9% vs. 18.2%) and lower non-hemorrhagic neurological deficit rates (0% vs. 27.3%) than symptomatic CVR cases, [82]; however, obliteration is still recommended in these cases [78]. Although dAVFs are commonly treated with endovascular embolization techniques, they can also be treated using stereotactic radiosurgery when indicated, as is the case for cavernous dAVF without cortical venous drainage or with surgery, which is frequently employed for ethmoidal dAVF [84-87].

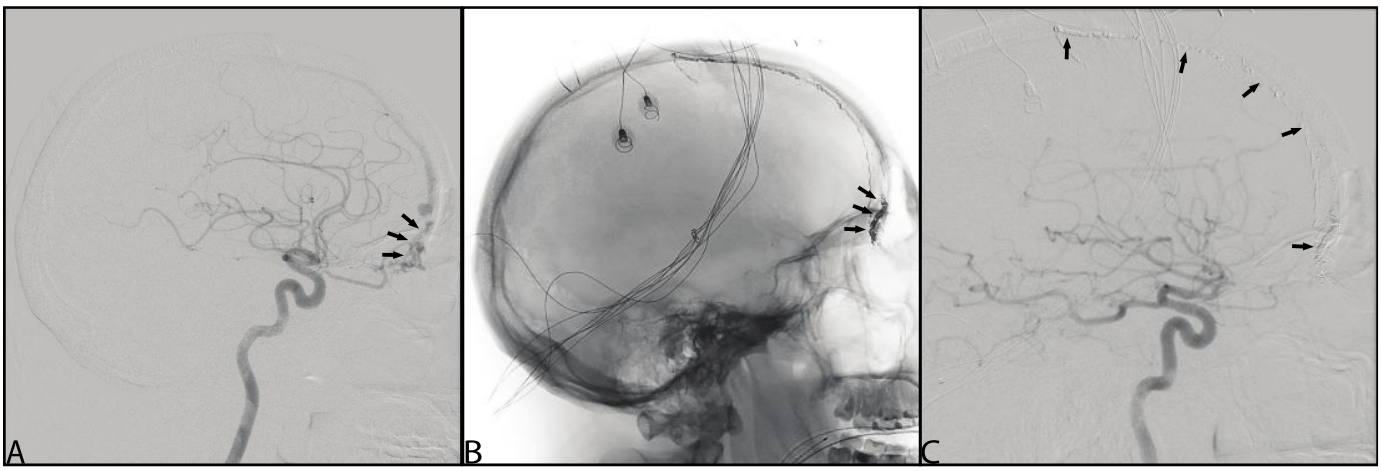


Figure 3. Digital subtraction angiography scans illustrating the embolic agents Onyx and microcoils for the embolization of a cerebral ethmoidal dAVF. A) Right anterior ethmoidal artery angiogram before embolization, arrows point to the dAVF; B) Right anterior ethmoidal artery angiogram after embolization with coils. Arrows point to the microcoils; C) Right anterior ethmoidal artery angiogram after transcranial embolization with Onyx.

Embolization for dAVF is performed transarterial, transvenous, a combination of the two, or through direct percutaneous approaches [78]. Transarterial embolization is performed by placing a microcatheter into an artery in the leg or arm and leading it to the brain through the ophthalmic, ethmoidal, or middle meningeal artery, amongst others [88-90]. For a transvenous approach, the catheter is inserted into the femoral vein and advanced to the brain using intraoperative cone beam CT in much the same way as the transarterial approach [91-93]. Direct percutaneous embolization is often employed for dAVF of the anterior cranial fossa, hypoglossal canal, or upon failure or prior contraindication of a transarterial or transvenous approach [94-98]. The customary practice involves percutaneous or transorbital needle puncture or direct surgical access to expose the vessel, followed by catheterization [78, 94, 99-102]. Several variant

procedures exist using balloon-assisted techniques. Jagadeesan et al. report a technique in which a balloon is inflated proximal to the microcatheter tip at the nidus of the dAVF to negate the need to form an onyx plug [103]. When navigating from larger parent vessels into smaller or tortuous vessels, a common problem occurs where the catheter cannot successfully navigate the bend and transition into the distal vessel. Zhao et al. report a technique where a balloon is inflated to enlarge the parent vessel's lumen, thereby facilitating the microcatheter's escort into the smaller, distal vessels, on the way to the nidus and prior to embolization [104]. The transarterial approach is most frequently indicated of all embolization techniques due to a lower incidence of intra- and postoperative complications and a higher frequency of success [78, 105].

The primary embolic agent used is "Onyx," a non-adhesive polymer made of ethylene vinyl alcohol, dimethyl sulfoxide (DMSO), and micronized tantalum powder (Figure 1) [67, 78, 106-108]. The use of Onyx first appeared in the literature in 1990 and 1991 [109, 110]. As a non-adhesive, one benefit of Onyx is that it doesn't adhere to the microcatheter tip to the extent of traditional acrylic glues. Before Onyx, this limitation complicated retrieval of the microcatheter following the obliteration of the dAVF [111, 112]. One drawback to Onyx is the need for DMSO during its administration which can cause chemical irritation to the endothelium or, in rare cases, may result in vasospasms [113-115]. One alternative is the embolic agent n-butyl-2-cyanoacrylate, commonly known as acrylic glue, although its use has decreased with the advent of Onyx [116-119]. One of the beneficial features of acrylic glue is its quick preparation and administration capabilities [112, 120, 121]. This can play an especially important role in emergent cases. Acrylic glue is cheaper than Onyx, making it more accessible for low-income countries [117]. However, one drawback of acrylic glue is its rapid polymerization following contact with ionic solutions [122].

Additionally, unlike Onyx, acrylic glue is not radiopaque, which can make intraoperative visualization more laborious. Coils are another embolic agent (Figure 1). Coils are typically made of steel or platinum and can be created according to different specifications. Coils are frequently combined with Onyx to achieve complete obliteration [123-125]. Risks include coil migration and subsequent obstruction of adjacent lumina [126]. The advantages of coils include quick preparation, administration, and greater visualization. Some coils come with coatings like hydrogel, which increases the diameter of coils upon contact with blood [127]. Many coils are also surrounded by wool or other thrombogenic agents. Of all the embolic agents available, Onyx has quickly expanded to become the primary choice for dAVFs, with reported complete obliteration rates between 55 and 60 percent [128, 129]. The odds of avoiding surgery following embolization with Onyx are also significantly higher at 81.80 percent compared to 22.22 percent with acrylic glue [130].

Surgical management of dAVF The anatomical location of the fistula and its effect on the venous drainage flow dynamics produces symptoms that require treatment. The presenting clinical features are variable and depend on the location of the fistula and include seizures, myelopathy, cranial nerve palsies, and sensory or motor deficits. In about 20%–33% of dAVFs, the presentation is intracranial hemorrhage [131]. Management lines include both endovascular and surgical approaches. While the endovascular approach is widely used now, surgery is still a safe available option [70]. Over 80 years, the technique of surgery for AVM has progressed steadily since the first resection. Introducing cerebral angiography in y in 1927 by Antonio Caetano de Abreu Freire Egas Moniz has accelerated the surgical progress [132]. Surgical treatment is always considered for all aggressive dural AVF [133]. In most cases of cranial dural AVF involving the transverse sinus, the AVF is drained by the sinus itself or the dural veins. These types of AVF tend to develop new collateral venous routes with the sinus or dural veins. Therefore, surgical excision is considered the definitive therapy in these cases [134]. Other indications for surgery include abnormal tortuous arterial feeders not suitable for embolization, feeders involved in normal brain structures feeding or difficult to localize or a fistula that persists after endovascular or radiosurgery [135].

Surgery is always considered for high-grade dural AVF with retrograde cortical lesions. These lesions have a high rate of complications than the lower grade [136]. Surgical-wise, dural AVFs are classified into two types direct and indirect. The direct types usually have a specific arterial feeder to a draining vein, while the indirect types have numerous small arterial feeders that pass through the dura [135]. Most of the surgical techniques for dural AVM are like those involved in the treatment of dural AVF except for the nidus which is a feature of dural AVM only [68]. In 1974, Hugosson and Bergstrom described sinus skeletonization for the indirect type [137]. Lucas et al also described the same technique in two cases of dural AVF involving the tentorial apex [138]. While sinus skeletonization preserves

the patency of the sinus, it still has some burdens. Brain retraction, air embolism, and bleeding are possible identified common complications [139].

Spinal Dural AVF (Figure 4); Spinal dAVFs are direct communication between a radiculomeningial artery and a radicular vein [140-143]. They represent only 1-2% of vascular neurologic pathologies, though are the most common spinal vascular malformation [142, 144-147]. Spinal dAVFs occur primarily in the thoraco-lumbar region, and rarely in the cervical region [148-150]. Cases are primarily idiopathic, though may develop following trauma or surgery.[150, 151] Classically, spinal dAVFs present with progressive spastic motor weakness, sensory deficits, sphincter disturbances, and back pain [148, 150-153]. Other patients may present with hemorrhage or an acute exacerbation referred to as "Foix-Alajouanine syndrome."[152]. Myelopathy, intramedullary edema, and chronic hypoxia develop due to the venous hypertension from arterial blood shunting into the valveless veins of the spinal cord [144, 153-155]. Subsequently, a decrease in arterial supply, arterial steal, and ischemia are observed [153-155]. This myelopathy can be irreversible if left untreated [153-155].

Initially, both T1-weighted and T2-weighted MRIs often provide the first evidence suggestive of a spinal dAVF via a serpentine pattern of low signal in the subarachnoid space [150]. Spinal angiography is the diagnostic gold standard, as these studies precisely distinguish the type of fistula, position of fistula, and its angioarchitecture [156, 157].
From a treatment standpoint, the goal in spinal dAVFs is eliminating the venous congestion and giving the spinal cord an environment to recover [154, 158]. Spinal dAVFs can be managed successfully in the majority of patients if the diagnosis is made before irreversible neurological deficits develop [159]. Available options include surgical disconnection, endovascular embolization, or a combination of the two. Surgery is largely pursued due to the simplicity of the procedure, as well as the low morbidity and recurrence rates observed [150]. Recently, advances in endovascular techniques have drastically increased the number of patients pursuing embolization options[147, 148, 152, 154, 160], though literature seems to continue to favor primary surgical treatment from failure rate and late recurrence standpoints [161, 162]. Microscopic direct surgery is considered in cases when endovascular access is not safely possible [163].

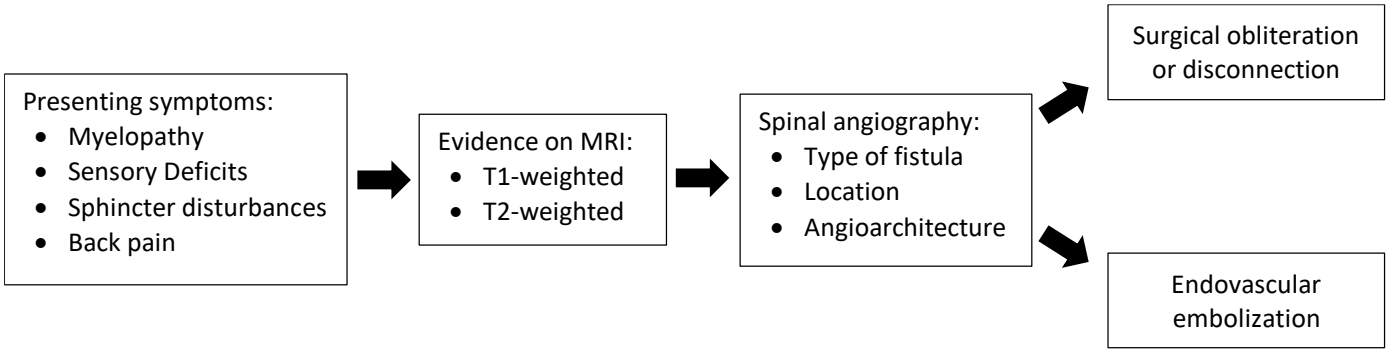


Figure 4. Patient Course of Spinal dAVF. Patient presents with classic symptoms and undergoes an MRI that suggests spinal dAVF. MRI findings prompt spinal angiography, the gold standard diagnostic tool for spinal dAVF. Based on findings from the angiography, as well as evidence based and shared decision making between the provider and patient.

Previous meta-analysis by Steinmetz and colleagues found improvements of symptoms to favor surgical management [160]. In 2021, Fiaschi and colleagues found improvement of motor symptoms in >80% of patients following surgical obliteration and improvement of sphincter dysfunction in >30%. Additionally, their cohort demonstrated higher patient quality-of-life perceptions following surgical management when compared to embolization [164].

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