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Review Article

Update of embolization of intracranial dural arteriovenous fistula

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Abstract

Intracranial dural arteriovenous fistulas (DAVFs) are abnormal arteriovenous communications within the dura locating near a major venous sinus and are supplied by pachymeningeal arteries. DAVFs represent 10–15% of all intracranial arteriovenous malformations. The natural history and clinical manifestations are determined by location of the DAVFs and their angioarchitecture. Aggressive DAVF is usually associated with leptomeningeal venous drains or reflux. It may present with hemorrhagic or nonhemorrhagic stroke. The goal of embolization of DAVFs is total fistula occlusion without interfering with the normal dura–venous drains. Embolization can be performed by transarterial and/or transvenous routes or direct puncture of affected dural sinus. Selection of embolic materials depends on access route and angioarchitecture of the fistula. With the involution of endovascular devices, embolic materials, and high-quality angiography, endovascular embolization of DAVFs has been proved a safe and effective method of treating these complex cerebrovascular lesions.

Keywords: arteriovenous fistula; dura; embolization

1. Introduction

Dural arteriovenous fistulas (DAVFs) can occur in any intracranial dural regions, most commonly in the cavernous sinus or transverse-sigmoid sinus.[1–3] DAVFs are usually fed by dural branches of the external carotid artery. The etiology of DAVFs is not fully understood; some are congenital and others are acquired, but most are thought to be acquired. Different etiologies have been implicated including thrombosis, trauma, or surgery or in the *postpartum* period.[4–6] Patients may be asymptomatic or may experience symptoms ranging from mild symptoms to fatal hemorrhage.[7–11] DAVFs have been classified to benign type with dural sinus drain or aggressive type with leptomeningeal venous drain and/or reflux which may associate with hemorrhage or nonhemorrhagic neurologic deficits.[12–14] Management of DAVFs includes open surgery, radiosurgery, embolization, or combined treatment. In this article, we will discuss the method of embolization, with emphasis on the access routes, embolic materials, and individual DAVF embolization.

2. Symptoms, natural history, and classification of intracranial DAVFs

The clinical presentations of DAVF are variable, depending on the location of the shunt and angioarchitecture of the shunting flow. These symptoms include exophthalmos, bruit, cranial nerve deficits, tinnitus, hemorrhagic or nonhemorrhagic neurological deficits, increased intracranial pressure, papilledema, and cerebrospinal fluid reabsorption abnormalities resulting in ventricular enlargement or congestive heart failure. Several classification systems have been developed to grade the risks and natural course of DAVFs.

Conflicts of interest: The authors declare that there are no conflicts of interest related to the subject matter or materials discussed in this article.

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Those devised by Cognard et al[15] and Borden et al[16] are the most widely used. All these classifications emphasize the fistula venous drains. DAVFs with antegrade dural sinus drains and only association with sinus reflux usually present with benign symptoms. By contrast, DAVFs with retrograde leptomeningeal cortical venous drainage channel show a significantly high rate of aggressive symptoms such as hemorrhagic and nonhemorrhagic neurologic clinical manifestation. Although DAVF location is not directly correlated with aggressive behavior, the propensity for dangerous drainage patterns found at initial diagnosis does vary with location.

3. Goal of embolization of DAVFs

The treatment strategy of DAVFs should be tailored individually to patients considering the angioarchitecture, natural history of the DAVFs, and the severity of symptoms. The definitive treatment of DAVFs requires the occlusion of the arteriovenous shunt without interfering with the normal venous drains. Treatment can be performed by conventional open neurosurgery, radiosurgery, or endovascular embolization, or a combination of these modalities. The first reported series of embolization cases was by Halbach et al in the 1980s.[17] With the development of newer techniques and embolic materials in the field of interventional neuroradiology, endovascular therapy has now been developing as the primary treatment strategy to manage DAVFs, particularly in high-risk DAVFs, reserving surgery only if endovascular techniques fail or are unfeasible. The goal of embolization is to achieve an angiographic cure by obliteration of all feeders and proximal draining veins with preservation of the patency of the affected sinus. In those difficult and complex DAVFs with a difficulty of achieving total occlusion of the fistula, the goal of embolization is to achieve partial treatment with reversal of the aggressive type of DAVF to benign type, to facilitate subsequent radiosurgery or neurosurgery.[18–20] Embolization should usually fulfill the following requirements. If a sinus is occluded or severely stenosed, treatment should be directed to reestablish sinus patency[21,22]; if that fails, sinus occlusion with angiographic cure of DAVF is considered. If there is a functional venous pathway that was not previously occluded, it should not be sacrificed. Treatment should not induce redirection of flow to other cortical veins.

4. Embolization access routes

There are three embolization routes to manage DAVFs: transarterial embolization (TAE) of arterial feeders (Figs. 1 and 2), transvenous embolization (TVE; Figs. 3 and 4) or direct puncture of the affected sinus. Selective TAE requires

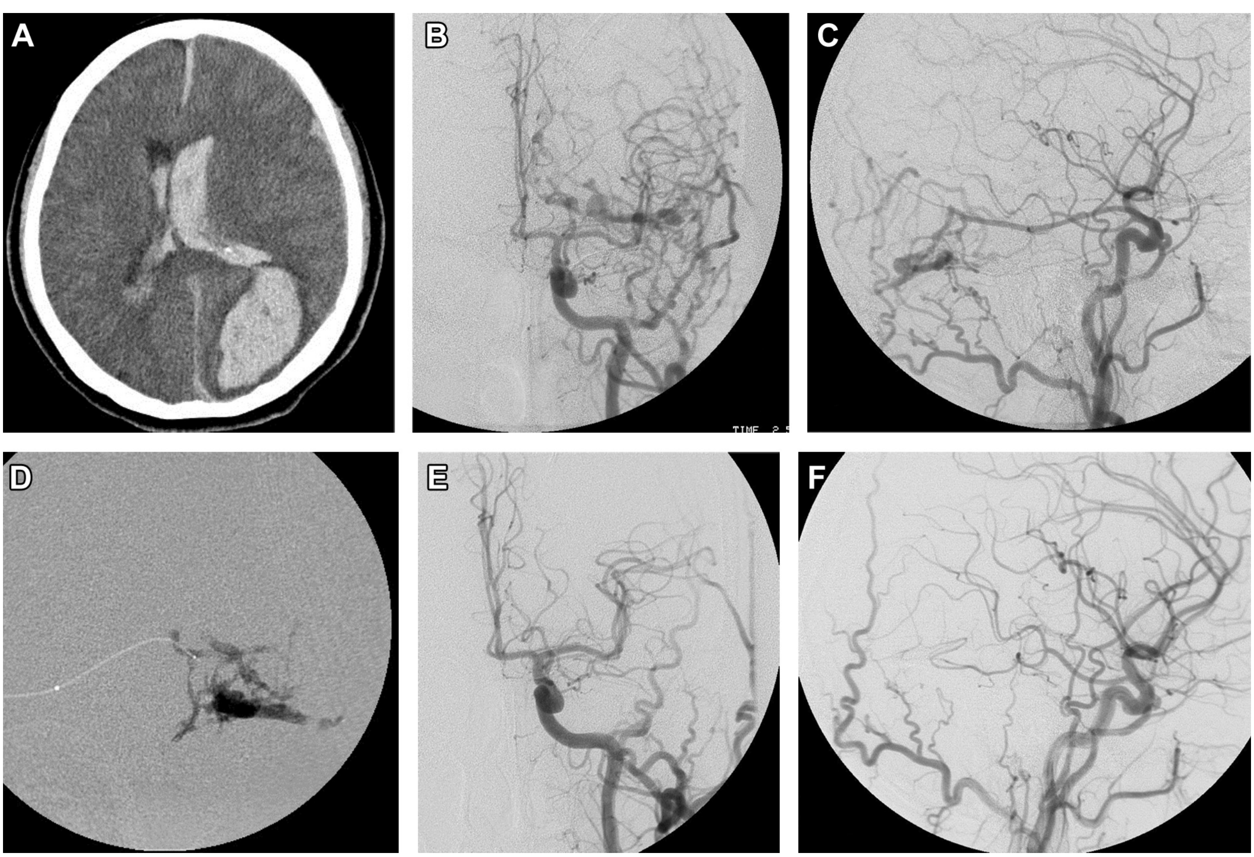

Fig. 1. A 61-year-old woman presented with consciousness change. (A) Brain computed tomography demonstrated intracerebral hemorrhage with extension to ventricular systems; subdural hematoma was found as well; (B, C) left carotid angiograms depicted Cognard type IV dural arteriovenous fistulas at the left posterior tentorium; (D) the patient underwent transarterial (middle meningeal artery) n-butyl-2-cyanoacrylate embolization; and (E) postembolization angiograms demonstrated total fistula occlusion.

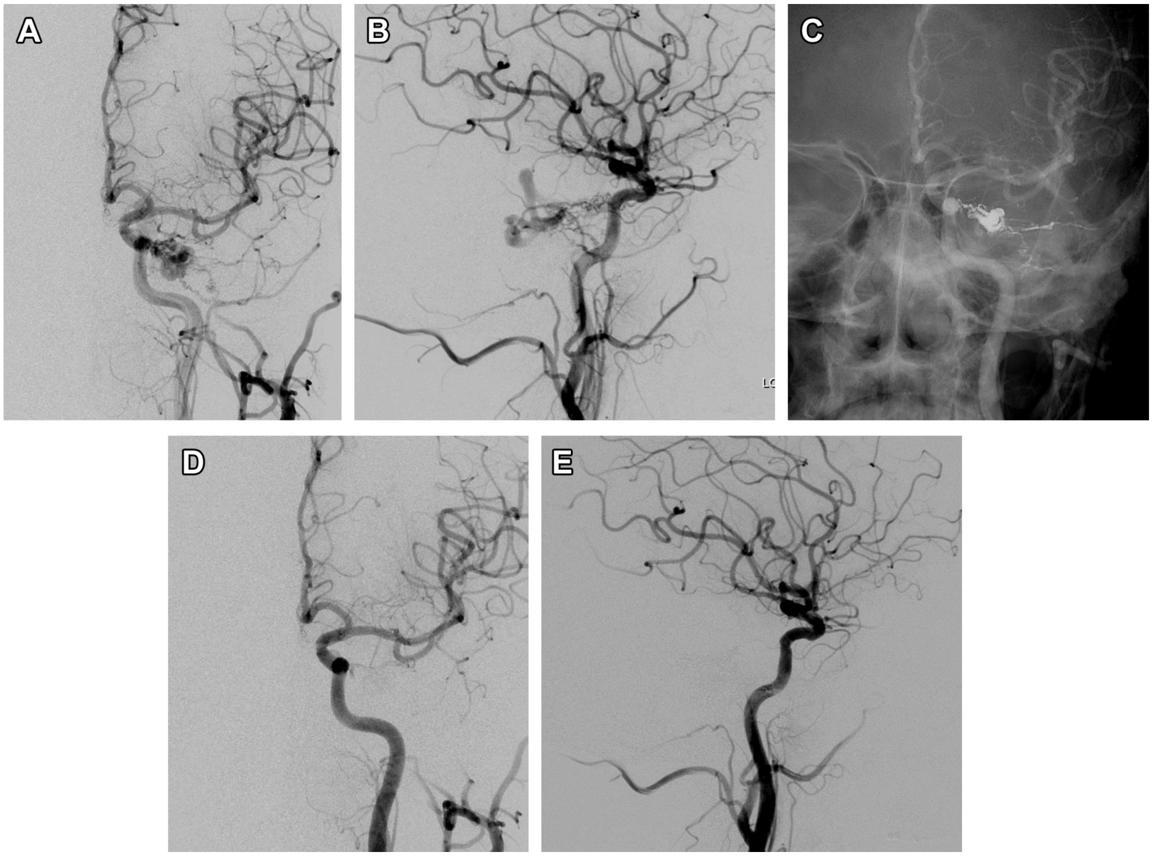

Fig. 2. A 61-year-old man presented with limb weakness and diplopia. (A, B) Left carotid angiograms demonstrated Cognard type IV left tentorium dural arteriovenous fistulas; and (C−E) the patient underwent transarterial Onyx embolization. Postembolization carotid angiogram showed total fistula occlusion.

that the microcatheter be positioned into the ideal distal aspect of the feeding artery, with delivery of the embolic material through a feeding artery to the shunt and then into the most proximal venous outlet. Although some studies have shown good outcome with low complication rates for occluding DAVFs via a transarterial route using liquid embolic agents,[23−26] this route may not always be feasible, because of multiple, small, and tortuous feeders. Furthermore, some risks may occur by inadvertent embolization of carotid or vertebral territory with ischemic stroke via dangerous anastomosis of the external−internal carotid artery or vertebral artery anastomoses or embolization of feeders of cranial nerves. In addition, proximal occlusion of feeding artery allows the persistent arterial flow and recruitment of collateral blood supply; therefore recurrence is likely. Furthermore, embolic material migration to the venous site may result in venous occlusion exacerbating venous hypertension. In the past, to achieve an angiographic cure with complete fistula occlusion, TVE with coil packing of the affected dural sinus was selected. This technique is logical as long as the diseased segment of the sinus is occluded completely or does not participate in normal venous return. However, TVE is not always feasible in cases with difficult fistula anatomy, such as in isolated or high-grade stenotic dural sinus. Embolization of DAVFs by direct puncture of affected sinus via transorbital (e.g., cavernous sinus)[27−29] or via craniotomy (e.g., lateral, superior sagittal sinus, or sinus confluence)[30−32] has been reported, with promising results. The advantages of transcraniectomy access are ease of fistula access after direct surgical exposure, ease of direct sinus coil packing in a small craniectomy, and ability to performed embolization without any intradural surgical manipulation. The disadvantage of this access route is the potential complication of craniectomy, including blood loss or infection. Generally, the affected sinus should be in a superficial location close to the cranium, isolated, and near another sinus. Direct puncture to access the affected sinus is an alternative. Because of the procedure's inherent invasiveness, it has only been considered when endovascular routes fail based on angioarchitecture analysis. In some complex DAVFs, a combination of TAE and TVE is considered.

5. Selection of embolic materials

Different embolic materials have been used. These include n-butyl-2-cyanoacrylate (NBCA; Codman, Raynham, MA, USA), particles (polyvinyl alcohol; PVA), Onyx (eV3; Neurovascular Inc., Irvine, CA, USA) or detachable coils. Coils usually are selected for transvenous occlusion of an affected nonfunctional dural sinus. TAE with PVA usually does not produce a durable cure, therefore, it can only be used as a supplement to eliminate residual minimal flow after embolization, to induce thrombosis of residual fistula, or for palliative management of benign DAVFs. NBCA is a cheap, fast embolic

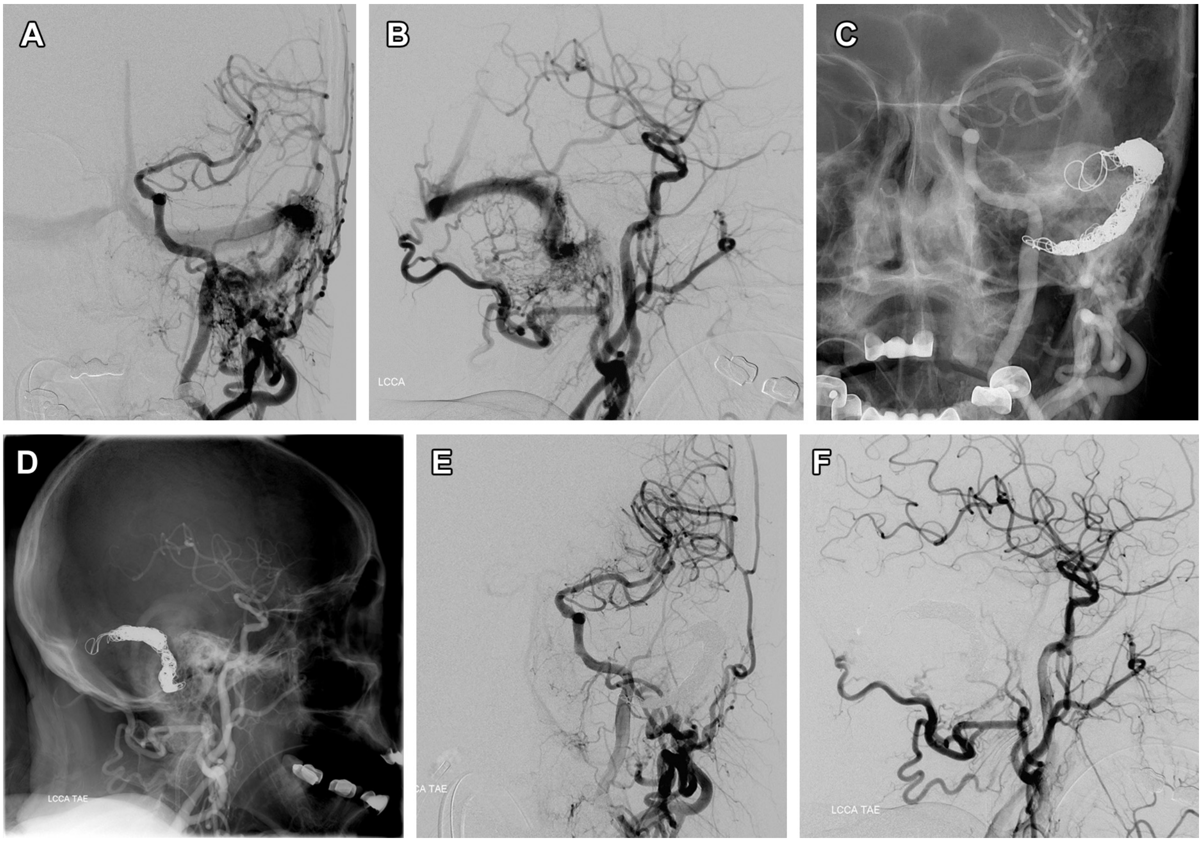

Fig. 3. An 82-year-old man had Cognard type IIb right transverse-sigmoid sinus dural arteriovenous fistula and presented with seizures. (A, B) Left common carotid angiograms revealed aggressive dural arteriovenous fistulas with cortical reflux; occlusion of left sigmoid sinus–jugular vein was found as well; and (C–E) the patient underwent transvenous coil packing of the affected sinus. Postembolization angiograms demonstrated total fistula occlusion.

material. Prior to the advent of Onyx, NBCA was widely used to occlude DAVFs transarterially.[33–36] Because NBCA is a liquid adhesive, this procedure requires a highly experienced operator with the ability to determine the appropriate concentration of NBCA (about 20–25%). The major disadvantages of NBCA are short injection time (within minutes) and limited amount of NBCA mixture injection leading to insufficient embolization and lower cure rate (30–50%).[34–36] The angiographic occlusion rate is statistically higher with Onyx compared with NBCA.[33] Another concern is that it is difficult to control NBCA flow after injection, resulting in proximal arterial occlusion in highly concentrated NBCA or migration to a draining vein, resulting in a risk of venous infarction or hemorrhage in low-concentrated NBCA. Onyx, a nonadhesive liquid embolic system, consists of ethylene vinyl alcohol as the embolic material, dimethyl sulfoxide as the solvent to deliver ethylene vinyl alcohol, and tantalum powder for radiographic visualization. Onyx was initially introduced in 1999 to treat brain arteriovenous malformation. In 2006, the first case of a DAVF successfully occluded by Onyx was reported.[24] Subsequently, other case reports and case series were published.[25,26,37,38] Onyx provides advantages by the ability to perform a slow, controlled injection. Injection of larger amounts of Onyx can result in filling of the fistulous network and allow for reflux into other arterial feeders in a single feeder embolization. The development of Onyx was a milestone in the endovascular treatment of DAVF, with promising results with a relatively high morphologic cure rate of 62.5–80%.[25,26,37,38] However, there are some disadvantages of Onyx. The long-term patency of Onyx embolization is unknown. Onyx itself may leave microchannels within the cast that allow a small fistula of flow, resulting in recurrence after endovascular embolization. In addition, Onyx has been thought to cause significant inflammation within the vasculature, particularly with treatment of cavernous carotid fistulas in which the surrounding cranial nerves are often irritated, resulting in various cranial nerve palsies.

6. Embolization of individual DAVFs

6.1. Transverse–sigmoid sinus DAVFs

The transverse–sigmoid sinus (TSS) represents the most common location for DAVFs. Spontaneous regression of TSS DAVFs is uncommon and usually occurs following hemorrhagic events.[39] The arterial supply originates from the dural branches of the occipital artery, middle meningeal artery, ascending pharyngeal artery, and/or dural branches of the vertebral artery or tentorial branches of the meningohypophyseal trunk. Although their most common symptoms are benign (e.g., pulsatile tinnitus and headache), TSS DAVFs may present with hemorrhagic and nonhemorrhagic aggressive

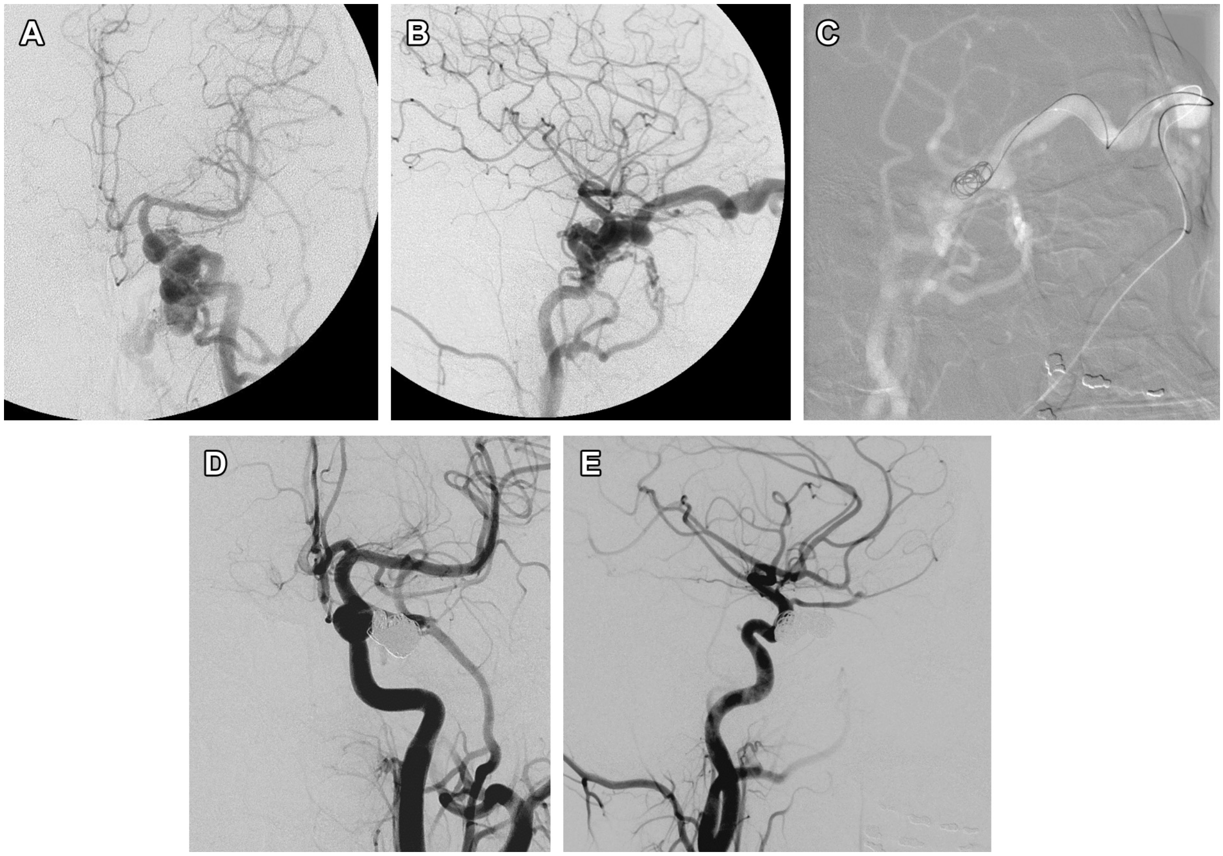

Fig. 4. A 53-year-old man had left cavernous sinus dural arteriovenous fistula and presented with chemosis and proptosis. (A, B) Pre-embolization carotid angiograms showed cavernous sinus dural arteriovenous fistula fed by dural branches of the left carotid arteries with exclusive drainage to the ophthalmic vein; (C) the patient underwent transvenous coil packing of left cavernous sinus via facial vein; and (D, E) postembolization carotid angiograms demonstrated total fistula occlusion.

neurologic symptoms. All TSS DAVFs are considered to require treatment because of the low rate of spontaneous regression without symptomatic events and the relatively high rate of aggressive symptoms. The selection of endovascular management of TSS DAVFs depends on the patient's symptoms and angioarchitecture analysis. If patients present with benign DAVFs with functional antegrade TSS drainage, TAE with preservation of the patency of the TSS and symptomatic treatment is the first choice. In those aggressive TSS DAVFs with leptomeningeal venous drainage/ reflux, the goal of treatment is total occlusion of the DAVFs by TAE or TVE. Transvenous coil packing of the affected TSS is usually safe and highly effective if the affected sinus does not participate in normal TSS venous return. Angiographic cure can be achieved in 80–100% of patients.[39–41] This procedure requires sacrifice of sinus flow, therefore, occlusion of the nearby normal antegrade cortical venous drainage should be avoided. The rate of permanent complications is approximately 4%.[39–41] Transarterial access by liquid embolic materials is selected for those patients with functionally affected TSS or failure to access by transvenous route. The reported cure rate for using TAE with NBCA is approximately 60%.[1] Recently, TAE with ONYN has replaced the NBCA as the first choice of liquid embolic material because of better morphologic cure. The cure rate may be up to 100% in some selected patients.[42] Another option for managing TSS DAVFs with high-grade stenosis or occlusion is endovascular stent placement. Recanalization, angioplasty, and stenting of stenosis/occlusive TSS can restore antegrade sinus flow and close shunts within the sinus wall, however, only a few successful cases have been reported.[21,22] Further investigation of the effectiveness of stent placement in the treatment of DAVFs is necessary.

6.2. Cavernous sinus DAVFs

Cavernous sinus (CS) DAVF represents the second most common location for DAVFs; it usually occurs in middle-aged women and is fed by multiple small dural branches of the internal and/or external carotid arteries. Because of their slow-flow shunts, some CS DAVFs have a tendency to undergo spontaneous thrombosis, observed in 10–50% of cases.[43] Regarding management of CS DAVFs, if patients present with mild clinical symptoms with no angiographic risk of hemorrhagic or nonhemorrhagic neurologic deficits, conservative treatment, or a less invasive procedure such as observation, manual compression of the carotid artery,[44] or radiosurgery[45] can be applied. If patients have pial reflux and/ or are at risk of clinical deterioration such as gradual loss of visual acuity or cranial nerve palsy, embolization is required. The most common symptoms of CS DAVFs AVF are ocular symptoms (e.g., chemosis, exophthalmos) caused by anterior venous drainage; posterior drainage to inferior or superior petrous sinus may lead to bruit or cranial nerve palsy or reflux to cerebellar veins; superior drainage to superficial middle cerebral vein may occasionally have a risk of impairment of local cerebral venous drain. Aggressive neurologic symptoms such as intracranial hemorrhage are extremely rare; inferior drains to the pterygoid plexus may lead to bruit or nasal congestion. Transvenous coil packing of affected CS has been proved to be an effective and safe method to manage the CS DAVF. The cure rate varied from 80% to 100%.[46] However, dense packing of the CS with coils should be avoided because of the risk of cranial nerve deficits due to coil compression. There are many transvenous routes to reach the CS, and these include the inferior petrous sinus, basilar plexus, facial vein, angular vein, and superior ophthalmic vein, or through the pterygoid plexus. Recently, TAE with Onyx of CSDAVFs has been performed, with better morphologic outcomes than those treated by NBCA. However, embolization via the middle meningeal artery with occlusion of blood flow to facial or trigeminal nerve has been reported. Another concern is the migration of Onyx cast to the pial artery via dangerous anastomoses leading to ischemic stroke and/or blindness. On rare occasions with aggressive CS DAVF, in which there is failure to access via the endovascular route, a transorbital approach is an alternative. Successful treatment of geometrically difficult CS DAVF by transorbital direct puncture of the ophthalmic vein or anterior CS under fluoroscopic guidance has been reported.[28–30] The potential risks of these procedures include orbital hemorrhage, injury to the optic nerve or other cranial nerves in the CS, globe puncture, and infection. Therefore, the operator should be familiar with orbital anatomy and equipped with high-quality digital subtraction angiography.

6.3. Tentorial DAVFs

Tentorial DAVFs represent 4–8% of all intracranial fistulas.[1,47,48] Because tentorial DAVFs drain only via the leptomeningeal vein, they carry a high risk of aggressive neurologic symptoms. The reported aggressive clinical behavior in 79–92% of cases,[12,16,48] the occurrence of intracranial hemorrhage is about 70%[14]; in some cases, this hemorrhage consists of fatal bleeding in the posterior fossa. The arterial supply mainly comes from the tentorial artery, such as dural branches of the meningohypophyseal trunk and/or middle meningeal artery. Venous drainage is usually through the superior petrosal sinus or through the pontine, perimesencephalic, and basal veins into the Galenic system. Because of the aggressive behavior of tentorial DAVFs, the goal of embolization is total fistula occlusion. In the past, TAE was effective by using NBCA occlusion of the proximal draining vein, whereas TVE with coils was preferred when the lesion was anatomically suited.[48] Now, TAE with deposition of liquid embolic material such as NBCA or Onyx to occlude the fistula is commonly selected. The angiographic cure rate by TAE with Onyx is better than that for treatments with NBCA. Huang et al[49] reported 12 patients presenting with intracranial hemorrhage who were treated by Onyx embolization. Complete cure was achieved in 11 patients. All of the patients recovered well, except one who died of severe infection caused by ventricular drainage.

6.4. Superior sagittal sinus DAVFs

Superior sagittal sinus (SSS) DAVFs represent approximately 3–8% of all intracranial DAVFs.[1,15] They are frequently associated with restrictive drainage of the SSS and retrograde cortical venous drainage, therefore, aggressive neurologic symptoms are seen in 50% of cases. The reported bleeding rate of SSS DAVFs is about 15–40%.[1,15,50] Venous congestion and ischemia of the bilateral frontal lobes due to SSS DAVFs can cause dementia. These fistulas are usually supplied by dural branches of bilateral middle meningeal arteries and/or the anterior falcine artery from the ophthalmic arteries. Therefore, SSS DAVFs commonly possess multiplicity in arterial feeders and critical venous drainage. Endovascular management of SSS DAVFs is similar to that for treating TSS DAVFs. However, SSS DAVFs are more frequently associated with hemorrhagic and nonhemorrhagic stroke and therefore often require aggressive treatment. TVE with coil packing of affected SSS is the first choice if the SSS is not functional for normal cortical vein drainage. Nevertheless, SSS DAVFs are usually associated with SSS occlusion or stenosis; the TVE is often difficult. In cases with difficult access via the transvenous route, or if the SSS has participated in normal venous drainage, TAE with NBCA or Onyx is indicated; however, the feeders of meningeal arteries are usually distally and tortuously located, leading to difficulty in navigating the microcatheter to the fistula site. TAE with occlusion of feeding arteries is commonly associated with insufficient embolization, with fistula recurrence by recruitment of the feeders. In the past, TAE using NBCA was selected to achieve angiographic cure or as an adjunct therapy for subsequent radiosurgery or open surgery. Recently, NBCA was replaced by Onyx as a transarterial embolizer because of better angiographic and clinical outcomes. Chai et al[51] reported four cases in which successful angiographic cure was achieved with Onyx via TAE.

6.5. Anterior cranial fossa DAVFs

Anterior cranial fossa DAVFs constitute rare lesions, representing < 10% of all DAVFs.[52] DAVFs located on the anterior fossa drain through the retrograde leptomeningeal–cortical venous drainage system only, associating with a high risk of hemorrhagic or nonhemorrhagic aggressive symptoms. The occurrence of intradural hemorrhage is about 84%[15]; therefore, a complete angiographic cure is necessary. The arterial supply is mainly from the posterior and anterior ethmoidal branches of the ophthalmic arteries. They may also recruit supply from the ethmoidal branches of the external carotid artery. Because there is no dural sinus in this area, the main venous drainage is into the frontal cortical veins and secondarily into the SSS. In the past, this type of fistula was considered as difficult for endovascular treatment, as the supplying vessels are small and tortuous, precluding catheterization with current technology. By contrast, surgical approaches are relatively easy and safe. When TAE with selective catheterization through the ophthalmic artery is performed, it is paramount to verify that the microcatheter tip is located beyond the origin of the central retina artery to avoid complications such as stroke and blindness. Few anterior fossa DAVFs can be safely treated with TVE, because transvenous routes are also tortuous and often associated with venous aneurysms. However, this approach is more complicated and rarely used when treating this type of DAVF. Some authors strongly oppose embolization as it poses numerous technical difficulties and represents more costly treatment. Since the advertent of Onyx, TAE with angiographic cure has become feasible.[52,53] Li et al[52] successfully managed 10 out of 11 cases with Onyx embolization. No signs of rebleeding or symptom progression were observed at a mean follow-up of 18.5 months.

6.6. Other DAVFs

DAVFs can also occur at other intracranial locations, including the marginal sinus, inferior and superior petrosal sinuses, major sinus wall, and hypoglossal canal. TVE by coil packing dural sinus is preferred if the affected sinus is nonfunctional and does not participate the normal venous drains. TAE by using Onyx to manage these DAVFs is indicated when the feeder is accessible.

In conclusion, endovascular management of DAVFs includes a number of options with varying risk and effectiveness for individual lesions. TAE, TVE, or direct puncture can be used depending on angioarchitecture and individual clinical scenarios. Endovascular management of DAVFs is a proven safe and effective method of treating these complex cerebrovascular lesions.

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References

1. Santillan A, Nanaszeko M, Burkhandt JK, Patsalides A, Gobin YP, Riina HA. Endovascular management of intracranial dural arteriovenous fistula: a review. *Clin Neuro Neurosurg* 2013;**115**:241–51.
2. Kirsch M, Liebig T, Kühne D, Henkes H. Endovascular management of dural arteriovenous fistulas of the transverse and sigmoid sinus in 150 patients. *Neuroradiology* 2009;**51**:477–83.
3. Meyers PM, Halbach VV, Dowd CF, Lempert TE, Malek AM, Phatouros CC, et al. Dural carotid cavernous fistula: definitive endovascular management long-term follow-up. *Am J Ophthalmol* 2002;**134**:85–92.
4. Watanabe A, Takahara Y, Ibuchi Y, Mizukami K. Two cases of dural arteriovenous malformation occurring after intracranial surgery. *Neuroradiology* 1984;**26**:375–80.
5. Bitoh S, Hasegawa H, Fujiwara M, Nakata M. Traumatic arteriovenous fistula between the middle meningeal artery and cortical vein. *Surg Neurol* 1980;**14**:355–8.
6. Sarma D, ter Brugge K. Management of intracranial dural arteriovenous shunts in adults. *Euro J Radiol* 2003;**46**:206–20.
7. Kim MS, Han DH, Kwon OK, Oh CW, Han MH. Clinical characteristics of dural arteriovenous fistula. *J Clin Neurosci* 2002;**9**:147–55.
8. Lasjaunias P, Chiu M, ter Brugge K, Tolia A, Hurth M, Berenstein M. Neurological manifestations of intracranial dural arteriovenous malformations. *J Neurosurg* 1986;**64**:724–30.
9. Stiebel-Kalish H, Setton A, Nimii Y, Kulish Y, Hartman J, Huna Bar-On R, et al. Cavernous sinus dural arteriovenous malformations: patterns of venous drainage are related to clinical signs and symptoms. *Ophthalmology* 2002;**109**:1685–91.
10. van Dijk JM, terBrugge KG, Willinsky RA, Wallace MC. Clinical course of cranial dural arteriovenous fistulas with long-term persistent cortical venous reflux. *Stroke* 2002;**33**:1233–6.
11. Brown Jr RD, Wiebers DO, Nichols DA. Intracranial dural arteriovenous fistulae: angiographic predictors of intracranial hemorrhage and clinical outcome in nonsurgical patients. *J Neurosurg* 1994;**81**:531–8.
12. Davies MA, Ter Brugge K, Willinsky R, Wallace MC. The natural history and management of intracranial dural arteriovenous fistulae, part 2: aggressive lesions. *Interv Neuroradiol* 1997;**3**:303–11.
13. Söderman M, Pavic L, Edner G, Holmin S, Andersson T. Natural history of dural arteriovenous shunts. *Stroke* 2008;**39**:1735–9.
14. Lasjaunias P, Berenstein A. *Surgical neuroangiography: endovascular treatment of craniofacial lesions*. Berlin: Springer-Verlag; 1987.
15. Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, et al. Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. *Radiology* 1995;**194**:671–80.
16. Borden JA, Wu JK, Shucart WA. A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment. *J Neurosurg* 1995;**82**:166–79.
17. Halbach VV, Higashida RT, Hieshima GB, Hardin CW, Pribram H. Transvenous embolization of dural fistulas involving the cavernous sinus. *AJNR Am J Neuroradiol* 1989;**10**:377–83.
18. Friedman JA, Pollock BE, Nichols DA, Gorman DA, Foote RL, Stafford SL. Results of combined stereotactic radiosurgery and transarterial embolization for dural arteriovenous fistulas of the transverse and sigmoid sinuses. *J Neurosurg* 2001;**94**:886–91.
19. Bertalanffy A, Dietrich W, Kitz K, Bavinzski G. Treatment of dural arteriovenous fistulae (dAVF's) at the superior sagittal sinus (SSS) using embolization combined with micro- or radiosurgery. *Minim Invasive Neurosurg* 2001;**44**:205–10.
20. Lewis AI, Tomsick TA, Tew Jr JM. Management of tentorial dural arteriovenous malformations: transarterial embolization combined with stereotactic radiation or surgery. *J Neurosurg* 1994;**81**:851–9.
21. Troffkin NA, Graham 3rd CB, Berkmen T, Wakhloo AK. Combined transvenous and transarterial embolization of a tentorial-incisural dural arteriovenous malformation followed by primary stent placement in the associated stenotic straight sinus: case report. *J Neurosurg* 2003;**99**:579–83.
22. Murphy KJ, Gailloud P, Venbrux A, Deramond H, Hanley D, Rigamonti D. Endovascular treatment of a grade IV transverse sinus dural arteriovenous fistula by sinus recanalization, angioplasty, and stent placement: technical case report. *Neurosurgery* 2000;**46**:497–501.
23. Luo CB, Teng MMH, Chang FC, Chang CY. Traumatic indirect carotid cavernous fistulas: angioarchitectures and results of transarterial embolization by liquid adhesives in 11 patients. *Surg Neurol* 2009;**71**:216–22.
24. Carlson AP, Taylor CL, Yonas H. Treatment of dural arteriovenous fistula using ethylene vinyl alcohol (onyx) arterial embolization as the primary modality: short-term results. *J Neurosurg* 2007;**107**:1120–5.
25. Maimon S, Nossek E, Strauss I, Blumenthal D, Frolov V, Ram Z. Transarterial treatment with Onyx of intracranial dural arteriovenous fistula with cortical drainage in 17 patients. *AJNR Am J Neuroradiol* 2011;**32**:2180–4.

26. Lv X, Jiang C, Li Y, Wu Z. Embolization of intracranial dural arteriovenous fistulas with Onyx-18. *Euro J Radiol* 2010;**73**:664–71.
27. Luo CB, Teng MMH, Chang FC, Guo WY, Chang CY. Transorbital direct puncture of posterior cavernous sinus through the ICA for embolization of isolated cavernous sinus dural arteriovenous fistula. *J Neurointerv Surg* 2013;**5**:e1.
28. Teng MM, Lirng JF, Chang T, Chen SS, Guo WY, Cheng CC, et al. Embolization of carotid-cavernous fistula by means of direct puncture of superior orbital fissure. *Radiology* 1995;**194**:705–11.
29. White JB, Layton KF, Evans AJ, Tong FC, Jensen ME, Kallmes DF, et al. Transorbital puncture for the treatment of cavernous sinus dural arteriovenous fistulas. *Am J Neuroradiol* 2007;**28**:1415–7.
30. Luo CB, Chang FC, Wu HM, Chung WY. Transcranial embolization of a transverse-sigmoid sinus dural arteriovenous fistula carried out through a decompressive craniectomy. *Acta Neurochirurgica* 2007;**149**:197–200.
31. Houdart E, Saint-Maurice JP, Chapot R, Ditchfield A, Blanquet A, Lot G, et al. Transcranial approach for venous embolization of dural arteriovenous fistulas. *J Neurosurg* 2002;**97**:280–6.
32. Endo S, Kuwayama N, Takaku A, Nishijima M. Direct packing of the isolated sinus in patients with dural arteriovenous fistulas of the transverse-sigmoid sinus. *J Neurosurg* 1998;**88**:449–56.
33. Rabinov JD, Yoo AJ, Ogilvy CS, Carter BS, Hirsch JA. Onyx versus n-BCA for embolization of cranial dural arteriovenous fistulas. *J Neurointervent Surg* 2013;**5**:306–10.
34. Kim DJ, Willinsky RA, Krings T, Agid R, Terbrugge K. Intracranial dural arteriovenous shunts: transarterial glue embolization-experience in 115 consecutive patients. *Radiology* 2011;**258**:554–61.
35. Nelson PK, Russell SM, Woo HH, Alastra AJ, Vidovich DV. Use of a wedged microcatheter for curative transarterial embolization of complex intracranial dural arteriovenous fistulas: indications, endovascular technique and outcome in 21 patients. *J Neurosurg* 2003;**98**:498–506.
36. Guedin P, Gaillard S, Boulin A, Condette-Auliac S, Bourdain F, Guieu S, et al. Therapeutic management of intracranial dural arteriovenous shunts with leptomeningeal venous drainage: report of 53 consecutive patients with emphasis on transarterial embolization with acrylic glue. *J Neurosurg* 2010;**112**:603–10.
37. Cognard C, Januel AC, Silva Jr NA, Tall P. Endovascular treatment of intracranial dural arteriovenous fistulas with cortical venous drainage: new management using Onyx. *Am J Neuroradiol* 2008;**29**:235–41.
38. Luo CB, Chang FC, Teng MMH, Wu HM, Lin CJ, Guo WY, et al. Transarterial Onyx embolization of intracranial dural arteriovenous fistulas: a single center experiences. *J Chin Med Assoc* 2014;**77**:184–9.
39. Luciani A, Houdart E, Mounayer C, Saint Maurice JP, Merland JJ. Spontaneous closure of dural arteriovenous fistulas: report of three cases and review of the literature. *Am J Neuroradiol* 2001;**22**:992–6.
40. Dawson 3rd RC, Joseph GJ, Owens DS, Barrow DL. Transvenous embolization as the primary therapy for arteriovenous fistulas of the lateral and sigmoid sinuses. *Am J Neuroradiol* 1998;**19**:571–6.
41. Urtasun F, Biondi A, Casaco A, Houdart E, Caputo N, Aymard A, et al. Cerebral dural arteriovenous fistulas: percutaneous transvenous embolization. *Radiology* 1996;**199**:209–17.
42. Jiang C, Lv X, Li Y, Wu Z. Transarterial Onyx packing of the transversesigmoid sinus for dural arteriovenous fistulas. *Eur J Radiol* 2011;**80**:767–70.
43. Kiyosue H, Hori Y, Okahara M, Tanoue S, Sagara Y, Matsumoto S, et al. Intracranial treatment of intracranial dural arteriovenous fistulas: current strategies based on location and hemodynamics, and alternative techniques of transcatheter embolization. *Radiographics* 2004;**24**:1637–53.
44. Kai Y, Hamada J, Yano S, Nakamura H, Nakino K, Takeshima H, et al. Treatment of cavernous sinus dural arteriovenous fistulae by external manual carotid compression. *Neurosurgery* 2007;**60**:253–7.
45. Guo WY, Pan DH, Wu HM, Chung WY, Shiau CY, Wang LW, et al. Radiosurgery as a treatment alternative for dural arteriovenous fistulas of the cavernous sinus. *AJNR Am J Neuroradiol* 1998;**19**:1081–8.
46. Klisch J, Huppertz HJ, Spetzger U, Hetzel A, Seeger W, Schumacher M. Transvenous treatment of carotid cavernous and dural arteriovenous fistulae: results for 31 patients and review of the literature. *Neurosurgery* 2003;**53**:836–56.
47. Zhou LF, Chen L, Song DL, Gu YX, Leng B. Tentorial dural arteriovenous fistulas. *Surg Neurol* 2007;**67**:472–81.
48. Tomak PR, Cloft HJ, Kaga A, Cawley CM, Dion J, Barrow DL. Evolution of the management of tentorial dural arteriovenous malformations. *Neurosurgery* 2003;**52**:750–62.
49. Huang Q, Xu Y, Hong B, Li Q, Zhao W, Liu J. Use of Onyx in the management of tentorial dural arteriovenous fistulae. *Neurosurgery* 2009;**65**:287–92.
50. Houser OW, Baker Jr HL, Rhoton Jr AL, Okazaki H. Intracranial dural arteriovenous malformations. *Radiology* 1972;**105**:55–64.
51. Chai EQ, Wang J. Transarterial Onyx embolization of sagittal sinus dural arteriovenous fistulae. *Neurol India* 2011;**59**:262–5.
52. Deng JP, Li J, Zhang T, Yu J, Zhao ZW, Gao GD. Embolization of dural arteriovenous fistula of the anterior cranial fossa through the middle meningeal artery with Onyx. *Clin Neurol Neurosurg* 2014;**117**:1–5.
53. Li Q, Fang YB, Huang QH, Zhang Q, Hong B, Zhao WY, et al. Transarterial embolization of dural arteriovenous fistulas of the anterior cranial fossa with Onyx. *J Clin Neurosci* 2013;**20**:287–91.