Review Article

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Quick Response Code:

Website: http://www.braincirculation.org

DOI: 10.4103/bc.bc_12_24

Intracranial dural arteriovenous fistulas with pial arterial supply: A narrative review

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Abstract:

Intracranial dural arteriovenous fistula (DAVF) is a relatively complex intracranial condition, and its clinical presentation and treatment strategies often vary significantly due to various factors. Although the cure rate of intracranial DAVF is currently high, there is still a lack of understanding of its etiology and pathogenesis. There is ongoing controversy regarding the treatment strategies for DAVF associated with the pial arteries, and there is a lack of understanding of its pathogenesis. The author conducted a brief literature review on DAVF with pial arterial supply and presented some treatment experiences from their own medical center. Large-scale retrospective cohort studies and prospective research in future are expected to address these issues.

Keywords:

Dural arteriovenous fistula, dural branch, pial artery supply, pial supply, pure supply, review

Introduction

Dural arteriovenous fistula (DAVF) refers to abnormal connections between the meningeal arteries and the dural venous sinuses and/or the subarachnoid veins. DAVFs account for 10% to 15% of intracranial arteriovenous malformations.[1,2] The main distinction between DAVFs and parenchyma-related lesions (such as arteriovenous malformations) is that DAVFs do not involve the parenchyma. It has been recognized that in rare cases, DAVFs can be supplied by branches of the pial arteries.[3-11] They accounts for about 10%–20% of all DAVFs.[8-10] DAVFs with pial arterial supply are more likely to have treatment-related complications compared to those without pial arterial supply.[6,7,9,11] However, some studies have shown that there is no significant difference in the incidence of treatment-related complications (postoperative hemorrhage and ischemia) between DAVFs with and without pial arterial supply.[8,10] Further understanding the incidence, clinical features, angioarchitectures, and pathogenesis of DAVFs with pial arterial supply is crucial for guiding treatment and preventing surgical complications. The author provides a narrative review of current research progress on this topic.

Epidemiology

DAVF is a rare vascular disorder of the nervous system. The estimated incidence of DAVF is 0.15–0.29/100,000 adults per year.[12-14] Some DAVFs may be asymptomatic or resolve on their own, leading to the possibility that the true incidence of DAVF may be underestimated.[15] Moreover, the incidence of DAVF may vary depending on the study population, diagnostic methods, and classification criteria used.[16] A multicenter surveillance study conducted in Japan found that the incidence of DAVF increased from approximately 0.5/100,000 person-years in 2009 to around 1.4/100,000

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Submission: 25-02-2024
Revised: 29-04-2024
Accepted: 09-05-2024
Published: 26-09-2024

How to cite this article: Su X, Ma Y, Song Z, Zhang P, Zhang H. Intracranial dural arteriovenous fistulas with pial arterial supply: A narrative review. Brain Circ 2024;10:205-12.

person-years in 2019, surpassing arteriovenous malformations (0.805/100,000 person-years).[14] The increase in the incidence of DAVF may be attributed to several reasons. First, advancements in imaging diagnostic technologies have enhanced the detection and diagnosis of DAVF, enabling radiologists and clinicians to identify cases that may have previously gone undiagnosed. Second, the aging population may have played a role, as DAVF is more common among the elderly.[14,16]

Most DAVFs occur in middle-aged and older adults, although a small percentage can be seen in children. These lesions are often more extensive in children, with poorer treatment outcomes and prognosis.[17-19] DAVFs are slightly more common in males,[2,20] but in certain specific locations such as the cavernous sinus region, they are more common in females.[2,21,22]

DAVFs with pial arterial supply account for approximately 10% to 20% of all DAVFs.[8-10] Patients with DAVFs supplied by the pial artery tend to have a younger age, but they are still mainly in middle and older age groups. The male-to-female ratio is approximately equal, with slightly more male patients.[8,10]

Anatomy

The dura mater of the cerebral convexity is mainly supplied by branches of the middle meningeal artery, occipital artery, ophthalmic artery, vertebral artery, etc.[23] The dura mater, which covers the skull base, is supplied in a more complex way than the cerebral convexity. Several branches of the internal carotid, external carotid, and vertebrobasilar artery systems supply the skull base dura mater in a complex and overlapping way. The internal carotid artery system supplies the midline dura mater of the anterior and middle cranial fossa, as well as the anterior boundary dura mater of the posterior cranial fossa. The external carotid artery system supplies the lateral segments of the anterior, middle, and posterior cranial fossa. The vertebrobasilar artery system supplies the midline portions of the posterior cranial fossa and the area of the foramen magnum dura mater. The main areas where the blood supply overlaps are the parasellar region, the tentorium cerebelli, and the falx cerebri dura mater. In addition, the tentorium cerebelli and the falx cerebri also receive blood supply from the cerebral arteries, making these structures a communication pathway between the dural and cerebral arterial systems.[5,6]

Common blood supply from cerebral arteries to dura mater includes:[3,23]

1. Anterior cerebral artery-olfactory branches,[24,25] pericallosal artery, and the anterior falcine artery[3]
2. Posterior cerebral artery – the artery of Davidoff and Schechter[26,27]
3. Superior cerebellar artery – the medial dural tentorial branch[5]
4. Anterior inferior cerebellar artery – the subarcuate artery[3]
5. Posterior inferior cerebellar artery – posterior meningeal artery and the artery of the falx cerebelli.[3]

The pial arteries, located on the surface of the brain, give rise to branches that supply blood to the brain parenchyma. In DAVFs, the anastomoses mentioned above allow for the possibility of supplying the fistula through the pio-dural arterial connections.[8] The bridging veins are located at the transition between the subarachnoid space and the subdural space, and they are covered by the dura mater and thickened arachnoid mater.[28] For bridging vein type DAVF, the fistula is thought to be at the point where the bridging vein is covered by the dura mater,[28,29] making it more prone to pial arterial supply.[8,10,28] This explains why most DAVFs with pial arterial supply are found near the tentorium [Table 1], as the majority of tentorial region DAVFs are the bridging vein type.

DAVFs with pial arterial supply are classified into two types:[8,10] one type arises from anastomoses between the aforementioned pial and dural arteries, located within the dura mater, displaying a linear morphology on angiography resembling common dural artery supply [Figure 1a and b]; the other type is fed by pial arteries without any connection to dural arteries, exhibiting a tortuous, ramified, and glomus-like structure on angiography [Figure 1c-e].[8,30] Due to the lack of dural covering, the latter is more fragile compared to the former. A DAVF supplied by pure pial artery may extend into the subdural space along the drainage pathway.[7] The treatment strategy for this type of DAVF may differ from traditional dural artery-supplied DAVFs.[6,11,30] In this type of DAVF, the risk of intraoperative hemorrhage may be higher. The high risk of hemorrhage may be due to the lack of dural covering around the bridging vein fistula extending into the subdural space. The fragile glomus-like structures may rupture due to increased pressure after arterial embolization restricts venous drainage, similar to the principle observed in arteriovenous malformation embolization, in which feeding arteries rupture if the draining vein is completely occluded first.[31,32]

Classification

Rizzoli first described DAVF in 1881, whereas Sachs provided the first description using cerebral angiography in 1931.[33,34] In 1977, Djindjian and Merland first described the relationship between the angioarchitecture

Table 1: Summary of study characteristics

Study	Total population (DAVF with pial arterial supplies)	Location	Pial arterial supply	Treatment	Complication/ suspected causes	Recommended treatment strategy
Wu *et al*., 2016[6]	6	Tentorium (6)	PCA (6), ACA (1)	TAE (6)	Intraoperative hemorrhage (2)/ pial arterial supply (2)	Embolize the pial artery supply first
Hetts *et al*., 2017[9]	29	Not specified	Not specified	Embolization (17), surgery (3), embolization+surgery (9)	Ischemia stroke/ pial artery supply	Transvenous embolization
Li *et al*., 2018[11]	26	Tentorium (26)	SCA (16), PICA (9), AICA (3)	TAE (21), TAE+surgery (5)	Rebleeding (2)/pial artery supply (2), cranial nerve palsy (1)/dangerous vessels	Embolize the pial artery supply first
Osada and Krings, 2019[8]	23	Tentorium (12), TTS (8), torcular (1), SSS (1), FM (1)	ACA (2), SCA (10), AICA (4), PCA (9), PICA (4), MCA (1)	Observation (2), TAE (8), TVE (2), γ knife (3), TAE + γ knife (1), TVE + γ knife (1), surgery (1), TAE + surgery (1), TAE + surgery + γ knife (1), TAE + TVE (3)	No complications	Not necessary to embolize the pial arterial supply first
Brinjikji *et al*., 2021[10]	27	TSS (1), tentorium (21), SSS (2), multiple (2), other (1)	PCA (20), PICA (3), SCA (8), ACA (3), MCA (1), AICA (1)	Confusing description	Minor complications (2)/embolus and perforation of the dural artery	Not necessary to embolize the pial arterial supply first
Okamoto *et al*., 2020[70]	8	Tentorium (5), TSS (1), ethmoid (1), convexity (1)	PCA (3), SCA (2), MCA (2), ACA (1)	Surgery	Worsening of occipital edema/ venous edema	Surgery
Miyamoto and Naito, 2022[33]	10	Tentorium (10)	PCA (8), SCA (3), PICA (2)	TAE (10)	Intraoperative hemorrhage (2)/ pial artery supply	Embolize the pial artery supply first

ACA: Anterior cerebral artery, AICA: Anterior inferior cerebellar artery, DAVF: Dural arteriovenous fistula, FM: Foramen magnum, MCA: Middle cerebral artery, PCA: Posterior cerebral artery, PICA: Posterior inferior cerebellar artery, SCA: Superior cerebellar artery, SSS: Superior sagittal sinus, TAE: Trans-arterial embolization, TSS: Transverse sigmoid sinus, TVE: Transvenous embolization

of DAVF and the risk of hemorrhage.[34,35] This later led to the development of two commonly used classification systems, the Borden classification[36] and the Cognard classification.[2] These classifications can help guide treatment decisions because DAVF with cortical venous drainage may necessitate treatment to avoid potential complications such as intracranial hemorrhage. There are also other famous classifications such as those for craniospinal epidural venous anatomy,[37] the Barrow classification for carotid-cavernous fistula and cavernous sinus DAVFs,[38] the Lasjaunias classification for pediatric DAVFs,[39] and classification based on the leptomeningeal venous drainage.[29] These various classifications help us gain a comprehensive understanding of the different subtypes of DAVFs from perspectives such as embryonic development, pathogenesis, and anatomy.

As mentioned earlier, DAVFs with pial arterial supply are mainly classified into two types: One where the blood supply is from a physiological anastomosis between pial arteries and dural arteries, and the other type where the blood supply originates from pial arteries themselves.[3,8,10,23] Due to the different physiological structures of the arteries involved, the treatment strategies for DAVFs supplied by these two types of arteries will also vary.[6,7,11,33]

Pathophysiology

While multiple theories have been proposed to describe the pathophysiology of intracranial DAVF, the true underlying reasons behind their occurrence and development remain a topic of debate. The primary reasons that may lead to the formation of a DAVF are venous sinus thrombosis and venous hypertension.[40-42] In conditions of venous hypertension and hypoxia, the high expression of vascular endothelial growth factor, hypoxia-inducible factor-1, and other molecules is closely associated with the occurrence and development of DAVF.[30] Moreover, the elevation of D-dimer in DAVF patients can account for the thrombotic abnormalities.[43] In patients with cerebral venous thrombosis, thrombosis leading to venous hypertension may result in subsequent dilation of preexisting physiological arteriovenous anastomoses or the development of *de novo* arteriovenous shunts.[16,42,44,45] Similarly, secondary angiogenesis caused by trauma, malignancies, intracranial inflammatory reactions, or a history of craniotomy can result in

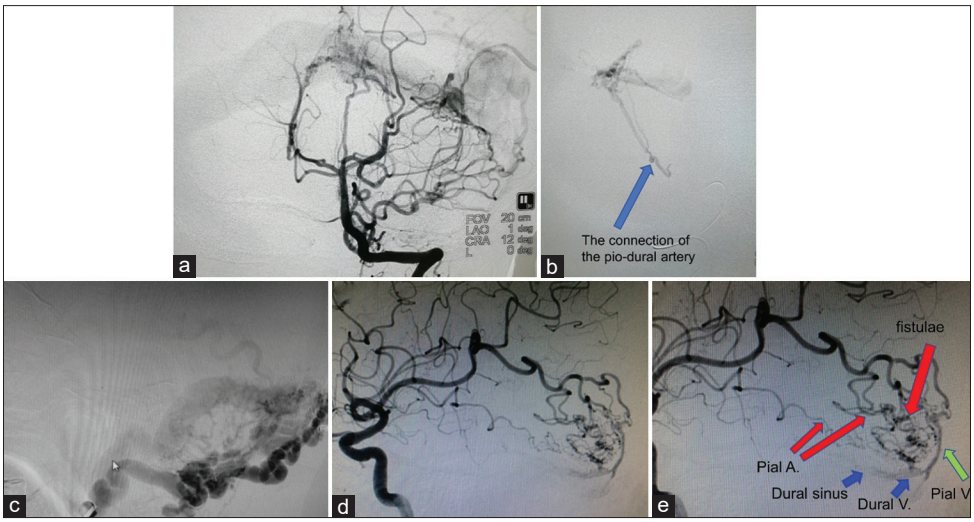

Figure 1: (a and b) A vertebral angiogram showing a transverse sinus dural arteriovenous fistula. A selective angiogram of the right posterior cerebral artery showing the dural branch (b). Note the transition from tortuous pial artery to linear dural artery (arrow). (c-e) A case of transverse sinus dural arteriovenous fistula supplied by multiple dural and pure pial branches. Note the tortuous pure pial arteries from the middle cerebral artery with glomus-like structure and the fistulous point in the pial part of the blood supply may be located around the pial mater rather than within the dura mater (e). A: Artery; V: Vein

the formation of DAVF.[16] In addition, venous sinus thrombosis may also result from DAVF, possibly due to alterations in the hemodynamics of the venous system, leading to the activation of blood coagulation factor and subsequent thrombus formation.[46]

The presence of pure pial arterial supply cannot be explained by physiological anastomoses. The speculated mechanism of occurrence may be associated with abnormal vascular proliferation in the mentioned above. There are reports of multiple cases of DAVFs with associated pial arterial supply following venous sinus thrombosis[47,48] and acquired pial arteriovenous fistulas following cerebral venous thrombosis.[49,50] In the vein of Galen malformation, pial arterial supply may be induced due to hypoxia.[51] To validate the hypothesis of "venous hypertension-hypoxia-inducible factors – vascular endothelial growth factor – neovascularization," studies have shown that the dilation of veins within DAVF, which signifies venous hypertension, is an independent predictive factor for associated pial arterial supply.[8,10] In addition, younger age is also a predictive factor for associated pial arterial supply in DAVF[8,10] and patients with DAVF accompanied by pure pial arterial supply tend to be younger compared to those with dural branches from the pial arteries.[8] This may be related to the high sensitivity of younger individuals to hypoxic conditions.[52] Venous hypertension caused by various reasons may be the main cause of the pial and dural arterial supply in DAVF.[53] The current hypothesis regarding the pathogenesis of intracranial DAVF is shown in Figure 2.

Clinical Presentations

The clinical presentation of DAVF depends on the anatomical location of the DAVF, the pattern of venous drainage, blood flow, the extent of the lesion, and other factors.[2,29,36,39] Even in high-grade DAVFs (Cognard IIb/III/IV and Borden II/III), many patients may remain asymptomatic until hemorrhage occurs.[54] Therefore, for patients with atypical or progressive neurological deficits, a history of trauma, a history of venous sinus thrombosis, and cranial surgeries, a high suspicion for DAVF should be maintained. Research shows that the most common clinical manifestations include orbital symptoms (conjunctival congestion, proptosis, and visual impairment), pulsatile tinnitus, hemorrhage (brain parenchymal hemorrhage, subarachnoid hemorrhage, and subdural hematoma), and nonhemorrhagic neurological deficit (cortical venous reflux leading to cortical venous and deep cerebral venous system hypertension).[20,55]

Research has shown that DAVFs with pial arterial supply have a higher proportion of hemorrhage compared to those without pial arterial supply.[10] In one study, the proportion of hemorrhage at presentation was approximately three times higher in DAVFs with pial artery supply than in those without.[10] In another study, the proportion of hemorrhage at presentation was slightly higher in DAVFs with pial artery supply than in those without.[8] This could be closely related to the higher proportion of high-grade DAVF and more dilated drainage veins in the group of DAVF with pure arterial supply.[8,10] Because of study biases, it is difficult to accurately determine the specific risk of hemorrhage in this population.

Radiological Diagnosis

Non-enhanced computed tomography (CT) is commonly used for imaging examination before or after treatment

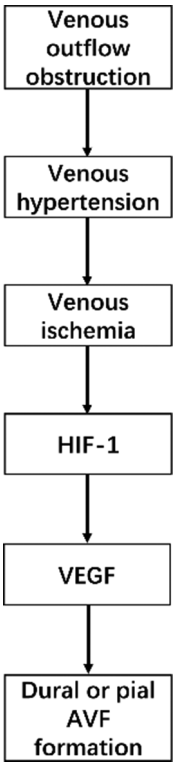

Figure 2: The current hypothesis regarding the pathogenesis of intracranial dural arteriovenous fistula. An obstruction to venous outflow, such as a sinus thrombus or tumor compression, would produce venous outflow obstruction. AVF, Arteriovenous fistula; HIF-1, hypoxia-inducible factor-1; VEGF, vascular endothelial growth factor

of DAVF to diagnose cerebral parenchymal hemorrhage, subarachnoid hemorrhage, hydrocephalus, and vascular source edema caused by venous hypertension.[56] In addition, CT can also reveal bone defects caused by dilated emissary veins.[31] Compared to CT, magnetic resonance imaging (MRI) can detect more in DAVF, such as white matter edema, venous sinus thrombosis, venous sinus stenosis, arterial supply, and dilated draining cortical veins.[56,57] In arteriovenous fistula, the "flow void sign" is commonly seen on T2 MRI, which is caused by the abnormal dilation of the draining veins. However, both CT and MRI can still lead to missed diagnosis of DAVF, and digital subtraction angiography (DSA) remains the gold standard for diagnosing DAVF.[31,16] DSA can identify the specific feeding arteries, fistula location, dilation draining veins, venous stasis, and more, providing guidance for the treatment of DAVF. In addition, DSA is one of the main methods used for follow-up after DAVF treatment. However, due to its invasive nature, some DAVF patients often opt for MRI as a primary method for follow-up after curative embolization.

DSA is necessary for the diagnosis of DAVF with accompanying supply from the pial artery. The specific blood supply and morphological structure of such DAVFs have been described previously.

Management

DAVF can be treated in various ways, including conservative management, endovascular therapy, surgical resection, and stereotactic radiosurgery. The choice of treatment for a DAVF is often based on the patient's clinical symptoms and the angioarchitecture of the DAVF. Currently, the most commonly used treatment method is endovascular embolization with the goal of occluding the fistula between the feeding artery and draining vein, as well as the proximal draining vein if possible.[55,58-62] However, in specific locations such as anterior cranial fossa DAVF, foramen magnum DAVF, and petrosal region DAVF, surgical ligation of the draining veins remains a primary treatment option.[4,63-65]

DAVFs with pial arterial supply are often high-grade aggressive lesions, therefore requiring endovascular therapy or surgical treatment.[6-10,33]

Complications

Endovascular complications are mainly associated with the infiltration of embolic agents into intracranial-extracranial anastomoses, leading to brain infarction and cranial nerve palsy, thromboembolic complications, vessel perforation, venous congestion, and procedure-related complications.[20,66-68] In a recent meta-analysis on endovascular treatment for cavernous sinus DAVFs, the treatment-related complication rate was 7.75%, with the majority being transient cranial nerve palsies.[69]

In the treatment of DAVF with pial arterial supply, common complications are related to intracranial hemorrhage or ischemic stroke.[6-10,33] One study found that if the pial artery supply was not embolized first, the complication rate was roughly 15 times higher than in DAVFs without pial artery supply.[6] However, two other larger studies yielded different results.[8,10] The causes of ischemic stroke may be related to the reflux of embolic agents into the pial arteries or postoperative retrograde thrombosis of pial arteries, leading to cerebral ischemia.[9] These complications may be more likely to occur in DAVFs with pure pial arterial supply.[33]

Does the Presence of the Pial Artery Have an Impact on the Treatment Strategy for Dural Arteriovenous Fistula?

In a literature report based on 53 patients treated over a 5-year period at a single center, there were six cases of tentorial DAVFs with pial arterial supply. Among these six patients undergoing treatment, two experienced intraoperative hemorrhage, and one of them died as a result. Compared to DAVFs without pial arterial supply,

there may be a higher probability of intraoperative hemorrhage in cases with pial arterial supply.[6] Therefore, the authors recommend performing embolization of the pial arteries first to prevent intraoperative hemorrhage. Subsequent case reports and small-sample studies also support the authors' suggestion.[7,11,33]

A study involving 122 patients with and without pial arterial supply in DAVF showed that DAVF with pial arterial supply is more prone to develop postoperative ischemic stroke compared to those without pial arterial supply. To avoid embolic agents refluxing into the pial arteries during arterial embolization, transvenous embolization is commended.[9]

Two studies of 201 and 204 DAVF patients, where no pre-embolization of pial arteries treatment strategy was used, found no statistically significant difference in the rates of hemorrhage and ischemia between the two groups.[8,10] As a result, the researchers do not recommend pre-embolization of the pial arteries to treat this type of DAVF. In addition, embolization of tortuous and fragile pial arteries increases the risk of rupture. The current relevant large-sample literature is summarized in Table 1.

In the medical center where the author is based, there were several cases of intraoperative hemorrhage during routine treatment of this type of DAVF in earlier years. The primary reason was believed to be the rupture of pial arteries. Therefore, we are more inclined to recommend starting with the embolization of pial arteries when treating this type of DAVF. Furthermore, only those pure pial arteries with extensive supply may result in hemorrhage if not embolized beforehand.[33] For those with a very limited supply of the lesions, pre-embolization is considered unnecessary. The reasons why the problems were not elucidated by the first two studies may primarily include the following aspects:[8,10] (1) the sample size was too small, with only about 20 cases of DAVFs supplied by the pial arteries; (2) there was no subgroup analysis between DAVFs supplied by pial arteries and those with dural branches from the pial arteries; and (3) even in DAVFs supplied by pure pial arteries, not all pial arteries would necessarily rupture without pre-embolization, which might be closely related to the range and morphological structure of them. In conclusion, large-scale retrospective studies and prospective research are needed to address these issues.

Conclusion

Intracranial DAVF is a relatively complex intracranial condition, and its clinical presentation and treatment strategies often vary significantly due to various factors. Although the cure rate of intracranial DAVF is currently high, there is still a lack of understanding of its etiology and pathogenesis. There is ongoing controversy regarding the treatment strategies for DAVF associated with the pure pial arteries, and there is a lack of understanding of its pathogenesis. Large-scale retrospective cohort studies and prospective research in future are expected to address these issues.

Author contributions

XS: Design, Literature search, data analysis, manuscript preparation and manuscript editing; YJM: Concepts, manuscript review and guarantor; ZHS: Definition of intellectual content and manuscript editing; PZ: Definition of intellectual content, clinical studies and manuscript editing; HQZ: Concepts, manuscript review and guarantor.

Ethical statements

Not applicable.

Declaration of Helsinki

Not applicable.

Data availability statement

Data sharing not applicable to this article as no datasets were generated and/or analyzed during the current study

Financial support and sponsorship

This study was funded by the National Natural Science Foundation of China (No. 82101460).

Conflicts of interest

There are no conflicts of interest.

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