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Review Article

Endovascular Treatment of Anterior Cranial Fossa Dural Arteriovenous Fistula

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Anterior cranial fossa (ACF) dural arteriovenous fistula (DAVF) is a rare lesion among cerebral DAVFs. This lesion shows significant bleeding risk because of the angioarchitecture, involving direct leptomeningeal retrograde venous drainage, as a nonsinus-type DAVF. Over the years, direct surgery has been considered the primary treatment for ACF DAVF, offering favorable clinical outcomes compared to a low complete obliteration rate with endovascular treatment and the relatively high risk of blindness due to central retinal artery occlusion with transophthalmic artery embolization. In recent years, however, significant improvements in DSA and 3D reconstruction imaging quality have allowed a much more precise understanding of the angioarchitecture of the shunt and vascular access route. In addition, advances in endovascular devices, including catheters and embolic materials, have facilitated microcatheter navigation into more distal vessels and more reliable closure of the fistulous point. Supported by such technological innovations, endovascular approaches to the treatment of ACF DAVF have been becoming successful first-line treatments. This article reviews the evolution of treatment strategies and the current status of endovascular treatment for ACF DAVF, with a particular focus on transarterial embolization.

Keywords ▶ anterior cranial fossa, dural arteriovenous fistula, transarterial embolization, Onyx

Introduction

Anterior cranial fossa (ACF) dural arteriovenous fistulas (DAVFs) are rare lesions, accounting for only 5.8% of all cerebral DAVFs.[1–3] This entity is recognized for an aggressive behavior and a high rate (62–91%) of hemorrhagic presentations.[1,4] The significant bleeding risk of this entity is likely associated with the angioarchitecture, depicted as direct leptomeningeal retrograde venous drainage classified as Borden type III and Cognard type III or IV.[5,6] An analysis of the Japanese Registry of Neuroendovascular Therapy 2 (JR-NET2) showed that ACF DAVF was significantly associated with a hemorrhagic presentation (odds ratio, 4.1; 95% confidence interval, 1.0–14.7, p = 0.036) in multivariate analysis.[7] Historically, surgical treatment has been considered the primary treatment option for ACF DAVFs. This is because direct surgery has consistently shown excellent angiographic and clinical outcomes,[4,8] whereas endovascular treatments for ACF DAVFs have encountered significant technical challenges resulting in a low complete obliteration rate and a high risk of blindness due to central retinal artery occlusion.[9–11] In recent years, however, the endovascular approach has been becoming a successful first-line treatment, as the precise anatomical structure of the lesion can now be fully understood due to significant improvements in DSA imaging quality, while advances in endovascular devices have facilitated microcatheter guidance into more distal vessels. This article reviews the evolution of treatment strategies and the current status of endovascular treatment for ACF DAVF, with a particular focus on transarterial embolization.

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Angioarchitecture and Clinical Symptoms of ACF DAVFs

ACF DAVFs are usually supplied from uni- or bilateral ethmoidal arteries. The ethmoidal arteries originating from

the distal ophthalmic artery in the orbit enter the ethmoid sinus and pass through the ethmoid foramen at the lateral edge of the cribriform plate.[12] Some patients present with blurred vision, attributed to a steal phenomenon from both central retinal arteries due to ethmoidal arterial feeding of the ACF DAVF.[13] Supply from the external carotid artery is usually via the internal maxillary artery, superficial temporal artery, or distal cribriform branch of the anterior medullary artery.[14] Some papers have reported ACF DAVF symptomatic cases with epistaxis. Those patients showed microaneurysms associated with a feeding artery or aneurysmal-like dilatation of drainage vein in the ethmoid sinus and nasal cavity, which were suggested as likely sources of epistaxis.[14] Some ACF DAVF cases have a pial artery supply.[15,16] A pial arterial supply has two potential etiologies: a physiological dural branch of the pial artery or a pure pial arterial supply. Although the pathophysiologies underlying the development of DAVF has not yet been confirmed, a pure pial supply is hypothesized to involve de novo development after the induction of angiogenesis.[17] Recent studies have reported a high risk of perioperative complications in DAVFs with a pial arterial supply.[18,19] The cribriform plate, which is embryologically the shunt point of ACF DAVFs, is derived from neural crest cells, and the dura mater around the cribriform plate in the anterior skull base comprises only intrinsic dura mater. Therefore, there is basically no venous sinus in the space between the intrinsic dura mater, and shunt blood flow is expected to flow back into the cortical veins via adjacent bridging veins.[20] ACF DAVFs then drain secondarily into the superior sagittal sinus and/or through the olfactory vein to the basal vein. Some papers have reported unique ACF DAVF cases with drainage into the frontobasal vein with reflux into the superficial cerebral vein, resulting in aphasia.[21,22] Diplopia due to cavernous sinus venous congestion via the sphenoparietal sinus and myelopathy due to venous congestion in the brainstem caused by ACF DAVF have both been reported.[23] ACF DAVF can be a diagnostic pitfall, as presentation can involve a wide variety of symptoms depending on the anatomical variations.

Developmental Mechanisms in ACF DAVF

The etiology of DAVF in the anterior cranial base remains uncertain. Clinical observations and serial angiographic findings have shown that many DAVFs are acquired and that sinus thrombosis, sinus hypertension, trauma, and cranial surgery might contribute to their formation.[24,25] Most DAVFs arising after intracranial surgery are located in the transverse sigmoid sinus, although a case of ACF DAVF that developed remote from the craniotomy site after intracranial surgery has also been described.[24,26] ACF DAVFs are sometimes also identified with other cerebrovascular pathologies, including ruptured aneurysm, cavernous malformation, arteriovenous malformation, and internal carotid artery occlusion. However, the association with these other vascular disorders can be incidental and triggers a diagnosis of asymptomatic DAVFs.[27–29] An analysis of the JR-NET2 reported that the incidence of aggressive symptoms was higher in the transverse sigmoid sinus, superior sagittal sinus, craniocervical junction, and ACF.[7] ACF DAVFs are recognized for an aggressive behavior and a high rate (62%–91%) of hemorrhagic presentations including intracranial hemorrhage (ICH), and subdural and epidural hematoma.[1,4] Catastrophic ICH in an ACF DAVF often occurs in the setting of increased hemodynamic pressure in the draining venous channel. Some cases of an increase in the size of a varix of ACF DAVF have been reported,[23,30] and the development of venous dilatation may be part of the natural history of DAVFs with cortical venous reflux and may contribute to the occurrence of ICH.

Radiological Characteristics of ACF DAVFs

Venous varix near the fistulous site without obvious venous outflow might be mistaken for aneurysm on CT or MRI in patients with ACF DAVF.[31,32] Since the development or enlargement of venous varix due to increased hemodynamic stress associated with progressive steno-occlusion of the venous drainers might contribute to catastrophic intracerebral hemorrhage, this finding could lead to misdiagnosis. DSA is a standard radiological examination to evaluate the exact angioarchitecture of a fistula. However, early diagnosis of DAVF is very helpful to avoid progression and subsequent ICH in patients with this lesion as a screening. Dilated cortical draining veins exist in the frontobasal area in most patients with ACF DAVF and can be depicted as flow voids on T2-weighted MRI.[33,34] Susceptibility-weighted MRI identifies retrograde venous reflux and venous congestion.[35] For these reasons, MRI is convenient for detecting clinically silent or incidental abnormalities. In addition, the new MRI technique of four-dimensional flow phase-contrast MRI to identify abnormal blood velocity in the draining vein of ACF DAVF[36] and arterial spin-labeling (ASL) MRI diagnose obliteration of the ACF

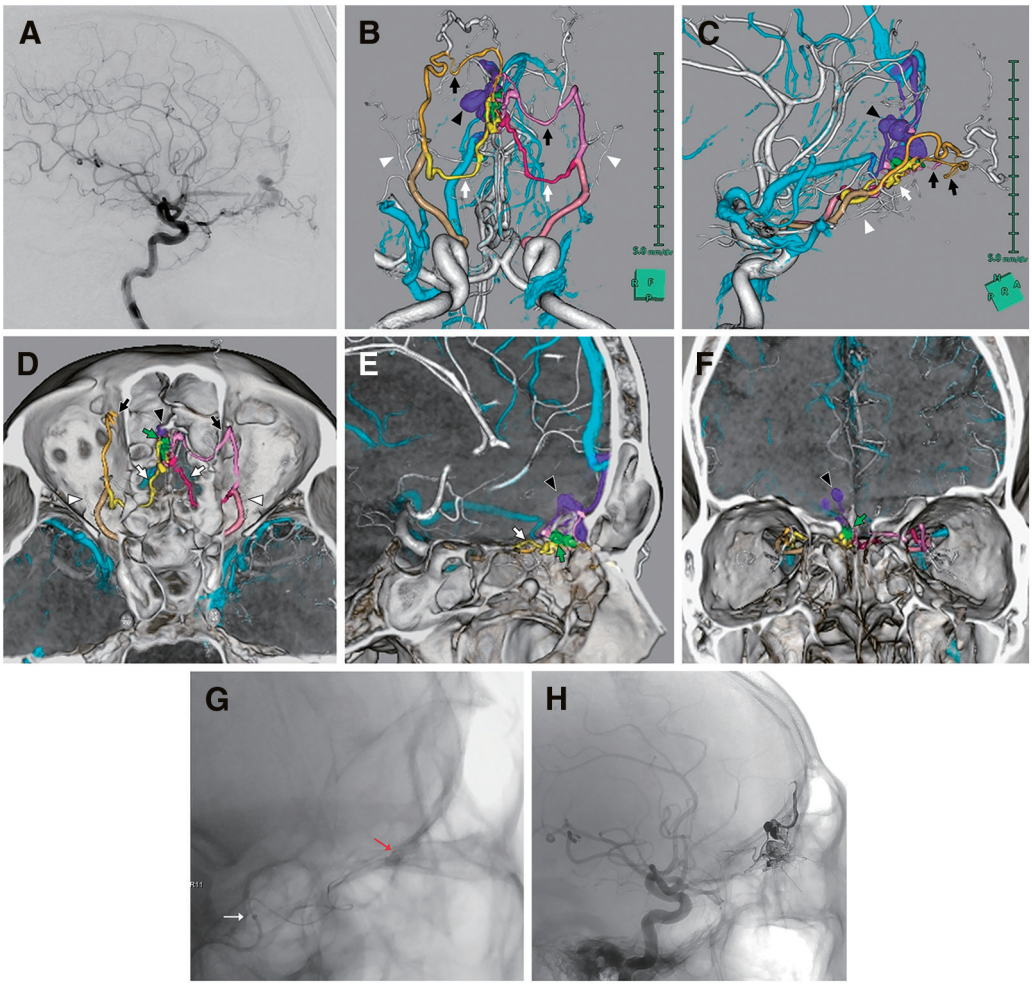

Fig. 1 Representative case of ACF DAVF. Right internal carotid angiography depicts the ACF DAVF with venous dilatation (**A**). Axial (**B**) and lateral (**C**) views of 3D reconstructed vascular imaging visually aid in understanding the 3D angioarchitecture of the shunt. Bilateral anterior and posterior ethmoid arteries feed the shunt and drain the superior sagittal sinus and frontal basal vein through dilated cortical veins. A 3D fusion image of vascular and surrounding structures likewise aids the anatomical understanding of the shunt (**D**–**F**). Black arrows: anterior ethmoidal artery, white arrows: posterior ethmoidal artery, white arrowheads: central retinal artery, black arrowheads: venous varix, and green arrows: fistulous portion. Lateral view from the right internal carotid angiography shows the location of catheters navigated to the shunt through the right ophthalmic artery (**G**). Red arrow indicates that the tip of the microcatheter close to the shunt. White arrow shows the intermediate distal-access catheter located in the ophthalmic portion of the right internal carotid artery. Lateral view from the right internal carotid angiography shows complete obliteration of the shunt after transarterial embolization with Onyx in a single session (**H**). ACF: anterior cranial fossa; DAVF: dural arteriovenous fistula

DAVF based on the disappearance of a signal-hyperintense area on ASL.[37] Recently, 3D fusion imaging technology with 3D DSA, MR, and CTA has been developed for the preoperative evaluation of angioarchitecture. We show a representative 3D fusion image of ACF DAVF for preoperative evaluation using SYNAPSE VINCENT software (Fujifilm, Tokyo, Japan) in **Fig. 1**. This diagnostic imaging tool visualizes the 3D positional relationships between the skull, dura mater, brain, and nerves, facilitating an understanding of the precise 3D vasculature of ACF DAVF, and allowing more detailed and specific treatment plans to be discussed within the treatment team.

Treatment Strategy for ACF DAVF

Over the years, surgical treatment has been considered the primary treatment option for ACF DAVF, because direct surgery has shown favorable clinical outcomes.[4,8,38] Direct surgery is somewhat invasive but can reliably obliterate the shunt regardless of vascular anatomy and tortuosity. Endovascular therapy, on the other hand, is less invasive, but clinical outcomes are influenced by vascular anatomy and the access route to the shunt, and advances in catheter technology have been necessary to improve the obliteration rate.[38–40] A recent meta-analysis comparing endovascular

versus surgical treatment for ACF DAVF indicated that surgical treatment was superior to endovascular therapy in terms of complete obliteration and overall good outcome, although adverse event rates were similar between groups (postoperative hemorrhage, 3% vs 7%; postoperative stroke rate, 2% vs 3%; new-onset seizure, 3% vs 0%).[9] Indeed, previous reports have shown that transarterial embolization provides an occlusion rate of 13%–64%,[1,41] whereas transvenous embolization achieved a complete occlusion rate of 63%–91% in a larger series.[42] However, a recent systematic review indicated that 91.7% of 48 patients achieved complete angiographic cure, and 93.8% had a good outcome.[43] In addition, the postprocedural complication rate with endovascular treatment for ACF DAVF was 8.3% in 48 cases and no central retinal artery ischemia was identified, even in 2 cases with excessive reflux of Onyx (ev3/Covidien, Irvine, CA, USA) into the ophthalmic artery.[43] Therefore, recent endovascular treatments have thus allowed safe and effective treatment for ACF DAVFs.

Endovascular Treatment for ACF DAVFs

The therapeutic goal of endovascular treatment is for the embolic materials to penetrate through the fistulous point to obliterate the shunt. Transarterial embolization is the primary treatment option in cases with a nontortuous transarterial access via the ophthalmic artery to the fistula.[1,40] Recent advances in endovascular devices, however, have allowed the use of flexible guiding catheters and intermediate distal-access catheter to guide the microcatheter close to the fistulous point and deposit liquid embolic materials through the fistula.[39] Previously, diluted N-butyl cyanoacrylate (NBCA; B. Braun, Melsungen, Germany) was commonly used for transarterial embolization via the ethmoidal artery, because particle embolization carries a risk of unintended occlusion of the central retinal artery or internal carotid artery, resulting in visual loss and ischemic complications.[13,21,44] Recently, nonadhesive liquid embolic argents such as Onyx, Squid (Balt, Montmorency, France), and Phil (MicroVention, Aliso Viejo, CA, USA) have been employed for transarterial embolization, as these agents are easier to handle than adhesive liquid embolic argents, can be infused in a large bolus at one time, and show high permeability into the shunt.[14,42] However, physicians must pay attention to the risk of excessive reflux to the ophthalmic artery. ACF DAVFs with pial arterial supply can treat with transarterial embolization using liquid embolic argents; however, involvement of pial arterial supply can be associated with a higher risk of ischemic and hemorrhagic complications due to retrograde migration of embolic argents to pial feeders or restriction of the venous outlet.[16,45] Therefore, physicians should also be careful to ensure obliteration of pial feeders and not allow excessive reflux to pial feeders.

To avoid endovascular complications and achieve complete obliteration of the fistula, various innovations have been reported. Use of a micro-balloon catheter can enhance the safety and efficacy of transophthalmic artery embolization, providing additional protection to the retinal and ciliary arteries against unintended reflux of liquid embolic agent.[46] Short-segment detachable catheters can minimize traction on the feeding artery upon removal of the catheter and can reduce the risk of hemorrhage.[46] To reduce blood flow via internal maxillary artery feeders for curative transophthalmic artery embolization, insertion of a surgical gauze infiltrated with xylocaine and epinephrine into bilateral nasal cavities has also been reported.[47] Transarterial embolization via the middle meningeal artery (MMA) has been described as an alternative to transophthalmic artery embolization to avoid visual complications.[48,49] However, MMA is not the main feeding artery in ACF DAVFs and is a long distance from and occasionally tortuous to the shunt. Transvenous embolization can also provide an effective alternative for ACF DAVFs while avoiding ophthalmic complications, with a success rate of 80%.[42] However, transvenous approaches are limited to cases with a well-developed anterior third of the superior sagittal sinus without nontortuous venous pathways and display a higher risk of complications, including venous perforation.[3,50]

Conclusion

Endovascular approaches to the treatment of ACF DAVF have been becoming a primary treatment option due to radiological advances allowing an understanding of the precise angioarchitecture of the lesion and innovations in endovascular devices. Transarterial embolization has become a safe and effective treatment as long as the angioarchitecture, including the vascular access in each case, can be accurately identified and an appropriate approach selected.

Disclosure Statement

The authors have no conflicts of interest concerning the materials or methods used in this study or the findings specified in this paper.

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