



# Non-Sinus Type Dural Arteriovenous Fistula: Others

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The clinical manifestations of dural arteriovenous fistulas (dAVFs) are highly variable and dependent on the hemodynamic properties and location of the fistula. The locations of the fistula are numerous and include the cavernous sinus, transverse–sigmoid sinus, superior sagittal sinus, inferior and superior petrosal sinuses, anterior condylar confluence, tentorium, anterior cranial fossa, middle fossa, foramen magnum, cranio-cervical junction, convexity, and spinal cord. These dAVFs can be divided into two types, “sinus type” and “non-sinus type,” based on their communication with dural shunts and cerebral veins. The sinus type involves direct communication between the arterial dural branch and one dural sinus, sometimes leading to recruitment of cortical veins. On the other hand, the non-sinus type is embedded into the dura, with the drainage always involving a cerebral vein and no communication with any sinus. Treatment options for these types of dAVFs differ; sinus-type dAVFs require normally sinus obliteration and occlusion of recruited veins, while non-sinus-type dAVFs require embolization of the drainage vein. Accurately classifying the type of fistula, sinus type or non-sinus type, is critical for developing a proper treatment plan. This review describes clinical characteristics and treatment of those non-sinus-type dAVFs involving unusual locations with illustrative cases.

**Keywords** ▶ dural arteriovenous fistula, endovascular treatment, non-sinus type, sinus type

## Introduction

Dural arteriovenous fistulas (dAVFs) account for 15% of all cerebral arteriovenous shunts, with 7% located supratentorially and 35% located infratentorially.<sup>1)</sup> These malformations are acquired and defined by abnormal communication between arteries, dural venous sinuses, and/or subarachnoid veins within the dural leaflets. The feeding arteries are often external carotid artery tentorial branches, vertebral artery meningeal branches, or cerebral arteries pial branches.

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While dAVFs most commonly affect people in their forties and fifties, they can also affect youngsters and do not have a definite sex predilection or genetic component.<sup>2)</sup>

The clinical manifestations of dAVFs are highly variable and dependent on the hemodynamic properties and location of the fistula. Just 2% of patients with Borden categorization type I dAVFs would develop cerebral bleeding and neurologic loss, but the rate rises to 18% for Type II and 34% for type III.<sup>3)</sup> During four years, the mortality rate for individuals with cerebral venous drainage is 45%, with a 19.2% intracranial hemorrhage risk and a 10.9% new neurologic impairment rate. Patients who present with hemorrhage have a 35% chance of repeated hemorrhage within the first 2 weeks of the initial ictus.<sup>1,4-7)</sup> The cavernous sinus, transverse–sigmoid sinus, superior sagittal sinus (SSS), inferior and superior petrosal sinuses, anterior condylar confluence, tentorium, anterior cranial fossa, middle fossa, foramen magnum, cranio-cervical junction, convexity, and spinal cord are all possible locations for the fistula.

These dAVFs can be divided into two types, “sinus type” and “non-sinus type,” based on their communication with dural shunts and cerebral veins. The sinus-type dAVFs drain directly into the dural sinus, which can sometimes result in cortical vein recruitment. In contrast, the non-sinus

type is embedded into the dura, with the drainage always involving a cerebral vein and no direct drainage to the dural sinus. Treatment strategies for various forms of dAVFs differ; sinus-type dAVFs necessitate sinus obliteration and blocking of recruited veins, whereas non-sinus-type dAVFs necessitate drainage vein embolization.<sup>8)</sup> Accurately classifying the type of fistula, sinus type or non-sinus type, is critical for developing a proper treatment plan.

Regarding practical surgical considerations, D'Aliberti et al.<sup>8)</sup> recommended that dAVFs be categorized as either "sinus type" or "non-sinus type." The former type involves a direct connection between the dural shunt and a single sinus, and cortical veins may sometimes be incorporated. The latter type, in contrast, is enclosed within the dura and always causes cerebral vein drainage. Just as with any medical condition, treatment decisions for dAVFs should be per the surgeon's or neuro-interventionalist's expertise, the severity of the condition, and the available treatment options. Three different approaches can be applied in treating dAVFs: surgical, endovascular (transarterial/transvenous embolization), and stereotactic radiosurgery. The surgical management of sinus and non-sinus types differs and incorporates the resection or occlusion of the affected sinus, alongside the clipping or endovascular occlusion of cerebral veins that have been recruited. The recruited veins are characterized by inverted internal blood flow and are nonfunctional. It is crucial to spare any functional veins located distal to the affected sinus. However, the treatment of non-sinus types involves clipping or endovascular occlusion of the nonfunctional draining red vein.<sup>8)</sup> Non-sinus-type dAVFs are located at a distance from the dural venous sinus and drain exclusively via leptomeningeal veins. The aggressive neurological symptoms related to dAVFs in the tentorium cerebelli and the anterior cranial fossa are common, with tentorial dAVFs demonstrating intracerebral hemorrhage (ICH) rates ranging from 60% to 74% and anterior fossa dAVFs displaying rates ranging from 44% to 84%.<sup>9,10)</sup> It is logical to assume that non-sinus-type dAVFs that occur in uncommon locations such as the convexity, parasagittal region, and falx cerebri may also exhibit a significant risk of causing severe neurological symptoms and ICH. Hence, a comprehensive treatment approach is important for most of these cases, and different treatment options to disrupt abnormal venous drainage have been applied to attain a complete resolution of the condition. Nonetheless, their deep-seated locations can express a persistent risk of surgical differentiation. Research has revealed that transarterial Onyx embolization has a promising response rate of

90.9% for treating non-sinus-type dAVFs, rendering it a viable treatment option.<sup>11)</sup> At first, the principle of dAVF therapy involved endovascular treatment for the sinus type and surgical treatment for the non-sinus type, but with the introduction of Onyx as an embolic material, the majority of non-sinus-type dAVFs can now be successfully treated with it.<sup>12)</sup> Present endovascular therapy involves transvenous coil embolization of the sinus type and transarterial glue or Onyx embolization of the non-sinus type.<sup>12)</sup> This comprehensive review provides insights into the diagnosis and management of such exceedingly rare non-sinus-type dAVFs although dAVFs involving the convexity, parasagittal region, and falx cerebri AVF are rare and have only been reported in case studies. In the etiology of non-sinus-type dAVFs around SSS, the connection between the cortical and dural systems plays a significant role, as all brain surface veins must traverse the subdural space, referred to as "bridging veins" in this specific location, to reach the dura.<sup>13)</sup> Typically, most bridging veins directly drain into venous sinuses. However, there are notable variations, such as dural "venous lakes," which are commonly observed at the frontoparietal junction adjacent to the SSS. Shapiro et al. conducted a retrospective review of 100 dAVFs and identified 26 cases involving dural venous channels.<sup>13–15)</sup>

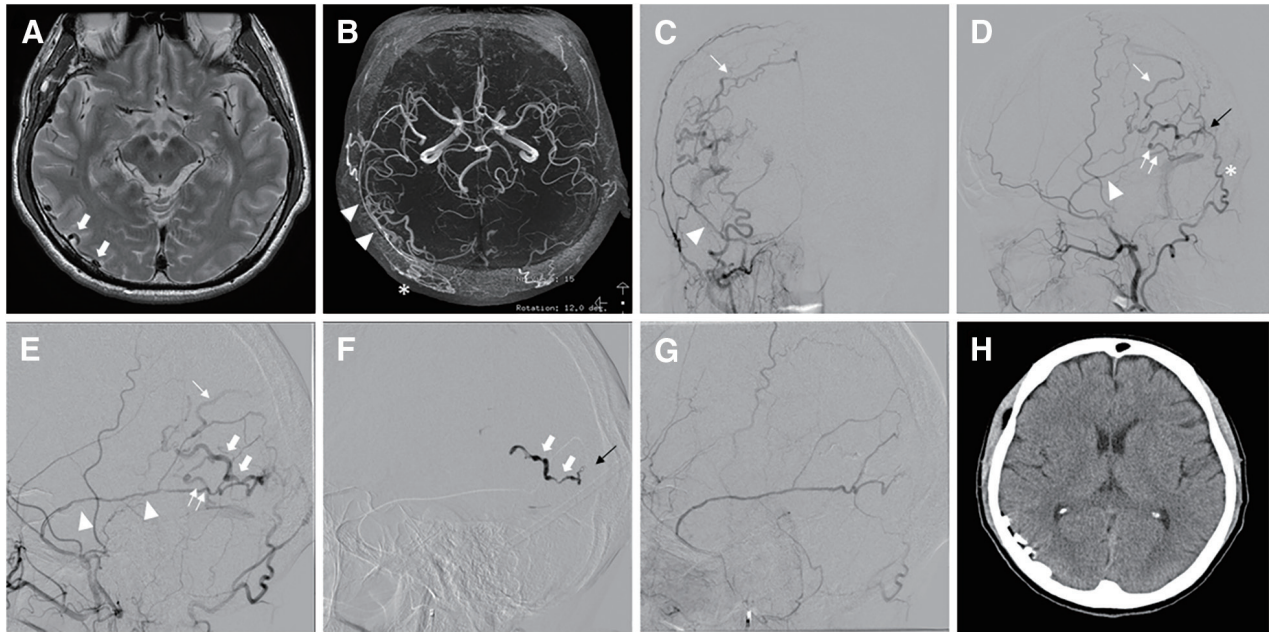
While most fistulas primarily involve a sinus, a significant minority appears to be situated either on or near a sinus but do not primarily drain into it. Instead, their drainage directly occurs into a cortical vein. Baltsavias et al.<sup>16)</sup> refer to these cases as shunts of the bridging veins. It is challenging to comprehend how a fistula unrelated to a sinus could gain access to a bridging or cortical vein unless that vein was initially associated with a dural segment before the subsequent formation of the fistula at that segment. A plausible explanation would be a fistula located adjacent to or draining into a dural venous channel.<sup>15)</sup>

When thrombosis occurs or there is a restriction of antegrade outflow into a draining sinus, it results in retrograde congestion within the cortical venous system. Fistulas that appear to drain exclusively into a cortical vein are believed to primarily involve a dural venous channel, providing an explanation for their formation around the SSS.

This review provides and discusses clinical cases of non-sinus-type dAVFs at rare locations.

## ■ Convexity dAVFs (Fig. 1)

Convexity dAVFs are supratentorial dAVFs that the fistulous points are situated in the dura mater encompassing the



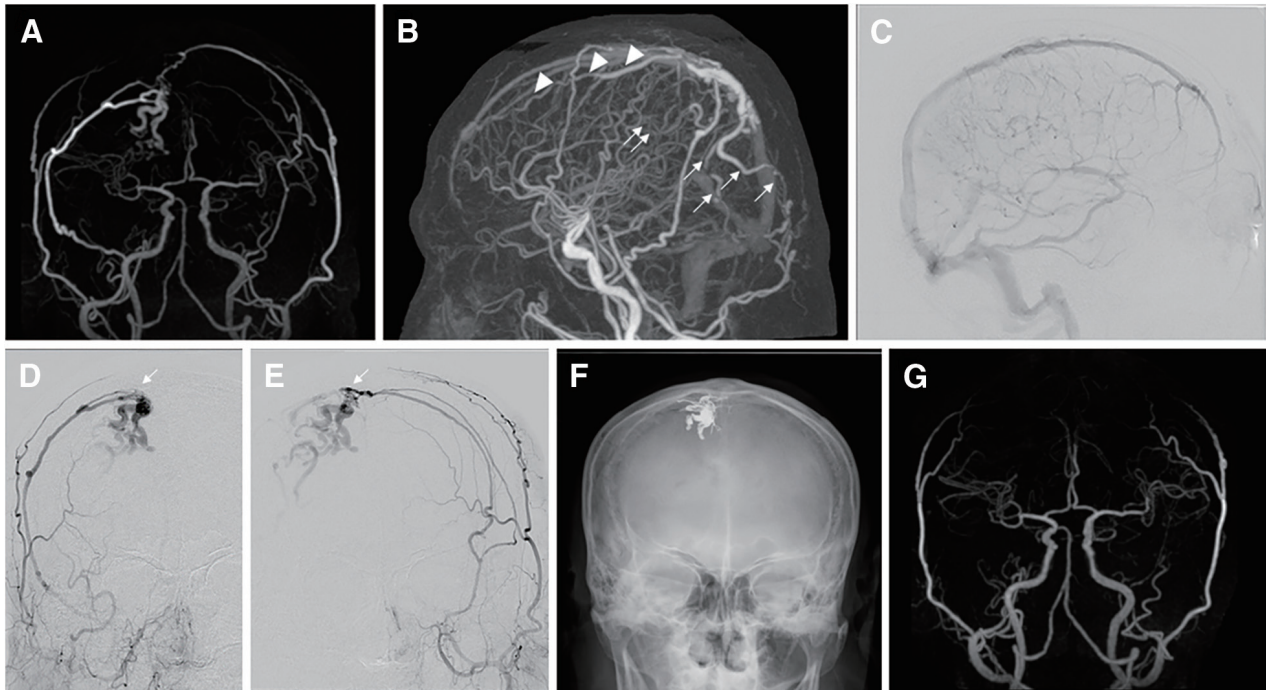
**Fig. 1** Convexity dAVF incidentally found on MRI in a 68-year-old man. (A) T2 weighted MRI showed flow voids (arrows) on the surface of his right occipital and temporal lobes. (B) MRA showed dAVF on the right occipital convexity, which was supplied by the right MMA (arrowheads) and OA (asterisk) with cortical venous reflux. (C–E) Frontal (C) and lateral (D, E) parts of the right external carotid angiography showed that the dAVF was nourished by the posterior convex branch of MMA (arrowhead) and the transosseous branch of OA (asterisk), with draining into the occipitoparietal cortical vein (white arrow) to the SSS and the vein of Labbe (double white arrows) to the transverse sinus. Black arrow indicated fistulous point. Endovascular embolization was performed because of a potentially high risk of cerebral hemorrhage for the dAVF with cortical venous reflux (white thick arrow) alone. A 1.5-F microcatheter (Marathon; ev3,

Irvine, CA, USA) was advanced into the posterior convex branch of the right MMA via a 4.2-F distal access catheter (Fubuki; Asahi Intecc Co., Ltd., Aichi, Japan) and a 6-Fr guiding catheter placed in the right ECA. (F) Lateral view of DSA during injection of 33% NBCA via the 1.5-Fr microcatheter whose tip positioned just proximal to the fistulous point (black arrow) showed NBCA cast filling into the venous side beyond the fistula (white thick arrow). (G) Lateral view of the right external carotid angiography soon after embolization showed complete occlusion of the convexity dAVF. (H) Postoperative CT revealed no brain edema. The patient was discharged without any complications, and no recurrence of the dAVF was observed for 5 years after treatment. dAVF: dural arteriovenous fistula; ECA: external carotid artery; MMA: middle meningeal artery; NBCA: n-butyl-2-cyanoacrylate; OA: occipital artery; SSS: superior sagittal sinus

convexity of the brain hemispheres and they do not have direct communication with dural sinuses like the SSS or transverse sinus (TS). ICH has been determined in 43% of convexity dAVFs.<sup>17)</sup> Curative treatment is required because of its high risk of aggressive symptoms. While it is acceptable to directly ligate the draining vein close to the shunt site as a treatment option, employing a liquid embolic material for transarterial embolization (TAE) is a less invasive alternative (**Fig. 1**). Presently, Onyx embolization for convexity dAVFs appears reasonable, as discussed previously. It is crucial to note that 11.3%–23.8% of intracranial dAVFs are supplied by pial arteries.<sup>18–20)</sup> Hetts et al.<sup>19)</sup> stated that dAVF patients with pial arterial supply who are treated endovascularly are at greater risk for ischemic stroke. Hence, it may be reasonable for patients with convexity dAVFs and pial arterial supply to undergo direct surgery. Recently, nevertheless, Korai et al.<sup>21)</sup> reported two cases of convexity dAVFs with pial arterial supply successfully treated with liquid embolic materials (n-butyl-2-cyanoacrylate [NBCA], Onyx). While both NBCA

and Onyx possess the potential to flow into the pial artery, Onyx is considered more effective in eliminating the fistula because the liquid material tends to flow toward the venous side, which is under lower pressure, before filling the pial arterial feeders. Thus, it is important to precisely determine the distance from the fistula to the injection site for Onyx and to stop the delivery of Onyx when the material reaches the predetermined location. Furthermore, Akamatsu et al.<sup>22)</sup> stated that convexity dAVFs and Borden type III classification were independent predictive factors for complete obliteration following embolization via the middle meningeal artery (MMA). NBCA could be employed as a substitute embolic material in emergency cases necessitating hematoma removal or when the microcatheter can be inserted near the fistulous site. A dAVF adjacent to a pial arteriovenous malformation (AVM) with a shared venous drainage may be mistaken for a dAVF with a pial arterial supply. It is a higher risk of hemorrhagic complications when endovascular embolization is performed under an incorrect diagnosis as dAVF. Due to the challenges





**Fig. 2** Parasagittal dAVF incidentally found on MRI in a 70-year-old man. (A) The silent MRA (frontal view) showed a dAVF in the right parasagittal region, which is being supplied by bilateral MMAs and drains into the cortical vein within the right parietal lobe. (B) Four-dimensional CTA showcased the presence of the dAVF within the right parietal cortex. The dAVF was fed by enlarged bilateral MMAs and directly drains into a cortical vein in the right parietal lobe. The identified venous drainage routes are connected to the vein of Labbe (arrow), parasagittal frontal vein (arrowhead), and superficial middle cerebral vein (double arrow). (C) Right internal carotid angiography revealed pseudophlebitic pattern, particularly in the right parietal lobe. Notably, the SSS remained patent. Considering the risk of cerebral bleeding associated with severe cortical venous reflux and pseudophlebitic pattern in the right parietal lobe, we opted for endovascular embolization as the treatment of choice. (D and E) Right (D) and left (E) external carotid angiography demonstrated the dAVF being supplied by bilateral MMAs. The shunt point (arrow) is situated on the

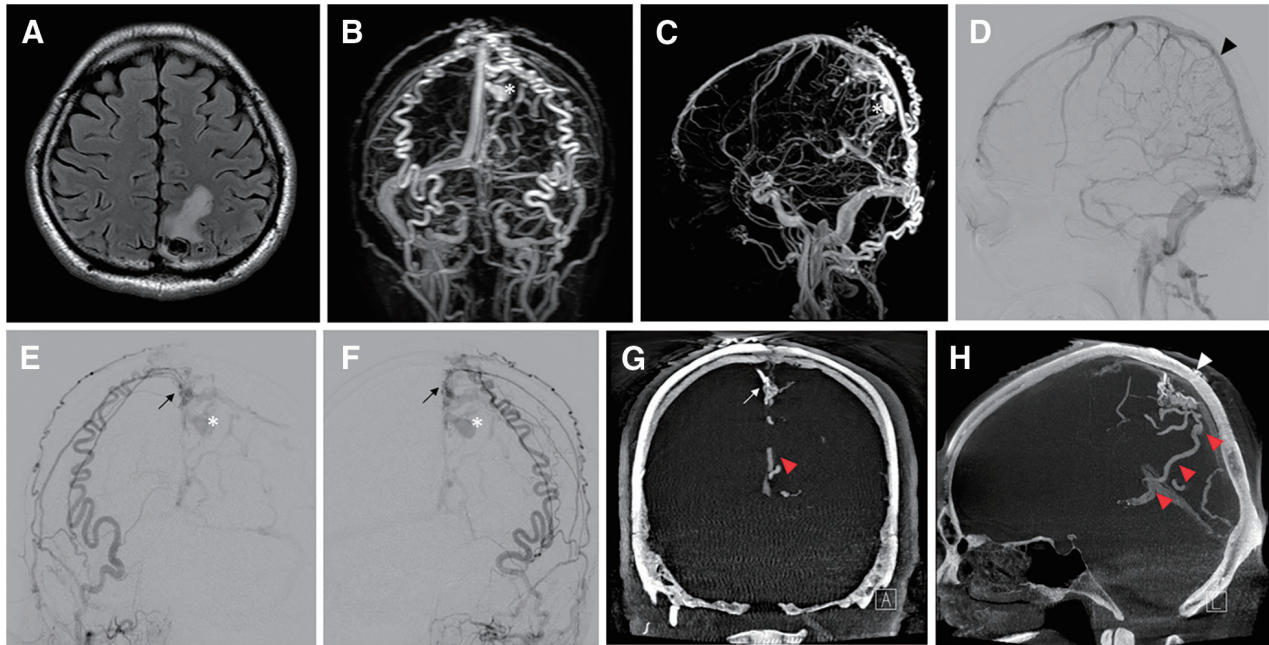
cortical vein within the right parietal lobe, rather than the SSS. Balloon catheters (Scepter XC; MicroVention, Aliso Viejo, CA, USA) were carefully navigated proximal to the foramen spinosum of both MMAs. Subsequently, a 1.3-Fr microcatheter (DeFricor; Sumitomo Bakelite, Akita, Japan) was advanced through a 3.4-Fr GuidePost (Tokai Medical Products, Aichi, Japan) into the right posterior branch of MMA, with the tip of the DeFricor positioned close to the shunt point. Under flow control utilizing both sides of balloons, Onyx was predominantly injected into a parasagittal venous lake. Postoperatively, left and right carotid angiograms confirmed the complete disappearance of the dAVF. (F) Frontal view of the post-embolization X-ray clearly displayed the presence of the shunt pouch, and the bilateral feeders from the MMA were filled with Onyx. (G) Postoperative silent MRA revealed the complete disappearance of the dAVF. The patient was discharged home without experiencing any complications. dAVF: dural arteriovenous fistula; MMA: middle meningeal artery; SSS: superior sagittal sinus

in intraoperative confirmation of the coexisting pial AVM, it becomes crucial to identify its presence during preoperative imaging diagnosis. Following endovascular treatment, a dAVF with a pial arterial supply exhibits a markedly higher occurrence of intracerebral hemorrhagic complications compared to a dAVF without a pial arterial supply.<sup>23)</sup> Direct surgery might be required especially when the dAVF with pial arterial supply is near an eloquent area such as the motor cortex to avert ischemic or hemorrhagic complications.

## Parasagittal dAVF (Fig. 2)

A parasagittal dAVF is considered a distinct entity within the spectrum of dAVFs, characterized by a shunt point located on adjacent to the SSS, draining directly into the cortical vein or falcine sinus. However, the precise anatomical explanation

remains elusive. Leveraging the advancements in recent imaging technology, we need to make a diagnosis based on accurate shunt points; however, a clear definition for parasagittal dAVF has yet to be established. Apart from parasagittal dAVF, alternative names such as falcine dAVF, dAVF at the falx cerebri, convexity dAVF (near the SSS), convexity-SSS dAVF, type 4 SSS dAVF, and dAVF in the SSS region have been used.<sup>21,24–29)</sup> Among them, falcine dAVF and dAVF at the falx cerebri must be classified as falcine dAVFs in this review. The other non-sinus-type dAVFs nearby or adjacent to SSS are included in the parasagittal dAVF. The parasagittal dAVF could be deemed extremely uncommon. The venous lake adjacent to the SSS may have served as a connection between the cortical vein and the dural system, ultimately leading to the formation of the fistula and dAVF. Several studies have proposed potential mechanisms to elucidate the



**Fig. 3** Falcine dAVF was incidentally found on MRI in a 55-year-old man. **(A)** The FLAIR MRI image indicated flow void and adjacent edema formation in the medial aspect of the left parietal lobe. **(B and C)** 3D MR-DSA **(B:** frontal view and **C:** lateral view) showed a dAVF in the falx cerebri fed by bilateral MMAs, STAs, and OAs, and primarily drained into cortical veins in the medial parietal lobe with varix formation (asterisk). **(D)** The lateral view of the left internal carotid angiography displayed pseudophlebatic pattern of medullary and cortical veins in the left parietal lobe. Note that the SSS is patent (arrowhead). **(E and F)** The right **(E)** and left **(F)** external carotid angiography showed the dAVF fed by bilateral STA, MMA, and OA. The shunt point (arrow) is situated on the falx cerebri, not on the SSS. The

asterisk represents the varix in the drainer. **(G)** The coronal reconstructed image of rotational angiography clearly showed the shunt point on the falx (white arrow) and midline drainage route (red arrowhead). **(H)** The coronal reconstructed image of rotational angiography clearly showed that the main drainage route (red arrowheads) on the medial parietal lobe with varix drained into the vein of Galen, which implies falcine sinus involvement. The option of endovascular embolization was chosen because of the possibility of intracranial hemorrhage resulting from the presence of cortical venous reflux and varix formation at the falcine sinus. dAVF: dural arteriovenous fistula; MMAs: middle meningeal arteries; OAs: occipital arteries; SSS: superior sagittal sinus; STAs: superficial temporal arteries

association between dAVF and cerebral venous thrombosis (CVT). One proposed mechanism is that CVT may trigger the development of a dAVF through angiogenetic activity induced by reduced cerebral perfusion due to venous hypertension or by the enlargement of preexisting arteriovenous shunts caused by elevated venous pressure. This mechanism has been supported by findings from various animal model studies.<sup>5,30</sup> The angioarchitecture of parasagittal dAVFs, which drain directly into a cortical vein, may be influenced by the presence of dural venous channels.

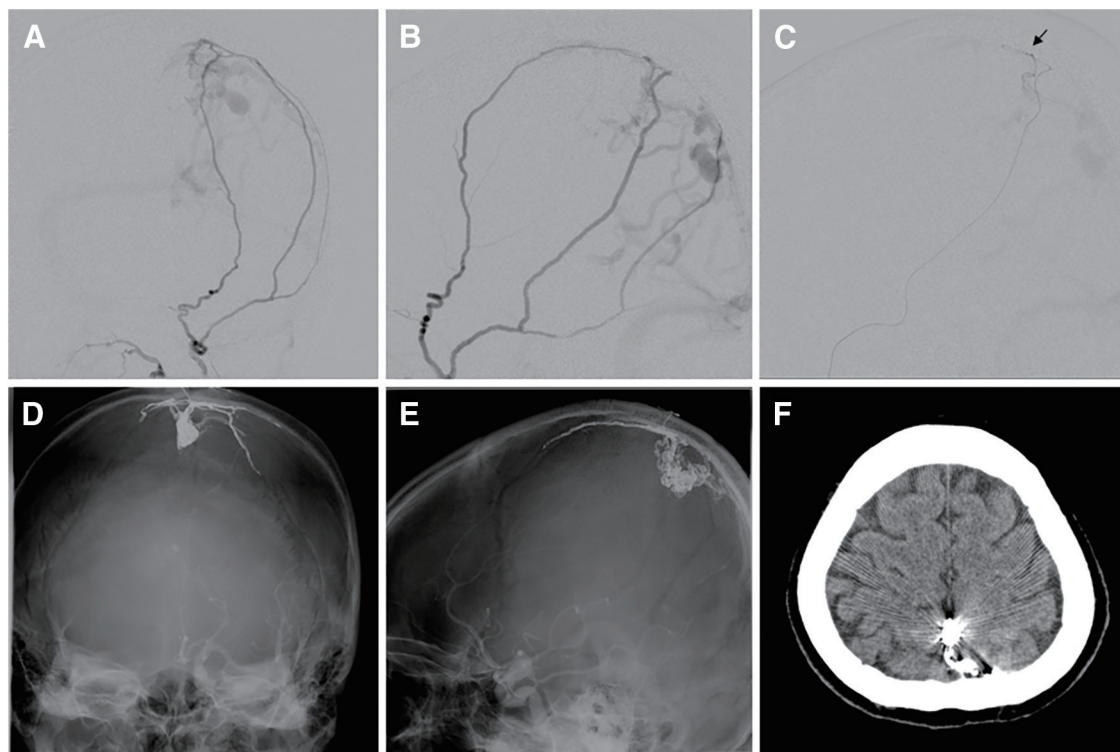
Regarding the treatment options, endovascular embolization can be a first-line treatment option for parasagittal dAVFs as well as other non-sinus-type dAVFs (**Fig. 2**). Although most cases have been treated by transarterial approach, transvenous embolization can be applicable for the cases with a short and non-tortuous approach route.<sup>31</sup> Similar to SSS dAVFs, the parasagittal dAVFs are usually fed bilaterally by feeders from the MMA, the superficial and occipital arteries, and embolization from those feeders are generally low risk for neurological complications. However, given that parasagittal dAVF as well as convexity dAVF also

receive blood supply from the anterior cerebral artery, surgical procedures necessitate a cautious approach. The artery of Davidoff and Schechter (ADS) is a dural branch of the posterior cerebral artery that can supply the meninges close to the falcotentorial junction. It is usually not identified on angiography except when enlarged in the setting of a dAVF or meningioma.<sup>32</sup> Endovascular treatment can be successfully undertaken by incorporating detailed anatomic assessment with risk-reduction strategies.

### ■ Falcine dAVFs (Figs. 3 and 4)

The falx cerebri is a midline partition that has venous plexuses, which are primarily concentrated in the posterior one-third and inferior two-thirds, as detected in cadaveric studies.<sup>33</sup> Additionally, the falcine sinus within the falx cerebri is a transit venous sinus that develops during embryonic development, connecting the SSS and inferior sagittal sinus (ISS) or straight sinus.<sup>34</sup> Although it is extremely rare, dAVFs can occur in the falx cerebri, involving either the falcine sinus or the falcine venous plexus. The falcine





**Fig. 4** Falcine dAVF incidentally found on MRI in a 55-year-old man (continued from **Fig. 3**). (**A** and **B**) Frontal (**A**) and lateral (**B**) views of the selected angiography of the left MMA revealed the angioarchitecture of the dAVF in the falx cerebri. (**C**) A 1.5-Fr microcatheter (Marathon; ev3, Irvine, CA, USA) was advanced through a 4.2-Fr distal access catheter (Fubuki; Asahi Intecc Co, Aichi, Japan) into the posterior convex branch of MMA. The tip of the microcatheter was positioned proximal to the shunt point and wedged (arrow). Onyx 18 was injected into the shunt point vascular channel and the feeders from MMAs, STAs, and OAs. (**D** and **E**) After embolization, the cortical draining vein and feeders were filled with embolic materials, and the shunt was successfully obliterated. Frontal (**D**) and lateral (**E**) views of postembolization X-ray showed the shunt pouch and the bilateral feeders from the MMA were filled with Onyx. (**F**) CT following embolization disclosed the Onyx cast in the shunt pouch in the falx cerebri, and the patient was discharged without any complications. Subsequent MRI scans conducted over 5 years demonstrated no recurrence of the dAVF. dAVF: dural arteriovenous fistula; MMA: middle meningeal artery; OAs: occipital arteries; STAs: superficial temporal arteries

sinus usually closes after birth, but persistent falcine sinuses have been reported in the pediatric population, although they are extremely rare without associated anomalies such as bifid cranium, vein of Galen aneurysmal malformation, agenesis of the corpus callosum, or Chiari malformation Type II.<sup>35)</sup> Furthermore, if the straight sinus is absent or underdeveloped, the falcine sinus may be reopened.<sup>36,37)</sup> Falcine sinuses are highly uncommon in adults, although Ryu<sup>38)</sup> noted that they were present in 2.1% of adults without associated anomalies. Tatarlı et al.<sup>39)</sup> observed that the falcine venous plexus demonstrated various anatomical patterns depending on its location in the anterior, middle, or posterior portions of the falx cerebri and that falcine sinuses portrayed various patterns as well. Nonetheless, it is crucial to distinguish between the rare persistent fetal falcine sinus and the falcine venous plexus. Yoshioka et al.<sup>24)</sup> described a case in which the falcine sinus functioned as the drainer for the dAVF, whereas Yamaguchi et al.'s analysis implies that

in some cases, part of the dAVF's drainer may involve the falcine sinuses.<sup>25)</sup> These dAVFs are developed owing to the recanalization of the falcine sinus/venous plexus following obstruction of one or more of the SSS, ISS, Galenic vein, and/or straight sinus, or the acquired obstruction of a congenital falcine sinus/venous plexus.<sup>25)</sup> Therefore, these dAVFs typically occur at a younger age and often possess venous pouches. Tubbs et al.<sup>33)</sup> observed that all falcine venous plexuses were connected to the ISS and that 63% were linked to the SSS. In our case, we discovered a dAVF in the falx cerebri that drained into the cortical veins and falcine sinus, but not into the SSS (**Figs. 3 and 4**). This implies that there was a disconnection between the SSS and the falcine venous plexus/falcine sinus. We believe that during the formation of the dAVF, the network channels between the SSS and falcine sinus may have become blocked, causing the reflux to the cortical vein and falcine sinus in our case. Treating a dAVF in the falcine venous plexus or falcine

sinus with transvenous embolization is difficult because of the challenges involved in accessing and embolizing the venous plexus. Rather, TAE utilizing liquid embolic materials can be a reasonable and effective technique for eliminating the dAVF in the falcine venous plexus or falcine sinus.<sup>34)</sup> It may be challenging to remove the falx cerebri extensively, in cases where the fistulous point is located deep within the falx cerebri including the falcine sinus. Yamaguchi et al.<sup>25)</sup> reported in 2015 that complete obliteration of the dAVF with sufficient coagulation on both sides of the falx is one option.

Intracranial dAVFs contain a diverse group of vascular malformations that display distinct pathophysiology, symptomatology, angioarchitecture, natural history, and treatment options.<sup>40)</sup> The presence of cortical venous drainage (CVD) is a warning sign that indicates a high risk of adverse neurological outcomes, although several classification schemes exist to stratify hemorrhage and ischemic neurological deficits risk.<sup>40)</sup> Recently, patients with dAVFs and CVD presenting with aggressive symptoms have been demonstrated to be at high risk for ICH and neurological deficits, especially if venous ectasia is present.<sup>41)</sup> Nevertheless, the etiology and pathogenesis of these lesions remain poorly understood, and their clinical presentation can be unpredictable and unspecific. The management of intracranial dAVFs can incorporate a range of conservative, surgical, endovascular, and/or radio-surgical options. Surgery may become less frequently used in the treatment of intracranial dAVFs with the emergence of endovascular therapies.<sup>12)</sup> A thorough understanding of the fistula's angioarchitecture is required before undertaking any intervention. This necessitates identifying the feeding arteries, fistulous point, venous drainage pathways, and the direction of venous flow. Endovascular approaches can be obtained from arterial, venous, or combined pathways, depending on the fistula's location and anatomy. This review focused on the diagnosis and management of rare types of dAVFs, such as convexity dAVFs, parasagittal AVFs, and falcine dAVFs. As these dAVFs are uncommon, their treatments must be considered on an individual basis.

## Disclosure Statement

The authors declare that they have no conflicts of interest.

## References

- 1) Davies MA, Saleh J, Ter Brugge K, et al. The natural history and management of intracranial dural arteriovenous fistulae. Part 1: benign lesions. *Interv Neuroradiol* 1997; 3: 295–302.
- 2) Brown RD Jr., Wiebers DO, Nichols DA. Intracranial dural arteriovenous fistulae: angiographic predictors of intracranial hemorrhage and clinical outcome in nonsurgical patients. *J Neurosurg* 1994; 81: 531–538.
- 3) Davies MA, Ter Brugge K, Willinsky R, et al. The natural history and management of intracranial dural arteriovenous fistulae. Part 2: aggressive lesions. *Interv Neuroradiol* 1997; 3: 303–311.
- 4) Liu JK, Dogan A, Ellegala DB, et al. The role of surgery for high-grade intracranial dural arteriovenous fistulas: importance of obliteration of venous outflow. *J Neurosurg* 2009; 110: 913–920.
- 5) Gandhi D, Chen J, Pearl M, et al. Intracranial dural arteriovenous fistulas: classification, imaging findings, and treatment. *AJNR Am J Neuroradiol* 2012; 33: 1007–1013.
- 6) Huang L, Ge L, Lu G, et al. Correlation of aggressive intracranial lesions and venous reflux patterns in dural arteriovenous fistulas. *World Neurosurg* 2017; 107: 130–136.
- 7) Takai K, Kin T, Oyama H, et al. Three-dimensional angioarchitecture of spinal dural arteriovenous fistulas, with special reference to the intradural retrograde venous drainage system. *J Neurosurg Spine* 2013; 18: 398–408.
- 8) D'Aliberti G, Talamonti G, Boeris D, et al. Intracranial dural arteriovenous fistulas: the sinus and non-sinus concept. *Acta Neurochir Suppl* 2021; 132: 113–122.
- 9) Kawaguchi T, Nakatani M, Kawano T. Study of dural arteriovenous fistula drains into leptomeningeal vein without sinus interposition. *Interv Neuroradiol*. 2004; 10 Suppl 1(Suppl 1): 127–134.
- 10) Kiyosue H, Hori Y, Okahara M, et al. Treatment of intracranial dural arteriovenous fistulas: current strategies based on location and hemodynamics, and alternative techniques of transcatheter embolization. *Radiographics* 2004; 24: 1637–1653.
- 11) Kim B, Jeon P, Kim K, et al. Predictive factors for response of intracranial dural arteriovenous fistulas to transarterial onyx embolization: angiographic subgroup analysis of treatment outcomes. *World Neurosurg* 2016; 88: 609–618.
- 12) Kuwayama N, Akioka N. Complications of endovascular treatment of intracranial dural arteriovenous fistulas. *Acta Neurochir Suppl* 2021; 132: 123–127.
- 13) Baltsavias G, Parthasarathi V, Aydin E, et al. Cranial dural arteriovenous shunts. Part 1. Anatomy and embryology of the bridging and emissary veins. *Neurosurg Rev* 2015; 38: 253–263; discussion, 263–264.
- 14) Baltsavias G, Spiessberger A, Hothorn T, et al. Cranial dural arteriovenous shunts. Part 4. Clinical presentation of the shunts with leptomeningeal venous drainage. *Neurosurg Rev* 2015; 38: 283–291; discussion, 291.
- 15) Shapiro M, Srivatanakul K, Raz E, et al. Dural venous channels: hidden in plain sight—reassessment of an under-recognized entity. *AJNR Am J Neuroradiol* 2020; 41: 1434–1440.

- 16) Baltasvias G, Kumar R, Avinash KM, et al. Cranial dural arteriovenous shunts. Part 2. The shunts of the bridging veins and leptomeningeal venous drainage. *Neurosurg Rev* 2015; 38: 265–271; discussion, 272.
- 17) Kobayashi E, Wakamatsu K, Tominaga S. A case of dural arteriovenous malformation on the convexity adjacent to the superior sagittal sinus. *No Shinkei Geka* 1994; 22: 643–648 (in Japanese).
- 18) Brinjikji W, Cloft HJ, Lanzino G. Clinical, angiographic, and treatment characteristics of cranial dural arteriovenous fistulas with pial arterial supply. *J Neurointerv Surg* 2021; 13: 331–335.
- 19) Hetts SW, Yen A, Cooke DL, et al. Pial artery supply as an anatomic risk factor for ischemic stroke in the treatment of intracranial dural arteriovenous fistulas. *AJNR Am J Neuro-radiol* 2017; 38: 2315–2320.
- 20) Osada T, Krings T. Intracranial dural arteriovenous fistulas with pial arterial supply. *Neurosurgery* 2019; 84: 104–115.
- 21) Korai M, Enomoto N, Satoh K, et al. Transarterial embolization for convexity dural arteriovenous fistula with or without pial arterial supply: a report of four patients. *Surg Neurol Int* 2022; 13: 340.
- 22) Akamatsu Y, Gomez-Paz S, Tonetti DA, et al. Middle meningeal artery: an effective pathway for achieving complete obliteration following transarterial ethylene vinyl copolymer (Onyx) embolization of dural arteriovenous fistulas. *J Cerebrovasc Endovasc Neurosurg* 2022; 24: 210–220.
- 23) Uda K, Izumi T, Kanamori F, et al. Coexistence of a dural arteriovenous fistula and pial arteriovenous malformation sharing a common drainer. *NMC Case Rep J* 2021; 8: 557–563.
- 24) Yoshioka S, Moroi J, Kobayashi S, et al. A case of falcine sinus dural arteriovenous fistula. *Neurosurgery* 2013; 73: E554–E556.
- 25) Yamaguchi T, Higaki A, Yokota H, et al. A case of dural arteriovenous fistula in the falx cerebri: case report and review of the literature. *NMC Case Rep J* 2016; 3: 67–70.
- 26) Cloft HJ, Kallmes DF, Jensen JE, et al. Percutaneous transvenous coil embolization of a type 4 sagittal sinus dural arteriovenous fistula: case report. *Neurosurgery* 1997; 41: 1191–1193; discussion, 1193–1194.
- 27) Hou K, Ji T, Guo Y, et al. Current status of endovascular treatment for dural arteriovenous fistulas in the superior sagittal sinus region: a systematic review of the literature. *World Neurosurg* 2019; 122: 133–143.
- 28) Hayashi K, Ohmori Y, Kaku Y, et al. Non-sinus-type dural arteriovenous fistula cured by transarterial embolization from middle meningeal artery: two case reports. *J Neuro-endovasc Ther* 2018; 12: 542–545.
- 29) Kawaguchi T, Kawano T, Kaneko Y, et al. Transarterial embolization with HEMA-MMA of variant convexity-superior sagittal sinus dural arteriovenous fistula—case report. *Neurol Med Chir (Tokyo)* 2000; 40: 366–368.
- 30) Wang SS, Li CH, Zhang XJ, et al. Investigation of the mechanism of dural arteriovenous fistula formation induced by high intracranial venous pressure in a rabbit model. *BMC Neurosci* 2014; 15: 101.
- 31) Yoshioka T, Kitagawa N, Yokoyama H, Nagata I. Selective transvenous coil embolization of dural arteriovenous fistula. A report of three cases. *Interv Neuroradiol*. 2007; 13 Suppl 1(Suppl 1): 123–130.
- 32) Bhatia KD, Kortman H, Walchli T, et al. Artery of Davidoff and Schechter supply in dural arteriovenous fistulas. *AJNR Am J Neuroradiol* 2020; 41: 300–304.
- 33) Tubbs RS, Loukas M, Louis RG Jr., et al. Anatomy of the falcine venous plexus. *J Neurosurg* 2007; 107: 155–157.
- 34) Satoh D, Sasaki T, Yako T, et al. A case of dural arteriovenous fistula in the falx with prominent falcine venous plexus. *J Neuroendovasc Ther*. 2021; 15: 444–448.
- 35) Sener RN. Association of persistent falcine sinus with different clinicoradiologic conditions: MR imaging and MR angiography. *Comput Med Imaging Graph* 2000; 24: 343–348.
- 36) Kesava PP. Recanalization of the falcine sinus after venous sinus thrombosis. *AJNR Am J Neuroradiol* 1996; 17: 1646–1648.
- 37) Strub WM, Leach JL, Tomsick TA. Persistent falcine sinus in an adult: demonstration by MR venography. *AJNR Am J Neuroradiol* 2005; 26: 750–751.
- 38) Ryu CW. Persistent falcine sinus: is it really rare? *AJNR Am J Neuroradiol* 2010; 31: 367–369.
- 39) Tatarlı N, Ceylan D, Canaz H, et al. Falcine venous plexus within the falx cerebri: anatomical and scanning electron microscopic findings and clinical significance. *Acta Neurochir (Wien)* 2013; 155: 2183–2189; discussion, 2189.
- 40) Reynolds MR, Lanzino G, Zipfel GJ. Intracranial dural arteriovenous fistulae. *Stroke* 2017; 48: 1424–1431.
- 41) Gross BA, Du R. The natural history of cerebral dural arteriovenous fistulae. *Neurosurgery* 2012; 71: 594–602; discussion, 602–603.