Endovascular Management of Falcine Dural Arteriovenous Fistula—A Case Report and Review of Literature

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Abstract
Cranial dural arteriovenous (AV) fistulas are abnormal connections between the branches of dural arteries to dural veins or venous sinuses. They are most frequently located at the transverse sinus and cavernous sinus. They can occur at every cranial dural sinus. Dural AV fistula of falx cerebri is rare. A 62-year-old female presented with signs and symptoms of raised intracranial pressure. Radiological imaging revealed a dural AV fistula at the posterior one-third falx cerebri. She underwent transarterial embolization, and complete obliteration of the fistula was achieved. A detailed digital subtraction angiography study is warranted in patients with seemingly benign complaints like recurrent headaches, and falcine dural AV fistula should be identified and treated in the nick of time. We describe a very rare falcine dural AV fistula case and its management.

Keywords ► dural AV fistula ► falx cerebri ► falcine sinus ► Onyx embolization

Introduction
Cranial dural arteriovenous fistulas (AVF) are the abnormal connection between the branches of dural arteries to dural veins or venous sinuses. They represent about 10 to 15% of intracranial vascular malformations.1 These lesions are most commonly located in the posterior fossa or around the cavernous sinus. The uncommon location includes the sagittal sinus, anterior cranial fossa, and craniocervical junction.2 Dural AVF located at the falx cerebri is very rare, and to date, only six cases have been reported (►Table 1).3–8 We report a rare and interesting case of falcine dural AVF and describe its management.

Case Report
A 62-year-old female patient was referred to manage signs and symptoms of raised intracranial pressure. Earlier, she had headaches, vomiting, and gait ataxia for the last 2 weeks, requiring hospitalization. Her symptoms improved with antiedema measures, so she was discharged. Her symptoms resurfaced after 10 days, and she was readmitted. She was conscious and oriented and had bilateral papilloedema grade II. Her computed tomography (CT) scan of the brain revealed abnormal small hyperdense lesions around the left peri mesencephalic cistern with contrast enhancement (►Fig. 1A, B). Magnetic resonance imaging (MRI) of the brain

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showed flow voids on T2-weighted images (►Fig. 1C). MR venogram of the brain showed nonvisualization of the inferior sagittal sinus and straight sinus (►Fig. 1D). Digital subtraction angiography (DSA) study of brain and neck vessels revealed a posterior falcine dural AVF, getting supplied from the posterior branch of the middle meningeal artery, a branch of the occipital artery on both sides, and meningeal branch from the left vertebral artery. This resulted in significant cortical venous reflux in the infratentorial compartment and drained into the superior sagittal sinus at its anterior end through basifrontal veins (►Fig. 2A–H).

The patient underwent embolization of the posterior falcine dural AVF (Cognard Grade IV). Under general anesthesia, a 6 French (F) arterial sheath was placed in the right femoral artery. About 7,000 units of heparin were given, and Activated Coagulation Time (ACT) was maintained at around 250 to 300 seconds throughout the procedure. With the help of a 5F angiography catheter, right external carotid angiography was performed. Under roadmap guidance, a 6F guiding catheter was replaced with a 6F Envoy (Cerenovus, Johnson and Johnson Mexico) guiding catheter and positioned into the distal external carotid artery. Through 6F guiding catheter, Sonic 1.2 F (Balt, Montmorency, France) microcatheter (167 cm length with 15 mm detachable tip) advanced over 0.08″ Mirage (Balt, Montmorency, France) microwire under digital roadmap guidance. The microcatheter tip was placed into the distal part of the posterior branch of the middle meningeal artery close to the mouth of the fistula. A super selective run was taken through the microcatheter (►Fig. 3A, B). The dead-space volume within the microcatheter was first filled with dimethyl-sulfoxide. Subsequently, under blank roadmap guidance, about 0.8 mL liquid embolic agent, Onyx (Medtronic), was injected very slowly in the fistula, and it was obliterated. The guide catheter and microcatheter were removed. An adequate glue penetration into the fistula was confirmed on fluoroscopic images (►Fig. 3C, D). Post Onyx injection angiography runs through the right external carotid artery, left external carotid artery, and left vertebral artery and revealed no filling of the fistula (►Fig. 4A–G). The postoperative course was uneventful. Her clinical symptoms significantly improved.

### Table 1: Reported cases of falcine dural AVF

<table>
<thead>
<tr>
<th>Sl. no</th>
<th>Authors</th>
<th>Age/Sex</th>
<th>Arterial feeder</th>
<th>Location</th>
<th>Venous drainage</th>
<th>Location</th>
<th>VP</th>
<th>CVR</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Kosnik et al 1974</td>
<td>34/F</td>
<td>Headache</td>
<td>Anterior</td>
<td>ACA, AFA</td>
<td>ISS</td>
<td>No</td>
<td>No</td>
<td>DS</td>
<td>Excellent</td>
</tr>
<tr>
<td>2</td>
<td>Agawa et al 1991</td>
<td>62/M</td>
<td>SAH</td>
<td>Posterior</td>
<td>MMA, AFA</td>
<td>FS, SSS</td>
<td>Yes</td>
<td>Yes</td>
<td>DS</td>
<td>Excellent</td>
</tr>
<tr>
<td>3</td>
<td>Kothbauer and Huber 1994</td>
<td>73/F</td>
<td>SAH</td>
<td>Posterior</td>
<td>MMA, OA</td>
<td>PPV, GV</td>
<td>Yes</td>
<td>Yes</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>4</td>
<td>Ratliff and Voorhies 1999</td>
<td>24/M</td>
<td>Headache</td>
<td>Anterior</td>
<td>ACA, VA</td>
<td>SSS, PPV GV</td>
<td>Yes</td>
<td>Yes</td>
<td>DS + RA</td>
<td>Excellent</td>
</tr>
<tr>
<td>5</td>
<td>Yoshioka et al 2013</td>
<td>60/M</td>
<td>Incidental</td>
<td>Anterior</td>
<td>ACA, MMA, AEA</td>
<td>FS, SSS/ISS</td>
<td>No</td>
<td>Yes</td>
<td>DS</td>
<td>Excellent</td>
</tr>
<tr>
<td>6</td>
<td>Yamaguchi et al 2016</td>
<td>67/M</td>
<td>Altered sensorium</td>
<td>Anterior</td>
<td>MMA, AFA and left pericallosal artery</td>
<td>SSS, posterior callosal vein, Vein of Galen</td>
<td>Yes</td>
<td>Yes</td>
<td>TAE + DS</td>
<td>Fair</td>
</tr>
<tr>
<td>7</td>
<td>Present case</td>
<td>62/F</td>
<td>Headache, gait ataxia</td>
<td>Posterior</td>
<td>MMA, VA</td>
<td>SSS</td>
<td>Yes</td>
<td>Yes</td>
<td>TAE</td>
<td>Excellent</td>
</tr>
</tbody>
</table>

Abbreviations: ACA, anterior cerebral artery; AEA, anterior ethmoidal artery; AFA, anterior falcine artery; CVR, cortical venous reflux; DS, direct surgery; F, female; FS, falcine sinus; GV, Galenic vein; ICH, intracranial hemorrhage; ISS, inferior sagittal sinus; M, male; MMA, middle meningeal artery; NA, not available; OA, occipital artery; PPV, posterior pericallosal vein; RA, radiosurgery; RVD, retrograde venous drainage; SAH, subarachnoid hemorrhage; SSS, superior sagittal sinus; TAE, transarterial embolization; VA, vertebral artery; VP, venous pouch.

**Fig. 1** Computed tomography scan brain plain study showing abnormal small hyperdense lesion around left peri mesencephalic cistern (A, white arrow) which is enhancing on intravenous contrast administration (B, white arrow). Magnetic resonance imaging brain coronal T2-weighted images showing flow void located at the lateral aspect of the left side of the mid brain in relation to tentorium cerebelli (C, white arrow). Magnetic resonance venogram of the brain showing non-visualization of inferior sagittal sinus and straight sinus (D).
improved, and she was discharged on the 4th postprocedure day. At 1-year follow-up, she was asymptomatic.

**Discussion**

Dural venous sinuses arise from the mesenchyme located in the mesencephalic flexure. During the growth of the embryo, the sagittal plexus is seen in the falk cerebri with two ends. Anterior superior sagittal sinus (SSS) arises from its dorsal end, and inferior sagittal sinus and straight sinus arise from its ventral end. Complete development of the occipital lobe results in the formation of the entire superior sagittal and straight sinus. Falcine sinus is formed by one of the caudal anastomotic loops of the sagittal plexus and disappears before birth after the complete development of SSS and straight sinus. Persistence of it can be a congenital or acquired phenomenon related to an abnormality of the straight sinus. According to an MRI-based study, persistent falcine sinus incidence is about 1% in pediatric patients and about 5.3% in a combined population. The incidence of recanalization of falcine sinuses is about 30%, and the etiological factors described are compression by tumor, cerebral venous sinus thrombosis, and hypertrophic meningitis. They demonstrate a strong association between falcine sinus recanalization and obliteration of major sinuses. A review of the literature revealed the rarity of falcine dural AVF. Only seven cases have been reported, including our case.

There are no outstanding characteristics about age, gender, or symptoms at onset. Variable symptoms can be observed in these patients, ranging from severe refractory headache, which can be due to raised intracranial pressure or subarachnoid and intraventricular hemorrhage. The fistula having direct cortical venous drainage is more prone to present with intracerebral hematoma.

Persistent or recanalization of falcine sinus results in vascular variation or anomalies. It can be easily missed on routine CT scans and MRI brain images, including CT- and MRI-based angiography. Unless found with secondary insults
like subarachnoid or intraparenchymal or intraventricular hemorrhage, these lesions are difficult to diagnose and often not sought after. MR or CT venogram studies in patients with features of raised intracranial pressure are instrumental in providing subtle hints of this uncommon pathology. Subsequently, they should be subjected to DSA. Patients presenting with hemorrhages should be directly subjected to DSA, which is the gold standard investigation for diagnosing this pathology, delineating its anatomy, and chalk out further management plans. It shows the exact morphology of the fistula, including its location, arterial supply, venous drainage, direction of the blood flow, and associated vascular abnormality. In view of significant cortical venous reflux and risk of hemorrhage, all the patients require treatment as early as possible.

Treatment options are endovascular, microsurgery, radiosurgery, or combination. While the endovascular procedure aims to inject glue in the fistula selectively, microsurgery aims to disconnect the arterial supply and excision the fistula in the falx. In patients with secondary insults like a large intraparenchymal hemorrhage, a decompressive surgery is warranted in patients with compromised consciousness. It serves the purpose of clot evacuation, and one can aim to obliterate the fistulous points. Interhemispheric approaches are often used to target their obliteration. Most previous cases were subjected to direct surgical obliteration of the fistulous point. The more recent form of their management is the transarterial embolization of the feeders. Only transarterial embolization has never been attempted for this pathology. Yamaguchi et al initially subjected their patient to partial transarterial embolization and was subsequently subjected to surgical occlusion. Ratliff and Voorhies patient had a similar presentation to our patient, and they managed it with direct surgical obliteration followed by subjecting him to radiosurgery. Ours was the first case where a complete obliteration was achieved with transarterial embolization.

**Conclusion**

One should consider DSA for patients with raised intracranial pressure with evidence of vascular channels around the falx and peri mesencephalic cistern. Awareness of the falcal sinus and falcal venous plexus is crucial in managing these rare forms of dural AVF. Through various treatment options, transarterial Onyx embolization can completely cure the fistula.

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**Conflict of Interest**

None declared.
References