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Case Report

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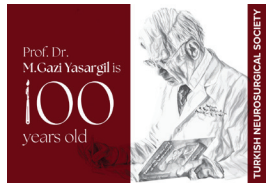

Bilateral Thalamic Edema Caused by Tentorial Galenic Dural Arteriovenous Fistula and Sinus Thrombosis: Successful Endovascular Therapy

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ABSTRACT

Bilateral thalamic edema is commonly caused by vascular, toxic/metabolic, neoplastic, and infectious factors. However, dural arteriovenous fistulas (DAVFs) are a relatively rare and often overlooked cause, with an incidence rate of about 8%. Tentorial dural arteriovenous fistulas (TDAVFs) represent a rare subtype. Cerebral angiography often shows TDAVFs with reflux into cortical or subarachnoid veins and retrograde deep drainage through the vein of Galen, which is associated with a high risk of hemorrhage—97% of cases involve hemorrhage and exhibit aggressive neurological behavior. Venous sinus thrombosis, high-flow arteriovenous malformations, or a combination of both can result in venous hypertension, leading to bilateral thalamic dysfunction. The arterial supply to TDAVFs is complex, involving meningeal arteries from the vertebral and internal carotid arteries, which are difficult to cannulate, increasing the risk of complications due to retrograde embolic flow compared to external carotid artery (ECA) feeders. Transvenous navigation to deep lesions around the tentorium is also challenging. Additionally, TDAVFs often drain into subarachnoid or cortical veins rather than their associated sinus (Borden Type III), making transvenous embolization impossible. The middle meningeal artery, which supplies more than two-thirds of the cranial dura, is the primary dural feeder. In this article, we presented a unique case of symptomatic bilateral thalamic edema caused by both a tentorial galenic DAVF and straight sinus thrombosis of the cerebral deep venous system, and we detailed our treatment approach and experience.

KEYWORDS: Tentorial dural arteriovenous fistula, TDAVF, Thalamic edema, Sinus thrombosis, Endovascular treatment

INTRODUCTION

Intrantracranial dural arteriovenous fistulas (DAVFs) account for approximately 10% to 15% of all intracranial arteriovenous malformations. They commonly occur in areas such as the cavernous sinus, transverse sinus, sigmoid sinus, and sagittal sinus. These fistulas typically form on the walls of the venous sinuses and are often multiple (1,9). Tentorial dural arteriovenous fistulas (TDAVFs) are a rare subtype of DAVFs, with an incidence rate of about 12% (12). They are generally considered to have the most life-threatening vascular structures, with an aggressive prognosis and natural history. Bilateral thalamic edema can have many causes, but DAVFs are a relatively uncommon and under-recognized cause, with an incidence rate of about 8% (6). Here, we presented a patient with bilateral thalamic edema caused by a TDAVF, successfully treated with transarterial embolization. The patient's deep venous drainage remained intact, and their cognitive function significantly improved over a few weeks.

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CASE REPORT

A 67-year-old healthy man was brought to the emergency department by his son, presenting with mental status changes after one month of progressively increasing somnolence and confusion. He had no focal neurological deficits and could follow simple instructions, but was unable to follow more complex commands. A subsequent magnetic resonance imaging (MRI) scan showed bilateral thalamic and left parietal lobe hyperintensity with minimal mass effect. The scan revealed a "butterfly" appearance, suggesting a lesion crossing the midline through the inter-thalamic adhesion (Figure 1). Magnetic resonance perfusion (MRP) showed that the lesion in the left occipital lobe was lower and longer than the contralateral side. Regional cerebral blood flow (rCBF) and regional cerebral blood volume (rCBV) were decreased, and mean transit time (MTT) was prolonged in both thalami. A preoperative sagittal magnetic resonance venography (MRV) scan demonstrated chronic thrombosis of the straight sinus and a tentorial galenic DAVF (Figure 2). Further angiography confirmed a TDAVF, with supply from branches of the right occipital artery and retrograde venous drainage into the vein of Galen, internal cerebral veins, and the right parietal cortical veins. No filling of the straight sinus was observed, consistent with thrombosis (Figure 3). Given that the middle meningeal artery is the primary dural artery, supplying more than two-thirds of the cranial dura(12), the fistula was embolized using Onyx (eV3) via a transarterial approach (Figure 4). Deep venous drainage remained intact, and the patient's cognitive abilities significantly improved within a few weeks (Figure 1-D).

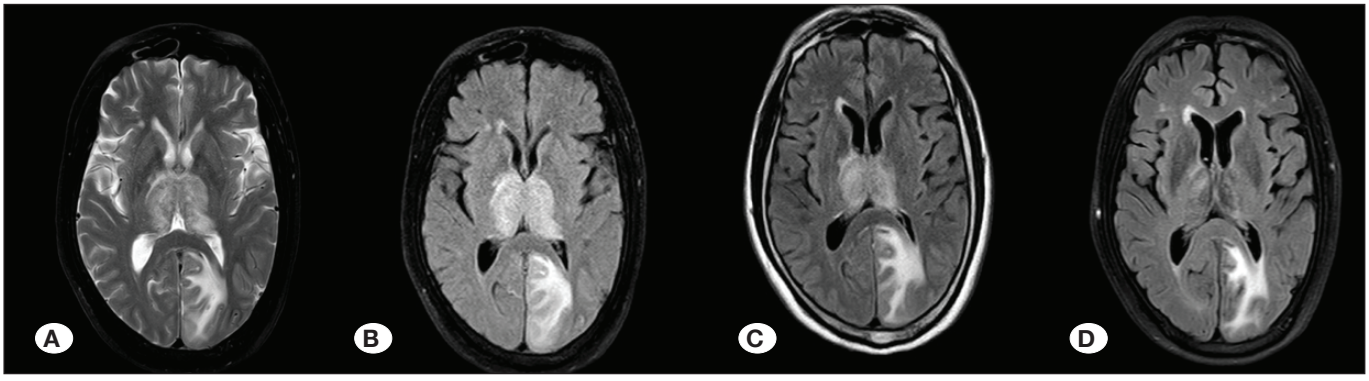

Figure 1: MRI **A)** T2-weighted axial and **B)** FLAIR demonstrating bilateral thalamic and left parietal lobe hyperintensity with minimal mass effect. Note the "butterfly" appearance suggesting a lesion crossing the midline through the interthalamic adhesion. **C)** FLAIR demonstrating bilateral thalamic and left parietal lobe hyperintensity reducing after postoperative one day. **D)** Images obtained 3 weeks after endovascular embolization of DAVF. FLAIR MRI showing resolution of bi-thalamic hyperintensities.

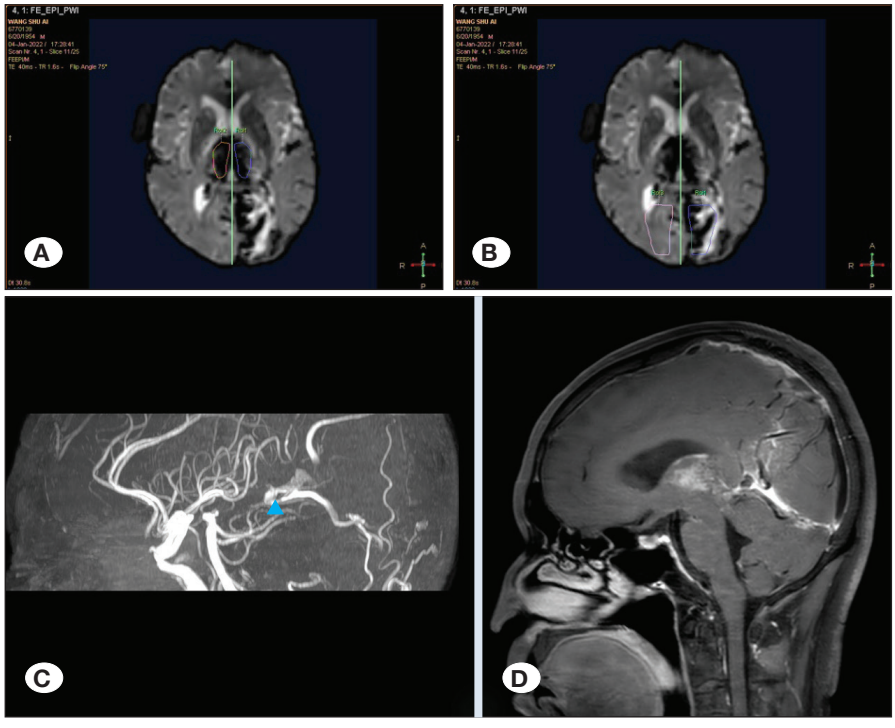

Figure 2: MRP (magnetic resonance perfusion weighted imaging) **A)** The rCBF, rCBV and MTT in the lesion area of left occipital lobe were lower and longer than contralateral. **B)** The rCBF, rCBV decreased and MTT prolonged in bilateral thalamus. **C, D)** Preoperative MRVs demonstrating occlusion of the straight sinus and Tentorial Galenic dural Arteriovenous Fistula (blue arrowhead).

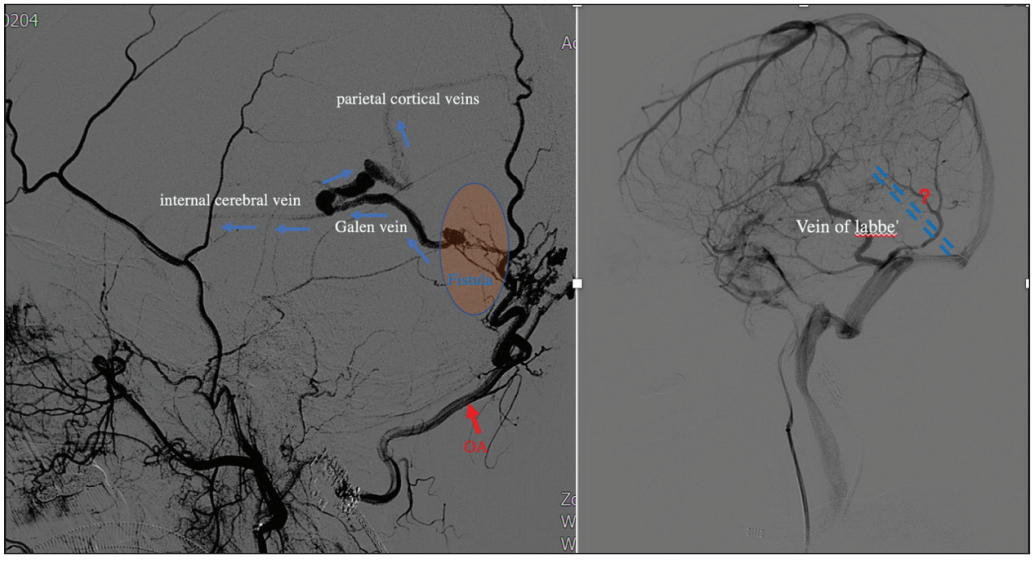

Figure 3: Right external carotid artery angiography shows a tentorial galenic DAVF (Borden type III,Cognard type III) with supply from the branches of the right occipital artery (OA red arrow), with retrograde venous drainage into the vein of Galen and internal cerebral veins as well as into the right parietal cortical veins. No filling of distal the straight sinus (dashed lines) was noted, consistent with thrombosis.

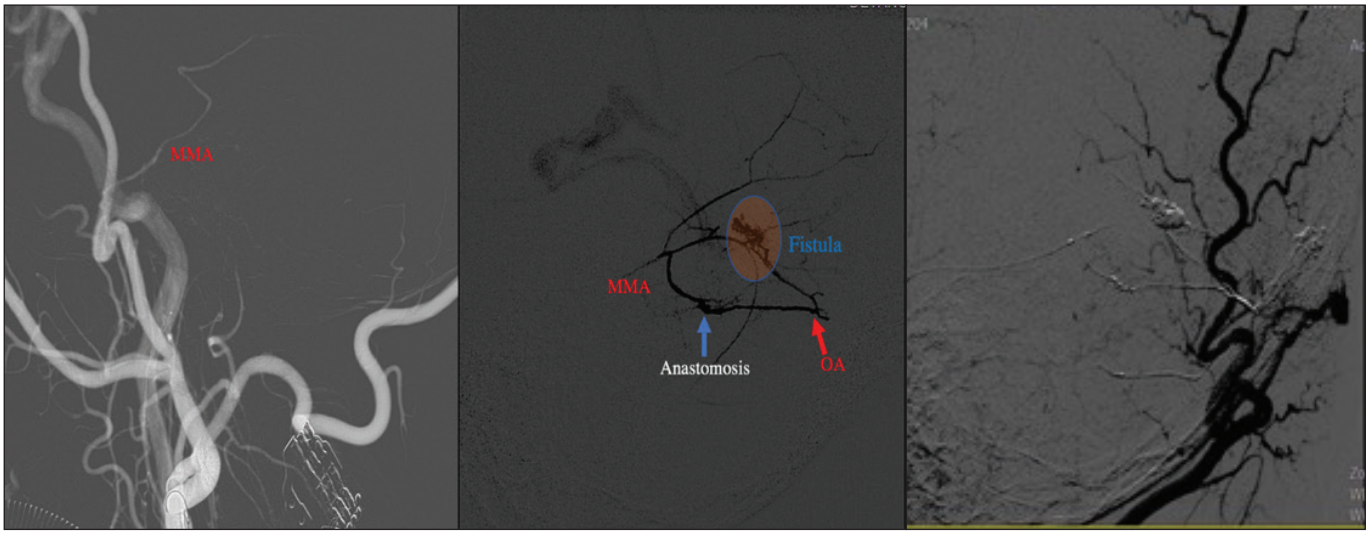

Figure 4: During the operation, Right middle meningeal angiography shows the middle meningeal artery and he branches of the right occipital artery exists collateral anastomoses (blue arrow). Selective angiogram of right external carotid artery demonstrates complete obliteration of the fistula.

DISCUSSION

Diseases affecting the thalamus are broad in their etiologies, including infectious, inflammatory, neoplastic, toxic/metabolic, and vascular causes, such as arterial cerebral infarction, venous thrombosis, arteriovenous malformations, and atypical posterior reversible encephalopathy syndrome (PRES). Typically, DAVFs are not commonly listed in the classic radiological differential diagnosis for thalamic lesions (5,17). DAVFs are relatively rare, with an incidence rate of about 8%, and are an often under-recognized cause of thalamic edema. The natural history, clinical presentation, and prognosis of TDAVFs are closely related to their venous drainage patterns (7,15). Clinically, Borden types IIa+b and IIITDAVFs are more frequently encountered, indicating retrograde leptomeningeal venous drainage, with or without sinus drainage (16,19). In our case, the patient had a DAVF involving the cortical veins of the right parietal cortex, the vein of Galen, and the straight sinus. Due to prolonged venous hypertension, patients with TDAVFs often present with progressive neurological deficits and/or intracranial hemorrhage (16,19). Therefore, there is a strong indication for treatment to prevent catastrophic complications (1). Neuroimaging is crucial for diagnosing TDAVFs. Plain computed tomography (CT) scans can rapidly identify subarachnoid or intraventricular hemorrhage, and three-dimensional CT reconstruction can reveal varicose draining veins. MRI can visualize the tentorial subarachnoid space, bulging at the tentorial edge, and distorted vascular flow voids, often indicating venous congestion and tumor-like changes. On T2-weighted images, the thalamus, midbrain, cerebellum, and high cervical spinal cord may show high-signal changes due to secondary edema from venous hypertension (4). However, these imaging

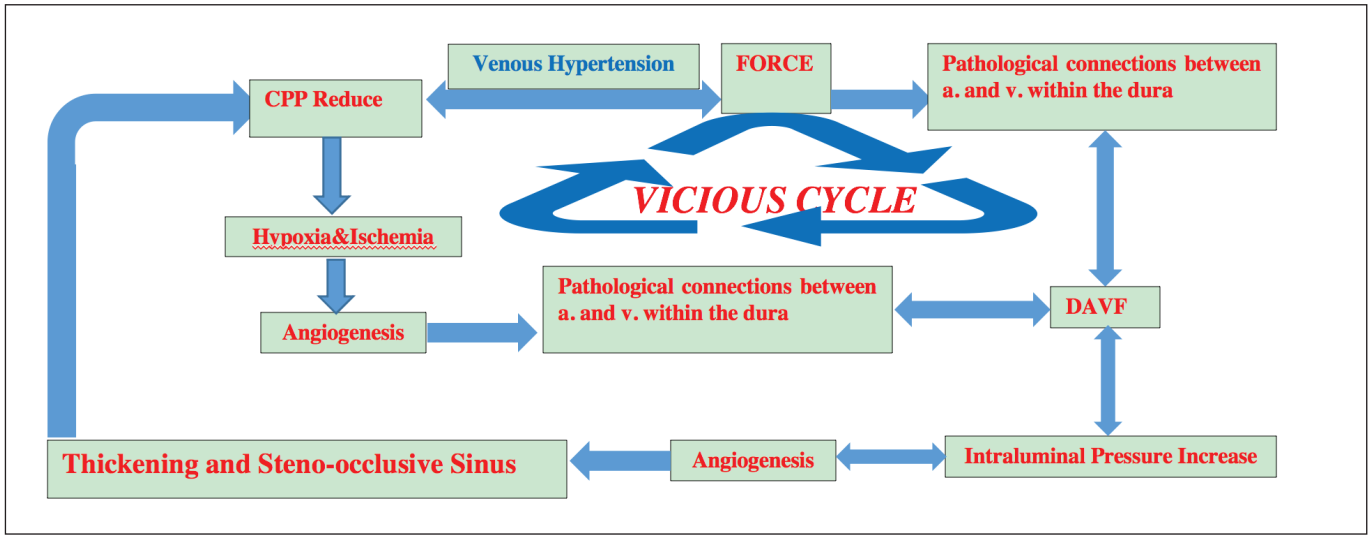

Figure 5: The "Vicious Circle" mechanism of dural arteriovenous fistula.

findings lack specificity and can be confused with intracranial demyelinating lesions, tumors, or inflammation. Angiography remains the gold standard for diagnosis. The feeding arteries and draining veins of TDAVFs are complex and often originate from six different sources: the artery of Bernasconi-Cassinari (11), the middle meningeal artery, the posterior meningeal artery, the artery of Davidoff-Schechter (2), scalp arteries, and miscellaneous branches of the external carotid artery (12). The venous drainage of the thalamus typically occurs via the cerebral deep venous system, with the anterior septal vein and thalamostriate vein draining into the internal cerebral vein, which joins the basal vein of Rosenthal to form the vein of Galen. The vein of Galen and inferior sagittal sinus unite to form the straight sinus, which drains into the torcular Herophili (14). This complex angioarchitecture often leads to progressive cognitive dysfunction, aphasia, ataxia, and focal neurological deficits (6,8,10,16). Given the complexity of TDAVFs, selective angiography should be performed on both internal and external carotid arteries, as well as the vertebral arteries, to identify all potential feeding routes and allow for more treatment options. In our case, conventional angiography did not initially show the feeding arteries of the middle meningeal artery. Based on the theory of anastomoses between meningeal arteries at the skull base, we performed selective angiography on the right middle meningeal artery, which revealed collateral anastomoses between the middle meningeal artery and branches of the right occipital artery. This case highlighted a successful endovascular approach for TDAVF, using the middle meningeal artery, which passes through the foramen spinosum. The fistula was super-selectively catheterized and successfully embolized via a transarterial approach. Anticoagulation is typically the primary treatment for sinus thrombosis (18). In this case, however, anticoagulation alone was not considered due to the "vicious cycle" mechanism of DAVF (Figure 5). Abnormal blood flow through the fistula drains via the pial veins, predominantly flowing retrograde into the straight sagittal sinus through the dilated parietal cortical veins at the tentorial incisura, with some flow directed into the internal cerebral vein. High-flow arterial blood enters the thin-walled cortical veins, causing passive dilation and high tension due to turbulent flow. This, along with turbulence at the junction of draining veins and venous sinuses, predisposes the area to thrombosis formation. Thrombosis further increases pressure in the proximal draining veins, leading to morphological adaptations such as tortuosity, venous bulbs, or venous lakes to compensate for the high pressure. Additionally, the high-pressure state triggers an inflammatory response that stimulates angiogenic and epidermal growth factors, resulting in further thickening and arterialization of the veins. This vicious cycle significantly increases the risk of rupture and hemorrhage in TDAVFs compared to other DAVFs, where abnormal blood flow enters the venous sinus directly. Because of the inherent risk of DAVF rupture and the limited likelihood of symptom improvement without intervention, we opted for endovascular treatment rather than anticoagulation alone. Furthermore, intracranial DAVFs combined with cerebral vein thrombosis were not typically associated with a worse prognosis (3).

CONCLUSION

Encouragingly, we have illustrated the potential formation mechanism of the DAVF, and the TDAVF was successfully embolized through the optimal channel of the middle meningeal artery ("All roads lead to Fistula"). Based on the anastomotic theory between the meningeal arteries at the skull base, this case demonstrates an excellent endovascular approach for TDAVF. The middle meningeal artery, emerging from the foramen spinosum, was super-selectively catheterized via angiography, allowing precise identification of the fistula shunt and successful embolization of the TDAVF via a transarterial approach. This method ingeniously overcame the challenges posed by the deep anatomical location of the TDAVF, as well as the difficulties of microsurgical exposure and resection. Moreover, it minimized the risk of severe complications, such as embolic material retrogradely flowing into branches of the internal carotid artery and vertebral artery.

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Consent to publication: This patient gave his informed consent for the publication and medical records for the figures and video recordings.

Declarations

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Availability of data and materials: The datasets generated and/or analyzed during the current study are available from the corresponding author by reasonable request.

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AUTHORSHIP CONTRIBUTION

Study conception and design: XL, AL, YH, CW

Data collection: XL, LW, AL, YH, JY, CW

Analysis and interpretation of results: XL, YY, AL, YH, CW

Draft manuscript preparation: XL, YH, LW, AL

Critical revision of the article: XL, LW, AL, DL, JY

Other (study supervision, fundings, materials, etc...): XL, LW, YH, JL

All authors (XL, LW, YY, AL, YH, JY, XL, CW, JL) reviewed the results and approved the final version of the manuscript.

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