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Tentorial dural arteriovenous fistulae presenting as transient ischemic attack. Case illustration

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ABSTRACT

Dural arteriovenous fistulae are vascular lesions of the dura mater, usually acquired, consisting of abnormal connections between the dural arteries with the venous sinuses or the cortical veins. A case report presents a case with an unusual form of presentation of the dural (tentorial) arterio-venous fistula simulating a transient ischemic attack in a 60-year-old male patient.

Abbreviations

DAVFs: Dural arteriovenous fistulas, tDAVFs: tentorial Dural arteriovenous fistulas
MRI: Magnetic resonance Imaging, MRA: Magnetic resonance Arteriography
CT: Computerized Tomography.

INTRODUCTION

As described and characterized angiographically, dural arteriovenous fistulas (DAVFs) are lesions generally acquired and progressive with an

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incidence of 10 to 15% Of all cerebral vascular lesions (1-3). They are divided into two main types: DAVFs that drain into a venous sinus with direct reflux to a cortical vein, or not of the venous sinus type (3).

The first type includes those that drain into the cavernous sinus, the sigmoid-transverse sinus, the sinuses confluence, superior sagittal sinus, and petrous sinuses. The Second type, include tentorial, ethmoidal, cranio-cervical DAVFs (4-9).

The most frequent DAVFs is at the transverse-sigmoid junction (38%), and the cavernous sinus, followed by deep veins, the Superior sagittal sinus, superior petrous sinus, ethmoidal sinus, inferior petrous sinus and ethmoidal DAVFs (2,10). The etiology is controversial and the location of the DAVFs in very specific sites, and not throughout the dura mater.

Case report

A 60-year-old male patient came to the emergency department with the complaint of acute onset of slurred speech that started 15 minutes prior to his arrival. He denied any associated headache or visual, motor, balance, sensory symptoms, or any previous similar episode, and his family denied witnessing any loss of consciousness, abnormal body movement or odd behavior when inquired. Relevant history included hypertension for 10 years for which the patient was prescribed a Lisinopril 10 mg once daily taken regularly as the patient emphasized.

The initial evaluation demonstrated a conscious, oriented patient with a blood pressure of 130/85, afebrile, with dysarthria. Other aspects of the neurologic examination were normal. The routine investigation showed normal blood sugar, electro-cardiography showed sinus rhythm and native brain CT-scan.

Meanwhile, the patient reported complete resolution of his speech difficulty and requested if he can go home. His initial ABCD2 score was 3 (1 for age, 1 for his dysarthria & 1 for less than 60 minutes duration of symptoms), and was deemed low risk for recurrent TIA (Transient Ischemic Attack) or stroke within the next 48 hrs., kept on antiplatelet and statin tablets, sent for full stroke workup including cardiac telemetry, given MRI/MRA appointment and was discharged home.

36 hours later, he presented again with disorientation that soon evolved into a stupor with a blood pressure of 200/110 mm Hg, and was suspected to have suffered from a hemorrhagic stroke. Aspirin was stopped and urgent CT scan was done, it showed left tempo-occipital hematoma. Evaluated by neurosurgeon and neuro-endovascular specialist. MRI/MRA and CT- Angiography were performed (Figure. 1 and 2), suggested the presence of tentorial vascular malformation.

The next day, Cerebral Digital Subtraction Angiography was performed, showed the dural arteriovenous fistula of the tentorium. During the procedure, histoacryl embolization was performed without any complications (Figure. 3). The patient was transferred to an intensive care unit. He was discharged after 15 days without neurological deficit.

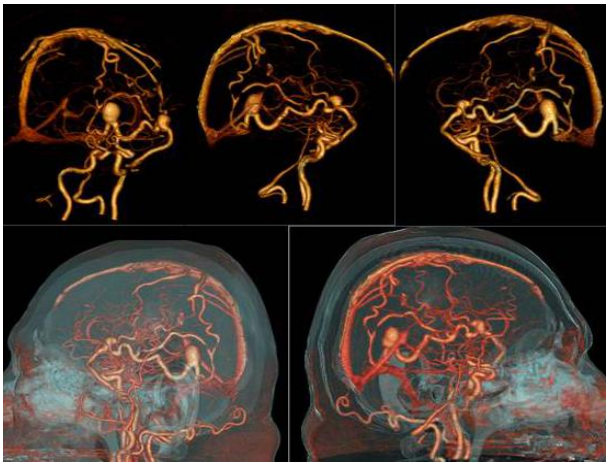

Figure 1. CT angiography 3D reconstruction study showed Tentorial DAVF, the fistulous connection was between the middle meningeal artery, tentorial branches of the meningohypophysial trunk of the Internal Carotid Artery with the transvers-sigmoid sinuses junction and the straight sinus, with venous varices that render the lesion as a high risk for haemorrhage DAVFs.

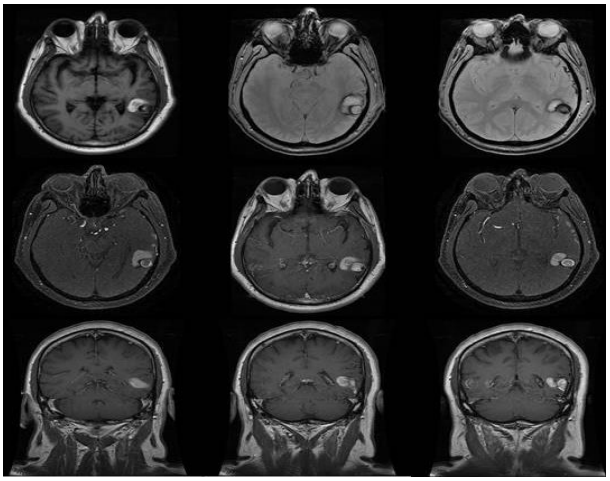

Figure 2. Cerebral MRI showing posterior left temporal intraparenchymal hematoma.

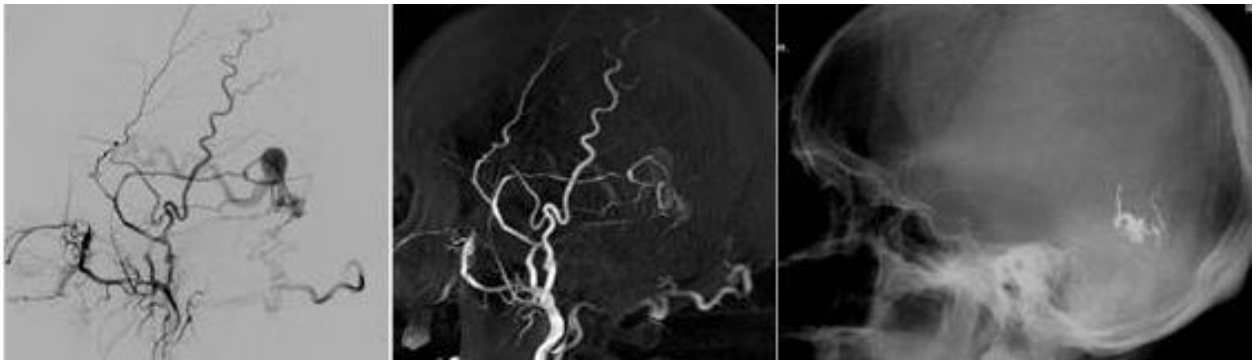

Figure 3. (**A, B**) Cerebral Digital Subtraction Angiography (DSA) of the left external carotid artery showing the medial tentorial DAVF supplied by the middle meningeal artery and drained into the transverse-sigmoid junction and the the straight sinus, with the venous varices. (**C**) Post embolization image showing the histoacryl used in relatively large amount due to multiple fistulous connections.

Discussion

Dural arteriovenous fistulas (DAVFs) are relatively rare, representing 10% to 15% of intracranial vascular malformations. Their usual locations are tentorium (26%), cavernous sinus (26%), transverse/sigmoid sinus (25%), convexity and superior sagittal sinus (11%), and frontal cranial vault (9%). The tentorial dural arteriovenous fistulas (tDAVF) clinically present with hemorrhage, with reported rates from 58% to 92%, and neurological deficits in 79% to 92% of patients. The pathophysiology is retrograde leptomeningeal venous drainage with ensuing venous congestion (1, 2, 11-13).

Common presentations include intracranial hemorrhage, urinary incontinence, paresis, and sensory loss affecting the extremities (14,15). Transarterial endovascular treatment of DAVFs showed symptomatic improvement in about 78% of the cases, with a complication rate of 5% (16).

In our patient, digital subtraction angiography revealed a fistulous connection located at the medial tentorium draining directly into cerebellar veins and finally into the straight sinus. DAVFs clinical presentation includes specific and nonspecific neurological symptoms. Non-specific manifestations such as headache are usually attributed to cerebrospinal fluid malabsorption due to increased pressure in the superior sagittal sinus, venous sinus thrombosis or meningeal scarring due to repeated small subarachnoid hemorrhages. Cranial nerve deficits are probably related to arterial steal phenomena. Specific neurological manifestations may be the result of venous ischemia, venous mass effect, venous rupture or venous thrombosis due to passive venous hypertension, congestion or both (17-20). Many non-hemorrhagic neurological deficits due to tDAVFs have been reported, including gait instability, bruit, personality changes, depression, trigeminal neuralgia, syncopal events, slurred speech, cranial nerve VII weakness (3), visual symptoms. (including bilateral proptosis, bilateral episcleral and retinal venous congestion, optic disc pallor, quadrantanopia and concentric narrowing of the visual field) (10), hemifacial spasm (3), progressive myelopathy, hemisensory disturbance, brainstem dysfunction and obstructive hydrocephalus (4). The mechanism of brainstem dysfunction may be related to arterial steal phenomena, brainstem venous congestion or compression due to dilated veins or cerebellar edema (15). The vast majority of these lesions can be successfully treated with selective endovascular embolization (3,17,21).

Conclusion

The clinical symptoms of DAVFs depend on the location and pattern of the venous drainage. The transient ischemic attack should be kept in mind as an uncommon presentation of tentorial DAVFs. Endovascular, surgical, radiosurgery, and even observation in certain cases are valid options in the treatment of different grades DAVFs.

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