Spontaneous Thrombosis of Dural Arteriovenous Malformation: A Case Report

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CASE REPORT

ABSTRACT

Dural arteriovenous malformation (DAVM) is relatively rare and defined as abnormal connections or shunts between the arterial and the venous side of vascular tree located within the dura mater. Spontaneous closures of DAVM are rare and have been scarcely reported. This case report will describe the neuroimaging findings and classification of DAVM. A 50 year old lady presented with headache. Neuroimaging showed prominent serpinginous flow-void structures, cerebral angiogram confirmed the presence of DAVM at the occipital region. She had defaulted treatment and follow-up for 3 years. On second admission, she had a cerebral angiogram which showed normal findings with no evidence of fistulas or malformation. She was discharged well. Causes of spontaneous closure of DAVM are discussed.

KEYWORDS: Dural arteriovenous malformation, Dural arteriovenous fistulae, Spontaneous closure

INTRODUCTION

Dural arteriovenous malformations (DAVM) are relatively rare lesions, constituting approximately 10-15% of all intracranial malformations.[1] It is defined as abnormal connections or shunts between the arterial and the venous side of the vascular tree located within the dura mater.[1] In the literature, this term is used interchangeably with Dural arteriovenous fistulas (DAVF). The main method of treatment nowadays, is endovascular intervention sometimes in combination with surgery.

Spontaneous closures of DAVM are rare and have been scarcely reported. Most of these spontaneously resolving lesions are type I DAVM.[2] This case report will describe the neuroimaging findings using different modalities, the classification of DAVM and hypotheses of how spontaneous closure could occur in this uncommon entity.

CASE REPORT

A 50 year old lady presented with complaints of headache for the past 6 months associated with vomiting. There was no focal neurological deficit. She is a known case of hypertension on atenolol 50 mg daily. On examination, there was no neurological deficit and her vital signs are stable. The blood pressure was normal.

Computed Tomography (CT) scan revealed no evidence of intracranial haemorrhage. Magnetic Resonance Imaging (MRI) showed prominent serpinginous flow void structures which represent the dilated draining veins or enlarged arterial feeders (Figure 1). Cerebral angiogram confirmed the presence of DAVM at the occipital region (Figure 2).

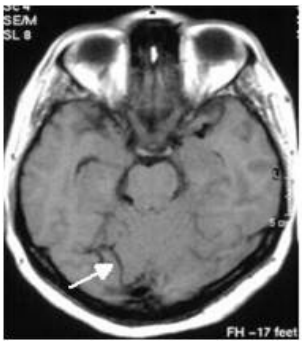

Figure 1a

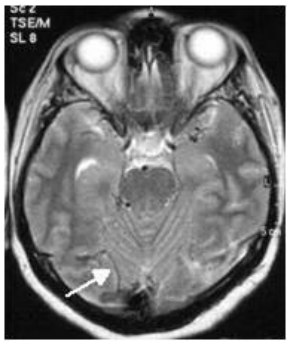

Figure 1b

MRI Brain: Axial T1 (Figure 1) and T2 (Figure 2) images showing serpenginous flow void structures representing dilated vessels in the posterior cranial fossa (white arrows).

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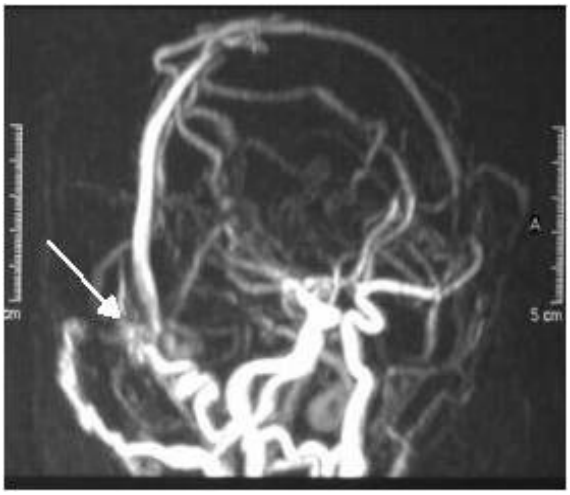


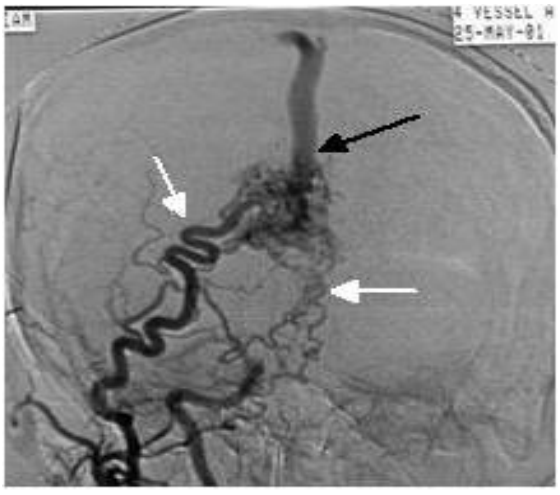
Figure 2

MR Angiogram showing multiple dilated vessels in the posterior cranial fossa which represents the dilated draining veins or enlarged arterial feeders (white arrow).

She was admitted for one day and the headache improved with intravenous Mannitol. She was discharged with embolization planned at a later date. Unfortunately, she was lost to follow-up until 3 years later. During that time, she still suffered nocturnal headache, on and off initially but later the headache improved without any treatment. She had no other complaint and there was no history of trauma.

On a second admission, she had diagnostic cerebral angiogram with a view towards embolization. However, the baseline cerebral angiogram showed normal findings with no evidence of fistulas or malformation (Figure 3). There was no headache or neurological deficits noted. She was discharged well.

Figure 3a



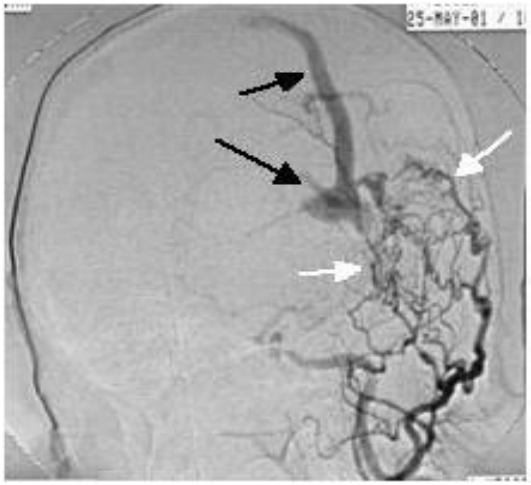


Figure 3b

Cerebral Angiogram: Right Common Carotid run (Fig 4) and Left Common Carotid run (Fig 5) showing the dural arteriovenous malformation supplied by both internal and external carotid arteries (white arrows) and draining into the torcular herophili with reflux into the superior sagital sinus (black arrows)

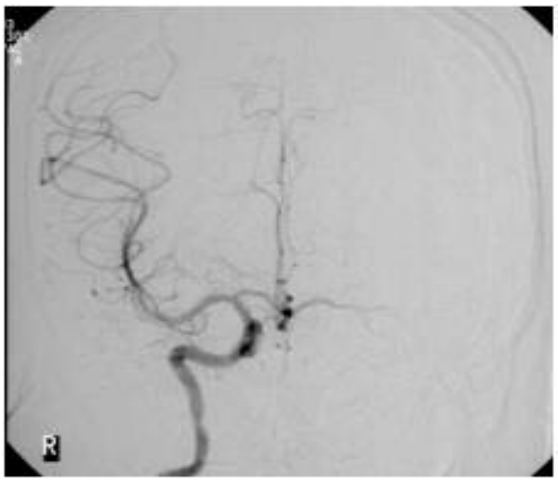


Figure 4a

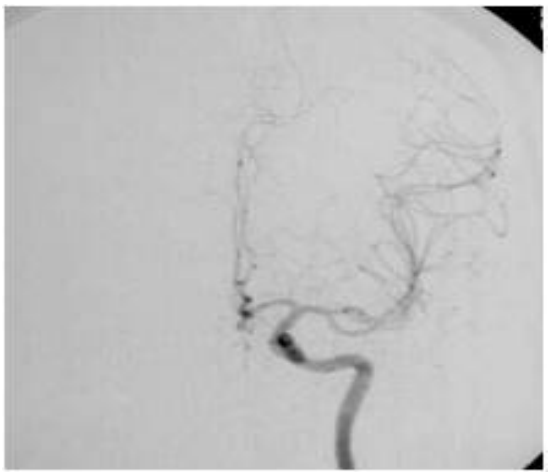


Figure 4b

Second cerebral angiogram : Right internal carotid run (Fig 6) and Left internal carotid run (Fig 7) shows normal findings with no evidence of vascular malformation or fistulas.

DISCUSSION

DAVM are thought to be acquired rather than congenital.[3] It has been postulated that factors which causes increased venous obstruction such as previous trauma, sinus thrombosis, hypercoagulable states, pregnancy and hormonal disease could lead to opening up of existing or formation of new microshunts leading to the formation of DAVM. Patients normally present with pulsatile tinnitus, cranial bruit with headache or hemifacial spasm. The most common sites include the transverse or sigmoid sinuses and cavernous sinus.

Non-invasive neuroimaging like CT scan and MRI may show dilated cortical veins without a nidus. There can be associated haemorrhage, either intraparenchymal, subarachnoid or subdural alone or in combination. The dilated cortical veins can simulate extra-axial masses on cross sectional imaging. These enhance vividly following intravenous contrast due to slow venous flow. These features may be mistaken for extra-axial tumours like meningioma. MRA might be useful particularly with contrast to highlight the vascular structures. Detection of venous contamination in the arterial phase of MRA indicating early venous drainage is an important feature in DAVM.

Catheter angiography is still the gold standard. It helps in understanding the angioarchitecture of DAVM and is important in treatment planning, evaluating the venous drainage and establishing the feeders. Catheter angiography should also include the external carotid artery (ECA) as meningeal branches may supply the DAVM.

Classification is important to prognosticate and plan the management of DAVM. Commonly used classifications include Borden and Cognard which emphasise the venous drainage of DAVM. Cortical venous reflux (CVR) is an important feature which denotes higher grade in both classifications and corresponds to poor outcome (Table I);

Table I. Classification of Cranial Dural Arteriovenous Fistulas

Borden classification	Type
Venous drainage directly into dural venous sinus or meningeal vein	I
Venous drainage into dural venous sinus with CVR	II
Venous drainage directly into subarachnoid veins (CVR only)	III
Cognard classification	
Venous drainage into dural venous sinus with antegrade flow	I
Venous drainage into dural venous sinus with retrograde flow	IIa
Venous drainage into dural venous sinus with antegrade flow and CVR	IIb
Venous drainage into dural venous sinus with retrograde flow and CVR	IIa+b
Venous drainage directly into subarachnoid veins (CVR only)	III
Type III with venous ectasias of the draining subarachnoid veins	IV

CVR = cortical venous reflux

Spontaneous closures of DAVM are rare and have been scarcely reported. The mechanism of these occlusions is unknown. However the postulations include thrombosis of the draining vein or sinus, which triggers the secondary closure of the DAVM.[5-7] Luciani et al, suggested that there are two types of spontaneously regressing DAVM which can be distinguished; these are posttraumatic and spontaneous dural AVM group.8 In the spontaneous group, mechanisms involved are postulated to be due to sinus thrombosis.

Intrinsic compression of the arteriovenous shunts within the sinus wall could also be one of the causes of spontaneous occlusion. Changes affecting the structure of the sinus wall, or a focal increase in sinus size could lead to focal compression of the arteriovenous shunts within the wall itself, thus leading to occlusion of the DAVM.

Haemorrhage could trigger the closure of an intracranial AVM, whether it is linked to haematoma-mediated mass effect or to secondary vasospasm of the feeding vessels. Similar mechanism may lead to spontaneous closure of DAVM.[8] This case documents spontaneous closure of a type IIa (2) DAVM involving the torcular herophili and superior sagital sinus with no intracranial haemorrhage or history of trauma.

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