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Case Report

A Twist of Fate: Spontaneous Closure of a Dural AV Fistula

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ABSTRACT

Dural arteriovenous fistulas (DAVFs) are rare vascular malformations characterized by abnormal connections between dural arteries and venous sinuses or cortical veins, with clinical presentation ranging from benign symptoms to seizures or hemorrhage1.

We report a case of a 35-year-old male who presented with a generalized tonic-clonic seizure. Magnetic Resonance Imaging (MRI) of the brain showed findings suggestive of an arteriovenous malformation. This was subsequently confirmed by digital subtraction angiography (DSA) as an anterior cranial fossa DAVF with cortical venous drainage, typically considered high risk. The patient was scheduled for endovascular embolization; however, during the procedure, no evidence of the fistulous connection or early venous drainage was identified. Follow-up imaging confirmed the spontaneous closure of the DAVF. Spontaneous regression of DAVFs is a rare occurrence, particularly in high-grade lesions and may be attributed to post-angiographic thrombosis, hemodynamic shifts or natural vascular remodelling2. This case reiterates the importance of re-evaluating patients prior to treatment, especially when there is a time lag between diagnosis and intervention. It also emphasizes the need for ongoing surveillance as there is always a potential risk of recurrence.

Keywords: Dural arteriovenous fistulas; Magnetic Resonance Imaging; Digital subtraction angiography

Introduction

Dural AV Fistulas are abnormal shunts between dural arteries and dural venous sinuses or cortical veins[3]. Their presentation ranges from benign symptoms like headaches to serious outcomes such as seizures or intracranial hemorrhage. Spontaneous closure of DAVFs is rare but recognized, typically associated with changes in hemodynamics or post-procedural effects[4]. This case highlights such an unexpected resolution.

Case Presentation

A 35-year-old male, chronic alcoholic, presented with a generalized tonic-clonic seizure following binge drinking. On examination, patient was conscious and oriented with Glasgow coma scale (GCS) E4V5M6 and no neurological deficit. His laboratory investigations were unremarkable. He was started on anti-epileptic medications.

Magnetic Resonance (MR) Brain was performed which was suggestive of few prominent flow voids in the sulcal spaces in left frontal region with mild parenchymal edema in the adjacent frontal lobe, suggestive of an arteriovenous malformation **(Figure 1 A-F)**.

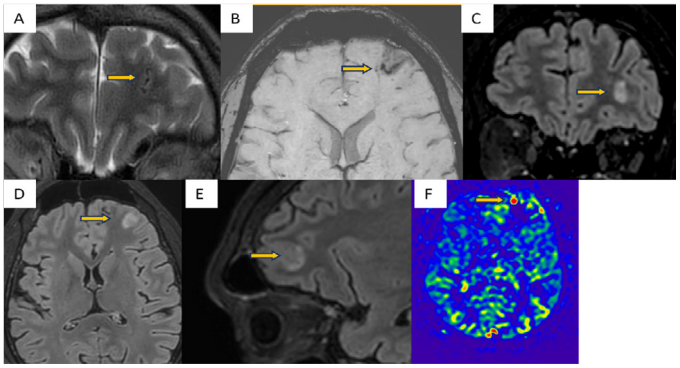

Figure 1: (A) - T2W coronal image showing hyperintense signal with a focus of hypo intensity within in left frontal region (ARROW)
(B) - SWI showing a prominent draining vessel in left frontal region (ARROW)
(C), (D) and (E) - 3D FLAIR images in all planes showing prominent flow void with mild parenchymal edema in left frontal region (ARROW)
(F) - ASL images showing hyper perfusion in the affected area (ARROW)

A digital subtraction angiography (DSA) was performed for further evaluation. Right femoral access was taken with a 5F short sheath. Selective catheterization was done and contrast was injected into the bilateral common carotid arteries, internal and external carotid arteries and vertebral arteries and images acquired. These revealed an anterior cranial fossa DAVF with arterial feeders from the posterior ethmoidal branch of the right ophthalmic artery and orbitofrontal branch of the left ACA. The fistula drained directly into an ectatic cortical vein. No feeders could be identified from either ECA or left ophthalmic artery **(Figure 2 A & B)**. The patient was advised fistula closure by endovascular embolization.

After a few days the patient was admitted for embolization under GA. Right CFA access was taken with 6 F short sheath using Envoy 5F guide catheter and wire combination, right ICA gram was obtained. 2mg infusion of Nimodipine was started slowly. Using marathon microcatheter and mirage microwire combination, right ophthalmic artery was cannulated. Fistulous branches were cannulated with marathon micro-catheter taken over mirage 0.08" microwire till the dural AVF nidus.

No early draining veins or fistulous connection was observed despite super selective catheterization. Super-selective cannulation of the right ACA was done however no early draining veins were found. Normal filling of intracranial circulation was seen **(Figure 3 A-C)**. Contralateral ICA injection confirmed spontaneous closure of the DAVF **(Figure 3D)**.

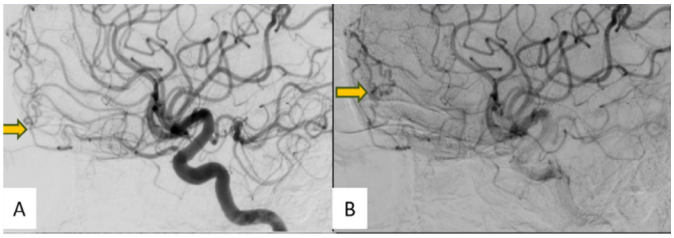

Figure 2: (A) &(B) - Digitally subtracted images through the right internal carotid artery showing anterior cranial fossa DAVF with arterial feeders from the posterior ethmoidal branch of the right ophthalmic artery and orbitofrontal branch of the left ACA (arrow)

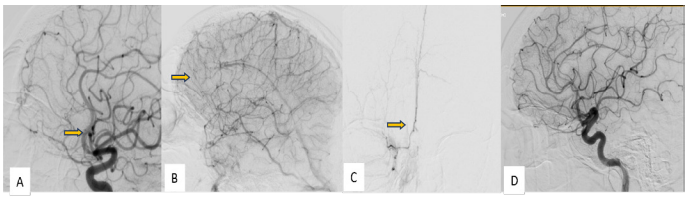

Figure 3: (A), (B) Selective digitally subtracted angiography images in arterial and venous phases through the right Internal Carotid Artery did not show any fistula at the previously documented site (arrow)
(C) - Superselective digitally subtracted angiography images through the branches of anterior cerebral artery did not any fistula (arrow)
(D)- Selective digitally subtracted angiography images through the contralateral (left) Internal Carotid Artery did not show any fistula at the previously documented site

Discussion / Summary

DAVFs represent abnormal vascular communications between meningeal arteries and dural venous sinuses or cortical veins[2]. While they account for only 10-15% of intracranial vascular malformations, their clinical presentation and potential for serious complications-such as intracranial hemorrhage, seizures or progressive neurological decline make early diagnosis and management critical[5].

The Cognard classification system is a widely used grading system for dural arteriovenous fistulas (DAVFs). It categorizes these vascular abnormalities based on their venous drainage patterns and clinical implications[6].

In the present case, the patient exhibited a DAVF located in the anterior cranial fossa with cortical venous drainage, which typically confers a higher hemorrhagic risk as per the Cognard classification (Grade III or IV). Prompt endovascular or surgical intervention is usually indicated in such high-grade lesions[1]. However, this case took an unexpected turn when the DAVF demonstrated spontaneous angiographic resolution just days after diagnostic workup and prior to embolization-highlighting a rare but well-documented phenomenon in neurovascular literature[1].

Four primary mechanisms have been proposed to explain the spontaneous closure of DAVFs: post-angiographic thrombosis,

hemodynamic alterations, venous remodelling and fibrosis and natural healing and immune response[1].

- **Spontaneous thrombosis:** Thrombosis can occur within a dural arteriovenous fistula (DAVF), often due to changes in blood flow dynamics, leading to reduced or ceased blood flow.
- **Hemodynamic influences:** Variations in systemic blood pressure, local vascular resistance and vascular remodelling can alter hemodynamics, potentially facilitating the spontaneous closure of the DAVF over time.
- **Venous remodelling:** Remodelling of the venous drainage pathways associated with the DAVF is another proposed mechanism. As the venous drainage pattern adapts to accommodate altered hemodynamics or increased venous pressure due to the fistula, it may lead to progressive narrowing or closure of the abnormal connection. This venous remodelling process could be influenced by changes in vascular endothelial integrity, collagen deposition or fibrotic changes within the venous walls.
- **Natural healing processes:** The body's innate healing mechanisms, including local inflammatory responses, tissue repair processes and vascular remodelling mechanisms, may also contribute to spontaneous closure of DAVFs. These mechanisms may act independently or in combination, with each potentially influencing the others in a given case. Recanalization of the occluded draining sinus could prompt regression of the dAVF[7]. Indeed, some authors have claimed haemorrhage could trigger the closure of intracranial arteriovenous malformations[8]. Although various factors may contribute to spontaneous closure, its occurrence and timing remain unpredictable. This underscores the importance of re-evaluating fistulas prior to treatment, especially if a delay occurs and maintaining follow-up due to the potential risk of recurrence[3].

In our case, the patient's DAVF may have closed due to altered hemodynamics or spontaneous thrombosis following diagnostic angiography.

Conclusion

DAVFs carry variable prognoses depending on their drainage pattern and location. While interventional therapy remains the mainstay of treatment, spontaneous resolution is a recognized rare outcome. This case reiterates the importance of follow-up imaging and clinical reassessment before planning invasive procedures.

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