Japan Journal of Research

Clinical Images

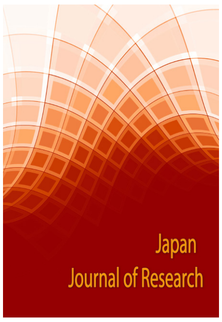

Image of Dural Fistulas

Gerardo Villegas López[1], Mónica A Rincón[2], Soledad Simont[2], Rocio Madrigal[2], Aurora Navarro[2], Jocelyn Hernández[2], Pedro Zarate[2], Sergio Barba[2], Fernando Cardenas[2], William Vicente D, Albano[2], Marco A Flores[3], Lizeth Nahomi Lopez[3], Sandra Landazuri[4], Josué David Ramírez[5]

[1]Pediatric Neuroradiologist, Medical Subdirector, Chopo Medical Laboratory, Northern Border, Mexico
[2]General Director of Diagnostic Centers, Chopo Medical Laboratory, Mexico
[3]Regional Medical Subdirectors, Mexico City and States, Chopo Medical Laboratory, Mexico
[4]Manager, Non-Radiological Diagnostic Centers and Medical-Commercial Projects, Mexico
[5]Head, Medical-Commercial Administration, Proa2mil Diagnostic Centers, Mexico

Correspondence
Gerardo Villegas López MD
Pediatric Neuroradiologist, Medical Subdirector, Chopo Medical Laboratory, Northern Border, Mexico

- Received Date: 06 Mar 2026
- Accepted Date: 16 Mar 2026
- Publication Date: 18 Mar 2026

Keywords
Angio MRI, Cerebral vascular malformations, Dural fistulas, Arteriovenous fistulas

Abstract

Cerebral vascular malformations, arteriovenous malformations (AVMs), are diagnosed incidentally or when presenting with clinical manifestations such as headache, acute sensorimotor deficit, and epilepsy, among others. Their detection ranges from 8–10% of patients admitted to our institution for magnetic resonance imaging (MRI) and cerebral magnetic resonance angiography (MRA). AVMs constitute a group of congenital or acquired diseases that represent morphological alterations affecting arteries, capillaries, and veins, or combinations thereof.

Several types of AVMs are known, including those with arteriovenous shunts and those with high or slow flow. The most frequent is the arteriovenous malformation (AVM), cavernous angioma (CA) or cavernoma, although it is also known as cavernous hemangioma or cavernous venous malformation. Venous angioma (VA), telangiectasia or capillary telangiectasia, and dural arteriovenous fistulas (DAVFs) are anomalous vascular communications located within the dura mater, such that the direct arteriovenous connections give the pathology its name. AVMs present with a parenchymal nidus, while DAVFs are direct communications with the dura mater. MRI, complemented by MRA, is essential for their classification and treatment planning.

Introduction

Although digital subtraction angiography (DSA) is the gold standard for hemodynamic characterization [1], magnetic resonance imaging (MRI) and MR angiography allow for detailed anatomical evaluation, detection of associated parenchymal changes, and morphological classification. Arteriovenous fistulas (AVFs) are considered acquired lesions and represent 10–15% of all cerebral and spinal cord vascular malformations. Several factors are considered in their pathophysiology [2]:

Theory of their congenital origin, which ensures the persistence of vessels from the embryological period, which can be associated with different anomalies such as phakomatosis and malformation of the vein of Galen [3].

Theory of secondary injury after traumatic brain injury (TBI): the most frequently observed traumatic fistulas are direct carotid-cavernous fistulas (CCFs), AVFs as a consequence of TBI.

Post-surgical theory, which develops secondary to the surgical approach, frequently affecting the venous sinuses.

Tumor theory due to extension to the venous sinuses.

AVFs are very common, observed in both computed tomography (CT) and magnetic resonance imaging (MRI). AVFs represent a group of disorders with very different clinical implications, depending on the area of brain involvement, ranging from benign behavior to motor and sensory neurological disabilities and/or even death.

The most common characteristic of ARVCs is that they almost exclusively affect the central nervous system (CNS), and the pivotal clinical finding is almost always an infarct-type vascular event with secondary hemorrhagic transformation. Depending on their location, they may be associated with focal neurological disorder or epilepsy.

ARVCs are venous lesions, and sometimes we fail to visualize them on catheter-guided cerebral angiography, characteristically due to the slowness of their flow. The occurrence of these lesions is estimated to be between 0.1% and 0.5% and is due to the dilation of blood

Citation: López GV, Rincón MA, Simont S, et al. Image of Dural Fistulas. Japan J Res. 2026;7(2):178

vessels, which are grouped and well-defined [3–5].

Other ARVCs are clinically silent and are found incidentally in asymptomatic patients or those with occasional headaches. More than half of ARVCs are diagnosed between the second and fourth decades of life, with a predominance in females.

In 1991, several MRI series were analyzed in a meta-analysis, estimating a prevalence of 0.39–0.47%; other authors have considered it a higher finding in the general population [2,5].

Objective

To understand and review the MRI, clinical, and neuroradiological findings of dural fistulas.

Findings and Analysis of Results

There is no known family history of the condition or similar symptoms in first-degree relatives.

In MRI images 1a, b, c, and d (axial T2-weighted images), dilated vascular structures are observed at the base and convexity of the brain.

Discussion

The vascular drainage pattern is of paramount importance in the natural history of the disease. ARVCs with cortical venous drainage demonstrate a worse prognosis, exhibiting a more aggressive natural course with a risk of hemorrhage and neurological deficits, with or without hemorrhage, in 7–8% of cases, and even a mortality rate of around 10% in affected patients. Upon diagnosis of ARVC, we must periodically review the imaging and clinical evolution of the patient, noting any changes in existing clinical symptoms. It is important to evaluate the following in our neuroradiological report:

1. Presence of subarachnoid, parenchymal, or subdural hemorrhage
2. Venous infarcts
3. Cerebral edema
4. Hydrocephalus
5. Asymmetry of venous sinuses or thrombosis
6. Thickening of venous sinuses or dilation of draining veins

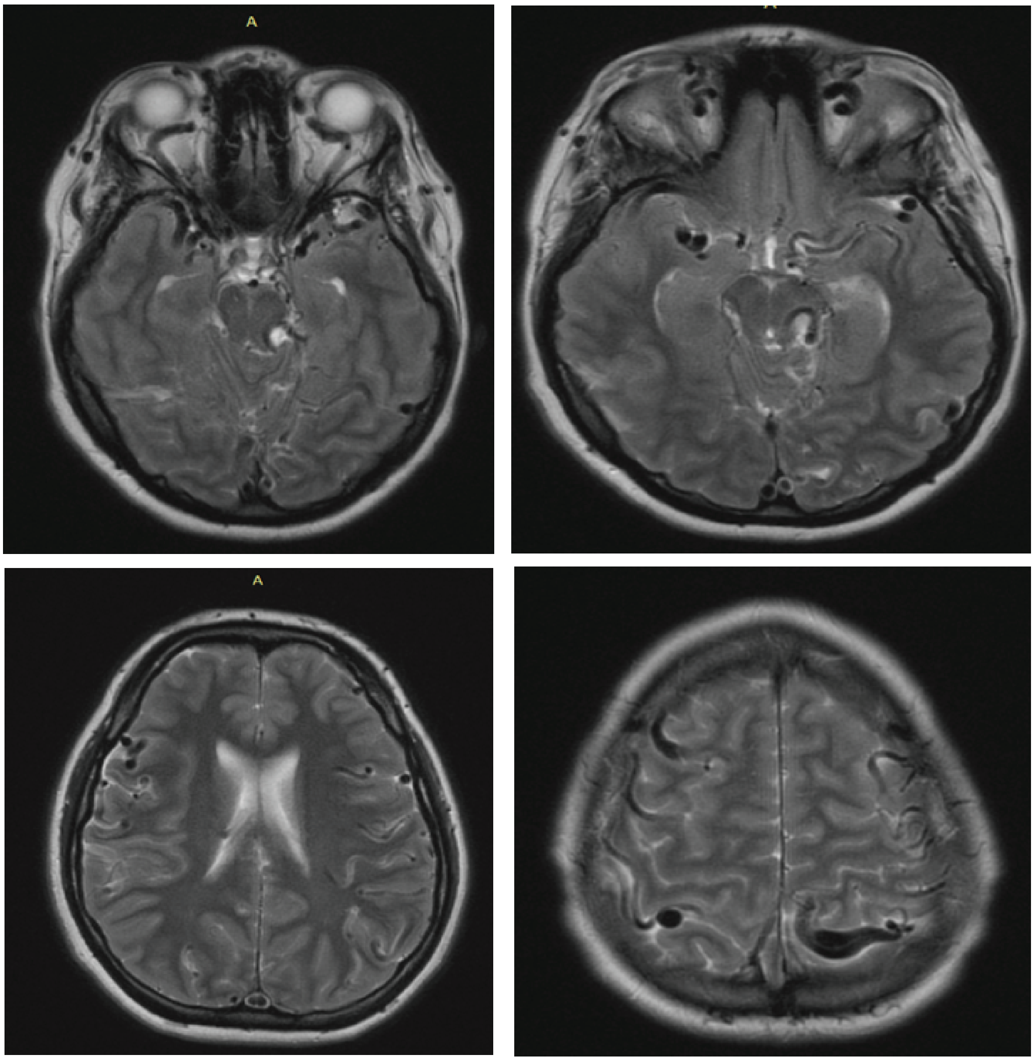

Image I *(a, b, c and d): Axial T2 MRI showing multiple dilated vascular structures, including ophthalmic, temporal and midbrain veins, a and b bilateral temporopaietal and bilateral asymmetric postcentral veins c and d*

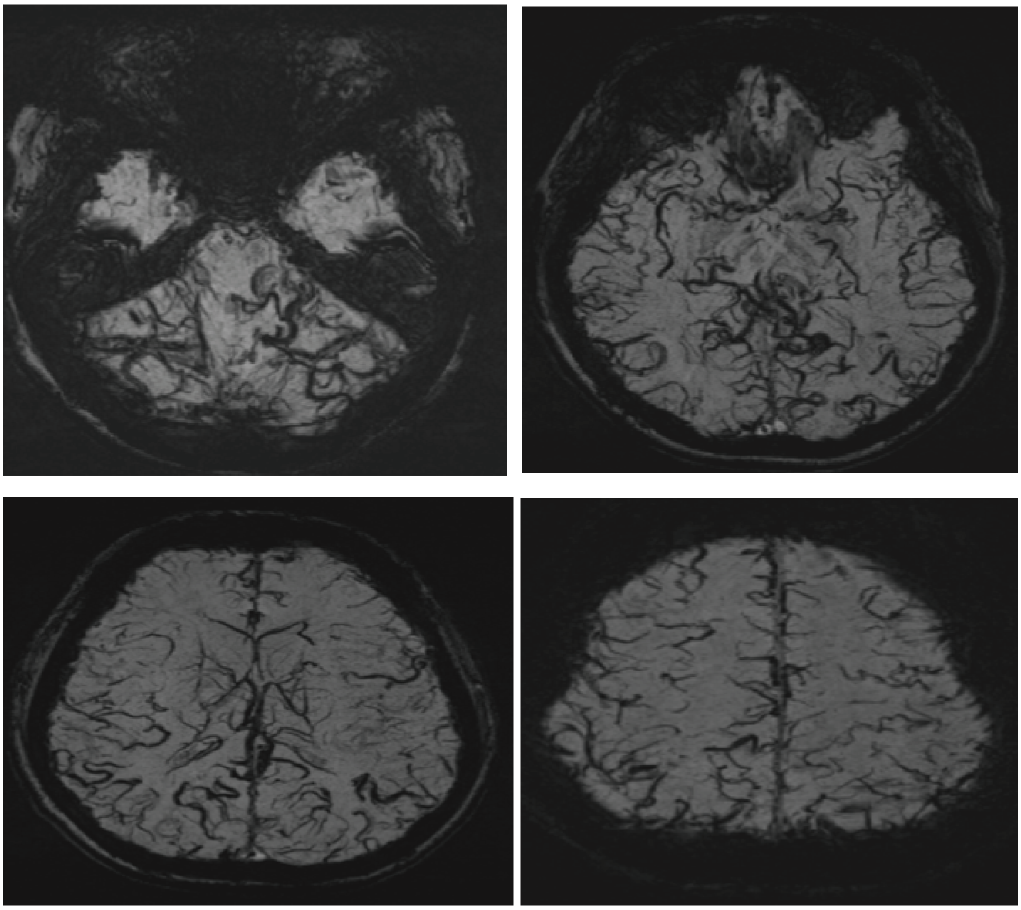

Image 2: *MIP SW MRI shows dilated venous structures in the cerebellum, posterior cranial fossa, midbrain a and b pre and postcentral c and d*

Conflict of interest

The authors declare no conflict of interest

Acknowledgment

The authors gratefully acknowledge Marifer and Naomi, whose illness inspired the presentation of this work.

References

1. Sánchez-Marco SB, Monge-Galindo L, López-Pisón J, García-Jiménez I, Peña-Segura JL, López-Lafuente A. Neurofibromatosis 1 y cavernomatosis múltiple familiar en un lactante con sialorrea profusa episódica. Rev Neurol. 2019;69(8):350-352. doi:10.33588/rn.6908.2019230
2. Lv X, Li Y, Jiang C, et al. Endovascular treatment of cerebral arteriovenous fistulas. AJNR Am J Neuroradiol. 2009;30(4):851-856. doi:10.3174/ajnr.A1436
3. Osborn AG. Diagnostic Cerebral Angiography. Lippincott Williams & Wilkins; 1999. ISBN:0397584040.
4. Congreso Nacional SERAM. Vol 2, No. 1. 2018:34.
5. Duffau H, Lopes M, Janosevic V, Sichez JP. Early rebleeding from intracranial dural arteriovenous fistulas: report of 20 cases and review of the literature. J Neurosurg. 1999;90(1):78-85.
6. Geibprasert S, Pongpech S, Jiarakongmun P, et al. Radiologic evaluation of cerebral arteriovenous malformations: what clinicians need to know. Radiographics. 2010;30(2):483-501.