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Dural Fistula of the Superior Cerebellar Artery: A Case Report

Y. Bouktib[1*], W. Adegbindin[1], B. Boutakioute[1], M. Ouali Idrissi[1], N. Cherif Idrissi Ganouni[1]

[1]Radiology Department, Neurosurgery Department, ER-RAZI Hospital, CHU Mohammed VI, Marrakech; Faculty of Medicine of Marrakech, Cadi Ayyad University, Marrakech, Morocco

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***Corresponding author:** Y. Bouktib
Radiology Department, Neurosurgery Department, ER-RAZI Hospital, CHU Mohammed VI, Marrakech; Faculty of Medicine of Marrakech, Cadi Ayyad University, Marrakech, Morocco

Abstract **Case Report**

Dural arteriovenous fistulas are rare intracranial vascular malformations with a propensity for hemorrhage. The Cognard classification system is the most widespread classification system wherein type IIB through V must be promptly treated to avoid the risk of hemorrhage. The case presented herein reports a 65 -year-old male presenting with vague non-hemorrhagic neurologic deficits found to have a Cognard type I right dural superior cerebellar arteriovenous fistula. Although quite obvious in retrospect, a DAVF can be missed even by an astute radiologist. This should be considered a "never miss" diagnosis as it carries a risk of intracranial hemorrhage and death.

Keywords: hemorrhage, Dural arteriovenous fistulas, DAVF, hemianopsia.

INTRODUCTION

Dural arteriovenous fistulas (AVDF) or intracranial dural fistulas consist of communications arteriovenous seated in the thickness of the dura mater of the skull. They can be encountered at any point of the dura mater but most sit on the dura mater of a sinus. In the Caucasian population, the lateral sinus is most frequently affected, followed by the cavernous sinus. With the exception of the rare dural fistulas in children, these lesions develop in adulthood from the fourth decade. Their etiology is unknown but, in some cases, it may have be established a link with a history of sinus thrombosis. The neurological prognosis of AVDF depends on their venous drainage studied by cerebral angiography.

CASE REPORT

A 65 year-old man with a history of a reported stroke three months previously without residual deficits presented to the emergency department after being involved in a minor traffic collision involving two cars without airbag deployment or damage to the windscreen or steering column. The patient was found to have mild dysarthria, tangential thoughts, confusion and difficulty answering questions. The patient was unable to answer whether there had been a loss of consciousness. Based on these findings, a code stroke was initiated. The patient's National Institutes of Health Stroke Scale (NIHSS) score was 3 for partial hemianopsia, mild loss of language and slurred but intelligible speech. An additional history obtained days later after improvement in mental status revealed two previous episodes of loss of consciousness during his lifetime, including a rock dropped on his head as a child and a kick to the back of the head about 50 years earlier.

A CT scan without injection was requested urgently which targets a spontaneously hyperdense right intra- cerebellar parenchymal hematoma measuring 46x24 mm, surrounded by a perilesional edema with doubt on a tonsil engagement (figure 1).

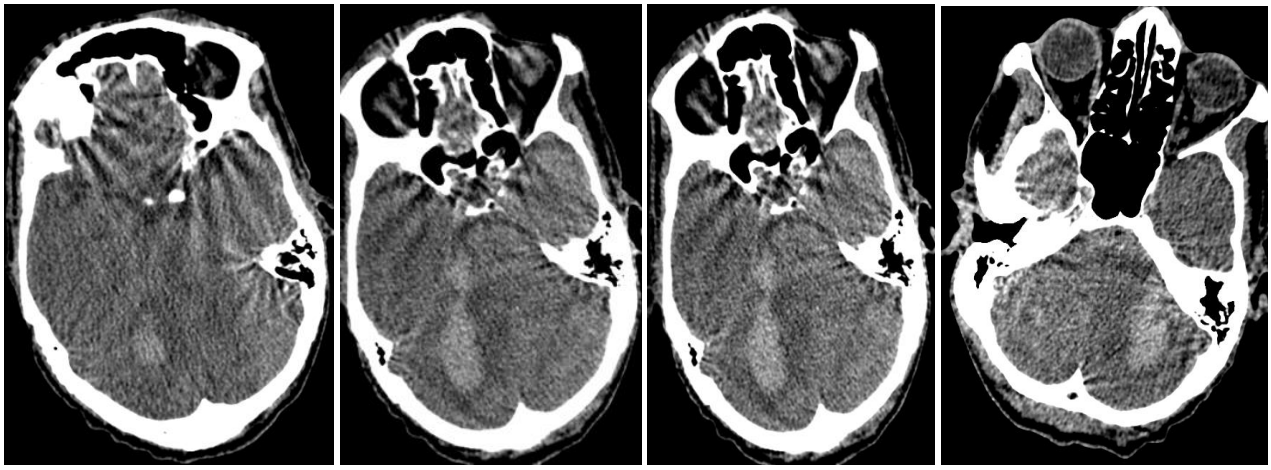

Figure 1: Cerebral CT scan without injection shows a spontaneously hyperdense right cerebellar hematoma Surrounded by a peri-injury oedema with tonsillar engagement

The patient was then taken to the neurointervention room for digital subtraction angiography (DSA) of the bilateral internal and external carotid arteries. Selective angiography of the ostium of the right vertebral artery highlights: spasm of the V4 segment with early opacification from the arterial phase of the right lateral venous sinus by a branch originating from the right superior cerebellar artery, with flow antegrade without venous reflux or associated cortical drainage (figure 2).

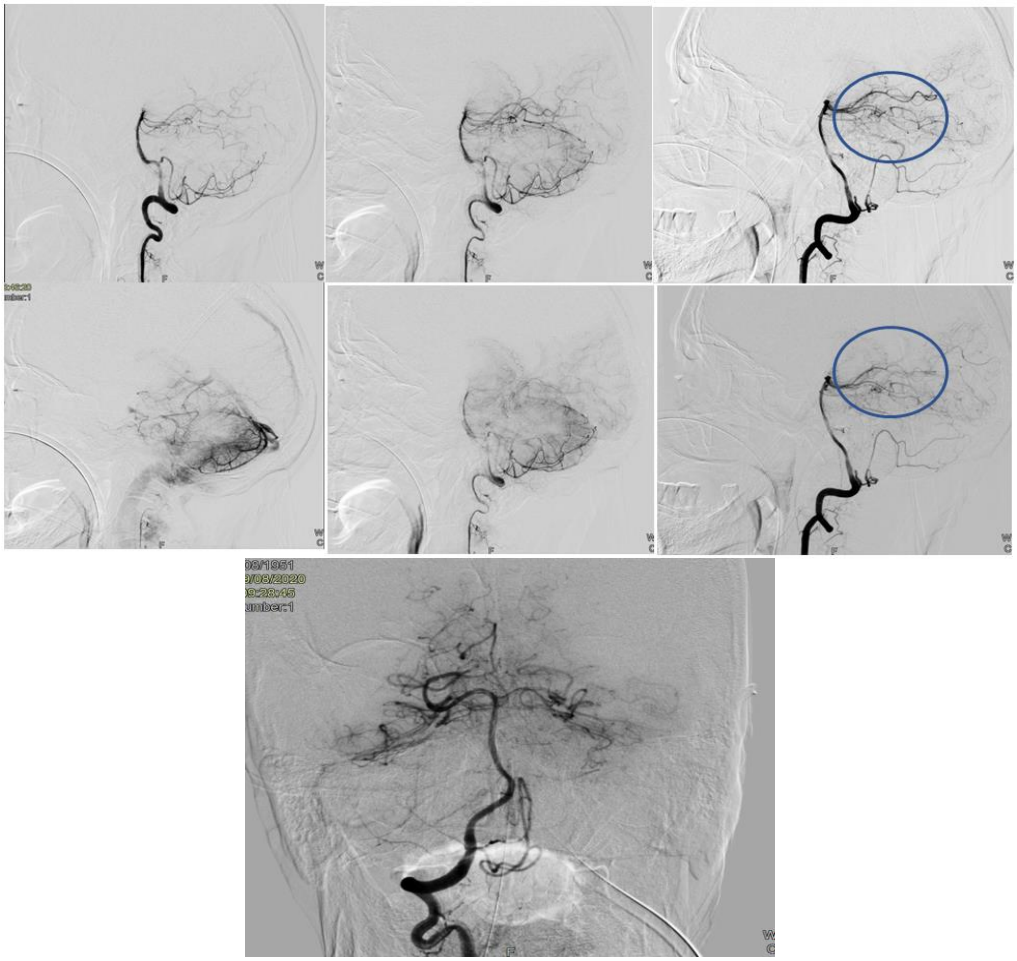

Figure 2: Cerebral arteriography with sagittal and coronal sections with catheterization of the internal carotid and right and left vertebral arteries, opacification of the right vertebral artery shows:

- **A spasm of the V4 segment.**
- **Early opacification of the right lateral venous sinus by a branch from the right superior cerebellar artery, with antegrade flow without venous reflux or associated cortical drainage.**

The patient was treated with transarterial embolization utilizing liquid embolization, control scanner shows a good radiological evolution with regression of the cerebellar hematoma and persistence of a hypodense plage without mass effect or engagement (figure 3).

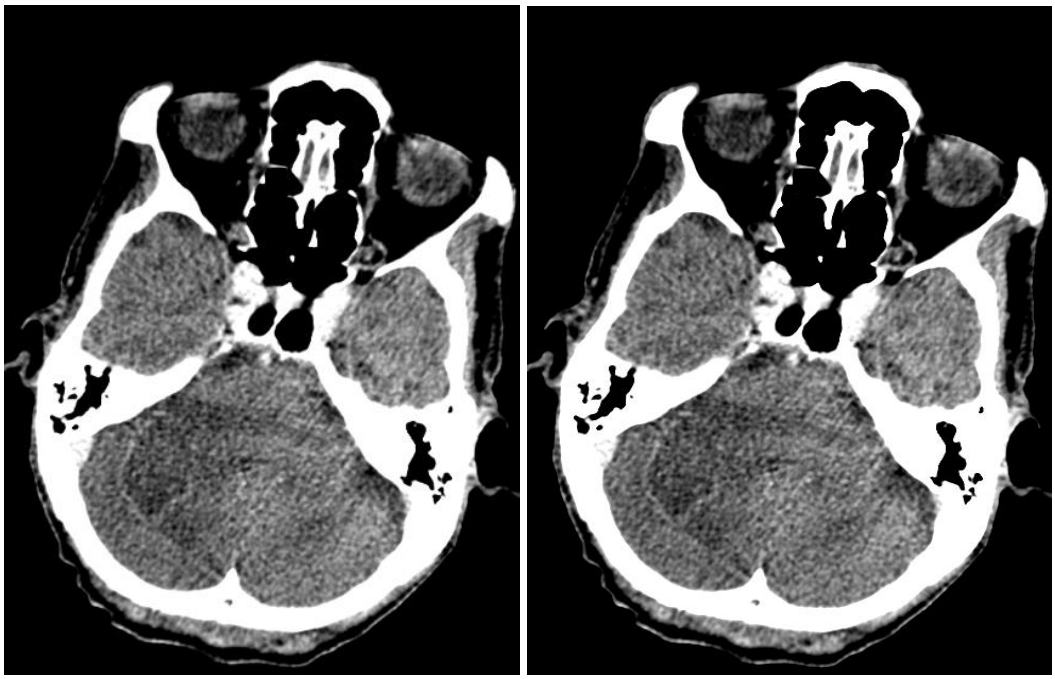

Figure 3: A CT scan show regression of the right cerebellar hematoma and persistence of a hypodense area

DISCUSSION

DAVFs are predominantly idiopathic, although a small percentage of patients have a history of previous craniotomy, trauma, or thrombosis of the dural sinus [2]. The pathogenesis of DAVFs remains unclear. Most occur as a result of neovascularization from a previously thrombosed dural sinus in patients with a documented antecedent [3].

The majority of patients with DAVFs present in the fifth and sixth decades. Signs and symptoms are related to the location of the lesion and the presence of complications. Ophthalmoplegia, proptosis, chemosis, retroorbital pain, or decreased visual acuity may be present in cavernous DAVFs. Transverse and sigmoid sinus DAVFs often present with pulsatile tinnitus. The most common complication of a DAVF is hemorrhage, which can lead to more severe neurological signs and symptoms.

However, the Cognard and Borden classification systems are based on the venous drainage pattern, which often determines the severity of symptoms. The Borden classification organizes lesions based on the site of drainage and the presence of cortical venous drainage [4]. The Cognard system was the original and more widely used classification scheme, which includes the direction of flow [5]. Type I lesions drain into the dural sinus. They have an antegrade flow direction and lack cortical venous drainage. Type II lesions are further subdivided into A, B, and A+B. Type IIA lesions drain antegradely without cortical venous drainage. Type IIB lesions drain antegradely into a dural sinus with cortical venous drainage. Type IIA+B lesions drain retrogradely into a cortical sinus. Type III and IV lesions drain into a non-dilated or dilated cortical vein (without dural sinus drainage). Type V lesions drain into a spinal vein (without dural sinus flow).

Approximately 20%-33% of DAVFs are associated with intracranial bleeding [6]. The risk of intracranial hemorrhage from Cognard type I and The risk of intracranial bleeding from Cognard type I and IIA (which lack cortical venous drainage) is extremely low [7]. In addition, the risk of conversion of type I or IIA to AVF with cortical drainage is low, reported at about 2% [8]. Spontaneous resolution of a DAVF is rare but possible [9]. The presence of cortical venous drainage (Cognard IIB-V) is an aggressive feature with 10.4% annual mortality and 8.1% annual risk of intracranial hemorrhage [10]. After an initial hemorrhage, the risk of rebleeding can be as high as 35% in the first two weeks [11]. Further subdivision of Cognard types IIB-V into symptomatic and asymptomatic cases shows a significant difference in the annual risk of intracranial hemorrhage (7.4% vs. 1.5%) [12]. In patients with retrograde cortical venous drainage, up to half of patients have parenchymal oedema; the addition of enhancement in areas of oedema indicates an aggressive fistula with a high bleeding rate [13].

Conventional digital subtraction angiography (DSA) remains the gold standard for detection and classification of DAVFs, but it is not without risk, as a complication rate of 2.6% has been reported [14]. Fortunately, with the increase in technological applications of CT and MRI, the detection and classification of DAVFs prior to angiography has been greatly improved. It is also important to note that a complete DSA, including angiography of the bilateral internal, external, and vertebral arteries and super-selective evaluation of the distal feeder branches, is required to fully evaluate all possible arterial supplies.

Treatment risks should be weighed against the patient's natural history of DAVF. Conservative management and close clinical follow-up are indicated for Cognard I and IIA lesions, and any change in symptoms warrants repeat imaging. High-grade lesions (Cognard IIB-V) should be treated early to avoid the risk of hemorrhage and non- hemorrhagic neurological deficits. Transarterial embolization with liquid embolic agents has become the mainstay of DAVF treatment to completely eliminate the arteriovenous shunt [15]. Inadequate treatment may result in recruitment of collateral vessels with continued risk of intracerebral bleeding. Transvenous embolization was an important aspect of treatment prior to the advent of liquid embolization, as only half of the cases were technically successful with arterial embolization alone [16]. There are some scenarios where transvenous approaches are preferable, e.g., when the AVF is supplied by small, tortuous arteries that preclude safe transarterial access, when the AVF is supplied only by branches directly from the internal carotid or vertebral arteries, when the AVF is supplied by arteries with hazardous extracranial- to-intracranial anastomosis, or when the AVF is supplied by the cranial nerve feeding arteries [17]. A variety of surgical options, including direct intraoperative embolization, resection of abnormal dura, packing of the diseased sinus, disconnection of the cortical venous drainage, and skeletonization of the dural sinus, are available for cases in which endovascular approaches have failed or are not feasible [18]. As a last salvage option, stereotactic radiosurgery can also be used to induce endothelial cell damage and thrombosis. DAVFs can take months to obliterate and there is still a risk of bleeding [19].

CONCLUSION

A Cognard type III DAVF with four arterial feeders (left occipital, right occipital, right ascending pharyngeal, and right middle meningeal arteries) with fistulization into the transverse dural sinus and a (non-dilated) cerebellar cortical venous drainage into a vein of Galen was found in a patient presenting with vague non-hemorrhagic neurological deficits. This was treated with transarterial embolization of multiple arterial feeders resulting in complete obliteration of the left occipital and near complete obliteration of multiple small tortuous right-sided arterial feeders. Unfortunately, the patient was lost to follow-up. However, the cortical venous drainage was completely eliminated, thus neutralizing the high-risk/aggressive factor.

Even an astute radiologist can miss a DAVF, although it is quite obvious in retrospect. This should be considered a "never missed" diagnosis, as the annual risk of bleeding and dying is significant. The Cognard classification is the most widely used classification system for DAVF and helps to determine how aggressive it is. It is also important for the angiographer to perform a complete evaluation of the entire vasculature, including the bilateral internal and carotid arteries, as well as the bilateral vertebral arteries. DAVFs may have multiple feeders that are not evident on CTA. Endovascular treatment is the mainstay of Cognard type IIB-V, although surgical and stereotactic radiosurgical techniques are used in selected cases.

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