Case Report

Clinical and Radiological Evaluation of a Spontaneously Occluded Intracranial Arteriovenous Malformation

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Abstract

Spontaneous obliteration of brain AVMs is extremely rare estimated less than 1%. We report the case of a 37 year-old female with a history of seizures treated with anticonvulsives, who sought medical attention because of recurrence of her symptoms. The MRI findings were attributed to a patent AVM located at the left occipital lobe. Further radiological exams (angiography, CT, plain radiograms) revealed a totally calcified lesion probably representing an occluded AVM. The prompt diagnosis of spontaneous obliteration of a brain AVM requires a high index of suspicion and multidisciplinary radiologic evaluation with MRI, angiography, CT and plain radiograms.

Keywords: AVM; spontaneous obliteration

INTRODUCTION

Arteriovenous Malformations (AVM) of the brain are congenital vascular lesions affecting approximately 0.01%-0.50% of the population. Their clinical symptoms present during the second and third decade of life and are mainly due to intracranial haemorrhage (50%). AVMs can also cause seizures (25%), focal neurological deficits, headaches or remain completely silent. Occasionally, brain AVMs may undergo occlusion, a process that has not been yet clearly delineated.

We present a rare case of a totally calcified, obliterated AVM and we discuss possible misleadings due to imaging sequence.

CASE PRESENTATION

A 37 year-old female referred to our hospital complaining of recent episodes of loss of consciousnes with metacritical amnesia. The patient had a history of seizures during childhood, well-controlled with anticonvulsives, and has been...
suffering from periodical migraine with acute onset during the last ten years. Both neurological examination and ECG were normal.

Magnetic Resonance scan (MR) excluded sclerosis of hippocampus but revealed a 15 mm lesion of tightly packed flow-voids, located at the left occipital lobe with no signal either on T1 or T2-weighted images. The finding was attributed to a patent AVM (Fig. 1).

The patient was admitted to our hospital for further evaluation. Angiography excluded the presence of a patent AVM while Computed Tomography (CT) demonstrated a strongly calcified lesion of the same shape at the site under question. Plain radiographs confirmed the CT findings, which were finally attributed to an occluded brain AVM (Fig. 2, 3).

The patient was managed conservatively and regular follow-up was recommended. For the last two years, she remains free of symptoms.

**Figure 1:** Proton density (a) and T1-weighted (b) images demonstrate a lesion of tightly packed flow-voids located at the left occipital lobe.

**Figure 2:** Angiography revealed no pathological findings at the same region.
Spontaneous obliteration of brain AVMs is extremely rare estimated less than 1%\(^{(8)}\). It has been reported that it mostly concerns patients with a mean age of 40 years, a parietal location and a small size of nidus (<6 cm)\(^{(5,8)}\). Our patient was 37 years old with an occipital lesion of 1.5 cm. Occasionally, thrombosed AVMs may exhibit dystrophic calcifications as residua of repeated previous microhaemorrhages\(^{(3)}\).

Many factors are considered to be responsible for the obstruction but none has been clearly defined. Mostly, it occurs after an intraparenchymal haemorrhage, presuming that mass effect may obliterate AVM's vessels with subsequent thrombosis. When subarachnoid haemorrhage is the only finding, significant flow decrease due to constriction of the feeding arteries may lead to thrombosis\(^{(5)}\). It has also been proposed that gliosis due to repeated episodes of microhaemorrhage results in tortuosity of vessels, decrease of flow and thrombosis\(^{(8)}\). The reported periodical, acutely started, episodes of migraine in our case probably indicate repeated microhaemorrhages in the area resulting in calcification of the lesion. Other predisposing causes of AVM occlusion are atherosclerosis of the feeders\(^{(4)}\), alterations of the vessel wall due to turbulent flow (called ‘fibromuscular cushions’) and emboli\(^{(8)}\).

Thrombosis has also been reported in children with hypercoagulable states and women using oral contraceptives, as well as, after intravenous administration of contrast material\(^{(5,6)}\).

Although angiography remains the gold-standard for evaluating intracranial vascular malformation, occluded AVMs may often have a normal angiogram as in our case.

MRI is the primary method for diagnosing an AVM. The virtual absence of intraluminal signal (flow-void) results in a high degree of inherent contrast between the vessel lumen and adherent tissues which obviates the need for contrast material. On the other hand, calcified foci also may exhibit no signal at all. Therefore, routine pair of sequences T1-weighted (TE=20 ms, TR=400 ms) and T2-weighted
(TE=1500 ms, TR=120 ms) do not permit this differentiation. In our case, the diagnosis of a patent AVM was initially established without considering the possibility of a totally calcified lesion. This situation can be resolved by shortening the pulse interval from 500 to 100 ms so that rapidly flowing nuclei in vessels will no longer be able to escape the imaging plane before signal detection. This produces high signals of flow-bearing structures while calcifications will continue to have no signal. The technique is used only selectively because it results in a more noisy image with little soft-tissue contrast, since short pulse intervals cause low amplitude of signal.

In our case, the diagnosis was completed with CT and plain radiographs which confirmed the presence of a typical pattern of calcification due to a spontaneously occluded AVM. The possibility of a strongly calcified tumor such as oligodendroglioma, astrocytoma or hamartoma was totally excluded because of the typical appearance of the lesion that is serpiginous pattern and absence of brain parenchyma between the alleged vessels.

One should keep in mind the exceedingly rare event of recanalization of the malformation, reported especially in children.

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