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# A Case Report: Parieto-Temporal Arteriovenous Malformation

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**Abstract** **Case Report**

A cerebral arteriovenous malformation (cAVM) is an arteriovenous shunt of congenital origin without interposition of an intermediate capillary bed. It involves one or more arteries or veins. A cluster of abnormal vessels (the ″nidus) is usually found around it. The lack of a capillary interface results in increased arterial blood flow and venous pressure downstream of the defect. The haunt is that of a spontaneous rupture. This is a rare condition. We report the case of a 45 year old female patient with an parieto-temporal arteriovenous malformation. The initial symptomatology consisted of consciousness disorders with intracranial hypertension syndrome. Cerebral arteriography revealed a single left parietal arteriovenous malformation of peripheral location with superficial venous drainage.

**Keywords:** arteriovenous malformation, veins, nidus, intracranial hypertension syndrome.



## INTRODUCTION

Cerebral arteriovenous malformation (cAVM) is a congenital lesion usually consisting of a cluster of abnormal vessels (the ″nidus) that has formed around one or more localized arteriovenous shunts in the cerebral vascular network [1]. Cerebral arteriovenous malformations are rare, with an estimated prevalence of 18 per 100,000 populations [2]. Management involves surveillance, microsurgical treatment, endovascular treatment and radiosurgery [3]. We report the case of a patient with an unresectable occipital cerebral arteriovenous malformation who was treated with radiotherapy under stereotactic conditions.

## CASE REPORT

A 45 years old female, married, without children with no particular pathological history. Her family medical history did not reveal any pathological history, particularly vascular. She was admitted to the emergency room for consciousness disorders with an intracranial hypertension syndrome, the clinical examination objective a normal blood pressure.

A cerebral scanner was requested, which showed a left parietal-temporal intra parenchymal hematoma with compression and subalpine cerebral herniation (Figure1).

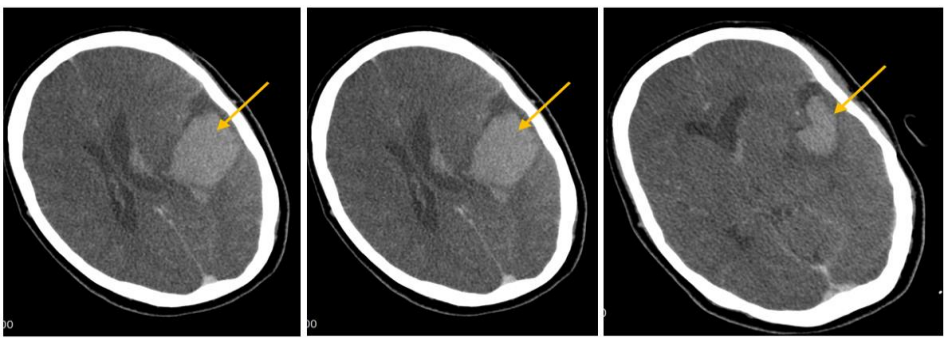

**Figure 1: Cerebral CT scan in axial section show a left parietal-temporal intra parenchymal hematoma with compression and subfalcine cerebral herniation**

The patient was then taken to the periinterventional suite for digital subtraction angiography (DSA) of the bilateral internal and external carotid arteries. Opacification of these different arteries shows the presence of a compact arteriovenous nidus with left parietal projection, fed by parietal branches of the homolateral middle cerebral artery.

Vascular void phenomenon at parenchymal time in the vicinity of the lesion in relation to the intraparenchymal hematoma. Venous drainage is towards the superior sagittal sinus via a dilated cortical vein (Figure 2).

The patient was proposed for surgery and a follow-up CT scan showed total regression of the intra parenchymal hematoma.

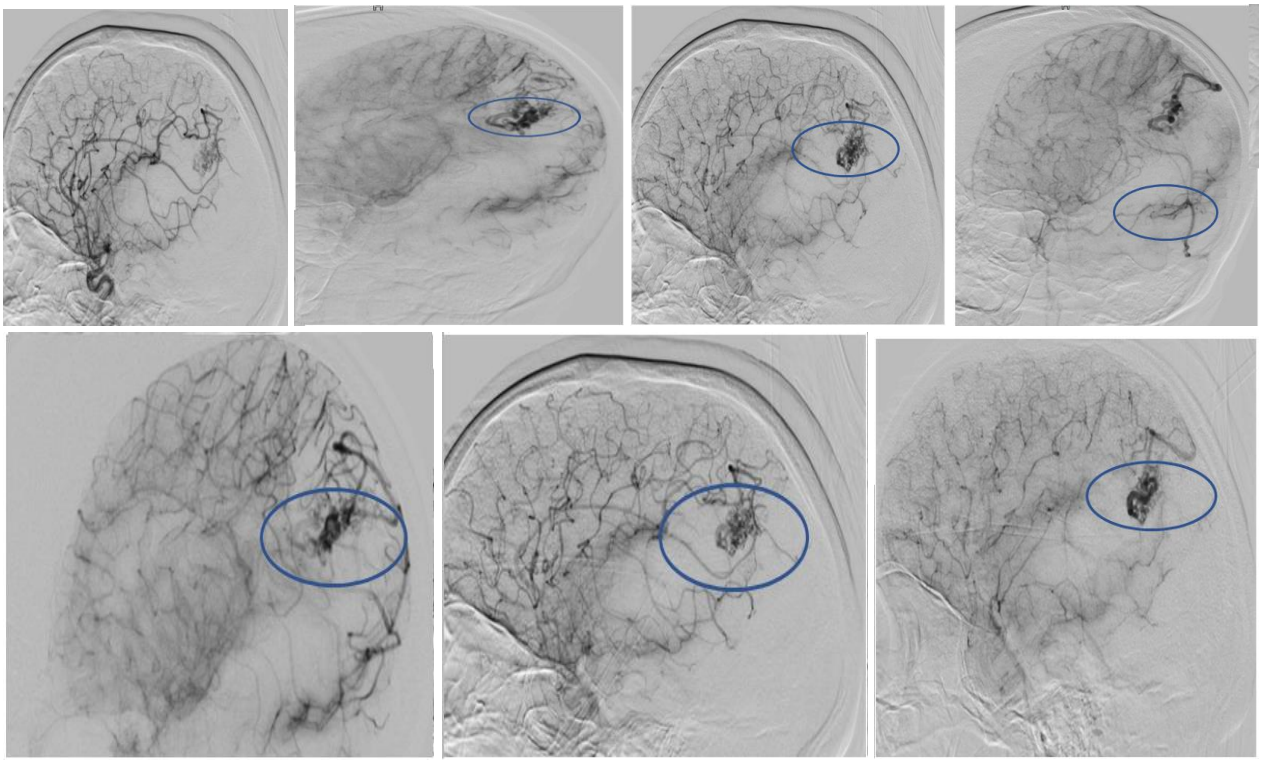

**Figure 2: Oblique and sagittal projection of a left internal carotid angiogram shows presence of a compact arteriovenous nidus with left parietal projection, fed by parietal branches of the homolateral middle cerebral artery**

**Vascular void phenomenon at parenchymal time in the vicinity of the lesion in relation to the intraparenchymal hematoma**

**Venous drainage is towards the superior sagittal sinus via a dilated cortical vein**

## DISCUSSION

Venous arterial malformation is a rare pathology. Its incidence and prevalence are therefore poorly understood. Available data are mainly from limited population-based studies and autopsy series. The latter would estimate the overall frequency of AVCs in the population at 4.3%. Its natural history is poorly understood [3, 4]. Over a period of 27 years, from 1965 to 1992, Brown *et al.*, reported 48 cases of intracranial vascular malformations, of which 26 were cerebral arteriovenous malformation (54%) and of these 22 patients were symptomatic [5]. The clinical symptomatology is dominated by hemorrhage, with seizures being less frequent, although this pattern tends to evolve [6]. The symptomatology in our patient was dominated by headache but with an inaugural seizure. Conventional brain magnetic resonance imaging sequences are used to locate the nidus in the brain parenchyma (T1 and T2 sequences), to analyze the morphology of the AVC and its relationship with neighboring brain structures (T1 gradient echo volume acquisition with gadolinium injection); to search for parenchymal abnormalities (T2 and FLAIR (fluid attenuated inversion recovery) sequences) and to detect bleeding (T2 gradient echo sequence) [7].

The most widely used system for the classification of cerebral arteriovenous malformation is the Spitzer-Martin system, which uses 3 criteria: the size of the nidus, the location of the AVC and the venous drainage. It assigns a score of 1 for small (<3 cm), 2 for medium (3-6 cm), and 3 for large (>6 cm) AVCs. The location of the nidus in a non-functional (0) or functional (1) territory. Venous drainage is scored as superficial only (0) or including drainage to the deep cerebral veins (1). The score obtained is used to divide

the AVCs into 3 classes and to guide the therapeutic attitude. Class A includes Spitzer-Martin grades I and II; class B includes grade III and class C includes grades IV and V [8]. Our patient belonged to group B. The decision to perform radiotherapy under stereotactic conditions was made. The scores for each feature are totaled to determine the grade. In the system described in this article, class A includes Spitzer-Martin grades I and II; class B includes grade III; and class C includes grades IV and V. Modified from Spetzler and Martin.

## CONCLUSION

Cerebral arteriovenous malformation is very rare. Intracerebral hemorrhage is the most serious complication. Magnetic resonance imaging allows the diagnosis to be evoked. Management is based on surveillance, microsurgery, endovascular treatment, and radiosurgery.

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