Case Report

**Spontaneous obliteration highlights the dynamic nature of cerebral arteriovenous malformations: A case report and review of the literature**

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**Abstract**

**Background:** Cerebral arteriovenous malformations (AVMs) are dynamic lesions and have been documented to arise de novo, enlarge, regress, obliterate, and even recur. Spontaneous obliteration of AVM is a rare and poorly understood phenomenon.

**Case Description:** We present a case of spontaneous obliteration of AVM in a 60-year-old gentleman who presented with intraparenchymal hemorrhage from a ruptured right parieto-occipital AVM. Angiography performed before gamma knife surgery 4 months after his initial presentation demonstrated complete absence of AVM.

**Conclusion:** In our center’s 20-year experience of treatment of cerebral AVMs (approximately 600 cases), this is the only case that has been aborted due to spontaneous obliteration leading us to infer that the incidence of spontaneous AVM obliteration is <1%. Spontaneous obliteration of AVM is a rare but well-established phenomenon that bears testimony to the dynamics of this vascular disorder.

**Key Words:** Cerebral arteriovenous malformation, dynamic lesion, spontaneous obliteration

**INTRODUCTION**

Gamma knife surgery is a well-established treatment modality for small and medium-sized cerebral arteriovenous malformations (AVMs). It has been used in Singapore since 1995, and around 600 AVMs have been treated. The following is the first case in our experience in which the AVM could not be visualized on stereotactic digital subtraction angiography in a patient scheduled for gamma knife surgery. This prompted us to explore the dynamics behind spontaneous obliteration of AVMs.

**CASE REPORT**

The patient is a 60-year-old gentleman who presented with acute intracerebral hemorrhage in the right parieto-occipital region [Figure 1]. Computer tomography angiography revealed an AVM in the right parietal lobe with a feeding vessel originating from the right posterior cerebral artery and a cortical draining vein leading into the superior sagittal sinus [Figure 2].

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Digital subtraction angiography performed 5 days after the initial hemorrhage revealed a 1.0 cm nidus of abnormal vessels, which was supplied by a single feeder from the right posterior cerebral artery and drained by a single vein into the superior sagittal sinus, confirming the presence of an AVM [Figure 3]. Digital subtraction angiography also confirmed fetal origins for both posterior cerebral arteries. The AVM was classified as a Spetzler-Martin Grade 2 AVM (size smaller than 3 cm, located in an eloquent region of the brain with superficial venous drainage).

The patient’s intracerebral hemorrhage was managed conservatively and he was discharged with minor deficit (left inferior quadrantanopia). He opted to treat his AVM with gamma knife surgery which was scheduled 4 months after his initial presentation. However, the stereotactic digital subtraction angiography performed just before gamma knife surgery planning demonstrated absence of the AVM nidus and resolution of the network of abnormal draining vessels [Figure 4]. Gamma knife surgery was aborted and the patient discharged. His visual deficit remains stable and he is well otherwise 18 months posthemorrhage.

**DISCUSSION**

The classic belief that all AVMs are congenital lesions arising from vascular dysgenesis during early fetal development has been challenged.[17] Recent evidence showing that they may develop later in life has been published.[9] Interval enlargement,[16] self-remodeling,[16] and spontaneous obliteration[1,3,12,19,21] of these lesions in children and adults bear testimony to their dynamic nature and suggest that a complex interplay of both mechanical and genetic factors influence the natural history of these vascular malformations.

Spontaneous obliteration is defined as the disappearance of an AVM in the absence of any intervention or therapy.[3] Spontaneous obliteration of an AVM is a rare but well-recognized phenomenon with a suggested prevalence of 0.3–1.3%.[1,3,12,19,21] In our center’s 20-year experience of treatment of cerebral AVMs (approximately 600 cases), this is the only case that has been aborted due to spontaneous obliteration leading us to infer that the incidence of spontaneous AVM obliteration is <1%.

Current hypotheses for spontaneous AVM obliteration are centered on the interruption of arterial feeders and venous outflow occlusion. Compressive effects of mass lesions, such as intracerebral hematomas due to AVM rupture,[8,10,20,22,26,27] postsurgical brain swelling,[14] and posthemorrhage gliosis,[10] have been associated with spontaneous obliteration of AVM. Other events linked with spontaneous obliteration of AVM include premature atherosclerosis,[2,11,14,23] embolic phenomenon causing occlusion of feeders,[11,25] turbulence of flow within feeders causing endothelial shearing and thrombus formation,[7] hypercoagulable states,[24] and genetic disturbances in angiogenesis pathways, such as in cases associated with hereditary hemorrhagic telangiectasia.[5,6,13] Spontaneous AVM obliteration is also associated with certain angiographic features, including a small nidus, single feeding artery, and solo draining vein.[14,21] As the proposed mechanisms of spontaneous obliteration are centered on intercepting AVM blood flow, AVMs with single entry and exit channels may thus be
the easiest to occlude. Furthermore, there are reports of AVM recurrence after spontaneous obliteration.\(^{1,13,15,21}\)

**CONCLUSION**

Spontaneous obliteration of AVM is a rare but well-established phenomenon that bears testimony to the dynamics of this vascular disorder.

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**Conflicts of interest**

There are no conflicts of interest.

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