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CASE REPORT

Revisiting normal perfusion pressure breakthrough in light of hemorrhage-induced vasospasm

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Abstract

Cerebral arteriovenous malformations (AVMs) have abnormally enlarged arteries and veins prone to spontaneous hemorrhage. Immediately following surgical excision of a cerebral AVM, even normal brain tissue surrounding the lesion is subject to hemorrhage, a phenomenon termed normal perfusion pressure breakthrough (NPPB) syndrome. According to this theory, arteries supplying cerebral AVMs become dilated and lose their capacity to dilate or constrict to autoregulate pressure. Acutely after removal of a cerebral AVM, excessive blood pressure in these arterial feeders can cause normal brain tissue to bleed. However, this theory remains controversial. We present a patient with a cerebral AVM that demonstrated cerebrovascular reactivity and argues against an assumption underlying the theory of NPPB syndrome.

Key words: Arteriovenous malformation; Autoregulation; Normal perfusion pressure breakthrough; Subarachnoid hemorrhage; Vasospasm

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INTRODUCTION

Cerebral arteriovenous malformations (AVMs) have abnormally enlarged arteries and veins prone to spontaneous hemorrhage. Immediately following surgical excision of a cerebral AVM, the remaining normal brain tissue surrounding the lesion is subject to hemorrhage, a phenomenon termed normal perfusion pressure breakthrough (NPPB) syndrome[1]. The theory proposed to explain this phenomenon remains controversial. We present a patient with cerebral AVM that demonstrated cerebrovascular reactivity and argues against an assumption underlying the theory of NPPB syndrome.

CASE REPORT

A 47 year-old woman with chronic headaches and a previously diagnosed right occipital cerebral AVM developed a severe headache followed 5 d later by left-sided weakness and neglect. Computed tomography and magnetic resonance imaging brain scans showed subacute subarachnoid hemorrhage and ischemic changes in the right middle cerebral artery distribution (Figure 1A and B).

Catheter arteriography demonstrated a 12 mm an-

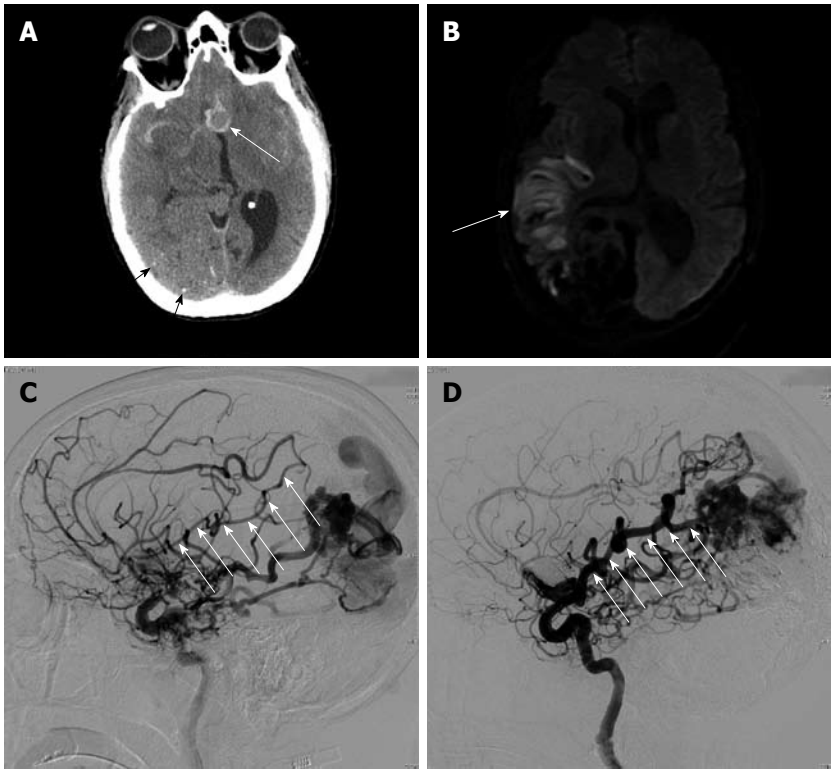

Figure 1 A 47-year-old woman with ruptured anterior communicating artery aneurysm associated with a large arteriovenous malformation. A: Computed tomography brain scan shows the 12 mm anterior communicating artery aneurysm in relief surrounded by high-density subarachnoid hemorrhage (white arrow). Also note small stippled calcifications in the large parietal-occipital arteriovenous malformation (black arrows); B: Diffusion-weighted magnetic resonance imaging shows area of restricted diffusion in the posterior right frontal lobe (white arrow) corresponding with the patient's left-sided weakness and hemineglect; C: Catheter arteriography of the right internal carotid artery in the lateral projection at the time of initial presentation approximately 1 wk following subarachnoid hemorrhage shows significant narrowing in the angular and Rolandic branches of the right middle cerebral artery, both of which supply the arteriovenous malformation (white arrows); D: Catheter arteriography of the right internal carotid artery in the lateral projection 4 mo after initial presentation and following complete neurological recovery shows resolution of vasospasm in the angular and parietal branches of the right middle cerebral artery supplying the arteriovenous malformation (white arrows).

terior communicating artery aneurysm, a 5 cm right occipital cerebral AVM, and relatively slow flow through small caliber arteries to the cerebral AVM representing vasospasm (Figure 1C). Endovascular coil occlusion of the aneurysm was immediately performed, and the vasospasm was treated with verapamil. Subsequent arteriography for treatment of the cerebral AVM showed interval resolution of the narrowed arteries in the distribution of the stroke (Figure 1D). Despite her deficits at the time of original presentation with stroke, the patient made a full neurological recovery, even after embolization and resection of the cerebral AVM.

DISCUSSION

Cerebral AVMs can cause pathological states including hemorrhage, vascular steal, chronic hypoperfusion, and low-grade ischemia, all of which were demonstrated by the patient reported here. In 1978, Spetzler *et al*[1] published the theory of NPPB, positing that chronic hypoperfusion causes branches of the AVM feeding arteries, that supply normal tissue, to become markedly dilated, remaining paralyzed due to lost autoregulatory capacity. After AVM extirpation, these branches in the remaining normal tissue experience increased perfusion but lack ability to constrict and autoregulate cerebral blood flow. They argued that, in some cases, this vascular dysfunction leads to hyperemia and compromise of capillary beds, resulting in massive edema and hemorrhage.

Several studies using various methodologies have supported the claims of NPPB[2-9]. However, more recent investigations contradict many aspects of the theory, casting doubt on the link between impaired vasoreactivity and postoperative complications. NPPB theory rests on the assumption that lost autoregulatory capacity persists following resection of a cerebral AVM. Nevertheless, several studies demonstrate a postoperative return to normal CO_2 reactivity in vessels with previously diminished reactivity[3,10-16]. Young *et al*[12,13] showed intact CO_2 reactivity both before and after resection. Similarly, Ogasawara demonstrated postoperative hyperperfusion in areas with normal response to acetazolamide in the preoperative period[17].

Young *et al*[18] reported that cerebral AVM removal improved perfusion in the hemisphere ipsilateral to the lesion, but increasing mean arterial pressure pharmacologically did not increase CBF, suggesting intact cerebral autoregulation. This was true for uncomplicated cases, as well as patients with NPPB-like complications. Demonstrating intact vasoreactivity both before and after resection, Young *et al*[13,18,19] suggested the true problem is a leftward shift of the autoregulatory curve such that normal pressure exceeds the upper limit of autoregulation.

Batjer demonstrated the ability of arteries to vasodilate but an inability to vasoconstrict in response to increased pressure, a finding confirmed in a rat model[11,20,21]. We suggest that the vasoconstriction in response to SAH in our patient demonstrated that the impairment of vasoconstriction in response to elevated pressure, which has been argued to underlie NPPB syndrome, is not due to some global inability of the vasculature to constrict. This agrees with both Batjer's findings and Young's proposal of a leftward shift of the autoregulation curve. However, we must maintain the possibility that vasospasm in response to SAH and the vessel properties underlying autoregulation, which have been argued are impaired in NPPB-like states, might result from two distinct mechanisms.

Another condition of Spetzler's theory that has come under scrutiny is that NPPB-like complications occur in areas adjacent to the malformation that formerly shared the arterial blood supply with it, and that such former feeders are prone to complications because of the chronic stress placed upon them by the cerebral AVM. Barnett *et al*[3] showed the worst steal effect exists in tissue 2-4 cm from the malformation, and higher flow occurred in tissue distal to the location of the cerebral AVM. To the contrary, Young *et al*[22] showed that increased cerebral blood flow following cerebral AVM resection occurs throughout the entire brain, not just in regions that shared vascular supply with the cerebral AVM. This suggests that mechanisms implicating preoperative focal hypoperfusion due to vascular steal are not the sole cause of postoperative complications. Indeed, our patient demonstrated vasospasm, infarction, and edema both ipsilateral and contralateral to her AVM, consistent with such findings.

The findings in this case illustrate that arteries surrounding cerebral AVMs can maintain vasoconstrictive capacity, although the contractile activity in response to subarachnoid hemorrhage and autoregulation might be different processes. Future studies should compare microvascular changes between procedures with uneventful cerebral AVM resection and those with these complications. However, comparison will prove difficult given the relative rarity of these complications and the resultant difficulty designing adequately powered investigations. Furthermore, better diagnostic definitions for these complications must be standardized. Differing interpretations of what constitutes an NPPB-like state have undoubtedly made studies difficult to compare. Better characterization of blood flow parameters surrounding cerebral AVMs will lead to improved prevention, prompt identification, and treatment.

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