

**Postoperative Hyperperfusion Associated With Steal Phenomenon Caused by a Small Arteriovenous Malformation**

—Case Report—

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**Abstract**

A 41-year-old woman presented with a small occipital arteriovenous malformation (AVM) manifesting as headache. Cerebral angiography showed an AVM in the right occipital lobe fed by the right temporooccipital artery and draining into the superior sagittal sinus and right transverse sinus. Single photon emission computed tomography showed the steal phenomenon in the ipsilateral temporal cortex fed by the main feeding artery preoperatively, and hyperperfusion in the same cortex after removal of the AVM. Postoperative systolic blood pressure was maintained between 100 and 120 mmHg to avoid disastrous hemorrhagic complications. Cerebral blood flow evaluation before and after surgery is important to avoid postoperative disastrous complications even in patients with small AVM.

Key words: cerebral arteriovenous malformation, steal phenomenon, postoperative hyperperfusion, cerebral blood flow, single photon emission computed tomography

**Introduction**

Arteriovenous malformations (AVMs) are congenital vascular malformations that cause direct arteriovenous shunts.4,12) Arterial blood is shunted through the low-resistance arteriovenous fistula of the AVM away from the normal brain tissue adjacent to and distant from the AVM, widely known as the steal phenomenon.4,7,14) AVMs are generally associated with large nidus, massive blood shunting, impaired autoregulation, venous congestion, and hyperperfusion in the surrounding tissues, which are all potential causes of postoperative hyperperfusion, resulting in complications such as cerebral edema and intracerebral hemorrhage.1,2,12) Hemodynamic changes associated with an AVM with a relatively small nidus, which may cause disastrous postoperative complications, are not well understood.

We report a case of a small AVM, which caused local cerebrovascular hemodynamic changes in the remote cortex fed by the main feeding artery before and after removal, and discuss the mechanism of hemodynamic changes and the importance of cerebral blood flow (CBF) evaluation before and after surgery to prevent postoperative complications.

**Case Report**

A 41-year-old woman presented with headache. Head computed tomography (CT) revealed a high density mass in the right occipital lobe (Fig. 1A). T2-weighted magnetic resonance imaging revealed abnormal signal voids in the right occipital lobe but no abnormal findings such as brain atrophy and edema in the right temporal lobe (Fig. 1B). Cerebral angiography demonstrated an AVM of 20 mm maximal diameter in the right occipital lobe, which was mainly fed by the right temporooccipital artery and drained into the superior sagittal sinus and right transverse sinus, classified as Spetzler-Martin grade...
Fig. 1 A: Preoperative computed tomography scan of the head showing a high density mass in the right occipital lobe. B: T2-weighted magnetic resonance image revealing an abnormal signal void in the right occipital lobe.

Fig. 2 A: Preoperative right carotid angiogram, lateral view, showing an arteriovenous malformation (AVM) mainly fed by the right temporooccipital artery and draining into the right transverse sinus. B: Right carotid angiogram 1 week after surgery showing marked stagnation of the right temporooccipital artery after resection of the AVM.

The conventional concept of the steal phenomenon associated with AVM is as follows. The massive arteriovenous shunt decreases the arterial blood pressure in the surrounding tissue, resulting in autoregulatory arteriolar vasodilation to maintain regional CBF. However, autoregulatory compensation will become insufficient with a greater decrease in pressure, and a low perfusion area will develop in the surrounding tissue. The mechanism of the steal phenomenon associated with AVM remains controversial.

Positron emission tomography and PtO2 electrode studies showed that the mechanism to compensate for low cerebral perfusion pressure involves reduced glucose and oxygen metabolism without increased oxygen extraction in the perilesional brain tissue.3,5 There is solid evidence that AVM-induced reduction of cerebral perfusion pressure does not cause maximum reduction of cerebrovascular resistance in the adjacent cortex because further arteriolar relaxation was possible in all patients with AVM.6,10 These observations are consistent with the hypothesis of adaptive autoregulatory displacement, that is, the lower limit of autoregulation shifts to a lower mean pressure in the chronic hypoperfusion area.15 Increased capillary density may be another structural mechanism of adaptation.13 Therefore, steal should possibly not be defined as cortical ischemia as a result of arterial hypotension.9 Common clinical steal phenomena...
such as progressive focal neurological deficits or impairment of higher cognitive functions are rather a consequence of neuronal deafferentation and diaschitic phenomena in distant regions of the brain. In our case, preoperative SPECT of the low perfusion area in the remote cortex fed by the main feeding artery demonstrated normal vasoreactivity to acetazolamide. Our observations are consistent with other investigations\(^{10,15}\) that found the hypoperfusion area maintained the autoregulatory system and was not in misery perfusion. In addition, the anatomical factor of poor collateral flow in the temporal lobe is involved with the asymptomatic steal phenomenon in the ipsilateral temporal lobe.

Areas of postoperative abnormal hyperperfusion are associated with areas of preoperative abnormal hypoperfusion. Two hypotheses for the cause of brain edema and hemorrhage during or after removal of the AVM have been proposed: normal perfusion pressure breakthrough and occlusive hyperperfusion.\(^{11}\) The normal perfusion pressure breakthrough theory suggests that postoperative edema and hemorrhage are caused by a failure in autoregulation in the ischemic brain around the AVM.\(^{4}\) However, vasomotor paralysis is not responsible for this phenomenon and uncoupling of the CBF and metabolism is more likely to be the cause of reperfusion injury.\(^{9,15}\) Our case of AVM manifested as hypoperfusion with normal vasoreactivity before surgery and stagnation of the main feeding artery with hyperperfusion after surgery. Strict blood pressure management and hypothermal treatment during and after the surgery, preoperative embolization, and staged operation are recommended to prevent complications.\(^{8,11,12}\) In our case, adequate blood pressure management during and after operation avoided disastrous injury in spite of the postoperative hyperperfusion.

The present case suggests CBF evaluation before and after surgery using SPECT and strict control of blood pressure in the postoperative period are important to prevent disastrous complications even in cases of small AVM.

References

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