

# Large cerebral arteriovenous malformation presenting with venous ischemia in the contralateral hemisphere

## Case report

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✓ The authors report a rare case in which a large cerebral arteriovenous malformation (AVM) located in the left parietooccipital region presented with venous ischemia in the contralateral hemisphere. A 74-year-old man was admitted to the hospital because he was experiencing a loss of appetite, disorientation, and left hemiparesis. Computerized tomography scans revealed a low-density area in the right temporal lobe. Angiography demonstrated a large AVM in the left parietooccipital lobe and dilation, stagnation, and meanders of cortical veins in the contralateral hemisphere. The authors speculated that the elevated sinus pressure caused by a huge venous return of blood from the AVM produced venous ischemia in the contralateral hemisphere.

**KEY WORDS** • arteriovenous malformation • venous ischemia • embolization

**A**LTHOUGH patients with AVMs can present with hemorrhage or seizure,<sup>1,7</sup> symptomatic venous ischemia is a very rare clinical condition<sup>4,6</sup> that is caused by arterial blood shunting into a major sinus, which impedes venous return of blood flow. We report the case of a patient suffering from a large cerebral AVM and a venous ischemic complication in the contralateral hemisphere; the arterial blood shunting into the SSS caused an elevation of sinus pressure and, consequently, disturbed the venous return of blood flow from the contralateral cortical hemisphere.

### Case Report

*History.* Seventeen years before admission this 74-year-old man was found to harbor a large AVM in the left parietooccipital lobe, which presented with intracerebral hemorrhage. External decompression, removal of the hematoma, and partial occlusion of the parent artery were performed. After surgery the patient suffered from mild right hemiparesis. From March 1995 onward, he often displayed general convulsions for which he received medication. Computerized tomography scans detected no new lesion.

*Examination.* On March 26, 1999, this patient was admitted to our hospital because he was experiencing a loss of appetite, disorientation, and left hemiparesis. A CT scan obtained at that time revealed a newly developed

*Abbreviations used in this paper:* AVM = arteriovenous malformation; CA = carotid artery; CT = computerized tomography; MR = magnetic resonance; SSS = superior sagittal sinus.

low-density area in the right temporal lobe that appeared to be unrelated to the unchanged large AVM in the left parietooccipital region (Fig. 1). A high-intensity area was revealed on both T<sub>1</sub>-weighted and T<sub>2</sub>-weighted MR images (Fig. 2).

Conventional angiography demonstrated a large AVM (6 × 7 × 6 cm) in the left parietooccipital region. The lesion was supplied by the left anterior, middle, and posterior cerebral arteries, the superficial temporal artery, and the middle meningeal artery. Venous drainage was primarily superficial to the SSS, with some deep drainage to the vein of Galen. Compared with the last angiogram that had been obtained in this patient (November 16, 1998, Fig. 3), the size of the AVM was unchanged. During the venous phase, angiograms of the left CA revealed arterial blood shunting into the SSS and the SSS was not occluded (data not shown). Remarkably, right CA angiograms revealed dilation, stagnation, and meanders of cortical veins in the contralateral hemisphere (Fig. 4).

We speculated that the arterial blood that shunted into the SSS caused an elevation of sinus pressure and, consequently, disturbed the venous return of blood from the contralateral cortical hemisphere. The patient was treated with transarterial embolization by using ethanol (100%) and *N*-butyl cyanoacrylate from the left middle and posterior cerebral arteries. Although we could not successfully complete the occlusion, partial embolization of the nidus reduced the cerebral blood flow to the AVM and the transitional time during the venous phase was improved. Two months after embolization, there was no sign of the lesion on either CT or MR images (Fig. 5), although the AVM

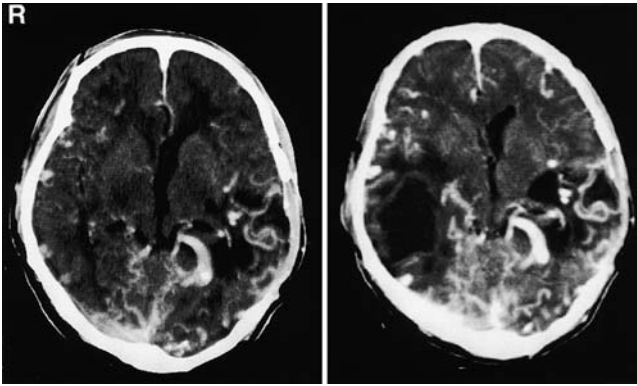


FIG. 1. Computerized tomography scans obtained 2 years before admission (*left*) and at admission (*right*) revealing a large AVM in the left parietooccipital region that remains unchanged although a new low-density area appears in the right temporal lobe (*right*).

had not been eliminated. The patient's left hemiparesis was improved and, moreover, his seizures were controllable by anticonvulsant medications. Although we had intended to perform embolization of the left external feeding artery, the patient left our hospital in response to his family's wishes.

### Discussion

Arteriovenous malformations cause hemorrhage, seizure, and focal neurological deficits.<sup>1,7</sup> The mechanisms behind these effects can be classified into three types: arterial contribution, nidus, and venous hypertension or congestion. Lasjaunias, et al.,<sup>7</sup> reviewed possible vascular mechanisms that may cause neurological deficits from AVMs involving the dura mater, but theirs is not a report of pial AVMs. Those authors indicate that the insignificant arterial contribution from the internal CA or vertebral artery circulations to dural AVMs cannot be considered to have any hemodynamic consequences for the CNS. They suggest instead that any evidence of CNS dysfunction associated with dural AVMs appears to be related to venous hypertension or congestion.<sup>9</sup> Potential mechanisms of venous dysfunction are classified as those occurring secondary to venous thrombosis, high venous pressure

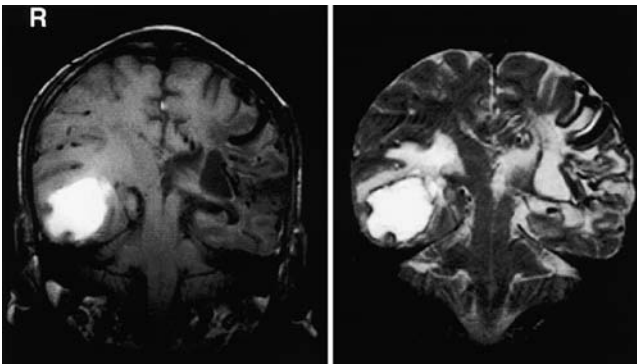


FIG. 2. Magnetic resonance T<sub>1</sub>-weighted (*left*) and T<sub>2</sub>-weighted (*right*) images revealing a new lesion in the right temporal lobe.

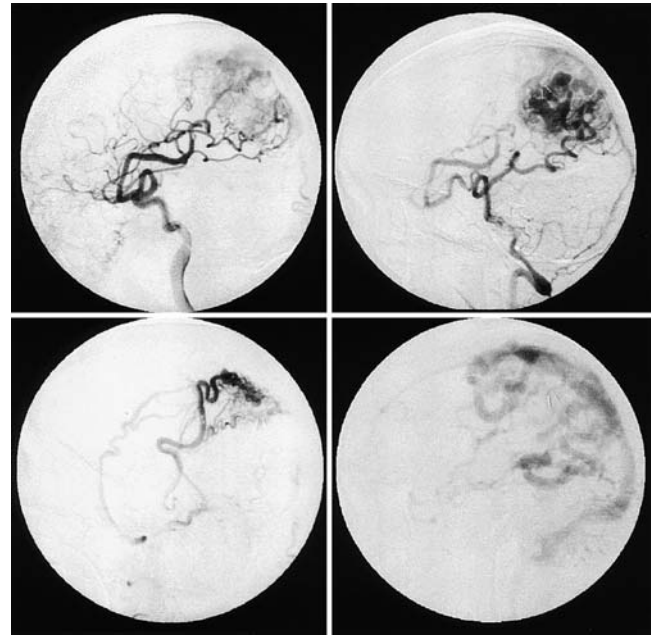


FIG. 3. Angiograms obtained 5 months before admission demonstrating the large AVM, which is fed by the left anterior, middle, and posterior cerebral arteries, the superficial temporal artery, and the middle meningeal artery. These lateral views show the left CA during the arterial phase (*upper left*), the left vertebral artery (*upper right*), the left external CA (*lower left*), and the left CA (*lower right*) obtained during the venous phase.

resulting in either increased SSS pressure and increased intracranial pressure or venous ischemia, venous mass effect, or venous rupture with hemorrhage.<sup>2,8,9</sup>

In our case, angiography demonstrated that the SSS was not occluded. We could therefore rule out the presence of venous thrombosis. Venous ischemia appears to be related to passive congestion due to increased retrograde venous pressure toward venous drainage routes and venous rupture causing cerebral hemorrhage. We speculated that, in this case, similar to cases of dural AVMs, the shunting of arterial blood into the SSS caused elevation of sinus pressure and, consequently, disturbed the venous return of blood from the contralateral cortical hemisphere.

Kamite and colleagues,<sup>5</sup> reported the case of a patient in whom pial AVMs presented with benign intracranial hypertension due to venous ischemia. Kurata and coworkers

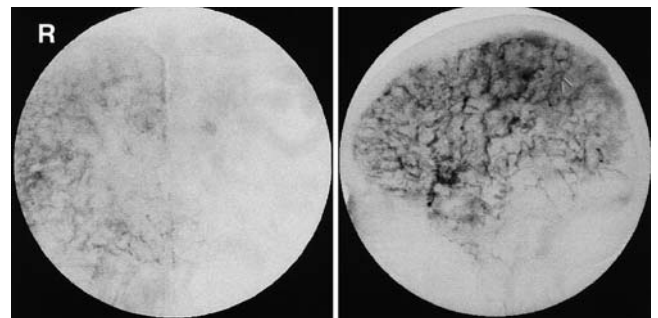


FIG. 4. Anteroposterior (*left*) and lateral (*right*) angiograms of the right CA obtained during the venous phase on admission, revealing congestive cortical veins.

## Large arteriovenous malformation presenting with venous ischemia

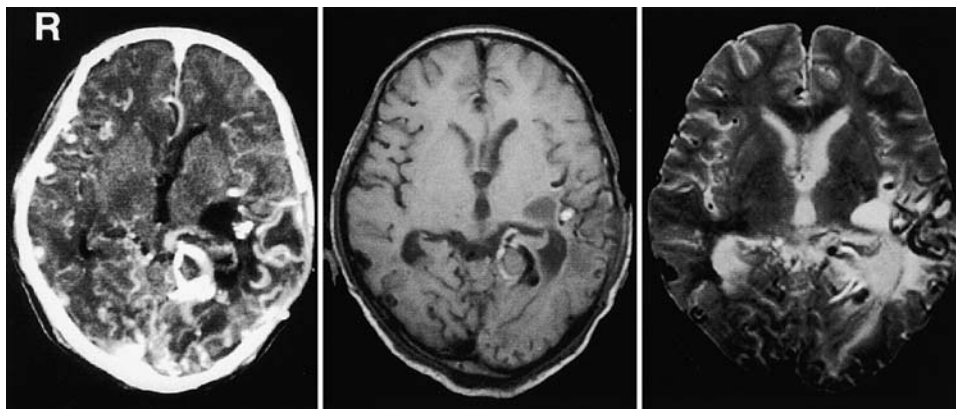


FIG. 5. Contrast-enhanced CT scan (*left*), T<sub>1</sub>-weighted MR image (*center*), and T<sub>2</sub>-weighted MR image (*right*) obtained 2 months after embolization, confirming that the venous ischemic lesion has vanished, although the AVM has not been eliminated.

ers,<sup>6</sup> and Inafuku, et al.,<sup>3</sup> reported on patients with tentorial dural AVMs that were associated with visual hallucinations and quadrant hemianopsia. In patients with dural AVMs, venous ischemia may often produce a neurological deficit; however, cases of pial AVMs in which ischemia occurs are rare. Moreover, a pial AVM that presents with venous ischemia in the contralateral hemisphere is extremely rare; to our knowledge there is no reported case in the literature.

In the patient reported on by Kamite and colleagues,<sup>5</sup> a pial AVM presented with benign intracranial hypertension. Although the underlying mechanism in such a case is unknown, there may be several factors: a large AVM; high blood flow; and drainage into the major sinus. In the present case, the AVM was large (6 × 7 × 6 cm) and there was a high flow of blood, which drained into the SSS.

The treatment of such a lesion is total surgical excision, intravascular embolization, radiosurgery, or combination therapy. In patient reported on by Kamite, et al.,<sup>5</sup> surgical excision of the AVM effectively reduced intracranial hypertension. Surgical excision of AVMs, if it can be accomplished with low risk, is thus the best treatment to decrease intracranial hypertension and venous ischemia. Some AVMs, however, are too large to be treated surgically. Kurata and coworkers,<sup>9</sup> and Inafuku, et al.,<sup>3</sup> reported cases in which the patient was treated with transarterial embolization. Preoperative embolization that is performed to decrease flow through the AVM also enhanced the open surgical approach. These treatments resulted in marked clinical improvement in the patient. In our case, the AVM was so large that we could not perform the operation and, thus, transarterial embolization was performed. Although we could not successfully achieve complete occlusion, partial embolization of the nidus reduced cerebral blood flow to the AVM. We wish to emphasize the importance of early diagnosis achieved using angiography and of appropriate therapy for patients with venous ischemia. If treated at an early stage, venous ischemia may be reversible.<sup>9</sup>

### Conclusions

In this paper we have presented a rare case in which the patient harbored a large cerebral AVM in the left pari-

etooccipital region accompanied by venous ischemia in the contralateral hemisphere. We speculate that the elevated sinus pressure caused by the huge venous return of blood from the AVM caused venous ischemia in the contralateral region. The prompt reversal of both focal and more generalized signs after endovascular therapy highlights the role of embolization in the management of neurological deficits secondary to elevated cerebral venous pressure.

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Manuscript received February 7, 2001.

Accepted in final form May 13, 2002.

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