

## Venous ischemia caused by dural arteriovenous malformation

### Case report

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✓ A case is presented of tentorial dural arteriovenous malformation (AVM) associated with visual hallucinations and quadrant hemianopsia. Computerized tomography (CT) and magnetic resonance imaging showed an ischemic region, mainly affecting the white matter of the right occipital lobe, that was defined as an area of increased blood volume on dynamic CT scans and as a decrease in cerebral blood flow on N-isopropyl-p-<sup>123</sup>I-iodoamphetamine single-photon emission CT scans. Angiography demonstrated venous congestion, probably because the retrograde arterial inflow from the dural AVM into the corticomедullary vein was direct and not via the sinuses. The symptoms and radiological findings improved immediately after endovascular treatment. The origin of these symptoms was fully evaluated and confirmed to be a reversible ischemic change caused by disturbance of the volume of venous return over an extensive area.

**KEY WORDS** • dural arteriovenous malformation • venous hypertension • venous ischemia

**H**EMORRHAGE is a well-known and dangerous complication in patients with dural arteriovenous malformations (AVM's),<sup>7,11,14,17,20</sup> especially in parts of the dura mater unrelated to the sinus.<sup>7,14</sup> However, venous ischemia<sup>2,4-6,9,10,13,15</sup> is not a common complication, and few reports have attempted to evaluate it fully. The criteria of venous ischemia are as follows: 1) angiograms showing venous congestion that can account for the clinical symptoms; 2) other radiological findings demonstrating that the ischemic lesion, mainly affecting the white matter, is caused by a disturbance of the venous circulation; 3) clinical symptoms and radiological findings revealing improvement immediately after appropriate early-stage treatment, indicating a reversible change and not an infarct; and 4) alternative mechanisms, such as brain compression and arterial steal with ischemia, do not adequately explain the relationship between clinical symptoms and radiological findings.

### Case Report

This 65-year-old man was admitted to our department complaining of frequent visual hallucinations that

had persisted for 2 days. Neurological examination on admission showed only left lower-quadrant hemianopsia. A full-day electroencephalographic recording showed low voltage at the right temporo-occipital lobe, but no paroxysmal discharge.

Plain computerized tomography (CT) scans revealed a low-density area in the right occipital lobe with edema, mainly affecting the white matter (Fig. 1 *left*). Contrast-enhanced CT scans disclosed patches of abnormal enhancement in the medial part of the low-density area, suggesting dilated vascular channels (Fig. 1 *center*). Dynamic CT scans showed an increase in blood volume in the low-density area (Fig. 1 *right*). Magnetic resonance imaging demonstrated an abnormal flow-void area and edema (Fig. 2 *left*). Internal carotid and vertebral angiograms of the venous phase showed marked irregularly shaped corticomедullary veins in the right occipital lobe, strongly suggesting venous congestion. All venous sinuses and both jugular veins were patent. Only external carotid angiography demonstrated a dural AVM fed by the middle meningeal, occipital, and posterior auricular arteries in the right cerebellar tentorium and drained by a dilated cor-

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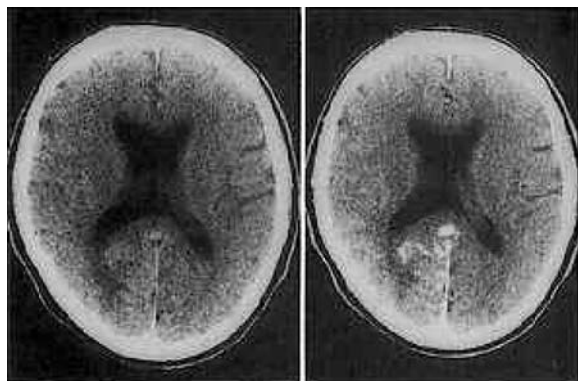


FIG. 1. Computerized tomography (CT) scans in a patient with a dural arteriovenous malformation. *Left:* Plain CT scan showing a low-density area in the right occipital lobe, mainly affecting the white matter. *Center:* Contrast-enhanced CT scan revealing abnormal enhancement in the medial part of the low-density area. *Right:* Dynamic CT scan showing an increase in blood volume in the right occipital lobe (3) as compared with the left lobe (2).

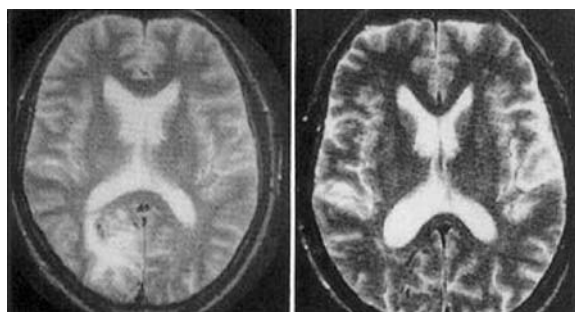
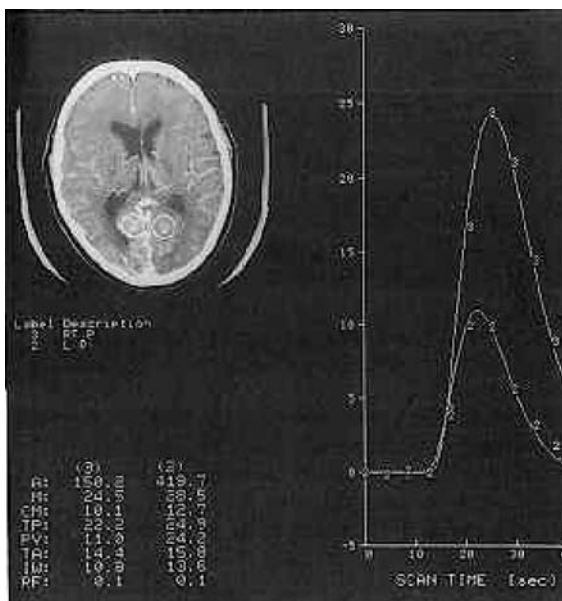


FIG. 2. *Left:* Pretreatment T<sub>2</sub>-weighted magnetic resonance (MR) image (TR 3000 msec, TE 110 msec) showing abnormal flow-void area and edema in the white matter. *Right:* Posttreatment MR image. The abnormal flow-void area and the hyperintense area in the white matter have disappeared.

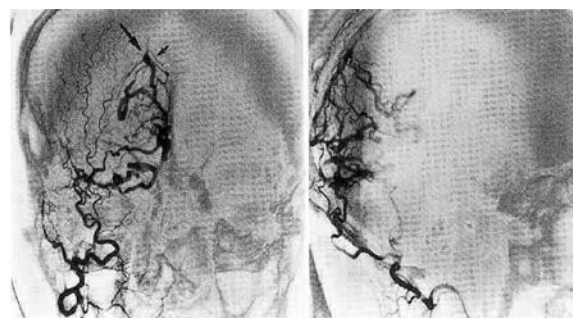


FIG. 3. Subtracted right occipital angiograms, anteroposterior (*left*) and lateral (*right*) views, showing a dilated draining vein narrowing abruptly (*large arrow*) before entering the superior sagittal sinus (*small arrow*). Retrograde multiple venous drainage into normal cortical veins and the vein of Galen via a dilated draining vein are clearly seen.

tical vein. This dilated vein narrowed abruptly near the superior sagittal sinus, then flowed into it. Multiple retrograde venous drainage routes into the normal cortical veins and the vein of Galen via a dilated draining vein were observed (Fig. 3). N-isopropyl-p-<sup>123</sup>I-iodoamphetamine single-photon emission CT disclosed an area of hypoperfusion in the right occipital lobe; a late-phase scan showed reduction of this area (Fig. 4 upper).

The patient was treated with transarterial embolization using polyvinyl alcohol particles (150 to 250  $\mu$ ) and microfibrillar collagen (Fig. 5). His visual hallucinations disappeared immediately, and quadrant hemianopsia and radiological findings improved over a period of 1 month (Figs. 2 right and 4 lower). The clinical course has been uneventful during the 1-year follow-up evaluation.

## Discussion

Stenosis or obstruction of venous drainage systems is recognized to be a cause of venous hypertension.<sup>11,16,22</sup> However, it is not as well known that arterial flow into the venous channels is another important cause. In cases of dural AVM, there are three routes for such inflow: direct antegrade inflow, direct retrograde inflow, and indirect retrograde inflow via a sinus. Antegrade anterior inflow into the normal venous channels is usually observed in cases of cerebral AVM and often in cases of dural AVM, especially when the AVM is located in the dura mater and is not related to the dural sinus. Direct angiographic antegrade flow into the venous channels is considered to be a dangerous condition because it frequently leads to hemorrhage due to venous hypertension.<sup>2,7,14</sup> However, there are few re-

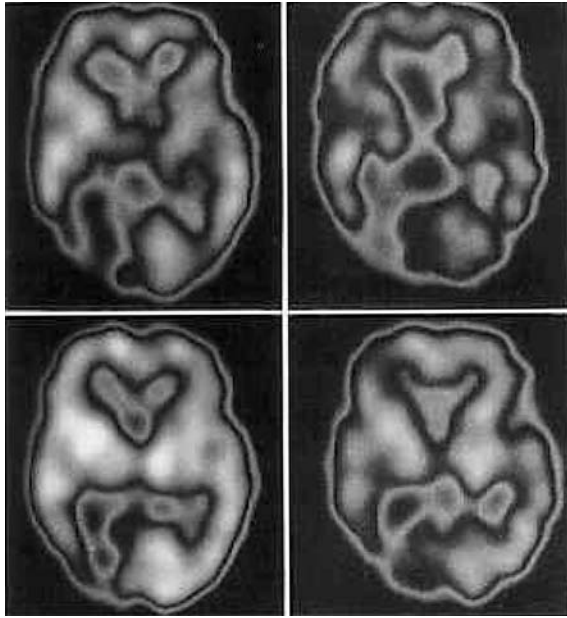


FIG. 4. Early- (left) and late- (right) phase N-isopropyl-p-<sup>123</sup>I-iodoamphetamine single-photon emission computerized tomography (SPECT) scans. Upper: Before treatment, hypoperfusion is apparent in the right occipital lobe in the early-phase scan (left); the late-phase scan (right) shows reduction of this hypoperfusion area. Lower: Both posttreatment SPECT scans show improvement of the hypoperfusion.

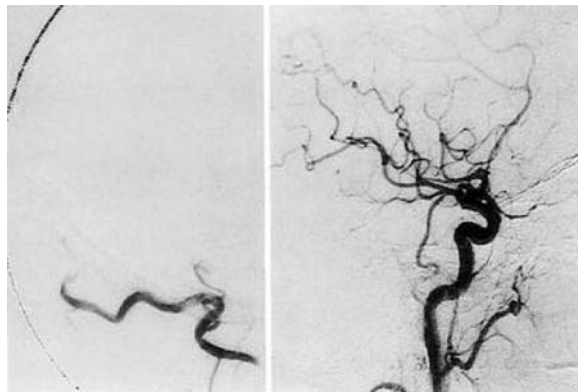


FIG. 5. Posttreatment right occipital angiogram (left) and right common carotid angiogram (right) demonstrating no evidence of the dural arteriovenous malformation.

ports that attribute cerebral focal signs related to venous ischemia to antegrade arterial inflow into the cortical venous drainage.

Retrograde cortical venous drainage has two routes: direct<sup>4-6,9</sup> and indirect.<sup>12,13,17,29</sup> Both have been reported to be dangerous conditions<sup>1,8,11</sup> and are often noted in cases of dural AVM with hemorrhage.<sup>17,20</sup> Moreover, the venous hypertension caused by retrograde venous flow reportedly produces venous ischemia with unusual manifestations.<sup>2,9-11,13,15</sup> Retrograde arterial flow

into the venous channels produces more severe disturbances of the venous circulation of the brain than the antegrade type.

The cause of venous ischemia in the present case was probably direct retrograde arterial flow into both superficial and deep venous systems. The high pressure of direct and retrograde arterial flow into the main venous systems is thought to be an important cause of venous ischemia. Narrowing of the draining cortical vein near the venous sinus caused direct retrograde venous drainage without stenosis or obstruction of the venous sinuses in the present series. Only Halbach, *et al.*,<sup>9</sup> have reported a similar case, in which the connection between the draining vein and the sinus was occluded. The cause of retrograde venous drainage despite the presence of patent venous sinuses has not been previously discussed,<sup>9,15</sup> and cases similar to ours may have been overlooked. The etiology of the abrupt narrowing of the draining vein was not clear. One possibility is that the draining vein became severely stenosed as a result of venopathy<sup>3,16,18,19,22</sup> caused by shearing stress due to long-term turbulent and high-pressure flow of arterial blood. Another explanation is that the collateral channels opened after severe stenosis or obstruction caused by venopathy or thrombosis.

Venous ischemia is reported to manifest symptoms of an unusual nature,<sup>2,4-6,9,11,13,15,23</sup> as in the present case. These symptoms include fluctuating or progressive focal signs, such as motor weakness,<sup>4-6,9,23</sup> aphasia,<sup>13</sup> cortical blindness,<sup>2,9</sup> and progressive dementia of long duration (several months to years) in middle-aged patients.<sup>2,9,11,15</sup> Venous ischemia should be kept in mind as a possible diagnosis in patients presenting with any of the unusual manifestations mentioned above. Finally, we would emphasize the importance of early diagnosis by angiography, including external carotid arteriography, and of appropriate therapy for patients with venous ischemia since this condition may be a warning sign of hemorrhage. However, if treated at an early stage, venous ischemia may be reversible.

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