AJNR

Venous congestion: an MR finding in dural arteriovenous malformations with cortical venous drainage.

R Willinsky, K Terbrugge, W Montanera, D Mikulis and M C Wallace

AJNR Am J Neuroradiol 1994, 15 (8) 1501-1507
http://www.ajnr.org/content/15/8/1501

Venous Congestion: An MR Finding in Dural Arteriovenous Malformations with Cortical Venous Drainage

Robert Willinsky, Karel Terbrugge, Walter Montanera, David Mikulis, and M. Christopher Wallace

PURPOSE: To present the MR findings of intracranial dural arteriovenous malformations with cortical venous drainage, emphasizing the parenchymal changes. **METHODS:** Conventional MR and x-ray angiograms in 13 patients with dural arteriovenous malformations and cortical venous reflux were reviewed. The site of the shunt, location of the venous reflux, and presence of venous stenosis were assessed on the angiograms. Parenchymal changes, dilated vessels, and venous occlusive disease were assessed on MR. **RESULTS:** On MR, 10 of the 13 patients (77%) had dilated pial vessels. Two patients had hydrocephalus. Two patients presented with parenchymal bleeds, one with a subdural component, both remote from the nidus. Two patients presented with subarachnoid hemorrhage. One patient had a parenchymal bleed 9 months after presentation. Venous occlusion was evident on MR in 2 patients. Diffuse white matter edema in the cerebellar or cerebral hemispheres was present on MR in 4 patients and correlated with neurologic deficits. In 2 of these 4 patients, gadolinium enhancement was seen in the periphery of the involved hemisphere. **CONCLUSIONS:** On MR a surplus of pial vessels suggests a dural arteriovenous malformation with cortical venous drainage. The MR finding of white matter edema deep in the cerebral or cerebellar hemispheres is direct evidence of a venous congestion.

Index terms: Arteriovenous malformations, intracranial; Veins, diseases; Brain, diseases; Brain, magnetic resonance

AJNR Am J Neuroradiol 15:1501–1507, Sep 1994

Intracranial dural arteriovenous malformations (AVMs) with cortical or deep venous drainage can cause hemorrhages, seizures, and neurologic deficits (1–5). Their pathophysiologic manifestations may be caused by a venous congestion that in turn may be exacerbated by venoocclusive disease (3, 5–7). We propose the term *venous congestive encephalopathy* to describe those patients who present with a neurologic deficit caused by venous hypertension. This clinicopathologic entity is analogous to the venous congestive myelopathy of spinal dural arteriovenous fistula (8). The purpose of this report is to present the magnetic resonance (MR) findings in 13 patients with dural AVMs with cortical venous drainage, highlighting those patients with venous congestive myelopathy.

Patients and Methods

We retrospectively reviewed the hospital charts, x-ray angiograms, and MR studies in 62 patients with intracranial dural AVMs. All patients were explored and/or treated at our institution between 1984 and 1993. Twenty-three had cortical or deep venous drainage, and 13 of these had conventional MR. The clinical features of these 13 patients are summarized in the Table. There were 10 men and 3 women ranging in age from 30 to 72 years, with a mean age of 55 years. Hemorrhage (4 patients) and neurologic deficits (4 patients) were the most common presentations.

All patients had selective biplane angiography. The following angiographic features were assessed: site of the shunt, location of the venous reflux, and presence of ve-

Received September 14, 1993; accepted after revision December 1.

Presented at the Annual Meeting of the ASNR, May 1993, Vancouver, Canada.

From the Department of Radiology (R.W., K.T., W.M., D.M.), Department of Surgery, Division of Neurosurgery (M.C.W.), and Brain Vascular Malformation Study Group (R.W., K.T., M.C.W.), The Toronto Hospital, Western Division, University of Toronto, 399 Bathurst St, Toronto, Ontario, Canada, M5T 2S8.

Address reprint requests to R. Willinsky, MD, Department of Radiology, The Toronto Hospital, Western Division, University of Toronto, 399 Bathurst St, Toronto, Ont, Canada, M5T 2S8.

AJNR 15:1501–1507, Sep 1994 0195-6108/94/1508–1501

Thirteen patients with dural arteriovenous malformations and cortical venous drainage

Patient/ Age/Sex	Clinical	Angiography			MR		
		Shunt location	Reflux	Venous stenosis	Venous stenosis	Dilated vessels	Features
1/42/M	Confusion, ataxia, hemiplegia	SSS	Bilateral cortical	Occluded SSS	Occluded SSS	Yes	Edema white matter
2/30/M	Vertigo	Tentorial	Subepen-dymal	Straight sinus stenosis	No	Yes	Hydrocephalus
3/72/M	Transient ischemic attacks	Vein of Galen	Cortical	No	No	Yes	. . .
4/50/M	Dementia; ataxia	TS	SSS, ICV, BVR	Occluded ipsilateral TS; stenotic contra-qlateral TS	Occluded ipsilateral TS	Yes	Edema white matter
5/60/M	Seizure	Anterior cranial fossa	Cortical	No	No	Yes	Dilated medullary vessels
6/58/F	Paraesthesias	Foramen magnum	Pontomes encephalic	No	No	No	. . .
7/60/M	Hemorrhage	TS	Cortical ICV, BVR	Stenosis, vein of Galen	No	Yes	ICH subdural
8/30/F	SAH	Vein of Galen	Cortical	No	No	Yes	Hydrocephalus, pial hemosiderosis
9/55/M	Hemorrhage	Foramen magnum	Pontomes encephalic	No	No	Yes	ICH tectum
10/68/F	Tinnitus; double vision	Cavernous	Contralateral sylvian	No	No	No	. . .
11/48/M	SAH	Tentorial	Cerebellar	No	No	No	. . .
12/67/M	Ataxia	Torcula	Contralateral cerebellar	No	No	Yes	Edema cerebellum
13/72/M	Ataxia; hemorrhage (9 months later)	Torcula	Cerebellar	No	No	Yes	Edema cerebellum

Note.—SSS indicates superior sagittal sinus; ICV, internal cerebral vein; BVR, basal vein of Rosenthal; ICH, intracerebral hemorrhage; SAH, subarachnoid hemorrhage; TS, transverse sinus.

nous stenosis or occlusion. Conventional spin-echo MR at 1.5 T was done in the 13 patients. MR included T1-weighted, 600–916/15–28/1–2 (repetition time/echo time/excitations); proton density, 2000–3000/15–29/1; and T2-weighted, 2200–3700/70–90/1 sequences. The matrix size was 238 × 256 in 7 patients, 256 × 256 in 5, and 256 × 128 in 1. Section thickness was either 4 or 5 mm. In 2 patients, flow-sensitive gradient-recalled images, 55–60/22–28/4, 50° flip angle, were done. Eleven patients had at least three sequences in either two or three orthogonal planes. Gadopentetate dimeglumine (0.1 mmol/kg) was given intravenously in 4 patients. On MR the following findings were noted: parenchymal changes, dilated vessels, and venoocclusive disease.

Results

MR and angiographic findings are summarized in the Table. The location of the shunts included transverse sinus (2 patients), torcula (2 patients), foramen magnum (2 patients), vein of Galen (2 patients), tentorial (2 patients), cavernous sinus (1 patient), superior sagittal sinus (1 patient), and anterior cranial fossa (1 patient). Apart from the shunts at the foramen magnum, which refluxed into the pontomesencephalic veins, all had cortical venous reflux.

Four patients with neurologic deficits (patients 1, 4, 12, and 13) had T2 hyperintensity on MR. Two of these patients (patients 1 and 4) had no parenchymal or venous sinus abnormalities on MR at their initial presentation. On follow-up MR, both patients had sinus occlusions, prominent cortical vessels, and T2 hyperintensity in the cerebral white matter (Fig 1). Angiography in these 2 patients revealed venous sinus occlusions, dural AVMs with cortical venous drainage, and a delayed cerebral circulation time with venous collaterals (Fig 1). These shunts were in the wall of the occluded sinuses. In patients 12 and 13, MR revealed dilated vessels, T2 hyperintensity, mass effect, and enhancement in the cerebellar hemispheres (Figs

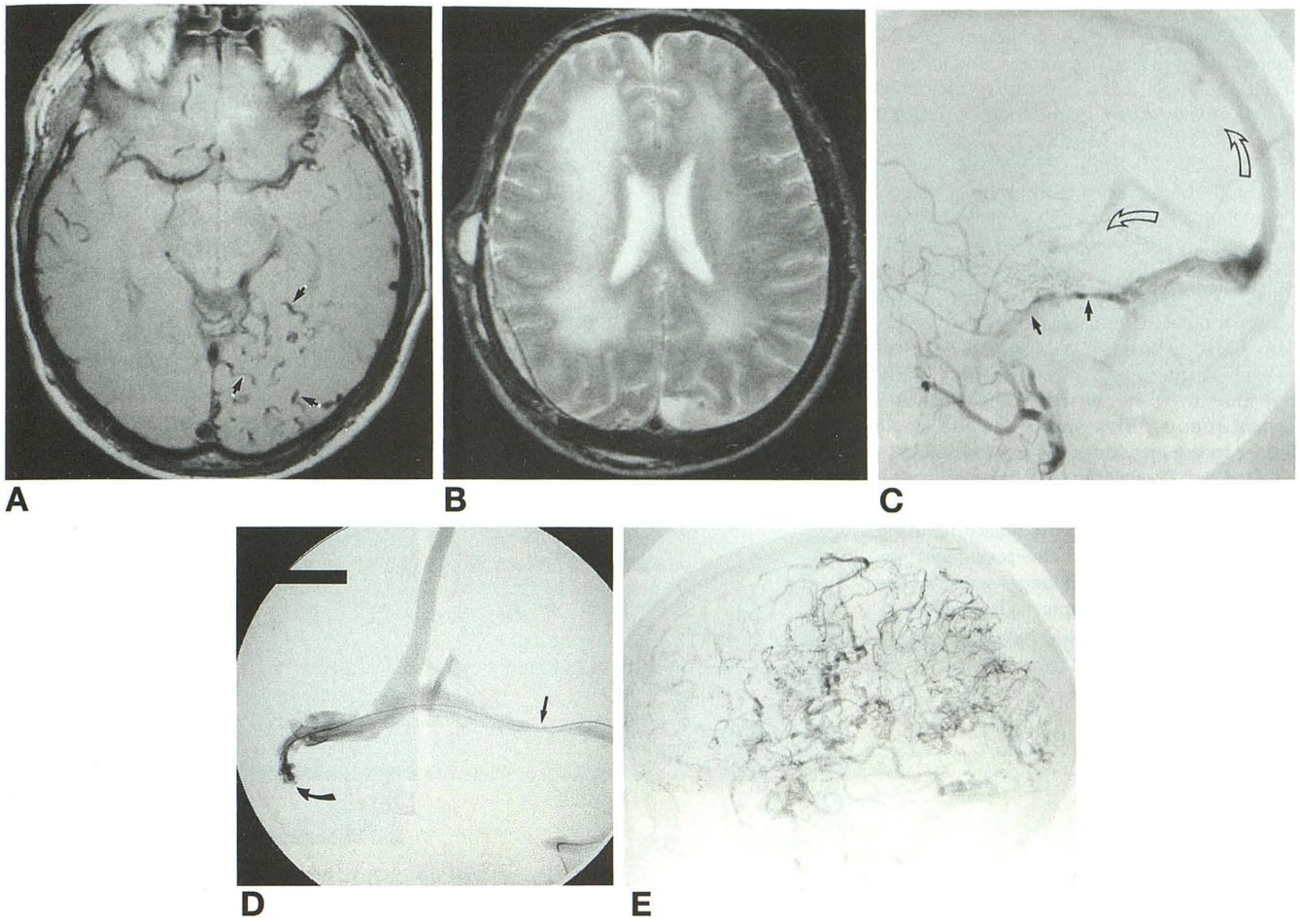


Fig 1. Case 4. *A*, Axial T1-weighted MR shows a plethora of tortuous vessels (*arrows*) in the left occipital region.

B, Axial T2-weighted MR shows hyperintensity in the cerebral white matter.

C, Lateral right external carotid angiogram shows a dural shunt in the superior petrosal sinus (*closed arrows*) and reflux into the superior sagittal sinus and basal vein of Rosenthal (*open arrows*). The distal ipsilateral transverse sinus is occluded.

D, Anteroposterior transverse sinus venogram shows occlusion of the distal right transverse sinus (*curved arrow*) and stenosis of the left transverse sinus (*straight arrow*). Pressure measurements revealed a 30-mmHg gradient across the stenosis.

E, Late phase of the lateral left internal carotid angiogram shows extensive venous collaterals and lack of filling of the superior sagittal sinus.

F, Axial T2-weighted MR 3 months after embolization and surgery shows less white matter hyperintensity commensurate with the clinical improvement.

2 and 3). Angiography in both these patients showed dural AVMs at the torcula with reflux into cerebellar veins (Fig 2).

Four patients presented with hemorrhages; two were subarachnoid and two had parenchymal hematomas. A subdural hematoma accompanied one of the parenchymal hematomas. The parenchymal hematomas were remote from the nidus (Fig 4). One patient (patient 13) died from a massive posterior fossa bleed 9 months after presenting with a neurologic deficit (Fig 3).

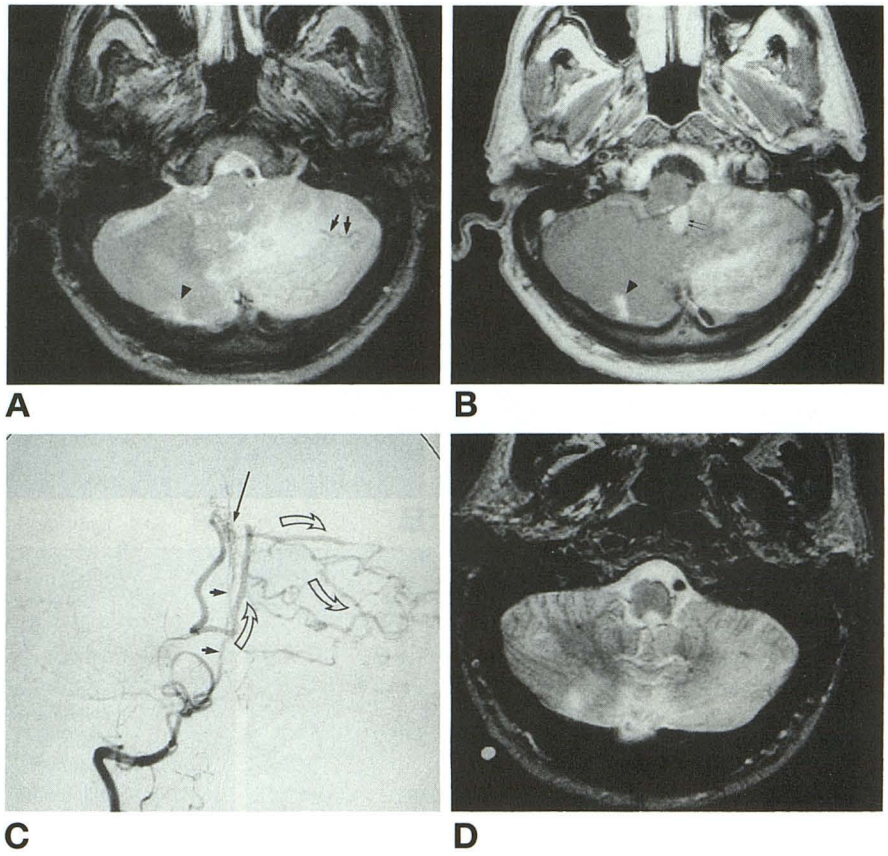


Fig 2. Case 12. *A*, Axial T2-weighted MR shows hyperintensity in the left cerebellar hemisphere with mass effect and a prominent superficial vessel (*arrows*). Increased signal is also seen in the right cerebellar hemisphere (*arrowhead*).

B, Axial T1-weighted MR with gadolinium shows enhancement in the periphery of the left cerebellum, the vermis (*double arrow*), and the right cerebellum (*arrowhead*).

C, Anteroposterior right vertebral angiogram shows a dural shunt at the torcula (*long straight arrow*), fed by the artery of the falx cerebelli (*small arrows*), and draining into the contralateral cerebellar veins (*open arrows*). This artery was the single feeder to the arteriovenous malformation.

D, Axial T2-weighted MR 5 months after surgery shows complete resolution of the mass effect and less hyperintensity in the left cerebellar hemisphere. The prominent superficial vessels were no longer evident. The patient's signs and symptoms had resolved.

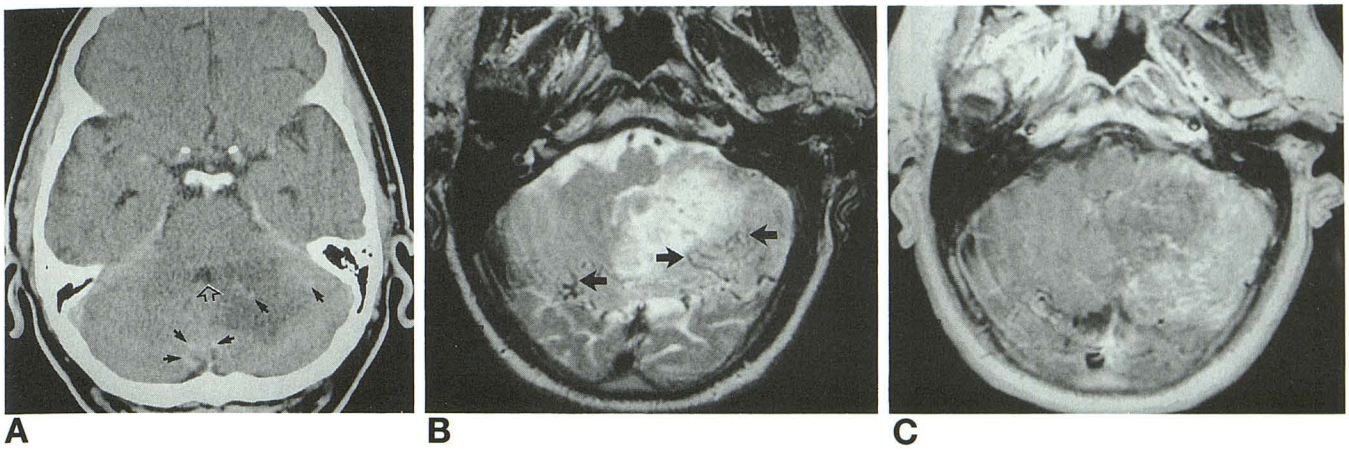
Venous sinus occlusion was evident on MR and arteriography in 2 patients (Fig 1). MR failed to reveal venous stenosis that was seen on angiography in 3 patients (Fig 4). An excess of pial vessels was evident on MR in 10 of our 13 patients (Figs 1–4). Two patients had hydrocephalus.

Discussion

From our series of 23 patients with dural AVMs and cortical venous drainage, 13 had MR, and in 10 a surplus of pial vessels was evident. De Marco et al (6) reviewed the MR findings in 12 patients with dural AVMs and found dilated cortical veins without a parenchymal nidus in 8. In De Marco's series, those with dilated cortical veins had venoocclusive disease that was better shown on x-ray angiograms than on MR. Five of our 13 patients had arteriograms showing venoocclusive disease, and this was evident on MR in only 2. The causal relationship of dural sinus thrombosis and dural AVMs is well established (9, 10).

Four (31%) of our 13 patients presented with neurologic deficits. At angiography, a delayed circulation time and venous collateral pathways were evident in all 4 (Figs 1 and 2). These findings are felt to be caused by a venous hypertension that may be accentuated by a venous stenosis or occlusion as illustrated by Ishii et al (7). Two of our 4 patients with neurologic deficits had venous sinus occlusions.

In our 4 patients with neurologic deficits, MR revealed a T2 hyperintensity in the parenchyma. We believe that this T2 hyperintensity reflects edema resulting from venous hypertension and passive congestion of the brain. This is analagous to the T2 hyperintensity seen within the cord in spinal dural AVMs (11). In the cerebral hemispheres, the deep white matter appears to be most vulnerable to this phenomena (Fig 1). The differential diagnosis of diffuse white matter edema in the cerebral hemispheres would include superior sagittal sinus thrombosis with a venous infarct or venous congestion, demyelination (ie, acute disseminated encephalomyelitis, progressive multifocal leukoencephalopathy), or dysmyelination (ie, the leukodystrophies) (12). In the posterior fossa, the edema was



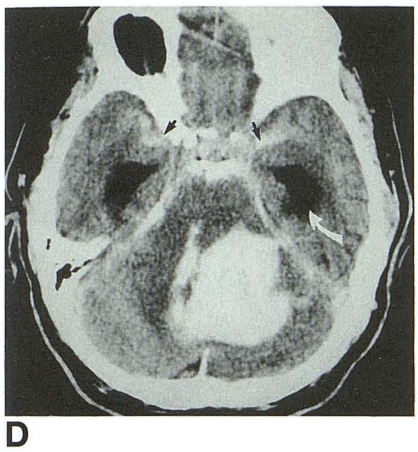


Fig 3. Case 13. *A*, Axial, enhanced CT shows an area of low density in the left cerebellar hemisphere with mild compression of the fourth ventricle (*open arrow*) and punctate areas of increased density (*arrows*).

B, Axial T2-weighted MR shows hyperintensity in the deep aspect of the left cerebellum with mass effect and tortuous superficial vessels (*arrows*).

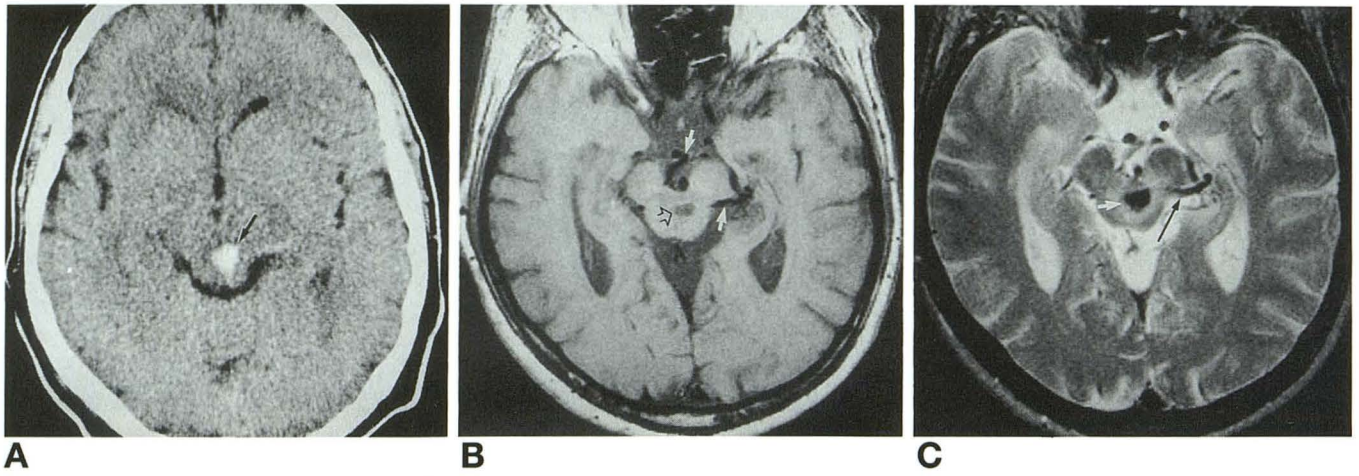
C, Axial T1-weighted MR with gadolinium shows enhancement in the periphery of the left cerebellum.

D, Axial noncontrast CT 9 months later shows a large left cerebellar hematoma with subarachnoid blood (*arrows*) and hydrocephalus (*curved arrow*).

predominantly deep in one cerebellar hemisphere (Figs 2 and 3). Gadolinium enhancement was peripheral, because of either a passive congestion or a breakdown of the blood-brain barrier (Figs 2 and 3). In 2 patients, the initial computed tomography (CT) interpretation suggested a neoplasm (Figs 2 and 3). MR revealed an excess of pial vessels and a pattern of edema and enhancement, which suggested a vascular malformation.

The parenchymal edema seen on MR in our 4 patients corresponds to the diffuse decrease in white matter density seen on CT, as reported by Chiras et al and Miyasaka et al (13, 14). In De Marco's review of 12 patients, venous infarcts were found in 3 and vasogenic edema in 1 (6). Two of these were illustrated; 1 exhibited a small pontine infarct, and the other had a small cerebellar hematoma with a diffuse T2 hypointensity in the cerebellar hemisphere believed to be related to hemosiderin deposition from chronic passive congestion.

In our 13 patients with dural AVMs and cortical venous drainage, there were initially 4 (31%) hemorrhagic presentations. A fifth patient died from a bleed 9 months after initially presenting with a neurologic deficit (Fig 3). He had refused the proposed surgical treatment. These hemorrhages emphasize the necessity to treat dural AVMs with cortical venous drainage (3, 7). In a series by Castaigne et al, 42% of dural AVMs with cortical venous drainage presented with a hemorrhage (1). In our series, 2 of the hemorrhages were subarachnoid. One of the bleeds was parenchymal and subdural, one parenchymal and subarachnoid, and the other bleed was isolated in the tectum. In a literature review of intracranial dural AVMs by Lasjaunias et al all types of intradural hemorrhages were encountered (3). Lasjaunias emphasized that dural AVMs of the anterior cranial fossa and tentorium almost all have cortical venous drainage and thus have a high frequency of intradural bleeding. In our report, the locations of the dural AVMs that bled were as follows: tentorium, straight sinus, vein of Galen, foramen magnum, and transverse sinus. In the patient with the parenchymal/subdural bleed, the dural AVM



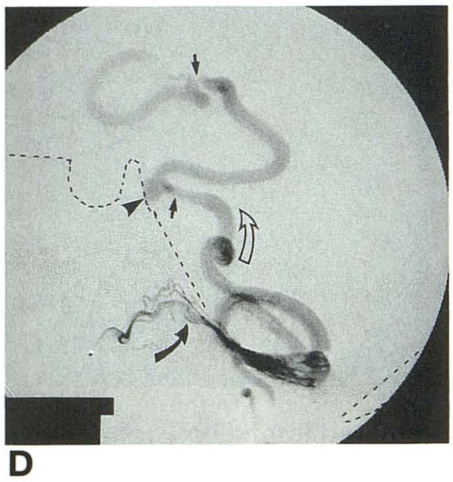


Fig 4. Case 9. *A*, Axial noncontrast CT shows a bleed into the tectum (*arrow*).

B, Axial T1-weighted MR shows the recent tectal bleed (*open arrow*) and dilated vessels around the midbrain (*arrows*).

C, Axial T2-weighted MR shows the bleed (*white arrow*) and better delineates the dilated vessels (*black arrow*) surrounding the midbrain.

D, Lateral ascending pharyngeal angiogram shows a dural shunt (*curved arrow*) draining into a tortuous pontomesencephalic vein (*open arrow*) with venous stenoses (*straight arrows*) and a venous aneurysm (*arrowhead*).

was evident only on the external carotid arteriogram. This highlights the need to include selective external carotid arteriograms in the workup of intracranial bleeds. The 2 parenchymal hemorrhages were far from the shunt, suggesting that the bleeds were related to the venous drainage (Fig 4). The remoteness of the bleeds has been stressed in the literature (5, 15, 16). The pathologic changes in the draining veins may be similar to the high-flow arteriopathy described by Pile-Spellman (17).

Raised intracranial pressure and papilledema can occur in dural AVMs with only sinosal drainage (18, 19). This is considered to be secondary to raised pressure in the superior sagittal sinus, resulting in impaired cerebrospinal absorption. Two of our patients had hydrocephalus, one of whom also had pial hemosiderosis. Hydrocephalus may result from decreased cerebrospinal absorption caused by the high pressure in the venous sinus or, in the patient with pial hemosiderosis, from blockage of the arachnoid villae from previous subarachnoid bleeds.

In summary, in patients with neurologic deficits caused by dural AVMs with cortical venous drainage (venous congestive encephalopathy), the correct diagnosis may be suggested on MR, by the presence of dilated pial vessels, and/or by a diffuse edema. Venous occlusion would support this diagnosis. In those patients with neurologic deficits in whom CT suggests a mass, the administration of gadolinium is suggested, because the diffuse enhancement in dural AVMs may differ from the enhancement pattern of many neoplasms. When the MR findings suggest a dural AVM, selective angiography including all dural branches is mandatory. CT is the primary modality in the diagnosis of spontaneous intracranial hemorrhage. In most intracranial hemorrhages, angiography is indicated. However, when a vascular malformation or tumor is suspected to be the cause of a bleed, we suggest an MR, because the finding of a surplus of prominent pial vessels should prompt angiography in a search for a dural AVM.

References

1. Castaigne P, Bories J, Brunet P, Merland J, Meninger V. Les fistules arterio-veineuses meningees pures a drainage veineux cortical. *Rev Neurol* 1976;132:169–181
2. Halbach V, Higashida R, Hieshima G, Wilson C, Hardin C, Kwan E. Treatment of dural fistulas involving the deep cerebral venous system. *AJNR Am J Neuroradiol* 1989;10:393–399
3. Lasjaunias P, Chiu M, Ter Brugge K, Tolia A, Hurth M, Bernstein M. Neurological manifestations of intracranial dural arteriovenous malformations. *J Neurosurg* 1986;64:724–730
4. Pierot L, Chiras J, Meder JF, Rose M, Rivierez M, Marsault C. Dural arteriovenous fistulas of the posterior fossa draining into subarachnoid veins. *AJNR Am J Neuroradiol* 1992;13:315–323
5. Vinuela F, Fox A, Pelz D, Drake C. Unusual clinical manifestations of dural arteriovenous malformations. *J Neurosurg* 1986;64:554–558
6. De Marco J, Dillon W, Halbach V, Tsuruda J. Dural arteriovenous fistulas: evaluation with MR imaging. *Radiology* 1990;175:193–199
7. Ishii K, Goto K, Ihara K et al. High-risk dural arteriovenous fistulae of the transverse and sigmoid sinuses. *AJNR Am J Neuroradiol* 1987;8:1113–1120
8. Aminoff M, Barnard R, Logue V. The pathophysiology of spinal vascular malformations. *J Neurol Sci* 1974;23:255–263
9. Houser O, Canpbell J, Campbell R, Sundt T. Arteriovenous malformation affecting the transverse sinus: an acquired lesion. *Mayo Clin Proc* 1979;54:651–661
10. Chaudhary M, Sachdev V, Cho SH, Weitzner I, Puljic S, Huang Y. Dural arteriovenous malformations of the major venous sinuses: an acquired lesion. *AJNR Am J Neuroradiol* 1982;3:13–19
11. Larsson EM, Desai P, Hardin CW, Story J, Jinkins JR. Venous infarction of the spinal cord resulting from dural arteriovenous fistula: MR imaging findings. *AJNR Am J Neuroradiol* 1991;12:739–743
12. Edwards M, Bonnin J. Magnetic resonance imaging of the brain and spine. In: Atlas SW, ed. *White Matter Diseases.* New York: Raven Press, 1991:467–500
13. Chiras J, Bories J, Leger J, Gaston A, Launay M. CT scan of dural arteriovenous fistulas. *Neuroradiology* 1982;23:185–194
14. Miyasaka K, Takei H, Nomura M, et al. Computerized tomography findings in dural arteriovenous malformations: report of three cases. *J Neurosurg* 1980;53:698–702
15. Reul J, Thron A, Laborde G, Bruckmann H. Dural arteriovenous malformations at the base of the anterior cranial fossa: report of nine cases. *Neuroradiology* 1993;35:388–393
16. Lasjaunias P, Merland J, Theron J, Moret J. Vascularization meningee de la fosse cerebrale moyenne. *J Neuroradiol* 1977;4:361–384
17. Pile-Spellman J, Baker K, Lizczal T. High flow angiopathy: cerebral blood vessel changes in experimental chronic fistula. *AJNR Am J Neuroradiol* 1986;7:811–815
18. Gelwan M, Choi IS, Berenstein A, Pile-Spellman J, Kupersmith M. Dural arteriovenous malformations and papilledema. *Neurosurgery* 1988;22:1079–1084
19. Lamas E, Lobato R, Esparza J, Escudero L. Dural posterior fossa AVM producing raised sagittal sinus pressure: case report. *J Neurosurg* 1977;46:804–810