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Dementia Resulting from Dural Arteriovenous Fistulas: The Pathologic Findings of Venous Hypertensive Encephalopathy

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PURPOSE: **Dural arteriovenous fistulas (DAVFs) are acquired arteriovenous shunts located within the dura. The highly variable natural history and symptomatology of DAVFs range from subjective bruit to intracranial hemorrhage and are related to the lesion's pattern of venous drainage and its effect on the drainage of adjacent brain. We examined the prevalence and features of DAVFs in patients with progressive dementia or encephalopathy.**

METHODS: **The records and radiologic studies of 40 consecutive patients with DAVFs treated at our institution were reviewed.**

RESULTS: **Five (12.5%) of 40 consecutive patients with DAVFs had encephalopathy or dementia. In each patient, high flow through the arteriovenous shunt combined with venous outflow obstruction caused impairment of cerebral venous drainage. Hemodynamically, the result was widespread venous hypertension causing diffuse ischemia and progressive dysfunction of brain parenchyma. Results of CT or MR imaging revealed abnormalities in each patient, reflecting the impaired parenchymal venous drainage. Pathologic findings in one patient confirmed the mechanism of cerebral dysfunction as venous hypertension. The hemodynamic mechanism and resulting abnormality appeared identical to that seen in progressive chronic myelopathy resulting from a spinal DAVF (Foix-Alajouanine syndrome). Remission of cognitive symptoms occurred in each patient after embolization.**

CONCLUSION: **Venous hypertensive encephalopathy resulting from a DAVF should be considered a potentially reversible cause of vascular dementia in patients with progressive cognitive deficits.**

Dural arteriovenous fistulas (DAVFs) consist of arteriovenous shunts within the dura and represent 10% to 15% of cerebrovascular malformations. A wide variety of signs and symptoms may arise from DAVFs, ranging from pulsatile tinnitus to intracranial hemorrhage (1, 2). The highly variable course and symptomatology of DAVFs have been convincingly related to their intracranial location and, more specifically, to their pattern of venous drainage (3–6). We report our experience with five patients with dementia or encephalopathy as the primary manifestation of DAVFs.

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Methods

We reviewed the records and radiologic studies in 40 consecutive patients with DAVFs treated at our institution from 1991 to 1997. In five (12.5%) of the patients, the primary manifestation was that of a diffuse encephalopathy or a chronic dementing process.

Results

All five patients experienced prominent deterioration of memory, judgment, and orientation during periods ranging from 3 months to more than 1 year (Table 1). In all cases, the degree of cognitive impairment was sufficient to severely change or limit the patient's daily activities. Impairment ranged from the loss of ability to drive a car (one patient) to the loss of ability to perform any self-care activities (two patients). Chronic headaches exceeding 6 months in duration were prevalent in all patients, and were described as either occipital or generalized in distribution and moderate to severe in intensity. Nuchal rigidity was not associated with headache in any of the patients. Although only two patients reported pulsa-

TABLE 1: Clinical course

Patient	Age, y/Sex	Clinical Course
1	54/M	Headache >12 mo; 12 mo of progressive confusion, disorientation; aphasia; R facial palsy; NP: generalized dementia with widespread cerebral dysfunction; Embo: transient (3 mo) improvement; recurrent cognitive decline; Embo ×2: minimal cognitive improvement; LP shunt placed; patient suffered herniation and cardiorespiratory arrest
2	64/M	Headache >12 mo; 11 mo of worsening confusion, memory loss, progressive left hemiparesis; stopped driving; Embo: improved (4 y); recurrent cognitive deterioration, Embo and surgery; improved cognition
3	70/M	Headache >12 mo; 1 y progressive mental status deterioration, memory loss, unresponsiveness; unable to care for self (bedridden); Embo: cognition improved, able to recognize family members; died (aspiration pneumonia)
4	65/M	Headache 9 mo; bruit; 6 mo of progressive confusion, memory loss, unresponsiveness; unable to care for self; NP: multifocal or generalized cerebral dysfunction; L temporal deficits, including anomia and verbal memory deficits; Embo: cognitive improvement (2 y); recurrent confusion; Embo: cognitive improvement
5	77/M	Headache 6 mo; 3 mo of confusion, memory loss; became unable to live alone; Embo: cognitive improvement, returned to independent living

Note.—Embo indicates partial embolization from the arterial circulation; NP, neuropsychological testing.

TABLE 2: Imaging Studies

Patient	CT Scan	MR Image
1*	Enlarged surface vessels; parietooccipital hypodensity	Enlarged vessels over hemispheric surface, patchy increased T2-weighted signal and enhancement within corona radiata; MR angiogram: high-flow veins over cerebral hemispheres
2	R parietoocciptial hypodensity and swelling	N/A
3	L temporal hypodensity and swelling	N/A
4	Hypodensity of cerebellar hemispheres; enlarged vessels over cerebral hemispheric surfaces	Enlarged vessels over hemispheric surface, increased cerebellar/cerebral hemispheric T2-weighted signal; MR angiogram: high-flow vessels within posterior fossa
5†	N/A	Enlarged vessels over R hemisphere, diffusely increased T2-weighted signal and swelling of R hemisphere

Note.—R indicated right; L, left; N/A, not applicable.

* See Figures 1 and 2.

† See Figure 3.

tile tinnitus, a bruit was present over the skull base in all patients, most often in the mastoid area.

Focal motor deficits were present in two patients (Table 1). In both patients, evidence of cognitive dysfunction predated the development of the focal deficit.

Neuropsychological testing was performed in two patients (Table 1), both of whom experienced severe deficits involving a number of cognitive domains, suggesting a generalized dementing disorder with widespread cerebral dysfunction.

Imaging studies, including CT scans, MR images, or both, were obtained in each patient. As outlined in Table 2, abnormalities were detected in each patient on CT or MR studies, and consisted of focal areas of parenchymal hypodensity (on CT scans) or of an abnormal signal associated with mild swelling (on T2-weighted MR images). The parenchymal abnormalities were remote from the site of the DAVF in each patient and were compatible with areas of edema or venous ischemia. Enlarged vessels over the surface of the brain were identified on the imaging studies of three patients. Angiographic correlation showed these vessels to be dilated veins draining the DAVF. Hydrocephalus was not noted in any of the patients.

MR angiograms were obtained in two patients, both of whom had abnormal vascularity representing increased flow velocity in veins draining the DAVF. The abnormal vessels were present over the cerebral hemispheres in one patient and over the posterior fossa in the other.

All patients were studied angiographically, the results of which are outlined in Table 3. Four of the patients had DAVFs involving the transverse and sigmoid sinuses, and the fifth patient had a DAVF involving the floor of the anterior fossa. The arterial supply was primarily from external carotid artery branches, although, in each case, minor contributions were present from branches of the internal carotid artery (ophthalmic or cavernous branches) or from the vertebral arteries (meningeal branches). In no case did major cerebral artery branches provide arterial supply to the DAVF. Consequently, an arterial steal phenomenon would be unlikely as a contributing mechanism to the neurologic deficits.

In all patients, early arteriovenous shunting from the DAVF refluxed into adjacent dural sinuses and into cortical veins. In the four patients with DAVFs of the transverse sinus, retrograde filling of both the superior sagittal sinus and straight sinus was present. Such retrograde flow indicates impairment of drainage from both the superficial and deep hemispheric venous systems and is considered to be a risk factor associated with more aggressive behavior of DAVFs (5). All four patients also had occlusion of the involved transverse and sigmoid sinus. In two patients, bilateral transverse and sigmoid occlusion was present, resulting in unusual drainage pathways via

TABLE 3: Angiographic findings

Patient	Arterial Feeders	DAVF Location	DAVF Venous Drainage	Brain Venous Drainage
1*	Left ECA (occipital ascending pharyngeal, MMA), left ICA (mht), VA (mb)	Left trv/sig	Retrograde into SSS/SS cortical veins; *occlusion* of both sigmoid sinuses	Delayed (>30 s)
2	Right ECA (occipital, ascending pharyngeal)	Right trv/sig	Retrograde into SSS/SS; *occlusion* of both sigmoid sinuses	Delayed (>10 s)
3	Basilar ECA (occipital, ascending pharyngeal), ICA, VA	Right trv/sig	Retrograde into right trv sinus, SSS/SS; *occlusion* of right transverse sinus; antegrade through left trv/sig and jugular vein	Delayed (>18 s)
4	Right ECA (occipital, ascending pharyngeal), ICA, VA	Right trv/sig	Retrograde into SSS/SS; *occlusion* of right transverse sinus	Delayed
5†	Basilar ECA, basilar ICA (ophthalmic)	Floor of anterior fossa	Retrograde into cortical veins; antegrade into SSS	Delayed over right hemisphere

Note.—ECA indicates external carotid artery; MMA, middle meningeal artery; mht, meningohypophyseal trunk; mb, meningeal branches; ICA, internal carotid artery; SSS, superior sagittal sinus; SS, straight sinus; trv/sig, transverse/sigmoid sinus; VA, vertebral artery.

* See Figures 1 and 2.

† See Figure 3.

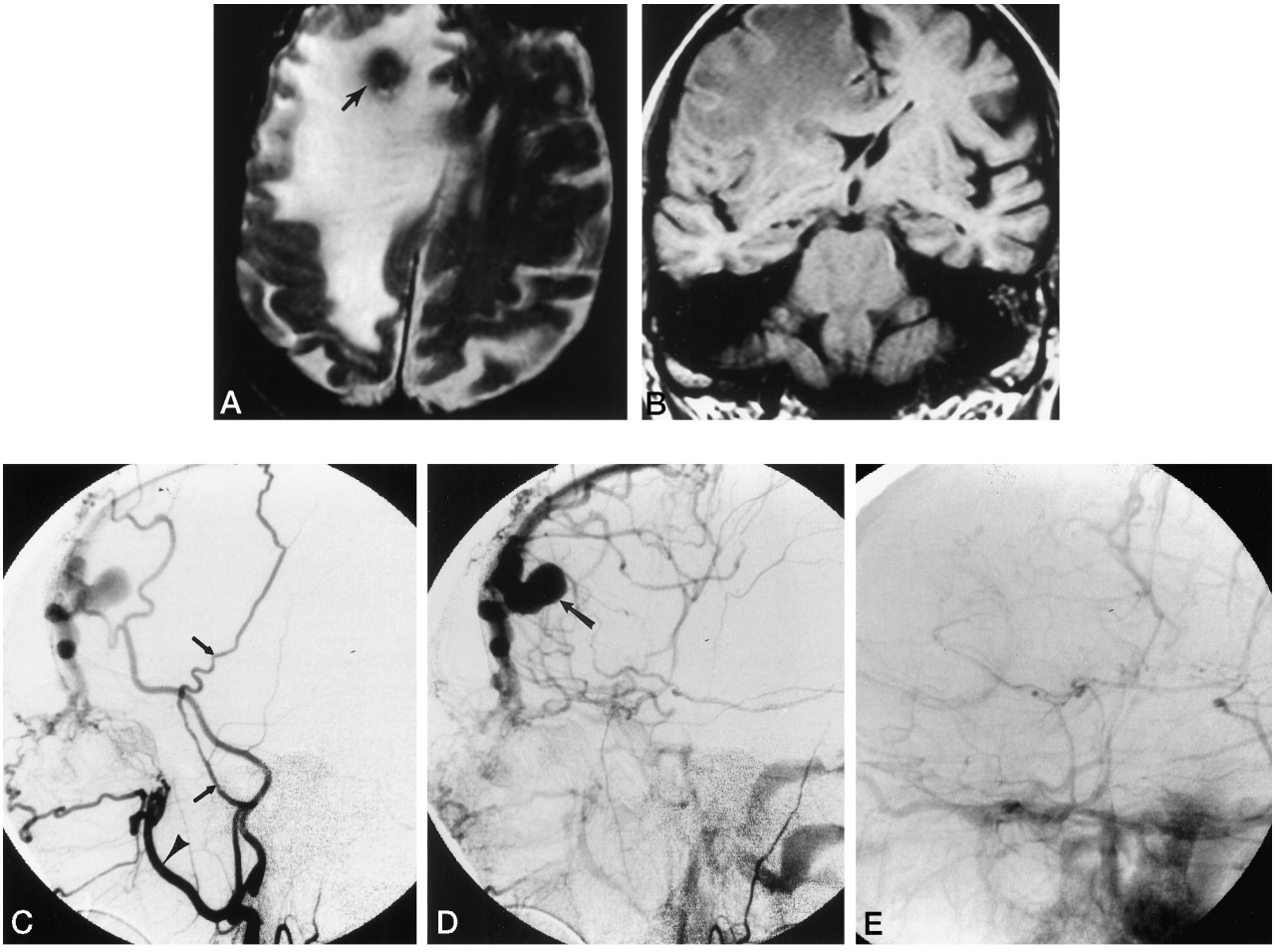


FIG 1. Patient 5.

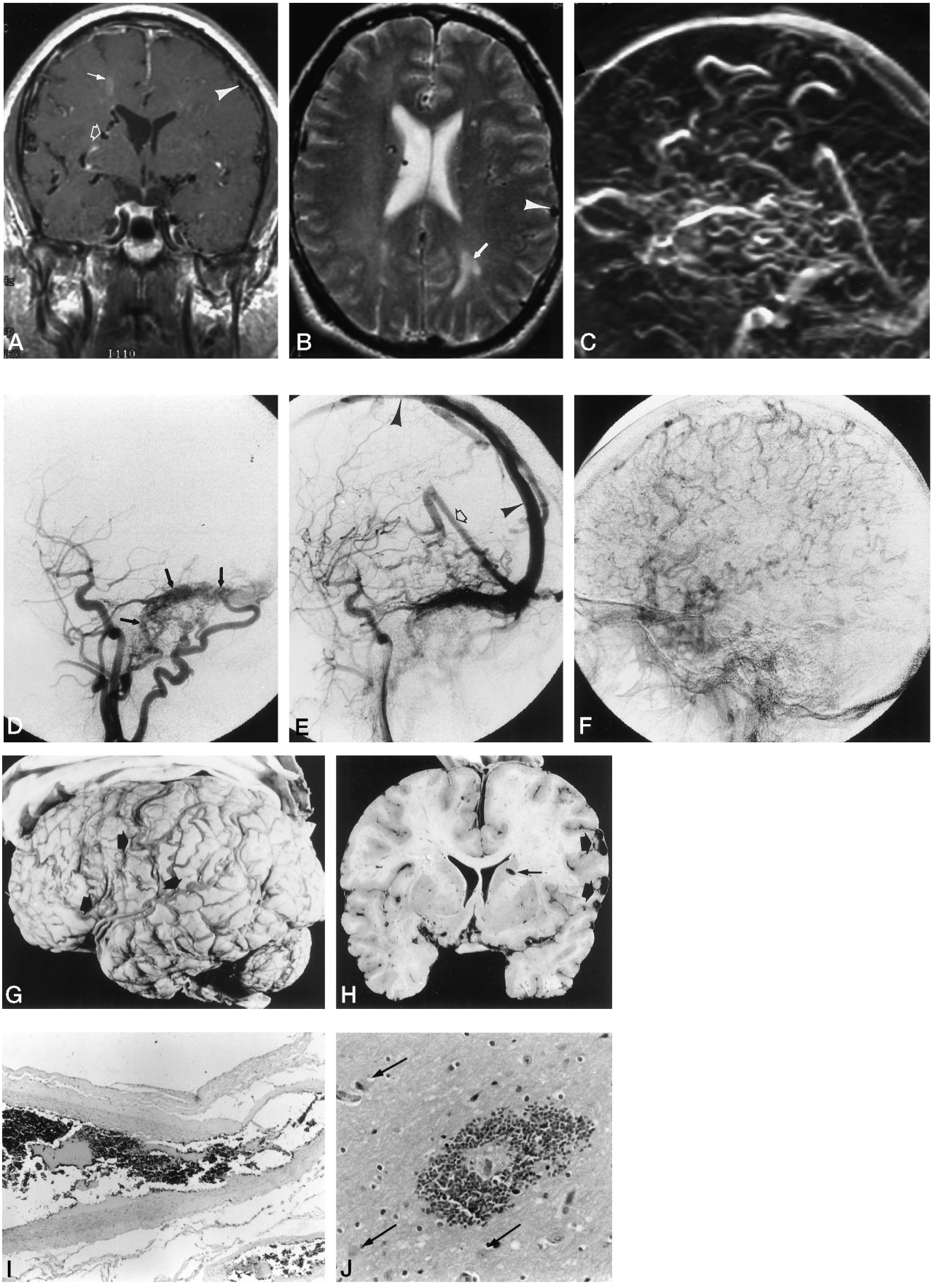
A, Axial T2-weighted MR image shows a signal abnormality throughout the entire right hemisphere with a dilated vein (*arrow*) draining a DAVF of the anterior fossa.

B, Coronal T1-weighted MR image with a signal abnormality and midline shift resulting from swelling of the involved hemisphere.

C, Lateral view of the right external carotid artery injection shows filling of the anterior fossa DAVF via the distal branches of the internal maxillary artery (*arrowhead*) and the middle meningeal artery (*arrows*).

D, Lateral view of the right external carotid artery injection, late phase, shows dilated cortical vein (*arrow*) (the same vein as in *A*) flowing across the right frontal lobe before draining into the superior sagittal sinus.

E, Lateral view of the right common carotid artery injection, late phase, with delay in emptying of parenchymal veins (>15 seconds).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the cavernous sinus, orbital venous system, and basal foramina into the pharyngeal venous plexus.

The shunt from the anterior fossa DAVF (patient 5) drained initially into a large right hemispheric cortical vein followed by flow into the superior sagittal sinus. An area of venous dilatation was present in the cortical vein, representing evidence of outflow obstruction to the venous drainage of the fistula (Fig 1).

The late views of the angiogram in each patient revealed a considerable delay of brain parenchymal venous drainage involving large areas of the cerebral hemispheres. The times for parenchymal venous emptying ranged from 19 to more than 30 seconds, well in excess of the normal cerebral circulation times of 4 to 6 seconds (7).

Embolization of arterial feeding vessels with polyvinyl alcohol was used in all cases to decrease flow to the DAVF, thereby decreasing venous congestion. Considerable but incomplete closure of the DAVF was achieved in each case. Despite the presence of residual DAVFs, all patients experienced clinical improvement in mental status, indicating a reversible effect of the DAVF on the brain parenchyma. In three of the patients, additional embolization was required from 3 months to 3 years later, after recurrent mental status deterioration, with each procedure resulting in remission of cognitive symptoms.

An autopsy, performed in one case (patient 1), disclosed both acute and chronic changes caused by venous hypertension (Fig 2). Chronic changes included marked dilatation of superficial and intraparenchymal cortical veins, some of which showed thickened and hyalinized walls. The centrum semiovale was mildly gliotic. The superior sagittal sinus showed evidence of both remote and recent thrombosis. Acute changes included diffuse cerebral edema, left uncal herniation, and numerous petechial hemorrhages within the gray and white matter.

Discussion

DAVFs most commonly develop in the region of the transverse and sigmoid sinuses, although they may involve dura in any part of the intracranial or spinal compartments of the CNS. DAVFs represent acquired arteriovenous shunts within the dura itself, usually involving the wall of a dural venous sinus. The lesions are thought to arise as a result of increased pressure within the dural sinuses, possibly as a sequela to sinus thrombosis (8, 9). When the sinus lumen remains patent, anterograde flow through the sinus persists without impairment of parenchymal venous drainage.

In some cases, however, significant increases in pressure develop within the sinus, either from large amounts of flow through the DAVF or from sinus outflow obstruction, or from both conditions. Under these circumstances, retrograde transmission of pressure into the superior sagittal sinus, straight sinus, and cortical veins may occur (2–4, 10).

The retrograde transmission of pressure causing enlargement of cortical veins has been identified as a major risk factor for aggressive behavior of DAVFs, including intracranial hemorrhage (4, 5, 11). While intracranial hemorrhage is the most dramatic and acutely life-threatening manifestation of DAVFs, retrograde transmission of increased venous pressure has a number of additional detrimental effects on brain parenchyma. Perhaps most important, as revealed by the findings in our patients, venous hypertension and congestion develops and impairs parenchymal venous drainage, thereby causing ischemia (12, 13).

A number of studies have investigated the factors affecting the development of venous hypertension and its effect on the brain parenchyma (14–17). The magnitude of venous hypertension created by a DAVF is primarily affected by two features of the fistula and the associated venous system. These features include the volume of arteriovenous shunting through the fistula and the resistance to venous outflow. A combination of increased flow into the venous system and increased resistance to venous outflow, as obstruction develops, can raise venous pressures to very high levels (18). Angiographic evidence of both high flow through a large DAVF and obstruction to intracranial venous outflow was revealed in each patient in this series.

The five patients in our study all experienced global cognitive dysfunction that was caused by venous hy-

FIG 2. Patient 1.

A, Enhanced coronal T1-weighted MR image shows dilated superficial veins (*arrowhead*) over the right cerebral hemisphere, an enlarged transcerebral vein (*open arrow*), and parenchymal contrast enhancement (*closed arrow*).

B, Axial T2-weighted MR image shows the increased signal that is diffusely present within the corona radiata bilaterally, as well as the more focal abnormality in the deep parietal white matter on the left (*arrow*). Several enlarged veins (*arrowhead*) are visible over the surfaces of the hemispheres bilaterally.

C, Compressed sagittal view of MR angiographic time-of-flight sequence (7.3/33.3) shows excessive vascularity over the entire brain, representing high flow within veins draining the DAVF.

D and *E*, Lateral view of the left common carotid injection, early phase, shows filling of the DAVF (*arrows, D*) involving the transverse and sigmoid sinuses via the branches of the occipital artery, with immediate retrograde filling of the superior sagittal (*arrowheads, E*) and straight (*arrow, E*) sinuses.

F, Angiogram, late phase (30 seconds), shows delay in emptying of parenchymal venous drainage.

G, Photograph at autopsy shows markedly dilated superficial cortical veins (*arrows*) overlying the cerebral hemisphere.

H, Coronal section reveals shows large intraparenchymal vein present in the head of the caudate nucleus (*thin arrow*) while others are seen on the surface of the brain (*thick arrows*). Gyral flattening and sulcal effacement caused by diffuse cerebral edema are present.

I, Photomicrograph shows dilated and thick-walled veins within the subarachnoid space (original magnification ×100).

J, Photomicrograph shows a perivascular petechial hemorrhage within the white matter, which also contains scattered reactive astrocytes (*arrows*) (original magnification ×200).

pertension and congestion resulting from DAVFs. In each patient, a progressive encephalopathy with dementia dominated the clinical presentation and course. This relatively nonspecific clinical picture may delay detection of the responsible lesion, permitting further deterioration in the absence of treatment.

Nevertheless, several aspects of the history and physical examination may provide important clues to the diagnosis. None of our patients was younger than 54 years of age. DAVFs are acquired abnormalities occurring in adults in whom development of sufficient arteriovenous shunts and venous outflow obstruction requires some time. These features may explain why we have not observed this process in younger patients.

In addition to cognitive deterioration, each patient reported headaches of stable or increasing frequency for periods of from 9 months to more than 1 year. At examination, a bruit was present in all five patients, which was usually best auscultated over the mastoid region, where enlarged occipital arteries provided supply to the DAVF. When detected in a patient with dementia, these clinical features should raise suspicion of an underlying vascular lesion.

Nonangiographic imaging findings, although nonspecific, were abnormal in each patient. Results of CT showed areas of hypodensity with mild swelling, most likely representing areas of gliosis, edema, or venous ischemia. Abnormally enlarged surface vessels were detected that could be identified angiographically as veins draining the DAVF. Results of MR imaging were also remarkable for revealing a parenchymal abnormality in each patient studied. The findings on MR images of parenchymal signal abnormalities and mild swelling are also compatible with the pathologic substrates of gliosis, edema, or venous ischemia. The MR imaging abnormalities in our patients arose as a consequence of venous outflow obstruction and correlate well with the potential for aggressive behavior from these high-flow DAVFs. Although nonspecific, the presence of parenchymal abnormalities in all patients studied aids in excluding other more common causes of cognitive decline, including Alzheimer disease or multiinfarct dementia.

The imaging changes corresponded well with the pathologic findings of the autopsy performed in patient 1. Vascular changes included markedly dilated superficial and intraparenchymal veins, some of which contained thickened or arterialized walls. These chronic vascular changes were accompanied by gliosis within the white matter, including the centrum semiovale. Evidence was present of an acute exacerbation of venous hypertension superimposed on the chronic changes, including thrombosis of the superior sagittal sinus, which most likely resulted in worsening cerebral edema, uncal herniation, and petechial hemorrhages within the gray and white matter. The pathologic findings were compatible with the acute decompensation seen in a patient with long-standing venous hypertension and explain the rapid clinical deterioration that preceded the patient's death. The pathologic findings in the brain mirror those found in the spinal cord in cases of spinal DAVFs and support venous hypertension as a causative mechanism (19). Arterialization of surface and intraparenchymal veins occurs with spinal DAVFs, attesting to long-standing elevated venous pressure. Edema and swelling of the cord parenchyma were also present, reflecting the effect of impaired drainage on the underlying neural tissue. Hemodynamic findings, including pressure measurements in patients with spinal DAVFs, have also confirmed venous hypertension and congestion resulting from arteriovenous shunting into veins draining the spinal cord. The changes found in the brain of our patient are evidence of an identical pathologic mechanism arising from DAVFs of the intracranial dura.

The widespread extent of the changes found in the brain is also analogous to the situation in the spine. Spinal cord compromise from an isolated DAVF involving a single radicular artery, mediated by engorgement of the extensive pial venous system, may extend from the conus to the midthoracic region. Neurologic deficits in both intracranial and spinal DAVFs most likely represent an identical mechanism of ischemic dysfunction of CNS parenchyma mediated by impairment of venous drainage.

A DAVF without venous outflow obstruction most often shows minimal or no abnormalities on CT or MR studies, and requires a high degree of suspicion to make the diagnosis (20). The location of the shunt within the dura and the lack of mass effect make it unusual that a nidus of a DAVF could be seen directly on an MR image or MR angiogram. Nevertheless, the large amount of flow through these lesions made it possible to detect the high-flow draining veins in the two patients in whom MR angiograms were obtained. The detection of the DAVFs in these cases suggests that a DAVF with sufficiently high flow to cause widespread venous hypertension and generalized cognitive dysfunction may be identifiable on MR angiograms.

Angiography remains the best technique for the diagnosis of a DAVF and also provides a route for endovascular therapy. Results of angiographic examination revealed rapid arteriovenous shunting through a large DAVF in each patient, resulting in the delivery of high flow into the cerebral venous system. In addition, direct angiographic evidence of outflow obstruction was present in each patient in the form of sinus occlusion or ectasia of draining veins. The retrograde flow into large cortical veins or dural sinuses causing obstruction of venous outflow from major portions of the brain parenchyma further confirmed the hemodynamic significance of outflow obstruction.

In each case, the angiographic appearance of brain parenchymal venous drainage was delayed, confirming widespread venous congestion (21). Normally, the veins draining the brain parenchyma are angiographically visible from 4 to 6 seconds after the beginning of the arterial phase. In several cases, opacification of the veins draining the cerebral parenchyma was delayed for more than 30 seconds, followed by extracra-

nial drainage via circuitous routes, including the orbital venous system.

Embolization via the arterial route with polyvinyl alcohol was used to treat each of the patients. Polyvinyl alcohol was chosen on the basis of the anatomy of each feeding pedicle to avoid potential skin necrosis and cranial nerve damage that may be associated with the use of liquid embolic agents in the specific locations involved by each DAVF. Although usually successful in decreasing flow through a DAVF, arterial embolization often results in subtotal eradication of the fistula regardless of the embolic agent used (2). Nevertheless, cognitive improvement was associated with the decrease in DAVF flow after embolization in each case. Should cognitive decline recur, additional arterial embolization may be performed to further decrease flow and venous hypertension. Alternatively, transvenous closure of the fistula may be successful in selected cases (22). Last, a combined endovascular-surgical approach should be considered, as had been planned in our most recent case (23).

The findings in our patients indicate that DAVFs may cause dementia or encephalopathy with significant frequency. The imaging changes and pathologic findings support the conclusion that the clinical course results from the delivery of excessive volumes of blood flow into a venous system with outflow obstruction. Venous congestion and hypertension result, with impairment of parenchymal venous drainage. The potential for recurrent and possibly permanent ischemic damage indicates an aggressive approach to closure of these lesions to relieve excessive venous pressure. Despite incomplete arterial embolization, cognitive improvement occurred in all patients. Should symptoms recur after an initial arterial embolization procedure, closure of the involved venous sinus or combined treatment should, if technically feasible, be strongly considered.

Conclusion

The five patients with encephalopathy or dementia resulting from intracranial dural arteriovenous malformations constituted 12.5% of patients with DAVFs evaluated at this institution. This clinical presentation has not been emphasized in the past and is an atypical clinical manifestation of a reversible vascular anomaly. A thorough examination of patients with relatively rapid dementia or deterioration of mental status, combined with an awareness of such a presentation of DAVFs, should lead to a consideration of venous hypertensive encephalopathy in the differential diagnosis.

References

1. Lasjaunias P, Chue M, Ter Brugge K. **Neurological manifestations of intracranial dural arteriovenous malformations.** *J Neurosurg* 1986;64:724–730
2. Vinuela F, Fox A, Pelz D, Drake C. **Unusual clinical manifestations of dural arteriovenous malformations.** *J Neurosurg* 1986;64:554–558
3. Awad I, Little J, Akrawi W, Ahl J. **Intracranial dural arteriovenous malformations: factors predisposing to an aggressive neurological course.** *J Neurosurg* 1990;72:839–850
4. Borden J, Wu J, Shucart W. **A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment.** *J Neurosurg* 1995;82:166–179
5. Cognard C, Gobin Y, Pierot L, et al. **Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage.** *Radiology* 1995;194:671–680
6. Malik G, Pearce J, Ausman J, Mehta B. **Dural arteriovenous malformations and intracranial hemorrhage.** *Neurosurgery* 1984;15:332–339
7. Leeds N, Terveras J. **Changes in local circulation time in frontal and parietal tumors by rapid serial angiography.** *Acta Radiol Diagn (Stockh)* 1963;1:332
8. Hamada Y, Goto K, Inoue T, et al. **Histopathological aspects of dural arteriovenous fistulas in the transverse-sigmoid sinus region in nine patients.** *Neurosurgery* 1997;40:452–458
9. Houser O, Baker H, Rhoton A. **Intracranial dural arteriovenous malformations.** *Radiology* 1972;163:55–64
10. Chaudhary M, Sachdev V, Cho S, Weitzner I. **Dural arteriovenous malformations of the major venous sinuses: an acquired lesion.** *AJNR Am J Neuroradiol* 1982;10:385–392
11. Brunereau L, Gobin Y, Meder J, Cognard C, Tubiana J, Merland J. **Intracranial dural arteriovenous fistulas with spinal venous drainage: relation between clinical presentation and angiographic findings.** *AJNR Am J Neuroradiol* 1996;17:241–249
12. Andeweg J. **Concepts of cerebral venous drainage and the aetiology of hydrocephalus.** *J Neurol Neurosurg Psychiatry* 1991;54:830–831
13. Lalwani A, Dowd C, Halbach V. **Grading restrictive disease in patients with dural arteriovenous fistulas of the transverse/sigmoid sinus.** *J Neurosurg* 1993;79:11–15
14. Bederson J, Otmar D, Wiestler M, et al. **Intracranial venous hypertension and the effects of venous outflow obstruction in a rat model of arteriovenous fistula.** *Neurosurgery* 1991;29:341–350
15. Kurokawa Y, Kazuo H, Okuyama T, Uede T. **Regional ischemia in cerebral venous hypertension due to embolic occlusion of the superior sagittal sinus in the rat.** *Surg Neurol* 1990;34:390–395
16. Nakase H, Heiman A, Kempski O. **Alterations of regional cerebral blood flow and oxygen saturation in a rat sinus-vein thrombosis model.** *Stroke* 1996;27:720–728
17. Nakase H, Kempski O, Heimann A, Takeshima T, Tintera J. **Microcirculation after cerebral venous occlusions as assessed by laser doppler scanning.** *J Neurosurg* 1997;87:307–314
18. Bederson J. **Pathophysiology and animal models of dural arteriovenous malformations. In: Awad I, Barrow D, eds.** *Dural Arteriovenous Malformations*. Park Ridge, IL: AANS; 1993:23–34
19. Hurst R, Kenyon L, Lavi E, Raps E, Marcotte P. **Spinal dural arteriovenous fistula: the pathology of venous hypertensive myelopathy.** *Neurology* 1995;45:1309–1313
20. DeMarco J, Dillon W, Halbach V, Tsuruda J. **Dural arteriovenous fistulas: evaluation with MR imaging.** *Radiology* 1990;175:193–199
21. Chiras J, Dubs M, Bories C. **Venous infarctions.** *Neuroradiology* 1985;27:593–600
22. Halbach V, Higashida R, Heishima G, Mehringer C, Hardin C. **Transvenous embolization of dural fistulas involving the transverse and sigmoid sinuses.** *AJNR Am J Neuroradiol* 1989;10:389–392
23. Barnwell S, Halbach V, Higashida R, et al. **Complex dural arteriovenous fistulas: results of combined endovascular and neurosurgical treatment in 16 patients.** *J Neurosurg* 1989;71:352–358

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