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422 Reversible Arteriovenous Malformation-induced Venous Hypertension as a Cause of Neurological Deficits Case Report

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ABSTRACT: A case of a dural arteriovenous malformation with prominent localizing neurological deficits is reported. The venous drainage of the lesion and the lack of a significant pial supply implicate venous hypertension as the mechanism of neurological dysfunction. This mechanism is supported further by the angiographic changes and the prompt resolution of the deficits after endovascular treatment. This case illustrates the potential for this frequently postulated but rarely confirmed pathophysiological mechanism to cause reversible neurological dysfunction.

KEY WORDS: Arteriovenous malformation; Dura; Embolization; Venous hypertension

INTRODUCTION

Multiple mechanisms have been postulated to explain the symptoms caused by arteriovenous malformations (AVMs) involving the central nervous system (CNS). Such mechanisms most commonly include hemorrhages, arterial steal phenomena, and diverse mechanisms resulting from interference with venous drainage such as thrombosis, mass effect of enlarged veins, and hydrocephalus. Venous ischemia, neurological dysfunction arising from the passive congestion of venous flow, represents an additional cause of the pathophysiological processes resulting from arteriovenous shunting⁽³⁾. Although the determination of a specific mechanism for symptom production may assist in the understanding of AVM pathophysiological processes and aid in their management, such knowledge is difficult to obtain. The complex angioarchitecture of most AVMs makes the delineation of the contribution from any single nonhemorrhagic mechanism nearly impossible. The rapid shunting of blood, which might otherwise supply adjacent normal brain tissue, and venous enlargement are almost universal findings in AVMs involving the nervous system. In addition, neurological manifestations often are temporally unresponsive to therapeutic manipulations or are of inadequate localizing value to be directly attributed to the AVM.

We report the case of a 61-year-old man with a primarily dural AVM whose clinical course and

lesion angioarchitecture strongly implicate passive venous congestion as the major mechanism resulting in reversible neurological dysfunction occurring at some distance from the AVM nidus. The embolization of the lesion resulted in dramatic improvement of the neurological deficits.

CASE REPORT

The patient, a 61-year-old man, first was evaluated for intermittent right-sided headaches of several years' duration in December 1989. For approximately 1 month before admission, he had been noted to exhibit peculiar behavior and poor judgment, frequently running red lights and getting lost in the neighborhood where he had lived for years. The examination at admission revealed him to be oriented in all spheres and was remarkable only for the presence of left homonymous hemianopsia. A computed tomographic (CT) scan demonstrated multiple linear enhancing structures involving the right temporo-occipital region. The angiographic examination revealed the presence of a dural AVM of the right transverse sinus fed primarily by multiple enlarged branches of the right occipital artery. The lesion was considered surgically untreatable, and no therapy was undertaken at that time.

The patient was admitted to another institution in September 1990 after he had experienced several months of progressive left arm and leg weakness as well as confusion followed by a single generalized seizure. A CT scan showed right posterior temporoparietal hypodensity involving the white matter with mild mass effect. Patchy streaks of high density were present along the cortical margin in the right posterior temporal region consistent with an area of hemorrhagic infarction (Fig. 1). No hydrocephalus was present. Electroencephalography demonstrated focal right-sided slowing with no epileptiform activity. He was treated with Dilantin and dexamethasone and transferred to our institution.

On admission, he was lethargic and disoriented to time, place, and person. Visual field testing was impossible because of an inability to cooperate. Positive neurological findings included left central 7th cranial nerve palsy, left-sided weakness affecting both the upper and lower limbs, a left-sided Hoffman's sign, and a left extensor plantar response.

On October 2, 1990, angiography demonstrated the dural AVM involving the proximal right transverse sinus. The major feeding vessels originated from an enlarged right occipital artery with multiple transmastoid branches. A small additional supply originated from the right middle meningeal artery and the right posterior auricular artery, as well as from several tiny branches of the posterior temporal arteries. Venous drainage flowed into the proximal right transverse sinus with rapid retrograde flow into the straight sinus and the superior sagittal sinus. By the 4-second film, the retrograde filling of cortical veins of both the right and left hemispheres was identified, which persisted late into the venous phase (Fig. 2). The retrograde venous filling was more prominent over the right hemisphere and was persistent, with extremely delayed venous drainage

over both hemispheres. No filling of the distal transverse sinuses was present. No venous dilatation was identified.

Embolization of the right occipital artery was performed using polyvinyl alcohol particles and silk sutures. Postembolization films revealed a considerable decrease in the flow to the lesion (Fig. 3). There was a marked decrease in the retrograde filling of the straight and superior sagittal sinuses. There was no retrograde filling of the right hemispheric cortical veins. The retrograde filling of the left hemispheric cortical veins was detected only after 7 seconds. The complete emptying of the venous phase occurred more rapidly than on pre-embolization films.

Within 12 hours after the embolization, the patient's hemiparesis had resolved. He was oriented in all spheres (i.e., with regard to person, place, and time) but continued to demonstrate left hemianopsia. At follow-up, 9 months after the embolization, there was no recurrence of hemiparesis or mental status abnormalities.

DISCUSSION

This 61-year-old man had a dural AVM that produced deficits including left hemiparesis and mental status abnormalities that resolved after a partial embolization. The character of the reversible deficits and the angioarchitectural features of the lesion make it possible to determine the probable pathophysiological mechanisms in this particular case.

Lasjaunias et al. ⁽³⁾ have reviewed the possible vascular mechanisms resulting in neurological deficits from AVMs involving the dura. The authors indicate that the insignificant arterial contribution from the internal carotid or vertebral circulations in dural AVMs, as was present in this case, cannot be considered to have any CNS hemodynamic consequences. They suggest instead that any evidence of CNS dysfunction associated with dural AVMs appears to be related to venous hypertension or congestion. Potential mechanisms of venous dysfunction are classified as those occurring secondary to venous thrombosis; high venous pressure resulting in either increased superior sagittal sinus pressure and increased intracranial pressure or venous ischemia; venous mass effect; or venous rupture with hemorrhage.

After developing new onset hemiparesis followed by a single episode of seizure activity, the patient demonstrated CT findings of posterior temporal venous infarction. The presence of venous infarction implies interference with the drainage from the posterior temporal region, possibly from dural sinus occlusion or passive increased venous pressure. The area of infarcted brain was, however, not one to which symptoms of hemiparesis would be referable.

The angiographic examination revealed that the supply to the lesion originated almost entirely from external carotid branches supplying the dura. Only a minimal supply originated from the inferior temporal pial vessels, and none originated from the pial supply, which, even if enlarged, would be expected to

result in hemiparesis. The arterial phase of the angiogram, therefore, indicated no significant participation in the symptom pathogenesis by the arterial "steal" of blood from normal brain tissue. Venous drainage from the lesion was retrograde via the dural venous sinuses and into the cortical veins of both cerebral hemispheres, more prominently on the right. No evidence of significant venous dilatation was found, and no venous structures that might be responsible for mass effect were present. The retrograde filling of bilateral cortical veins and delayed venous emptying, however, reflected a significant degree of interference with venous drainage from the cerebral hemispheres. The earlier, more prominent retrograde flow over the right frontoparietal area indicated a more significant disturbance on that side, accounting for the focal paresis.

Postembolization angiography revealed not only a striking diminution of the flow into the AVM but also a significant improvement in the venous flow draining normal brain tissue. The pre-embolization retrograde venous flow into the cortical hemispheric veins had resolved completely on the right and had significantly decreased on the left. The complete drainage of the brain occurred more quickly, which suggested a decrease in abnormally elevated venous pressure after the shunt occlusion.

It is postulated that the patient's left hemiparesis was the result of the passive congestion of veins draining distant normal tissue, specifically the right motor area. The congestion resulted from elevated venous pressure caused by the arteriovenous shunt through the AVM and consequent venous ischemia. This conclusion is strengthened by the angiographic findings and clinical events after the embolization. The absence of a significant cortical arterial supply allows the elimination of the arterial "steal" phenomenon as a participant in the pathophysiological processes, in keeping with the findings of Lasjaunias et al ⁽³⁾. The absence of hydrocephalus negates significant interference with the CSF drainage as a contributor to the patient's clinical picture. The absence of significantly dilated venous structures or any evidence of a hemorrhage involving the right frontoparietal area is significant in view of the report by Viñuela et al. ⁽⁶⁾ of 12 cases of dural AVMs whose CNS symptoms were attributed to mass effect or a hemorrhage originating from dilated veins. The prompt postembolization resolution of hemiparesis also speaks against a hemorrhage or infarction involving the area to which the motor symptoms were referable. The complete resolution of hemiparesis, therefore, provides additional support for the mechanism of passive cortical venous congestion with consequent venous ischemia in the production of the focal neurological deficit.

Venous hypertension resulting in focal neurological dysfunction is a frequently postulated but infrequently confirmed mechanism of intracranial pathophysiological processes in patients with dural AVMs. This contrasts with the situation involving a dural AVM-associated spinal cord dysfunction in which venous congestion has been demonstrated

unequivocally to elicit myelopathy ^(6,7). The probable role of venous congestion in the production of intracranial neurological deficits was discussed by Kosnik et al. ⁽²⁾ in their 1974 review of dural AVMs. Miyasaka et al. ⁽⁴⁾ emphasized the potential role of retrograde cortical venous drainage in the genesis of parenchymal edema, swelling, and venous infarction. They noted the reversal of these features on serial CT scans as well as the resolution of focal deficits over several months. The focal nature of our patient's deficit combined with the postembolization resolution of both angiographic and clinical abnormalities demonstrates the role of venous hypertension in the production of focal neurological deficits at a distance from the AVM nidus.

The improvement in mental status after embolization was striking and, although not supported by a quantitative neuropsychological evaluation, was identified readily on the mental status portion of the neurological examination. This less localized but nonetheless clinically confirmed mental status improvement also seems attributable to the overall improvement in the bilateral hemispheric venous drainage. Multiple reports documenting increased intracranial pressure associated with intracranial AVMs indicate that both venous hypertension and intracranial pressure abnormalities may resolve with the obliteration of the shunt ^(1,5). The retrograde filling of the major dural venous sinuses in this patient and the improved drainage after the embolization provide evidence to implicate increased venous pressure in the reversible abnormalities of his mental status.

In summary, this case provides evidence for venous hypertension as a cause for both focal neurological deficits referable to a locus distant from the AVM nidus as well as for the production of less-localized abnormalities of mental status. 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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Figure 1. CT scan without contrast enhancement shows edema involving the right posterior temporal region with cortical hyperdensity consistent with hemorrhagic venous infarction (*arrow*).

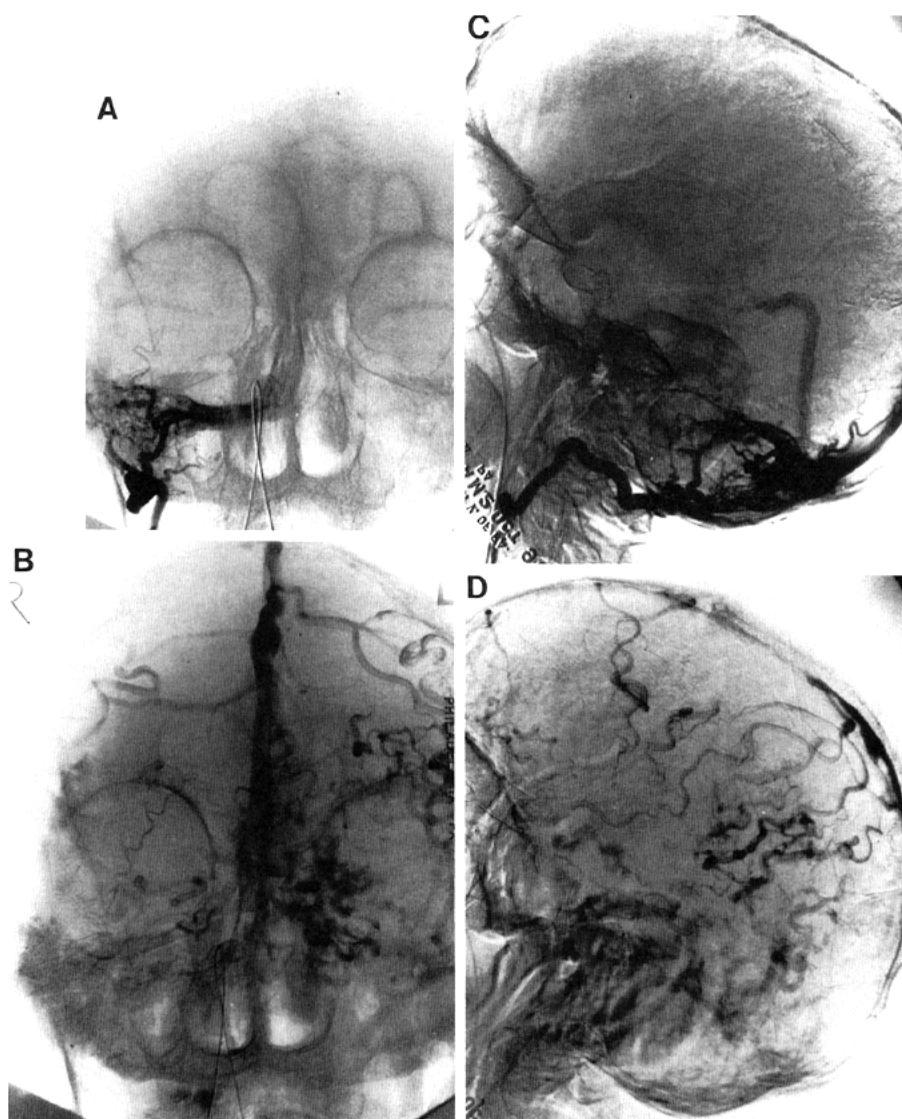


Figure 2. Anteroposterior (*A* and *B*) and lateral (*C* and *D*) arteriograms show a dural AVM fed by the right occipital artery with early retrograde venous drainage into cortical veins bilaterally.

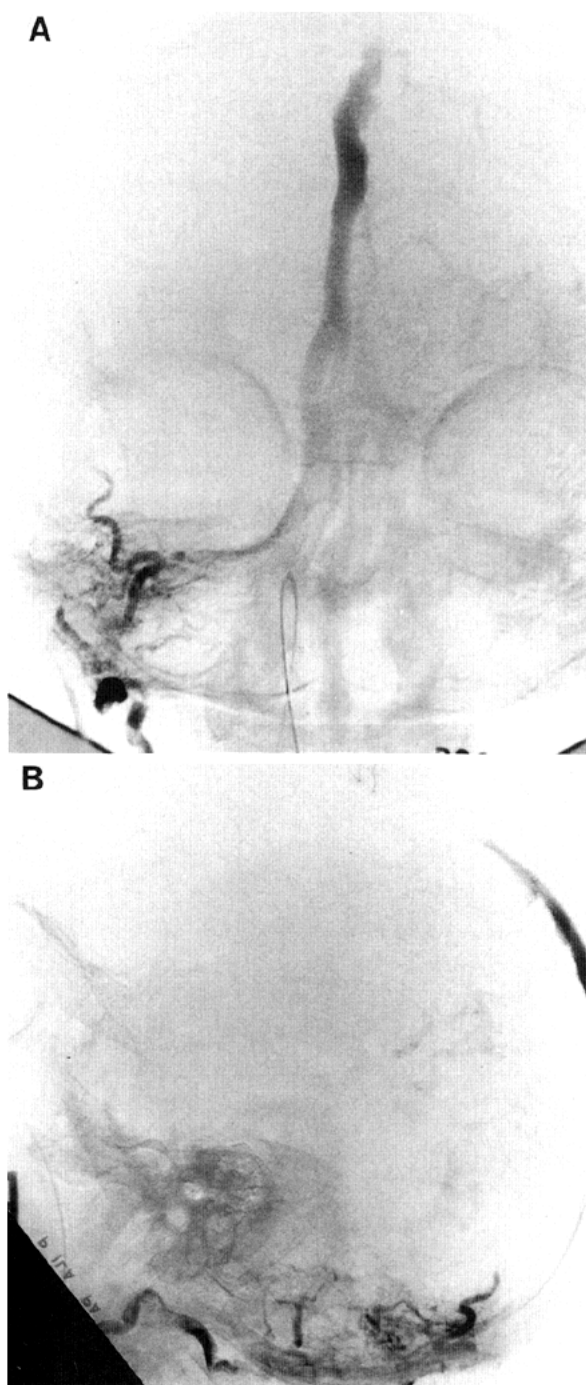


Figure 3. Anteroposterior (*A*) and lateral (*B*) postembolization films show a significant decrease in the arterial shunting and the resolution of cortical venous reflux to the right hemisphere.