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An Unruptured Arteriovenous Malformation with Edema

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Summary: We report a case of unruptured arteriovenous malformation in which an extensive zone of increased signal intensity in the brain parenchyma adjacent to the nidus is demonstrated on T2-weighted MR. This area of perilesional hyperintense signal exerts a compressive effect, suggesting that it represents perilesional edema.

Index terms: Arteriovenous malformations, cerebral; Brain, edema; Brain, magnetic resonance

Compressive effects in unruptured arteriovenous malformations have been reported since computed tomography (CT) scans became available (1–4). A high percentage of cases with compressive effects caused by unruptured arteriovenous malformations have been found (3, 4). Although multiple factors contribute to the compressive effect in arteriovenous malformations without hemorrhage (1–5), it is unusual for perilesional edema to exert such an effect (3).

Case Report

A 46-year-old woman presented with frequent psychomotor and generalized tonic-clonic seizures over a 12-year period.

The findings of a neurologic examination at the time of admission were normal. A right carotid angiogram demonstrated a large frontal arteriovenous malformation fed by the anterior cerebral artery. Dilatation of the draining vein was also shown (Fig 1). Obstruction of the venous drainage system was not demonstrated. CT scans showed an arteriovenous malformation-like lesion in the right frontal lobe and a low-density area adjacent to the lesion (Fig 2). Magnetic resonance (MR) was then performed on a 0.5-T unit. T1-weighted images (400/25/2 [repetition time/echo time/excitations]) demonstrated convoluted vascular signal voids of an arteriovenous malformation (Fig 3A), and T2-weighted images (2000/60) showed a large area of increased signal intensity in the brain tissue adjacent to the nidus (Fig 3B). The high-intensity lesion compressed the body of the right lateral ventricle (Fig 3B), suggesting that it corresponded to perilesional edema.

Local cerebral blood flow studies were performed using single-photon emission CT with N-isopropyl-p-[^{123}I]iodoamphetamine (^{123}I-IMP). IMP single-photon emission CT images 15 and 120 minutes after intravenous injection of ^{123}I-IMP (3 mCi) showed a defect of activity that indicated the position of the nidus and the decreased local cerebral blood flow around the malformation (Fig 4).

Removal of the nidus was achieved without complication. No evidence of hemorrhage from the arteriovenous malformation was noted. In order to avoid unnecessary damage to the brain tissue, no histologic study of the brain parenchyma adjacent to the nidus was undertaken.

Postoperative Course

The patient showed no neurologic deficit after the operation. Postoperative right carotid angiography demonstrated complete removal of the arteriovenous malformation. T2-weighted MR (2000/60) performed 2 years after surgery showed a reduction in size of the increased-signal zone in the brain parenchyma adjacent to the former location of the arteriovenous malformation. Also, no compressive effect caused by the increased-signal area was observed (Fig 5). This suggested that the perilesional high-intensity zone probably changed from edema to gliosis.

Discussion

Although arteriovenous malformations generally tend to replace brain tissue and do not, in the absence of hemorrhage, act as mass lesions, many exceptions occur. Compressive effects have been demonstrated in unruptured arteriovenous malformations by CT (1, 6–8). Kumar et al (3) reported that a compressive effect, demonstrated by CT, occurred in 55% of 60 patients with clinically unruptured arteriovenous malformations. Recently, Smith et al (5) observed such an effect on MR examinations of six out of 15

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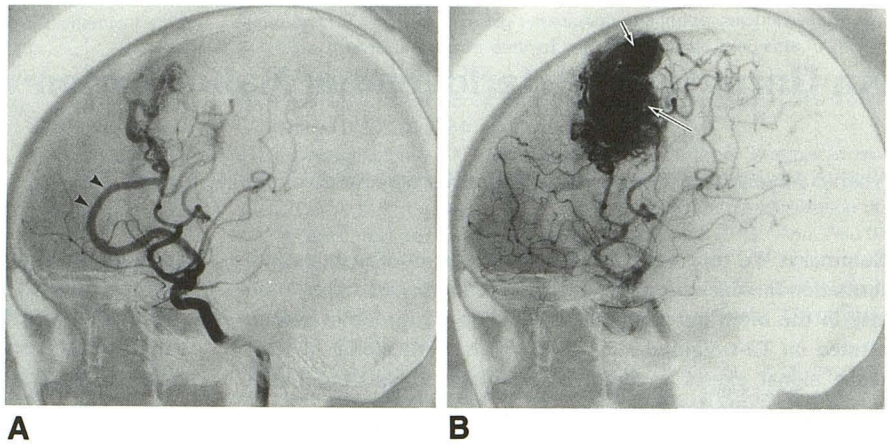

Fig. 1. Lateral view, arterial phase. Preoperative right carotid angiograms demonstrating a large frontal arteriovenous malformation (*long arrow*) fed by the anterior cerebral artery (*arrowheads*). Note the varicose dilatation of the draining vein (*short arrow*).

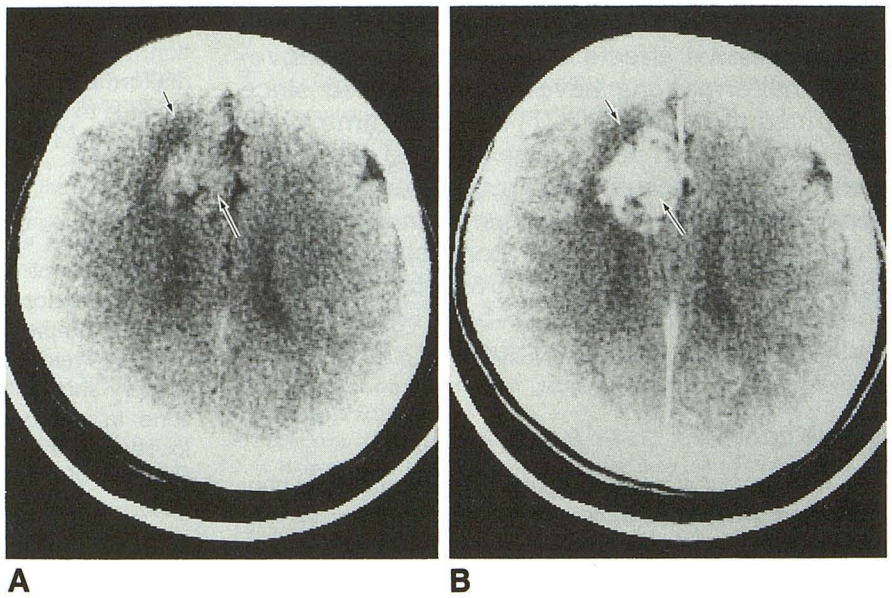

Fig. 2. *A*, Plain CT scan showing a poorly defined area of decreased density (*short arrow*) around a lesion of slightly elevated density (*long arrow*) in the right frontal lobe.

B, Contrast-enhanced CT scan showing racemose lesion (*long arrow*) in the right frontal lobe and a low-density area adjacent to it (*short arrow*).

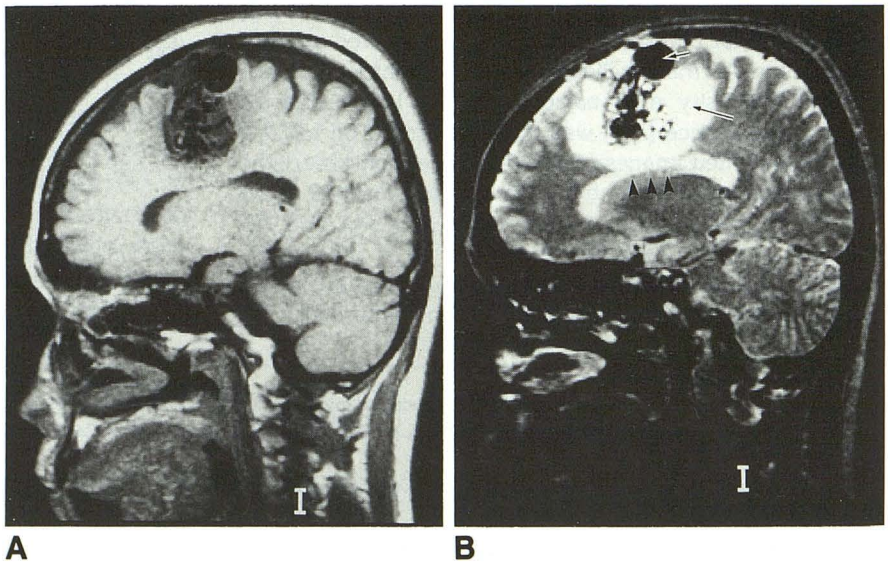

Fig. 3. *A*, Sagittal T1-weighted (400/25) MR image demonstrating the convoluted vascular signal voids of an arteriovenous malformation.

B, Sagittal T2-weighted (2000/60) scan. An extensive zone of high-intensity signal (*long arrow*) around the nidus, compressing the lateral ventricle (*arrowheads*), and a varix-like dilatation of the draining vein (*short arrow*) are shown. No evidence of hemorrhage is observed on MR.

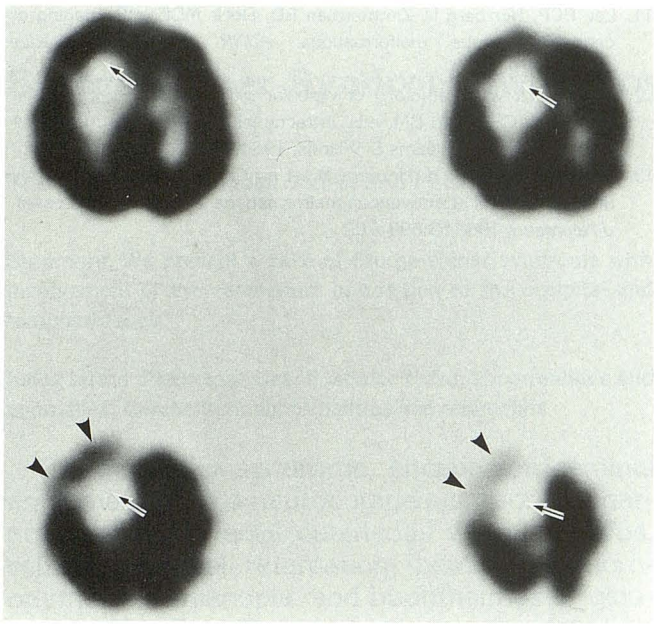

Fig. 4. Axial images of single-photon emission CT with ^{123}I-IMP taken 15 minutes after intravenous injection of ^{123}I-IMP. Note the defect of ^{123}I-IMP activity, representing the nidus itself (*arrow*) and the decreased local cerebral blood flow around the malformation (*arrowheads*).

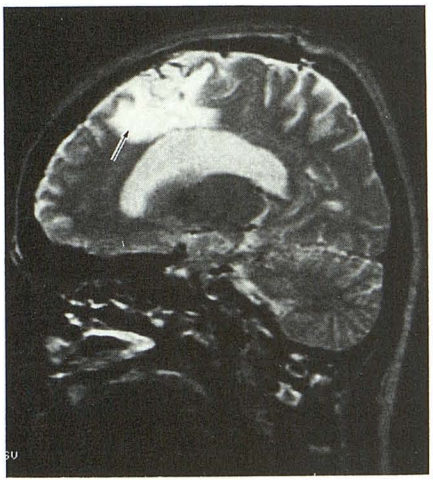

Fig. 5. Sagittal T2-weighted (2000/60) MR scan obtained 2 years after surgery showing an extensive zone of increased signal (*arrow*) in the brain tissue adjacent to the former location of the arteriovenous malformation. No compression of the lateral ventricle by the high-intensity lesion is observed.

arteriovenous malformations without hemorrhage.

The sizes of the arteriovenous malformations and ectatic veins are felt to contribute to the compressive effect in unruptured arteriovenous malformations (1–5). Perilesional edema associated with unruptured arteriovenous malformations has been recognized in CT scans (1, 2, 9), and two cases of massive edema contributing to compressive effect have been reported (3). In the present case, there was no evidence of hemorrhage from the malformation. Perilesional high-intensity signals, as shown by MR in the present study, may represent edema secondary to the marked compressive effect of the arteriovenous malformation (Fig 3B). A dilated venous sac was observed in the present case (Figs 1 and 3). However, compression of the ipsilateral lateral ventricle is probably the result of perilesional edema.

The pathophysiology of perilesional edema in the present case is uncertain. Effects of unruptured arteriovenous malformations on adjacent brain tissue have been documented on the basis of histologic, neuroradiologic, and cerebral blood flow studies (1–5, 7–13). Brain parenchyma around malformations may suffer ischemic effects, attributable to either a steal phenomenon (7, 12) or a decrease in cerebral perfusion pressure secondary to cerebral venous hypertension caused by an arteriovenous shunt (6, 13). Eventually, these ischemic effects result in edema or gliosis in the brain tissue around the nidus. T2-weighted MR images demonstrate these lesions dramatically as high-intensity signals (5, 8, 10, 11). Although it is sometimes difficult to differentiate edema from gliosis, the presence of marked compression by a high-intensity lesion on T2-weighted images strongly suggests the presence of edema (Fig 3B). On T2-weighted images, high-intensity lesions with no compressive effect probably represent gliosis (Fig 5).

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