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MR Imaging of Angiographically Occult Vascular Malformations

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Eleven patients with 15 angiographically occult arteriovenous malformations were studied by magnetic resonance (MR) imaging and computed tomography (CT). Five patients had biopsy proof; six were clinically diagnosed from the long-term clinical follow-up (more than 3 years) and imaging features. Of the 15 lesions, 11 were recognized by both CT and MR. Each method was falsely negative for two lesions. The most useful contribution of MR in the characterization of angiographically occult arteriovenous malformations was the depiction of hemorrhagic foci in 12 of 13 lesions seen on MR. High-attenuation foci indicative of hematomas were seen in only five lesions on CT; the rest were iso- or hypoattenuating. CT detected two very small lesions, in one case as punctate foci of enhancement and in the other as punctate calcification, that were not seen with MR. MR complements CT in characterizing angiographically occult arteriovenous malformations and in distinguishing them from similar-appearing lesions, in particular, small neoplasms. However, when such lesions are seen with only focal calcification and subtle enhancement on CT, routine MR may miss them.

The detection and accurate diagnosis of angiographically occult arteriovenous vascular malformations (AVMs) of the brain on the basis of clinical or radiologic manifestations was uncommon until the introduction of computed tomography (CT). Although CT criteria have now been well established and serve to improve detection of these lesions [1–3], preoperative discrimination from tumors remains problematic. The relative sensitivity and specificity of magnetic resonance (MR) imaging in these lesions is as yet unreported. However, this modality does have promising capabilities in depicting vascular structures with great anatomic detail and in identifying hemorrhagic lesions [4]. The purpose of our present study was to establish in a preliminary fashion the relative value of MR in the diagnosis and characterization of AVMs.

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Subjects and Methods

Nineteen patients in whom the diagnosis of an angiographically occult AVM was strongly considered were referred for MR imaging. The lesion was suspected on the basis of either clinical presentation and/or a characteristic CT appearance and an angiogram that failed to demonstrate abnormal vascularity. The patients were studied during a 36 month period ending December 1984. Eight patients were found to have tumors; all of the tumors were surgically confirmed. In the other 11 patients the diagnosis of AVM was established by biopsy (five patients) or was strongly considered on the basis of long-term follow-up (>3 years) and characteristic clinical and radiographic findings (six patients). Ages ranged from 2 to 56 years. Five were female; 6 were male. The clinical presentations were seizures, acute intracranial hemorrhage, or progressive focal deficit.

Nine patients had unenhanced CT examinations (GE 8800 or 9800); all had postenhancement studies. The lesions were analyzed on CT for density alterations indicating hemorrhage, enhancement, or calcification, as well as for low-density foci. Angiography was performed in all 11 patients. The MR examinations were performed on a Diasonics MT/S superconducting unit operated at 0.35 T [5]. Images were obtained using dual spin-echo (SE) multisection

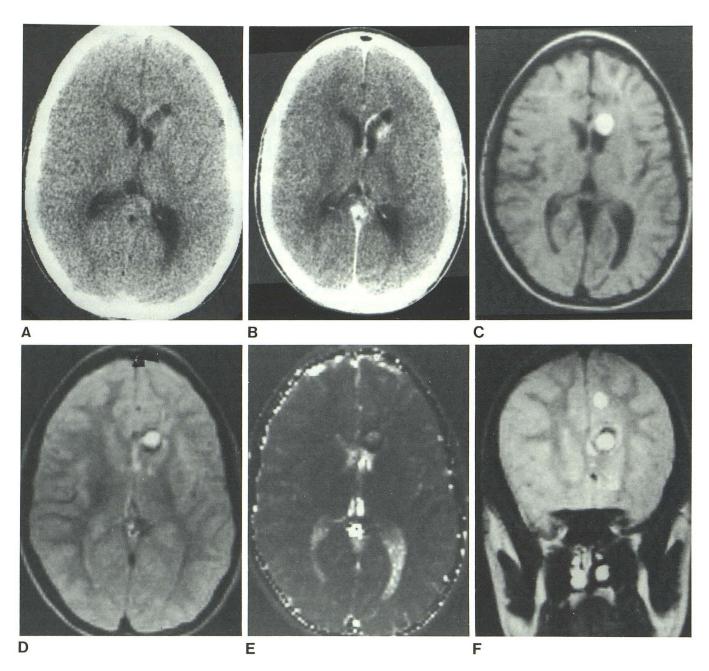


Fig. 1.—17-year-old girl with headaches, seizures, no focal deficit. A, Unenhanced CT scan. Area of slightly increased density adjacent to right frontal horn. B, Postcontrast. Ring enhancement of lesion suggests involvement of walls of ventricle. C, T1-weighted MR image (TR 500/TE 28). Well circumscribed area of high signal intensity reflects relatively short T1 characteristic of subacute or chronic hematoma. D, T2-weighted image (TR 2000/TE 56). Relative high signal in center of lesion reflects relative prolongation of T2, which may reflect hematoma of any age and is less specific. Low-intensity ring may

represent collagenous capsule. E, Calculated T2 image. This display technique will exaggerate any echo rephasing phenomena, which can be identified as areas of increased signal intensity. Examples in this image include internal cerebral veins, vein of Galen, and cortical veins. No slow flowing or rephasing areas are seen within lesion. F, T2-weighted image (TR 1500/TE 56) in coronal plane. Two additional parasagittal lesions are identified. Only cephalad and periventricular ones were seen on CT.

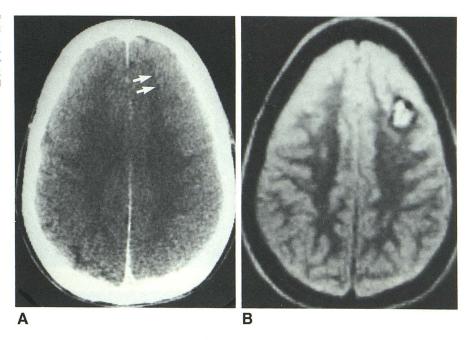
imaging techniques with both short (500 msec) and long (1500–2000 msec) pulse interval (TR) sequences. Echo delay (TE) in all the sequences was 28 msec for the first echo and 56 msec for the second. In one patient, the short TR sequence did not encompass the lesion because of inaccurate centering of the patient in the imager. T1 and T2 relaxation data were calculated from acquired data. These data were also used to create synthetic images indicating T1 and T2 relaxation times [6]. The images were analyzed for abnormal alterations of signal intensity and flow effects.

Results

Eleven patients were identified with angiographically occult AVMs. Nine patients had single lesions, one patient had two, and one had four, totaling 15 AVMs. Eleven of 15 lesions were visible on both CT and MR, two were visible only on CT, and two only on MR.

High-density components on CT, compatible with hematomas, were visible in five lesions. The density values were

Fig. 2.—17-year-old girl with 3 year history of seizures. No focal deficits. Presumed occult AVM with remote hemorrhage. A, Postcontrast CT scan. Area of equivocal increased density (arrows). B, T2-weighted image (TR 2000/TE 56). High-intensity circumscribed lesion surrounded by irregular dark rim. Findings consistent with old hemorrhage and fibrous capsule.



such that calcium deposition could not be reliably excluded. Three lesions had components that were definitely calcified on CT. All three of these lesions had a component that enhanced with intravenous contrast material. On MR, the presence of calcium was identified prospectively in one lesion as an area of signal void, and only retrospectively identified in another (fig. 3). The presence of calcium could not be identified in the third patient, resulting in a false-negative MR examination. Two additional lesions exhibited focal areas of central low density on CT.

In 12 of 13 lesions seen on MR, abnormal high-signal intensity foci were present in both T1- and T2-weighted MR images reflecting the short T1 and long T2 relaxation times of these foci (figs. 1 and 2). In the other case the T1-weighted sequence did not encompass the lesion, but on the T2-weighted image that did, it again appeared as high intensity similar to the other lesions. Eight of these foci corresponded to the five hematomas, to the two central low-density areas, and to a hyperdense area abutting definite calcification seen on CT (described above). The other five lesions exhibiting high intensity on the MR scan corresponded to isodense lesions on CT.

All 13 lesions seen on CT exhibited contrast enhancement, usually of minimal degree. No correlate of this enhancement could be detected by MR.

All MR images were examined for flow-related effects such as flow enhancement, high-velocity signal loss and even-echo rephasing. These effects were not observed in any angiographically occult AVMs but were routinely seen in large intracranial vascular structures such as the carotid arteries and dural venous sinuses.

Eight patients with small neoplasms had final diagnoses established by surgical biopsy. In seven of these eight patients whose initial presentation was consistent with either an angiographically occult AVM or a small neoplasm, CT showed foci of low density with homogeneous enhancement after

contrast administration. In these patients, MR showed more extensive tissue abnormality than did CT and excluded the presence of subacute or chronic hemorrhage by the absence of areas of increased signal on the T1-weighted images (fig. 4). In one patient, a 31/2 cm tumor did have an area of associated hemorrhage within it, which rendered both studies indeterminate for a specific diagnosis (fig. 5).

In one patient with normal pre- and postcontrast CT scans, MR depicted the presence of an old hemorrhagic collection in the pons. This patient had a history of progressive focal deficit, and at surgery a vascular malformation with associated chronic hematoma was found. In one patient with four surgically proven AVMs, all four were visible as high intensity on both T1- and T2-weighted MRI. Three were seen on CT; the fourth was isodense.

The calculated T1 and T2 images generated in each case were not particularly helpful. Lesions were seen equally well on acquired and calculated images. Qualitative information as to T1 and T2 relaxation times of demonstrated lesions could, with experience, be quickly derived from a combination of acquired T1- and T2-weighted images without referring to synthetic images.

Discussion

Angiographically occult AVMs encompass a diverse group of lesions with different histologic characteristics. The common diagnostic feature is the absence of abnormal vascularity on routine angiography. Histologically, these lesions are composed of vascular structures that contain slow flow, and their spectrum includes thrombosed AVMs, cavernous angiomas, capillary telangiectasias, and venous angiomas [7]. Associated pathologic findings include focal areas of dystrophic calcification, hemorrhage of various ages, associated cerebral atrophy, gliosis, and edema [8]. These lesions have on occasion been referred to in the literature as "cryptic AVMs"

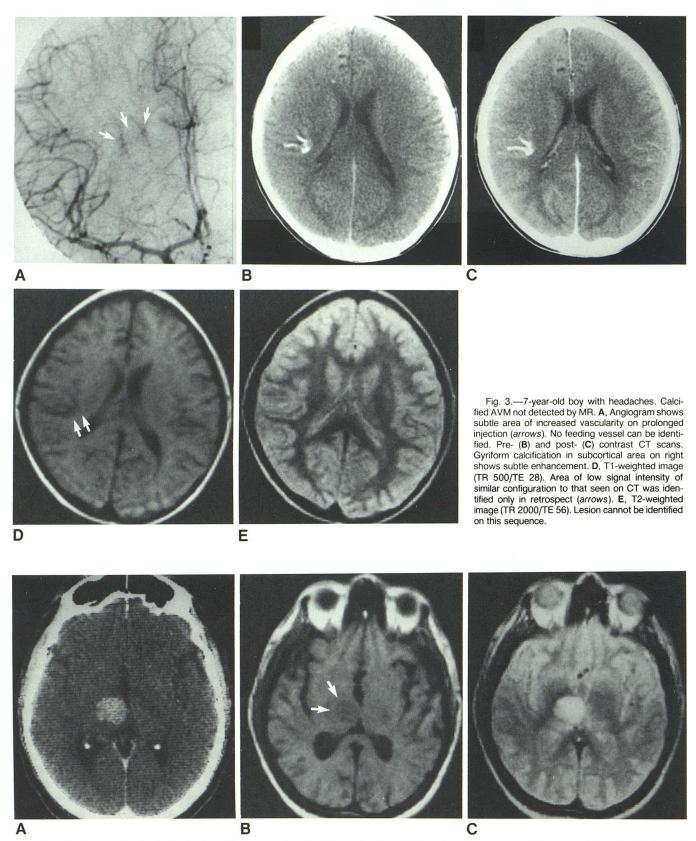
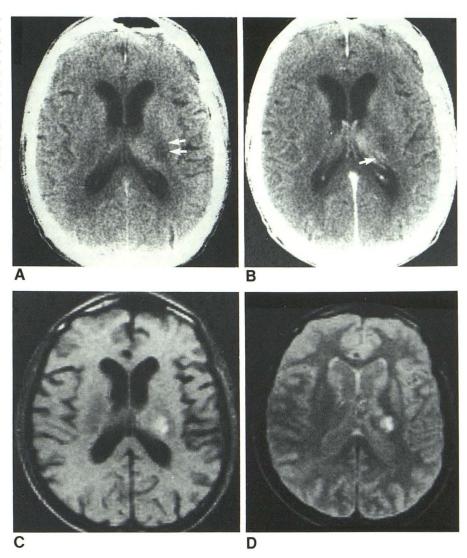


Fig. 4.—46-year-old man with relatively short history of progressive left-sided focal deficit. Biopsy demonstrated anaplastic astrocytoma. A, Contrast-enhanced CT scan. Enhancing lesion with minimal mass effect. Absence of surrounding edema. B, T1-weighted image (TR 500/TE 28). Lesion is depicted as area of low signal intensity reflecting relatively prolonged T1 (*arrows*). This

excludes the presence of subacute or chronic hematoma. C, T2-weighted image (TR 2000/TE 56). Relatively high signal intensity reflects relatively prolonged T2 values of lesion. Combination of prolonged T1 and T2 would not support diagnosis of AVM, but rather of malignant neoplasm.

Fig. 5.—67-year-old cello player with progressive right-sided focal deficit. CT and MR findings suggested AVM. Small hemorrhagic glioblastoma proven at necropsy. A, Noncontrast CT scan. Lowdensity lesion in posterior part of internal capsule (arrows). B, Contrast-enhanced scan. shows minimal enhancement on medial aspect seemingly related to choroid plexus (arrow). C, T1weighted image (TR 500/TE 28). High intensity (short T1) of lesions indicates presence of blood. D, T2-weighted image (TR 2000/TE 28). High signal intensity reflects prolonged T2. No increase in area of lesion supports diagnosis of cryptic malformation rather than malignant neoplasm. In latter, one would expect broader area of increased signal intensity because of frequently associated edema.



[9], "microangiomas" [10], and "small vascular malformations" [11].

The characteristic CT appearance of angiographically occult AVMs is usually that of a well circumscribed area of increased density that is either homogeneous or mottled. After administration of contrast material, there may be patchy, homogeneous, or, rarely, ring enhancement. One-third to one-half of the lesions will demonstrate a mass effect. Edema is generally absent [1, 2]. The characteristic locations of the lesions include brainstem, periventricular white matter, and the gray/white-matter junction. In a small number of cases, repeated hemorrhages, which may be subclinical, cause these lesions to grow and exhibit a ringlike enhancement pattern (fig. 1).

Although CT has in fact accounted for the detection of a larger number of these lesions than previously possible, the above characteristics are relatively nonspecific. The same findings can be attributed to a low-grade tumor that could be calcific and/or hemorrhagic. A significant number of patients with occult AVMs are not studied at the time of an acute

bleed. The low-density components of a lesion seen on CT often reflect the presence of a subacute or chronic hematoma. The diagnostic difficulty is that neoplasms or associated edema may have an identical appearance. The ability to distinguish between these diverse sources of low density could serve to improve specificity. This is where MR may prove to be of considerable value.

Subacute and chronic hematomas have a relatively short T1 relaxation time and a prolonged T2 relaxation time resulting in areas of relatively increased signal intensity when all three parameters are compared with normal brain tissue on both T1- and T2-weighted images. The only other relatively common tissue constituent that behaves in a similar fashion in the central nervous system is fat, but its T2 relaxation is shorter. Therefore, MR can be a remarkably sensitive means of determining the presence of hematomas and a fairly specific means of characterizing subacute and chronic hematomas or their breakdown products. It is in the latter area that CT is relatively weak. Therefore, on a theoretic basis and, in fact, in our initial experience, it would appear that MR is an effective

means of providing specificity in patients who have CT and/ or clinical findings that make them suspect for harboring an AVM.

All AVMs demonstrated on MR images, with the exception of a single lesion, displayed evidence of previous hemorrhage—high intensity on both T1- and T2-weighted images. (The single exception was hyperintense on the T2-weighted image but not included in the T1-weighted sequence because of a positioning error.) The absence of hemorrhage on MR, therefore, may be a sensitive indicator that one is dealing with pathology other than an angiographically occult AVM, in particular, a neoplasm. The presence of hemorrhage, on the other hand, is suggestive of but not specific evidence for an AVM. It is seen in a small percentage of neoplasms but is much more common in vascular malformations. Although the diagnosis of AVM was only presumptive in six of our cases, the MR findings strongly favor that diagnosis over a lowgrade neoplasm given the presence of hemorrhage. The latter event is a hallmark of higher grades of malignancy than those that occasionally mimic AVMs. Such more malignant lesions generally make themselves obvious in a shorter period than that (>3 years) used as a criterion in our presumed AVM cases.

It is premature at this time to project the relative sensitivity of the two methods. However, this is an important question, for in the future it will determine which technique will be the "screening" study of choice. There were several limitations of MR encountered with this preliminary experience. There was a relative inability to detect the presence of calcium, a fairly frequent component of AVMs [2]. The MR studies also failed to detect pathologic vessels. This shortcoming may reflect the limited spatial resolution of this first-generation unit. Inherent contrast resolution of vessels is excellent with MR. Smaller pixel displays and thinner sections should, on theoretic grounds, enable the imager to detect abnormal vessels with greater sensitivity. These same comments can also be applied to second-echo rephasing phenomena, which should serve to identify slow flow, either on the venous side or in the partly thrombosed vessels.

The MR correlate of the enhancement seen on CT is difficult to identify accurately. The enhancement seen on CT often reflects contrast agent in the relatively increased vascular bed of an AVM. This hypervascularity is not apparent on MR images if it occurs at the capillary level. The enhancement of AVMs on CT may also reflect a blood-brain barrier (BBB) defect known to be associated with large vascular malformations. The increased signal intensity seen on the MR studies presumably represents a combination of both subacute and/or chronic hematoma as well as increased water and decreased myelin content associated with dystrophic areas found in these lesions after BBB breakdown and/or hemorrhage.

In those patients in whom CT has difficulty distinguishing between a slow-growing tumor and cryptic malformation, MR may prove to be of additional assistance. Our experience with neoplasms in general, as well as with the eight patients in this series specifically, indicates that MR in the presence of a malignant neoplasm usually shows a much larger area of

abnormality than appreciated on CT. In the case of the cryptic malformations, the area representing the lesions was similar with both methods. In addition, nonhemorrhagic neoplasms have a relatively long T1 such that they appear as areas of low signal intensity on a T1-weighted image, differentiating them from a subacute or chronic hematoma, which appears as an area of increased signal intensity due to the relatively short T1 relaxation time.

The signal loss associated with calcific replacement of normal brain parenchyma was difficult to detect in our series. Inability to detect these areas reflects thickness of the sections, limited resolution in the x and y plane (pixel size of 1.6 \times 1.6 mm), as well as the early part of the diagnostic learning curve on which we were operating. At the present time, it is undoubtedly easier to identify calcific deposit on CT. It remains to be seen whether with improved resolution and experience with MR this will remain true in the future.

At the present time, MR is definitely a very useful technique in providing increased specificity. It is most useful in determining whether a lesion with the CT characteristics of a cryptic malformation is in fact an AVM, as evidenced by hematoma, or more likely representative of a low-grade glioma.

REFERENCES

- Kendall BE, Claveria LE. The use of computed axial tomography for the diagnosis and management of intracranial angiomas. Neuroradiology 1976;12:141–160
- Becker DH, Townsend JJ, Kramer RA, Newton TH. Occult cerebrovascular malformations. A series of 18 histologically verified cases with negative angiography. Brain 1978;102:249–287
- Bell BA, Kendall BE, Symon L. Angiographically occult arteriovenous malformations of the brain. J Neurol Neurosurg Psychiatry 1978;41:1057–1064
- Crooks LE, Mills CM, Davis PL, et al. Visualization of cerebral and vascular abnormalities. The effects of imaging parameters on contrast. *Radiology* 1982;144:843–852
- Crooks LE, Arakawd M, Hoenninger J, et al. Nuclear magnetic resonance whole body imager operating at 3.5 kilogauss. *Radiology* 1982;143:169–174
- Mills CM, Crooks LE, Kaufman L, Brant-Zawadzki M. Abnormalities: use of calculated T1 and T2 magnetic resonance images for diagnosis. *Radiology* 1984;150:87–94
- Russell DS, Rubinstein LJ. Pathology of tumors of the nervous system, 4th ed. Baltimore: William & Wilkins, 1977:129–134
- Newton TH, Troost TB. Arteriovenous malformations and fistulae. In *Radiology of the Skull and Brain*, Newton TH and Potts DG (eds). Volume 2, Book 4, pp. 2490–2565, St. Louis, Mosby, 1974
- Crawford JV, Russell DS. Cryptic arteriovenous and venous hematomas of the brain. J Neurol Neurosurg Psychiatry 1956;19:1–11
- Margolis G, Odom GL, Woodhall B, et al. The role of small angiomatous malformations in the production of intracerebral hematomas. J Neurosurg 1951;8:564–575
- Jensen HP, Brumlik J, Boshes B. The application of serial angiography to diagnosis of the smallest cerebral angiomatous malformations. J Nerv Ment Dis 1963;136:1–14