



Discover Generics

Cost-Effective CT & MRI Contrast Agents



FRESENIUS
KABI

WATCH VIDEO

AJNR

Magnetic resonance imaging of a venous angioma.

J A Scott, G T Augustyn, R L Gilmor, J Mealey, Jr and E W Olson

AJNR Am J Neuroradiol 1985, 6 (2) 284-286

<http://www.ajnr.org/content/6/2/284.citation>

This information is current as
of June 23, 2025.

Magnetic Resonance Imaging of a Venous Angioma

John A. Scott,¹ Gary T. Augustyn,¹ Richard L. Gilmor,^{1,2} John Mealey, Jr.,² and Eldon W. Olson¹

The appearance of intracranial venous angiomas at angiography and computed tomography (CT) has been well described [1-9]. We report the visualization of a venous angioma by magnetic resonance (MR) imaging.

Case Report

A 40-year-old man was admitted for evaluation of focal motor seizures of the right lower leg, progressing to generalized tonic-clonic convulsions. His first seizure occurred about 1 week before admission. He had been having occasional bitemporal headaches over the preceding 2 years, but was otherwise healthy. Physical examination and laboratory studies were unremarkable. An electroencephalogram and noncontrast CT scan at another hospital were normal.

MR imaging was performed using a Technicare Teslacon 0.15 T resistive system. On T2-weighted images (fig. 1) a cortically based, roughly oval region of high signal intensity was seen extending into the centrum semiovale. A circular structure with absent signal was seen to course from this lesion toward the deep venous system. T1-weighted images (fig. 2) were less helpful in defining the lesion. Estimated T1 and spin-density images were calculated by the Observed T1 (OT1) package supplied by Technicare. This program uses matched data from a spin-echo (SE)/inversion SE pulse sequence (echo time [TE]1 = 30, repetition time [TR]1 = 1250, inversion time [TI]1 = 400, TE2 = 30, and TR2 = 1250 msec) and a "two-point technique" to derive values for each pixel. These images demonstrated increased estimated OT1 and spin density of the lesion in comparison with white matter.

CT with intravenous contrast enhancement showed the typical findings of a venous angioma in the deep left parietal lobe (fig. 3). The presence of a venous angioma was confirmed at angiography

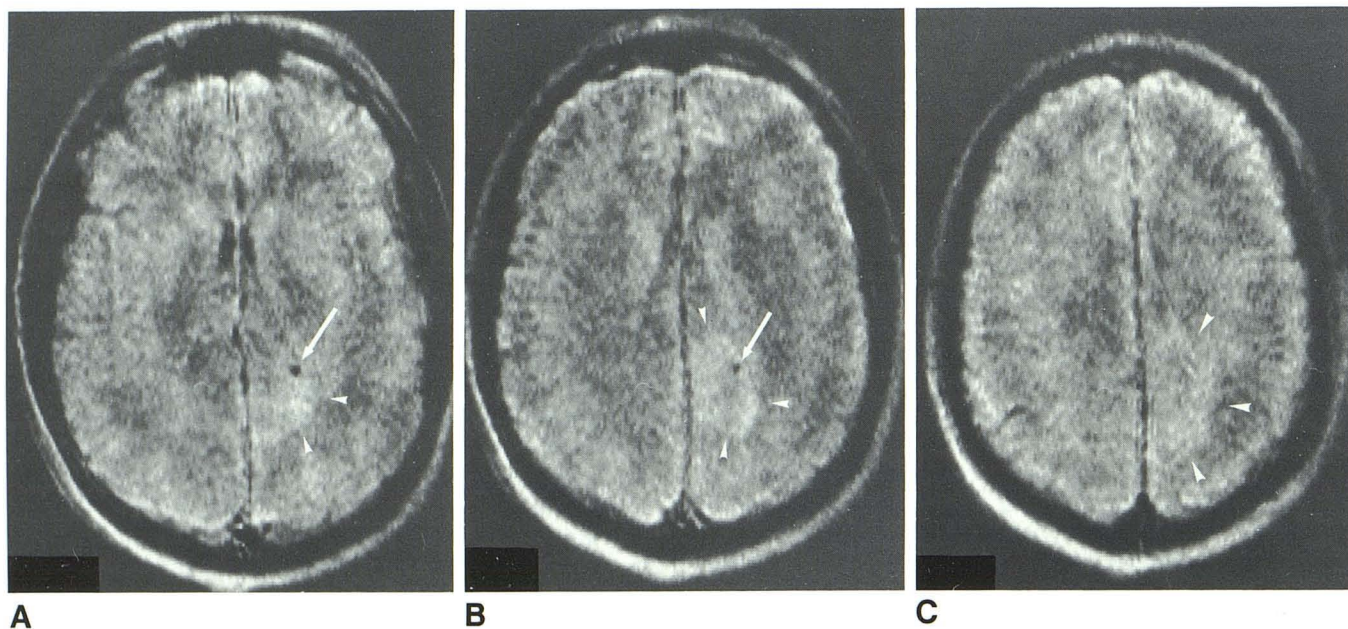


Fig. 1.—SE 1000/60. Venous angioma (arrowheads) has high signal intensity from prolonged T2. Draining vein (arrows) has absent signal due to blood flow.

Received April 23, 1984; accepted June 6, 1984.

¹ Department of Radiology, Indiana University School of Medicine, and Regenstrief Institute, 926 W. Michigan St., Indianapolis, IN 46223. Address reprint requests to R. L. Gilmor.

² Department of Neurosurgery, Indiana University School of Medicine, Indianapolis, IN 46223.

Fig. 2.—T1-weighted images. **A**, SE 250/30. Poorly defined venous angioma. **B**, IR 1250/400. Venous angioma is area of decreased signal (arrowheads).

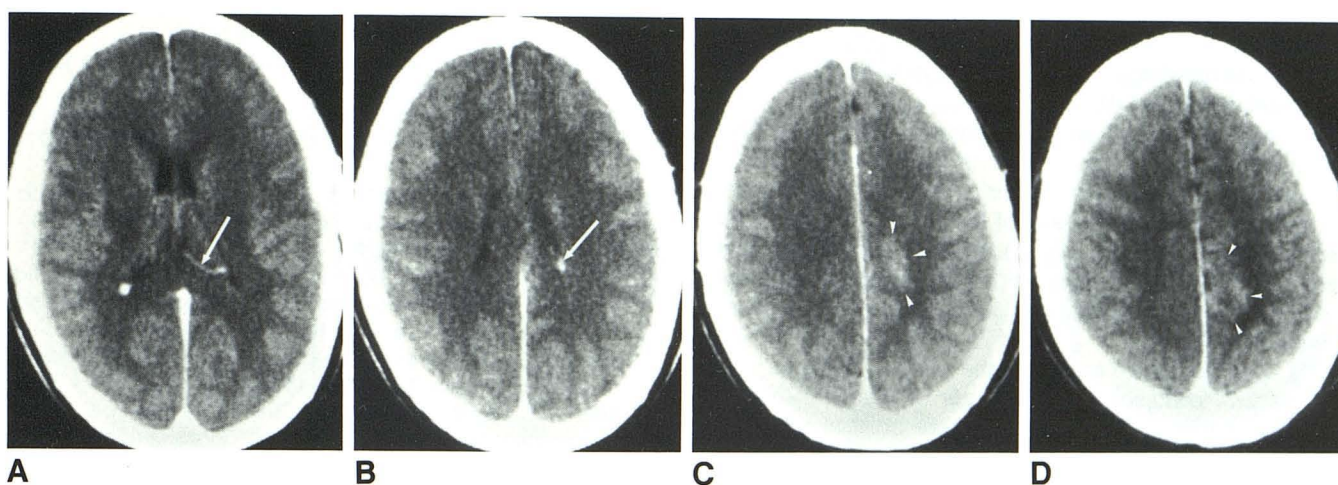
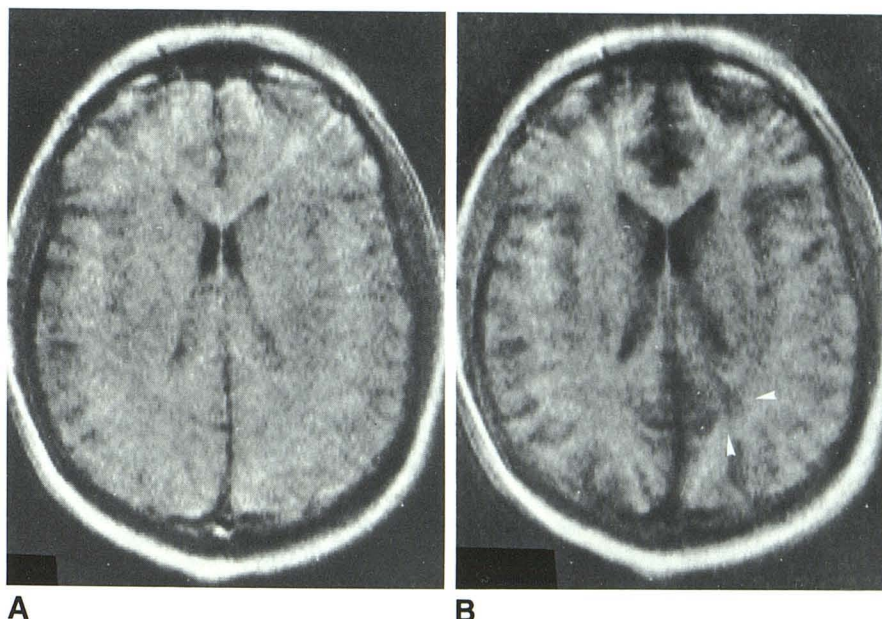


Fig. 3.—Contrast-enhanced CT scans. **A** and **B**, Enhancing dilated transcerebral draining vein (arrows). **C** and **D**, Enhancement in body of venous angioma (arrowheads).

(fig. 4). The patient was discharged on anticonvulsant medications, and did well during 7 months of follow-up.

Discussion

Venous angiomas are being recognized with increasing frequency as neuroradiologic techniques improve [1, 2]. They are composed of abnormal venous channels, probably caused by a disorder of embryogenesis [3]. While distinguishing these lesions from more serious conditions is the primary clinical benefit of accurate diagnosis, recent articles have emphasized the significant morbidity that they occasionally cause [4, 5].

Angiography is generally accepted as the diagnostic stan-

dard for venous angiomas. The classic angiographic appearance is one of dilated medullary veins converging toward an enlarged transcerebral draining vein, with no mass effect [6]. This is reflected on CT scans as an area of diffuse enhancement corresponding to the venous angioma itself, associated with an arcuate enhancement due to the transcerebral draining vein [7, 8].

To our knowledge, the MR appearance of a venous angioma has not been described previously. The MR findings we observed were similar to those found at CT and angiography. The absence of signal from the transcortical draining vein on the SE 1000/60 images (fig. 1) is undoubtedly due to blood flow within this vessel. A similar absence of signal was

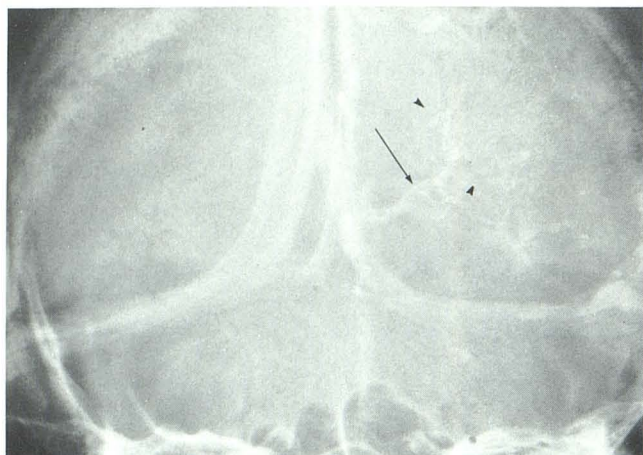


Fig. 4.—Late venous phase left internal carotid arteriogram, posteroanterior view. Characteristic pattern of dilated medullary veins (arrowheads) converging to enlarged transcerebral draining vein (arrow), which drains into galenic system. Arterial phase was normal.

not observed from the venous angioma itself, presumably because the slow blood flow within the innumerable venous channels composing this lesion does not move all excited nuclei out of the imaging plane during this pulse sequence. The effect of blood flow rates on MR signal intensity has been discussed by others [10, 11].

The large blood pool within a venous angioma has been postulated to account for aspects of its appearance on CT [9]. This may also account for our observation of prolonged T2 and increased spin density of the venous angioma with respect to adjacent brain (fig. 1). Similar observations have been made of other intracranial lesions with a large blood pool, such as acute intracerebral hemorrhages and subdural hematomas [12, 13].

The apparent prolongation of the venous angioma's T1 with respect to adjacent white matter on the IR 1250/400 and OT1 images (fig. 2B) may be more artificial than real. Interplay between the opposing effects of blood pool and blood flow may be the cause. A large blood pool within a lesion has been reported to increase signal on T1-weighted images because of a shortened T1 relaxation time and an increased mobile proton density [12–14], whereas blood flow may decrease MR signal, as discussed above. A similar

example of interplay between opposing effects has recently been described for cerebral edema, in which prolonged T1 and increased proton density have opposite effects on T1-weighted images [15].

Although we have demonstrated the ability of MR to image a venous angioma, the sensitivity and specificity of this imaging technique in making the diagnosis requires further investigation.

REFERENCES

1. Moritake K, Handa H, Mori K, Ishikawa M, Morimoto M, Takebe Y. Venous angiomas of the brain. *Surg Neurol* 1980;14:95–105
2. Olson E, Gilmor RL, Richmond B. Cerebral venous angiomas. *Radiology* 1984;151:97–104
3. Saito Y, Kobayashi N. Cerebral venous angiomas. Clinical evaluation and possible etiology. *Radiology* 1981;139:87–94
4. Numaguchi Y, Kitamura K, Fukui M, et al. Intracranial venous angiomas. *Surg Neurol* 1982;18:193–202
5. Rothfus WE, Albright AL, Casey KF, Latchaw RE, Roppolo HMN. Cerebellar venous angioma: "benign" entity? *AJNR* 1984;5:61–66
6. Wendling LR, Moore JS, Kieffer SA, Goldberg HI, Latchaw RE. Intracerebral venous angioma. *Radiology* 1976;119:141–147
7. Valavanis A, Wellauer J, Yasargil MG. The radiological diagnosis of cerebral venous angioma: cerebral angiography and computed tomography. *Neuroradiology* 1983;24:193–199
8. Lotz PR, Quisling RG. CT of venous angiomas of the brain. *AJNR* 1983;4:1124–1126
9. Michels LG, Bentson JR, Winter J. Computer tomography of cerebral venous angiomas. *J Comput Assist Tomogr* 1977;1:149–154
10. Mills CM, Brant-Zawadzki M, Crooks LE, et al. Nuclear magnetic resonance: principles of blood flow imaging. *AJNR* 1983;4:1161–1166, *AJR* 1984;142:165–170
11. Herfkens RJ, Higgins CB, Hricak H, et al. Nuclear magnetic resonance imaging of the cardiovascular system: normal and pathologic findings. *Radiology* 1983;147:749–759
12. Steiner RE. The Hammersmith clinical experience with nuclear magnetic resonance. *Clin Radiol* 1983;34:13–23
13. Mills CM, Crooks LE, Kaufman L, Brant-Zawadzki M. Cerebral abnormalities: use of calculated T1 and T2 magnetic resonance images for diagnosis. *Radiology* 1984;150:87–94
14. Buonanno FS, Pykett IL, Brady TJ, Pohost GM. Clinical applications of nuclear magnetic resonance (NMR). *DM* 1983;29:1–81
15. Brant-Zawadzki M, Bartkowski HM, Ortendahl DA, et al. NMR in experimental cerebral edema: value of T1 and T2 calculations. *AJNR* 1984;5:125–129