Appearance of venous malformations on magnetic resonance imaging

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The magnetic resonance (MR) imaging appearance of venous malformations, all angiographically verified, was evaluated in 11 patients. A venous malformation is characteristically depicted as a tubular area of decreased signal intensity in the white matter of the brain. In one patient, a histologically verified cavernous malformation was also present with a characteristic mixed signal-intensity core on the T2-weighted MR images. Care should be used when evaluating venous angiomas to exclude the presence of a lesion with associated prominent venous drainage, such as a glioma.

KEY WORDS • magnetic resonance imaging • venous angioma • venous malformation

Venous malformations, one of the four types of commonly described cerebrovascular malformations, are composed of radially arranged, anomalous veins that converge in a centrally located, dilated trunk. The malformation is surrounded by normal-appearing neural parenchyma. A few recent reports have discussed the use of magnetic resonance (MR) imaging in the diagnosis of venous angiomas. This paper will discuss additional diagnostic and clinical features in a series of 11 patients.

Clinical Material and Methods

During the course of workup for various neurological problems, 11 patients underwent four-vessel cerebral angiography that documented a cerebral venous malformation (Table 1). Cranial computerized tomography (CT) was performed in nine patients using a GE 9800 scanner.* All 11 patients underwent MR imaging on a 1.5-tesla GE unit,† using T1-weighted (TR 600 msec, TE 20 msec), mildly T2-weighted (TR 2500 msec, TE 40 msec), and T2-weighted (TR 2500 msec, TE 80 msec) spin-echo images.

Results

The clinical and neuroradiological findings for each patient are summarized in Table 1. In every case, angiography showed a normal arterial and capillary phase and a typical caput medusae appearance with prominent medullary venules in the venous phase (Fig. 1). Multiple venous malformations were found in two (18%) of 11 cases. In nine patients undergoing CT without contrast enhancement, the scans were negative in one, one case showed an area of low density, and in the remaining cases a linear (four cases) or punctate (two cases) area of increased density was visible (Fig. 1). After intravenous infusion of iodinated contrast material in eight patients, linear enhancement was found in each case (Fig. 1). In all 11 cases, MR imaging showed a linear area of decreased signal intensity that correlated with the venous malformation visualized angiographically (Figs. 1 and 2). The surrounding parenchyma had a normal signal intensity in seven cases, and an area of increased signal intensity characterized the surrounding parenchyma in two cases (Cases 2 and 6). One year later, one patient (Case 6) had a surgically confirmed glioblastoma multiforme at this location (Fig. 3). In Cases 3 and 7, a core of mixed increased and decreased signal intensity was noted on the T2-weighted images with a surrounding rim of hypointensity (hemosiderin-laden macrophages). In Case 7, a
venous and a cavernous angioma were confirmed at surgery.

Discussion

The characteristic angiographic appearance of a venous malformation has been described as “caput medusae,” because of its resemblance to the snake-covered head of the mythical creature killed by Perseus. \textsuperscript{1,4-8,10,12,14-19,21,25-31,33,36-43,45,46} The arterial and capillary phases of the angiogram are normal, with the malformation visualized best in the late venous phase. Wendling, \textit{et al.}, \textsuperscript{44} have suggested that enlarged arterial feeders may be present in some cases. In all of our cases, the malformation was seen late in the venous phase and no enlarged arteries were seen. Histologically verified venous malformations are rarely missed on angiography. \textsuperscript{2,9,13} We agree with those who question whether the pathological diagnosis of venous angioma is possible without angiographic verification. \textsuperscript{22} Venous malformations were considered uncommon prior to the advent of CT scanning, possibly because they are usually incidental findings that rarely seem to cause symptoms severe enough to warrant angiography.

The CT appearance of venous malformations has been extensively described in the literature. \textsuperscript{1,2,4-6,8,9,12-19,21,25-29,33,35,36,38,41,43,46} The most common finding, a linear or curvilinear enhancement after contrast administration, is present in more than 70% of cases. \textsuperscript{4-6,14-15,18,19,25-27,29,30,33,38,41,43,46} A nodular area of hyperdensity on the nonenhanced CT scan with faint enhancement after the administration of contrast material has been described. \textsuperscript{19,21,26,27,33} A nodular area of hyperdensity after the administration of contrast material is also frequently found. \textsuperscript{42} In our experience a precontrast study showing a faint punctate or linear hyperdensity with linear or curvilinear enhancement after contrast administration was the most common finding. While a nodular area of hyperdensity in the precontrast study was found in one patient (Case 7), we doubt that it is characteristic of a venous malformation and suspect it may represent the coexistence of another different le-
MR appearance of venous malformations

### TABLE 1
Summary of clinical and radiographic data for the 11 patients in this series*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>Symptoms &amp; Signs</th>
<th>Angiography</th>
<th>CT Scanning</th>
<th>MR Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M, 9</td>
<td>headache &amp; nausea; NE: negative</td>
<td>rt parietal VM</td>
<td>C−: punctate hyperdensity; C+: linear enhancement</td>
<td>linear ↓ SI</td>
</tr>
<tr>
<td>2</td>
<td>F, 68</td>
<td>sudden onset of diplopia &amp; gait ataxia; NE: 1 esotropia, nystagmus in Lt lateral gaze, Lt dysmetria</td>
<td>lt cerebellar VM</td>
<td>not available</td>
<td>linear ↓ SI</td>
</tr>
<tr>
<td>3</td>
<td>M, 42</td>
<td>complex partial seizures for 15 yrs, transient quadriaparesis after microvascular accident; NE: minimal incoordination in rt hand</td>
<td>lt temporal VM (double trunk)</td>
<td>not available</td>
<td>linear ↓ SI (two); core of mixed ↓ &amp; ↑ SI</td>
</tr>
<tr>
<td>4</td>
<td>F, 73</td>
<td>2 episodes of rt hand weakness &amp; aphasia lasting less than 1 hr; NE: negative</td>
<td>lt frontal VM; frontal linear contrast</td>
<td>C−: negative; C+: not available</td>
<td>linear ↓ SI</td>
</tr>
<tr>
<td>5</td>
<td>M, 54</td>
<td>periodic severe headache lasting few sec &amp; localized to lt frontal region; NE: minimal</td>
<td>lt frontal VM (multiple trunks)</td>
<td>C−: faint linear hyperdensity; C+: multiple curvilinear enhancement</td>
<td>linear ↓ SI</td>
</tr>
<tr>
<td>6</td>
<td>M, 31</td>
<td>2 episodes of loss of consciousness; NE: negative</td>
<td>rt frontal VM</td>
<td>C−: low density; C+: linear enhancement</td>
<td>linear ↓ SI surrounded by area of ↑ SI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 yr later</td>
<td>arteriovenous shunting</td>
<td>C−: low density; C+: irregular enhancement</td>
<td>multiloculated mass (hemorrhagic); ↑ SI from surrounding parenchyma</td>
</tr>
<tr>
<td>7</td>
<td>M, 11</td>
<td>lt frontal headache for 5 yrs; NE: negative</td>
<td>rt frontal VM</td>
<td>C−: nodular hyperdensity; C+: not available</td>
<td>linear ↓ SI; core of mixed ↑ &amp; ↓ SI</td>
</tr>
<tr>
<td>8</td>
<td>M, 11</td>
<td>headaches; NE: negative</td>
<td>rt cerebellar VM</td>
<td>C−: faint linear hyperdensity; C+: linear enhancement</td>
<td>linear ↓ SI</td>
</tr>
<tr>
<td>9</td>
<td>F, 43</td>
<td>headaches &amp; dizziness; NE: negative</td>
<td>lt cerebellar VM</td>
<td>C−: faint curvilinear hyperdensity; C+: curvilinear enhancement</td>
<td>linear ↓ SI</td>
</tr>
<tr>
<td>10</td>
<td>M, 12</td>
<td>VP shunt for hydrocephalus at 6 mos of age episode of loss of consciousness prior to admission; NE: rt ambylopia</td>
<td>rt frontal VM</td>
<td>C−: punctate calcification</td>
<td>linear ↓ SI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>C+: curvilinear enhancement</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>F, 46</td>
<td>headaches; NE: negative</td>
<td>rt frontal VM</td>
<td>C−: faint curvilinear hyperdensity; C+: curvilinear enhancement</td>
<td>linear ↓ SI</td>
</tr>
</tbody>
</table>

*CT = computerized tomography; MR = magnetic resonance; NE = neurological examination; VM = venous malformation; C− = without iodinated contrast medium; C+ = with iodinated contrast medium; ↓ SI = decreased signal intensity; ↑ SI = increased signal intensity.

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**FIG. 2.** Magnetic resonance (MR) images of venous malformations. **Left:** T₁-weighted (TR 600 msec, TE 20 msec) image showing abnormally prominent veins (signal hypointensity) in the deep right frontal white matter and putamen. **Center and Right:** T₂-weighted (TR 2500 msec, TE 80 msec; second echo) image revealing abnormally prominent veins (linear signal hyperintensity and hypointensity due to flow effects) in the same location as left. Subtle strands of linear hyperintensity representing medullary veins are seen in these high-resolution images.
An area of low density in the precontrast study associated with linear enhancement after contrast administration was found in a patient who later developed a glioblastoma (Case 6), suggesting that venous prominence should always be considered with a high degree of suspicion.

The use of MR imaging in the diagnosis of cerebral venous malformations has been described in recent reports. The typical appearance of a venous malformation on MR images is a linear or globular signal hypointensity on T1-weighted images and hyperintensity on T2-weighted images. One report suggests that in about 50% of cases the surrounding brain parenchyma exhibits signal hyperintensity. This is not corroborated by our experience. In one of our two cases in which there was an increased signal intensity of the brain surrounding the venous malformation, a glioma was pathologically verified 1 year after the initial MR image was taken (Case 6).

In two patients (Cases 3 and 7), MR imaging depicted the coexistence of tubular signal hypointensity (a venous malformation) with a core of mixed increased and decreased signal intensity characteristic of a cavernous malformation. One of these patients (Case 7) underwent surgery, and the coexistence of the venous and the cavernous malformation was verified histologically. This suggests that various types of vascular malformation may occur in the same patient and should be carefully sought when performing high-resolution MR imaging.

Early MR studies suggested that CT might be more sensitive for detecting venous angiomas. Our experience indicates that the sensitivity of MR imaging is equivalent to that of CT with intravenously injected contrast medium in detecting venous angiomas and does not entail the risk of contrast medium infusion. The presence of a tubular signal hypointensity in the white matter of the brain that exhibits even echophasing and is not associated with an adjacent area of increased signal intensity on T1-weighted images strongly suggests the diagnosis of venous malformation. No further imaging evaluation is necessary in this clinical setting, even though the intravenous infusion of a paramagnetic MR contrast medium (gadolinium-labeled diethylenetriamine penta-acetic acid dimeglumine) may even better characterize the caput medusae pattern of the venous angioma. It is recommended that any abnormalities found in association with venous prominences should be considered suspicious and a repeat study be performed after a few months.

References

FIG. 3. T2-weighted (TR 2000 msec, TE 100 msec) coronal magnetic resonance image of a glioblastic multiforme. A prominent superficial vein (signal hypointensity) is seen traversing a right frontal mass (signal hyperintensity), consistent with severe venous shunting of a high-grade malignancy.


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