Magnetic Resonance Imaging in Lateral Sinus Hypoplasia and Thrombosis

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Eric Meary, MD, and Marie-Germaine Bousser, MD

Lateral sinus thrombosis may be difficult to differentiate angiographically from lateral sinus hypoplasia, which mainly affects its proximal transverse portion. Using magnetic resonance imaging, we evaluated six patients who demonstrated poor filling or lack of filling of one or both lateral sinuses at angiography. In each patient, magnetic resonance imaging unambiguously demonstrated either lateral sinus thrombosis or lateral sinus hypoplasia. The latter was characterized by a frank asymmetry in size (surface of section) of the transverse portion of the lateral sinuses on parasagittal images without any abnormal signal in the course of the sinus. Lateral sinus thrombosis was indicated by increased intraluminal signal on all planes and with all pulse sequences. By virtue of its freedom from bone-related artifact, its multiplanar imaging capability, and its sensitivity to both blood flow and thrombus formation, magnetic resonance imaging is an excellent tool for the evaluation of lateral sinus thrombosis or hypoplasia. (Stroke 1990;21:1350-1356)

Dural sinus thrombosis is often difficult to diagnose because it presents with a wide spectrum of nonspecific clinical manifestations such as intracranial hypertension, focal signs, altered level of consciousness, and mental disturbances.1,2 One of the most frequent patterns, particularly when sinus thrombosis does not extend to the cerebral veins, is "benign intracranial hypertension," of which cerebral venous thrombosis is a well-established cause.

Lateral sinus thrombosis is almost as common as superior sagittal sinus thrombosis,1 but diagnosis of the former is more difficult. First, direct signs of lateral sinus thrombosis are difficult to assess on computed tomograms (CT scans).3-5 Second, it may be impossible at angiography to differentiate lateral sinus thrombosis from congenital hypoplasia, which mainly affects its proximal transverse portion. Recent reports6-13 have stressed the usefulness of magnetic resonance imaging (MRI) in the diagnosis of dural sinus thrombosis. We describe MRI findings in patients with lateral sinus hypoplasia or lateral sinus thrombosis that illustrate the value of MRI in differentiating these two conditions, a distinction that can have major therapeutic implications.

Subjects and Methods
Inclusion in this study was based solely on one criterion: the nonvisualization or poor visualization of one or both lateral sinuses at angiography. Each patient had selective opacification of both carotid arteries and a good-quality posterior fossa angiogram with opacification of the dominant vertebral artery or both vertebral arteries. In all patients MRI was performed on a Philips 0.5-T Gyroscan S15 (Eindhoven, The Netherlands) with a standard 30-cm head coil, using T1- and T2-weighted spin-echo pulse sequences with a slice width of 8 mm and an interslice spacing of 0.8 mm. Parameters for T1-weighted imaging included a repetition time (TR) of 450 msec and an echo time (TE) of 14 msec, whereas T2-weighted images had a TR of 1,800-2,000 msec and a TE of 50-120 msec. The acquisition matrix was 256x256 pixels. Sagittal T1- and T2-weighted sequences were performed in all patients. In addition, four patients had coronal T1-weighted imaging, three had coronal T2-weighted imaging, and three had axial T2-weighted imaging. At least three sequences were performed in each patient.

Lateral sinus signal and morphology were studied blindly by two radiologists. The transverse portion of the lateral sinus lies in the attached border of the tentorium, between the meningeal and perioseal layers of the dura. The transverse portion of the lateral sinus is triangular in cross-section and easily

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Figure 1. Patient 2. Top: Right carotid angiogram (frontal projection) showing nonfilling of right transverse sinus. Right sigmoid sinus (curved arrow) is opacified via cortical veins. Bottom: Sagittal T2-weighted magnetic resonance images (spin-echo sequence 2020/50 msec). Left: Right transverse sinus could not be identified (small arrow). Right: Normal signal void in large left transverse sinus (large arrow).

Identified on parasagittal MRI scans. Its posterior wall, composed of the tough fibrous tissue of the dura, may be identified (especially when a large window is used) as a thin linear signal between the hyposignal of flowing blood and the hyposignal of the inner table of the skull. The sizes (surfaces of section) of the transverse sinuses were compared on symmetric parasagittal slices (the second slice from the medial sagittal plane) and classified as grossly symmetrical or frankly asymmetrical.

Clinical presentation, findings on plain roentgenograms of the skull, and findings on plain and contrast-enhanced CT scans are summarized in Table 1. Cerebral angiography was performed as a control examination long after subarachnoid hemorrhage in patients 1 and 2, to rule out dural sinus thrombosis in patient 3 (who presented with benign intracranial hypertension), and to confirm cerebral venous thrombosis in patients 4–6 (who had histories and/or CT findings suggestive of hemorrhagic infarction).

Results

The MRI findings are summarized in Table 1. In patients 1–3 there was a frank asymmetry in sizes of the transverse portions of the lateral sinuses. One transverse sinus showed a normal signal void, while the other was not clearly identifiable and did not show any abnormal signal along its entire course. These findings pointed to transverse sinus hypoplasia on the left in patients 1 and 3 and on the right in patient 2 (Figure 1). In patients 4 and 5, an increased signal was found in vessels poorly visualized at angiography.
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<td>1/44/F</td>
<td>Control angiography 2 years after subarachnoid hemorrhage</td>
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<td>Nonfilling of L TS; L SS opacified via cortical veins; no indirect signs of CVT; normal sinus groove not identified</td>
<td>7 weeks</td>
<td>Asymmetry ++: L TS not clearly identified, normal signal void in R TS; L LS hypoplasia</td>
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<tr>
<td>2/32/M</td>
<td>Control angiography 1 yr after subarachnoid hemorrhage</td>
<td>. . . . .</td>
<td>Nonfilling of R TS; R SS opacified via cortical veins; no indirect signs of CVT; R sinus groove poorly identified</td>
<td>3 weeks</td>
<td>Asymmetry ++: R TS not identified, normal signal void in L TS; R LS hypoplasia</td>
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<td>3/20/F</td>
<td>Headache, vomiting, and diplopia for 6 weeks; bilateral papilledema; R 6th nerve palsy; obesity; oral contraceptives; tobacco abuse; cerebrospinal fluid opening pressure of 400 mm H2O</td>
<td>Small ventricular system 6 weeks</td>
<td>Very poor filling of L TS via straight sinus; L SS opacified via cortical veins; no indirect signs of CVT</td>
<td>6 days</td>
<td>Asymmetry ++: L TS not identified, normal signal void in R TS; L LS hypoplasia</td>
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<td>4/30/F</td>
<td>Sudden dysphasia following 5-day history of L temporal headache; oral contraceptives</td>
<td>L temporal hemorrhagic infarct 1 week</td>
<td>Very poor opacification of L TS; nonfilling of L SS and temporo-occipital veins; anastomotic cortical veins; L sinus groove poorly identified</td>
<td>6 days</td>
<td>Asymmetry --: isointense signal in L TS and high signal in L SS on T1-weighted images; high signal in L TS and SS on T2-weighted images; normal signal void in R TS; thrombosis of L LS</td>
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<td>5/20/M</td>
<td>Headache and vomiting for 2 weeks; alcohol abuse</td>
<td>R temporal hemorrhagic infarct, empty delta sign, abnormal enhancement of falk and tentorium 2 weeks</td>
<td>Nonfilling of SSS (posterior %), adjacent cortical veins, L TS, and SS; poor filling of R TS; delayed emptying and anastomotic cortical veins; both sinus groves clearly identified</td>
<td>5 days</td>
<td>Asymmetry --: high signal in SSS and L TS, small focus of high signal in proximal part of R TS on T1- and T2-weighted images; thrombosis of SSS, L TS, and proximal R TS</td>
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<td>6/39/M</td>
<td>Sudden L hemiparesis following severe dehydration in psychotic patient; occipital bone fracture</td>
<td>Normal 1.5 weeks</td>
<td>Nonfilling of SSS (posterior %) and R TS, SS, and jugular vein; poor filling of L TS; delayed emptying, anastomotic cortical and scalp veins; L sinus groove poorly identified</td>
<td>4 days</td>
<td>Asymmetry ++: normal signal void in small L TS; high signal in SSS and R LS and jugular vein on T1- and T2-weighted images; thrombosis of SSS and R LS and jugular vein; L LS hypoplasia</td>
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Pt, patient; F, female; M, male; CT, computed tomography; MRI, magnetic resonance imaging; R, right; L, left; TS, transverse sinus; SS, sigmoid sinus; CVT, cerebral venous thrombosis; LS, lateral sinus; SSS, superior sagittal sinus.
angiography (Figure 2). This increased signal was seen in all planes on both T1- and T2-weighted sequences. With T2-weighted sequences, little change was appreciated between the first and second echoes. These findings are diagnostic of sinus thrombosis.6-13

In patient 6, MRI findings pointed to the diagnosis of lateral sinus hypoplasia on the left and lateral sinus thrombosis on the right (Figures 3 and 4).

Discussion

Angiography has long been the key procedure in diagnosing cerebral venous thrombosis,14 and it remains the method of reference for evaluating new techniques. However, angiography carries a small but unquestioned risk, and there are a number of venous anatomic variations that could be misinterpreted as sinus occlusion. In particular, the transverse sinuses are often of unequal size, the sinus with the more direct connection to the superior sagittal sinus being the larger. The right transverse sinus is more often a direct continuation of the superior sagittal sinus, whereas the left transverse sinus frequently receives most of its blood supply from the straight sinus. In the study of Hacker,15 the proximal transverse por-
tion of the left lateral sinus was not opacified in 14% of normal left carotid angiograms, while its sigmoid portion failed to fill in only 4% of cases because it may be directly injected via cerebral veins, such as Labbe's vein. On normal right carotid angiograms, the proximal right transverse sinus was not injected in 3.3% of cases but the right sigmoid sinus was always demonstrated. Because the lateral sinus may receive most of its blood supply from posterior fossa vessels, vertebral angiography may be required when the lateral sinus is not opacified on carotid angiography. When technically perfect carotid and vertebral angiography fail to show properly a lateral sinus, either thrombosis or hypoplasia of the sinus is indicated. Lateral sinus thrombosis is easily diagnosed when angiography shows, in addition to nonvisualization or poor filling of the sinus, occlusion of cerebral veins or indirect signs of venous thrombosis such as delayed emptying and the development of a collateral circulation. However, when these latter signs are lacking or questionable, the diagnosis of lateral sinus thrombosis or hypoplasia can be only presumptive. A lack of filling of the proximal portion of the transverse sinus with normal opacification of the more distal portion of the transverse sinus and the sigmoid sinus suggests lateral sinus hypoplasia (Figure 1), while the reverse pattern favors lateral sinus thrombosis (Figure 2).

Plain roentgenograms of the skull are sometimes useful in differentiating lateral sinus thrombosis from lateral sinus hypoplasia. In particular, an arteriographically nonvisualized sinus contrasting with a well-delineated sinus groove is a strong argument for lateral sinus thrombosis. By contrast, the lack of a lateral sinus groove, classically in favor of lateral sinus hypoplasia, can be misleading. In our patient 4 the left sinus groove was not clearly identified on skull roentgenograms, whereas MRI demonstrated that the left lateral sinus was thrombosed and not hypoplastic.

In patients suspected of having cerebral venous thrombosis, CT is of crucial importance, mainly to rule out other diagnoses. Because of the close proximity of dense bony structures that may cause artifacts or partial volume averaging, direct imaging of lateral sinus thrombosis on CT is difficult and requires some technical refinements (the use of thin sections complemented by multiplanar reconstruction or direct coronal scanning, special window settings, and timing of the contrast bolus to obtain good vascular opacification). This particular CT procedure is likely to be carried out only in response to clinical suspicion. Because lateral sinus thrombosis is often unsuspected clinically, these refinements are not generally used and the correct diagnosis may be missed. Indirect CT signs of thrombosis (focal low-density areas of infarction, parenchymal hemorrhage, gyral enhancement, small ventricles, and intense tentorial enhancement) are more frequent but nonspecific. On the whole, CT can show typical changes, but in most cases it shows nonspecific abnormalities or is normal.
FIGURE 4. Patient 6. Sagittal and axial T2-weighted magnetic resonance images (spin-echo sequence 1880/50 msec) showing high signal of thrombosis extending from right transverse sinus (top left, filled curved arrow) to right jugular bulb (top right, filled straight arrow). Bottom left: Normal signal void in left hypoplastic transverse sinus (open curved arrow).
In our series, MRI was the most reliable procedure to differentiate lateral sinus hypoplasia from lateral sinus thrombosis. The diagnosis of the former was evident on sagittal MRI, which demonstrated frank asymmetry in sizes of the transverse portion of the sinuses without any abnormal signal in the expected course of the sinuses. This MRI pattern was easy to distinguish from that of transverse sinus thrombosis (viz., increased intraluminal signal on all planes of section and on both T1- and T2-weighted sequences, corresponding to an intermediate-stage thrombus\(^{1-13}\)). The diagnosis of lateral sinus thrombosis having major therapeutic implications, a careful study is essential to avoid false-negative or -positive images. With high-field units, the acute phase of venous occlusion presents on T2-weighted images with hypointense signal compared with normal brain parenchyma.\(^6\) The decreased signal may be confused with patency. In that case, it may be necessary to repeat the MRI examination after several days or at intermediate field strength to avoid this delay.\(^7\) Flowing blood, especially slowly flowing blood, can create artifactually high signals\(^{16-19}\) that can be mistaken for thrombus formation. Different MRI methods have been suggested to eliminate the possibility of flow-related artifacts. First and simplest, the suspected thrombosis should be seen on at least two different sequences, which may vary in plane of section or in TR. While most cases of thrombosis are obvious using these routine spin–echo techniques, in certain cases (especially those involving short segments of clot or partial thrombosis), interpretation of MRI scans may not be so easy and specialized acquisitions may be necessary.\(^12\)

In conclusion, our study indicates that, because of its multiplanar imaging capability, its sensitivity to both blood flow and thrombus formation, and its lack of bone-related artifact, MRI is an excellent tool for the evaluation of lateral sinus hypoplasia and lateral sinus thrombosis. This noninvasive technique is becoming the imaging modality of choice for the evaluation of suspected dural sinus thrombosis. However, it should be emphasized that this diagnosis requires a careful study to avoid false-positive findings. Also, further studies are necessary to better understand the evolution of MRI signs of thrombosis after a few weeks.

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References

17. Axel L: Blood flow effects in magnetic resonance imaging. AJR 1984;143:1157–1166

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