Early Diagnosis of Basilar Artery Occlusion Using Magnetic Resonance Imaging

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Three patients with a clinical diagnosis of pontine infarction probably due to basilar artery occlusion were studied with magnetic resonance imaging within 24 hours after onset or latest progression of symptoms. The earliest changes on magnetic resonance images were an absence of signal void in the basilar artery suggestive of severe reduction of blood flow or occlusion (flow-void phenomena). The presumed basilar artery thrombosis was best demonstrated as a linear structure isointense or hyperintense with the brainstem in the pontine cistern on T1-weighted parasagittal images and as either absence of flow-void phenomena or higher signal intensity at various levels corresponding to the course of the basilar artery on the axial T2-weighted images. Brainstem parenchymal changes characteristic of infarction were not obvious for at least 12 hours after onset or 90 minutes after latest progression of symptoms and were best shown by both axial and coronal T2-weighted images. Recognition of these magnetic resonance imaging findings may allow earlier diagnosis and treatment of acute ischemia in the vertebrobasilar system. (Stroke 1988;19:297–306)

Magnetic resonance imaging (MRI) appears to be superior to computed tomography (CT) in identifying posterior circulation infarctions. Although a great deal of research has involved the use of MRI, its ultimate role in the evaluation of patients with acute stroke is not defined. The questions of separation of the effects of ischemic necrosis from additional changes, or the MRI distinction between ischemia without infarction and edema, have not been resolved. The interval from the onset or progression of an ischemic cerebrovascular event until MRI shows abnormalities is not known. In particular, the usefulness of MRI in detecting lesions of <24 hours of age needs to be addressed. To evaluate this we studied three patients who on clinical grounds were suspected to have an acute occlusion of the basilar artery. We evaluated the ability of MRI to detect acute ischemic lesions in patients with ischemic events in the basilar artery territory within 24 hours after onset or latest progression of symptoms and the sequential changes in location or size of vascular lesions demonstrated by MRI in relation to changes in CT and clinical observation.

Subjects and Methods

Three patients were studied between January and July 1987. All had a clinical history and a physical examination that indicated a strong probability of basilar artery territory infarction of <24 hours’ duration. All three patients had sequential CT and MRI studies. The number of MRI examinations was determined by each patient’s clinical condition.

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Received August 14, 1987; accepted October 6, 1987.

All MRI examinations were performed with a 0.5-T superconductive Picker Vista MRI system (Cleveland, Ohio). The images were obtained with parasagittal T1-weighted (repetition time, TR 350–550 msec; echo time, TE 20–26 msec), axial T2-weighted (TR 2,000–2,016 msec; TE 100 msec), and coronal T2-weighted (TR 2,000 msec; TE 100 msec) pulse sequences. Slice thickness was 10 mm without gap.

The MRI criteria used to diagnose infarction were the presence of a focus of abnormally decreased signal intensity on the T1-weighted images and a corresponding area of increased signal intensity on the T2-weighted images. The MRI criteria suggestive of an occluded basilar artery were the presence of a linear structure isointense with the brainstem in the pontine cistern on T1-weighted parasagittal images and absence of flow-void phenomena with high signal intensity corresponding to the course of the artery on the T2-weighted axial images at various cuts.

CT images were obtained without infusion of contrast, using a Siemens DRH unit (Iseline, New Jersey) and 8-mm-thick axial slices.

Results

Case 1

A 50-year-old hypertensive man noted the sudden onset of bifrontal headache, intermittent confusion, vertigo, nausea, vomiting, bilateral tinnitus, and left-sided weakness 22 hours before admission.

On admission, he had a normal mental status. Coarse nystagmus in all directions of gaze was noted as well as a decreased left corneal reflex and bilateral weak eyelid closure. He had a left hemiparesis and left hypesthesia. Truncal ataxia was noted. Unenhanced CT was normal.

Twenty-four hours later he developed dysarthria, dysphagia, decreased hearing on the right, bilateral horizontal gaze palsies, and quadriplegia. MRI com-
Fig. 1. Parasagittal T1-weighted magnetic resonance images (repetition time, TR 483 msec; echo time, TE 20 msec) of Case 1 show linear structure (white arrows and black arrows) isointense with brainstem in pontine cistern suggestive of basilar artery thrombosis. Small linear area of higher intensity due to either slow flow or subacute intraluminal clot (arrowhead, upper right image) is noted.

completed within 90 minutes after the onset of neurologic deterioration demonstrated possible thrombosis (Fig. 1) and absence of flow-void phenomena in the basilar artery suggestive of complete occlusion (Figures 1 and 2). No evidence of cerebellar or cerebral parenchymal abnormalities was noted on either T1- or T2-weighted images. There was a faint midline high-signal-intensity area in the pons (Fig. 2). This was noted retrospectively, only after discovering the changes on follow-up MRI.

MRI obtained 3 days after initial symptoms, 2 days after the initial study, showed persistent lack of flow-void phenomena in the basilar artery. In addition, there was abnormal signal intensity, with the right greater than the left, with basis pontis involvement on both T1- and T2-weighted images. Collateral circulation was also demonstrated (Fig. 3).

Case 2

A 43-year-old man developed a sudden, brief, severe occipital headache the evening before admission. He went to bed only to awaken 2 hours later with agitation, confusion, dysarthria, and vomiting. He presented at the local emergency room with a very unsteady gait and was transferred to University of Iowa Hospitals. CT without infusion of contrast was normal.

Upon arrival, he was diaphoretic and had a blood pressure of 130/70 mm Hg, a regular pulse of 90/min, a rectal temperature of 37°C, and shallow respirations at 20/min. He required ventilatory assistance. He could voluntarily open and close his eyes, but he had only vertical eye movements in response to commands, to oculocephalic reflex testing, and to ice-water caloric stimulation. There was bifacial weakness, ocular bobbing, and absent corneal and gag reflexes. He exhibited flaccid quadriplegia with intermittent bilateral extensor posturing.

MRI obtained 12 hours after the initial symptoms demonstrated a linear structure on T1-weighted parasagittal images isointense with the brainstem in the pontine cistern (Fig. 4). On T2-weighted axial images, there was absence of flow-void phenomena and evidence of high signal intensity in the pontine cistern suggestive of an occluded basilar artery. No parenchymal abnormalities were noted, although in retrospect and only with the benefit of subsequent studies, a suspicious area of abnormal signal intensity involving the basis pontis was probably present on the T1- and T2-weighted images (Fig. 5).

Axial T2-weighted images obtained 16 hours after the initial symptoms showed new evidence of flow-void phenomena near the junction of the distal vertebral and proximal basilar arteries, with persistent absence of flow-void phenomena of the rest of the basilar artery. Areas of mildly increased signal intensity were noted in the middle of the basis pontis bilaterally. High signal
intensity was still identified in a segment of the basilar artery in the pontine cistern (Figure 6). Axial T2-weighted images 20 hours after the initial symptoms disclosed increased signal intensity and extent of involvement, with the right side more affected than the left (not shown). The level of occlusion and the flow-void phenomena of the proximal basilar artery were unchanged from the previous study. Axial T2-weighted MRI at 44 hours after the onset of symptoms demonstrated further increase in the extent of the flow-void sign of the basilar artery to the midpontine level and further increases in signal intensity and extent of involvement on both sides of the basis pontis (Figure 7). Axial T2-weighted images 10 days after the onset of symptoms revealed the highest degree of increased signal intensity in the previously involved areas and new areas of involvement in both cerebellar hemispheres (not shown).

Case 3
A 60-year-old man presented with a dull right-sided neck pain and severe gait difficulties. On examination he had a right Horner’s syndrome, primary gaze rotatory nystagmus, decreased sensation to light touch and pin-prick on the right side of his face and body, and a right central facial paresis. Dysmetria was noted in his right arm. He veered to the right while walking. Unenhanced CT was normal. Over the next 12 hours he developed bilateral internuclear ophthalmoplegia, ocular bobbing, bifacial weakness, and absent gag reflex.

Forty-eight hours after the initial onset of symptoms and 2 hours after progression, T1-weighted sagittal MRI demonstrated an occluded basilar artery (Figure 8), and axial T2-weighted images revealed absence of flow-void phenomena within the basilar artery, suggesting a thrombosis of the basilar artery (Figure 9). Areas of relatively mildly increased signal involving the inferolateral aspect of the right cerebellar hemisphere and a single small area of increased signal intensity involving the middle portion of the left cerebellar hemisphere and occipital lobe suggestive of infarctions were noted. No brainstem abnormalities were noted.

Discussion
Atherothrombotic disease in the vertebrobasilar system has a predilection for the distal vertebral artery and the lower or middle basilar artery. Atherosclerotic involvement of the intracranial portion of the vertebral basilar system frequently occurs in tandem with and is the common pathologic mechanism associated with the syndrome of vertebral basilar infarction. Although transcranial Doppler and dynamic CT may be useful, definitive diagnosis of intracranial vertebral or basilar artery thrombosis currently requires cerebral angiography.
FIGURE 3. Axial T2-weighted magnetic resonance images (repetition time, TR 2,000 msec; echo time, TE 100 msec) of Case 1 show high signal intensity in basis pontis (black arrows) and persistent absence of flow-void phenomena in basilar artery.
FIGURE 4. Parasagittal T1-weighted magnetic resonance images (repetition time, TR 483 msec; echo time, TE 20 msec) of Case 2 show linear structure isointense with brainstem along course of basilar artery in pontine cistern suggestive of basilar artery thrombosis (white arrows).

FIGURE 5. Axial T2-weighted magnetic resonance images (repetition time, TR 2,000 msec; echo time, TE 100 msec) of Case 2 show absence of flow-void phenomena and high signal intensity along course of basilar artery (white arrows). Faint area of increased signal intensity is present in basis pontis bilaterally (black arrows, lower left image).
FIGURE 6. Axial T2-weighted magnetic resonance images (repetition time, TR 2,000 msec; echo time, TE 100 msec) of Case 2 show areas of mildly increased signal intensity in basis pontis. Absence of flow-void phenomena and area of high signal intensity is again demonstrated along course of basilar artery (black arrows). New area of signal flow-void phenomena is visualized at vertebrobasilar junction (white arrow, lower right image).
FIGURE 7. Axial T2-weighted magnetic resonance images (repetition time, TR 2,000 msec; echo time, TE 100 msec) of Case 2 show pontine involvement much more clearly.
phy. However, knowledge of the vascular anatomy and pathology in each patient must be balanced against the associated risk of the procedure.

With its multiplanar ability, exquisite anatomic detail, and distinct tissue contrast, MRI is likely to be useful in evaluating the vertebrobasilar system. It is noninvasive, and images are obtained without the use of ionizing radiation, which permits sequential studies without the risks of repeated exposure to radiation. The contraindications to the use of MRI in patients with cerebrovascular disease are few. Because MRI is sensitive to changes in tissue water content, it may allow detection of early ischemic changes and brain edema. Furthermore, MRI is not subject to bone artifacts, which often hamper the interpretation of CT. Smaller lesions, particularly in the posterior fossa, may be shown by MRI but not by CT.

MRI becomes abnormal within a few hours after the onset of experimentally induced infarction in animals. Buonanno et al. observed prolongation of the T1 relaxation time and swelling of the affected hemisphere in symptomatic animals 2 hours after carotid artery ligation. In a previous study, one hypertensive diabetic patient with a pontine infarct had a normal MRI 12 hours after the onset of symptoms; no flow abnormality was identified in the

**TABLE 1. Sequential Magnetic Resonance Image Changes in Atherothrombotic Basilar Artery Oclusive Disease**

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>Time after onset of symptoms</th>
<th>Time after latest progression</th>
<th>Absence of flow-void</th>
<th>Brainstem parenchymal changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/50/M</td>
<td>24 hrs</td>
<td>1½ hr</td>
<td>Yes</td>
<td>Questionable</td>
</tr>
<tr>
<td></td>
<td>3 days</td>
<td>2 days</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2/43/M</td>
<td>12 hrs</td>
<td>NA</td>
<td>Yes</td>
<td>Questionable</td>
</tr>
<tr>
<td></td>
<td>16 hrs</td>
<td>NA</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>20 hrs</td>
<td>NA</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>44 hrs</td>
<td>NA</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>10 days</td>
<td>NA</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>3/60/M</td>
<td>48 hrs</td>
<td>2 hrs</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

NA, not applicable.
basilar artery. A second MRI obtained 6 days later demonstrated a well-defined area of abnormal hypointensity on T1-weighted images, with a corresponding area of abnormal hyperintensity on T2-weighted images consistent with a paramedian pontine infarction. This case suggests that in some instances there may be a delay of at least 12 hours before infarction can become obvious on MRI. Our recent experience seems to corroborate that report (Table 1). The interval from the onset of an ischemic event in the basilar artery territory until MRI depicted brainstem abnormalities in our cases was at least 12 hours, at least 2 hours after the latest progression.

A careful and systematic study of flow abnormalities corresponding to the course of the basilar artery indicates that the absence of flow-void phenomena may occur within 90 minutes after the onset of ischemic symptoms or latest progression and represents a sign that should allow earlier diagnosis of impending ischemic infarction. However, flow-void phenomena may not be noted when the arterial occlusion is incomplete. Axial MRI studies assume particular importance for the diagnosis of basilar artery occlusion in that they show either the absence of flow-void phenomena or higher signal intensity along the course of the artery, whereas parasagittal T1-weighted images are essential for the detection of possible thrombosis.

The potential exists that MRI may be able to provide a noninvasive way to diagnose early basilar artery occlusion, to detect the extent of the ischemic injury, and may become a safe and useful tool to monitor the potential beneficial effect of a therapeutic intervention in decreasing infarct size. MRI visualization of an isointense or hyperintense structure on parasagittal T1-weighted images and the absence of flow-void phenomena on axial T2-weighted images along the distribution of the basilar artery can alert the clinician to the potential for serious neurologic sequelae of a basilar artery occlusion. Follow-up MRI studies are needed to detect subtle parenchymal changes.

Acknowledgment
The authors wish to express their gratitude to Ms. Stacie Bretey for her help in manuscript preparation.

References

**Key Words** • basilar artery • cerebral artery diseases • magnetic resonance imaging
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doi: 10.1161/01.STR.19.3.297

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