Magnetic Resonance Imaging Study of Intracranial Vertebrobasilar Artery Dissections

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**Background and Purpose** The purpose of this study is to demonstrate the magnetic resonance features of intracranial vertebrobasilar artery dissections and to determine the potential and limitations of magnetic resonance imaging in their diagnosis.

**Methods** We studied five consecutive patients with angiographically verified intracranial vertebrobasilar artery dissection with magnetic resonance imaging (0.5 T) in regard to the shapes of the intramural hematoma and the chronologica change of its signal intensity. We also estimated the sensitivity of magnetic resonance imaging for diagnosing dissection.

**Results** We observed intramural hematoma in four patients on the first magnetic resonance scan and in all five patients during the course of the study. The shapes of the intramural hematomas were curvilinear, crescentic, "bamboo-cut," “band-like,” and spotty. The intensity of the intramural hematoma varied according to its age. On the T1-weighted and the proton images, the intramural hematomas appeared isointense to slightly hyperintense in the first few days and became hyperintense thereafter. The intramural hematomas became isointense or unrecognizable 2 months after onset. The T2-weighted image and the proton images were superior to the T1-weighted image in demonstrating the intramural hematomas.

**Conclusions** Magnetic resonance imaging is a sensitive tool for diagnosing intracranial vertebrobasilar artery dissection, particularly in the subacute to early chronic stage. Magnetic resonance imaging is complementary to angiography in that it can directly visualize intramural hematomas.

**Key Words** basilar artery • cerebral circulation • dissection • magnetic resonance imaging • vertebral arteries

**Subjects and Methods**

**Patients**
From 1991 to 1992, five consecutive patients with intracranial vertebrobasilar artery dissection confirmed by angiography were treated at Teraoka Memorial Hospital (three men, two women; age range, 33 to 64 years [mean age, 52.2 years]) (Table 1). None presented with subarachnoid hemorrhage or underwent surgery. All showed almost full recovery from symptoms.

**Serial Magnetic Resonance Imaging Follow-up**
All patients had undergone MR examination at least once in the acute stage (day 0 to day 3) before definite diagnosis was made by angiography. Three patients underwent follow-up MR examination in the acute stage, and all patients underwent follow-up MR examinations at least once in each of the subacute to early chronic (day 4 to day 60) and chronic (day >60) stages. The follow-up periods for MR examinations ranged from 84 to 256 days (mean, 154 days). MR imaging was performed with a 0.5-T superconducting Resona unit (Yokogawa Medical Systems) using a head coil. Multislice mode and spin-echo sequences were used. T1-weighted images were obtained with the following parameters: repetition time (TR), 350 to 700 milliseconds; echo time (TE), 15 or 20 milliseconds. Proton density images were obtained with TR of 2000 milliseconds and TE of 25 milliseconds. T2-weighted images were obtained with TR of 2000 milliseconds and TE of 100 milliseconds. The primary imaging projection was the axial section, and coronal or sagittal sections were added when necessary. The slice thickness ranged from 5 to 11 mm. Other MR imaging factors included a field of view of 25 cm and a 192×256 matrix.

**Interpretation of Magnetic Resonance Findings**
All MR images of the five patients were studied retrospectively by one of the authors (C.K.). The MR findings of each projection of each pulse sequence were evaluated in each case for the detectability of the dissection and the shape and signal intensity of the intramural hematoma when present. We classified the degrees of detectability into four categories: definite dissection, possible dissection, nondetectable,
TABLE 1. Clinical Summary of Five Patients With Vertebrobasilar Dissections

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y/Sex</th>
<th>Site</th>
<th>Presenting Symptoms and Signs</th>
<th>Angiography</th>
<th>Clinical Course and Outcome, Follow-up Angiography Period, y</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64/F</td>
<td>Bilateral VA-BA</td>
<td>HA, L hemiataxia</td>
<td>String sign of bilateral VA, double lumen of proximal BA</td>
<td>Complicated by severe lumbago but without recurrent attacks, angiographic resolution 1.5</td>
</tr>
<tr>
<td>2</td>
<td>55/M</td>
<td>L VA</td>
<td>HA, Wallenberg’s syndrome</td>
<td>Pearl and string sign of L VA</td>
<td>Uneventful recovery, spontaneous occlusion of L VA 1.5</td>
</tr>
<tr>
<td>3</td>
<td>54/M</td>
<td>L VA-BA</td>
<td>HA, L hemiparesis, L lingual paresis</td>
<td>Pearl and string sign of L VA, retention of contrast media at proximal BA</td>
<td>Uneventful recovery, angiographic resolution with mild stenosis of L VA 1</td>
</tr>
<tr>
<td>4</td>
<td>55/F</td>
<td>R VA</td>
<td>HA, vertigo, nausea, L hemidysesthesia</td>
<td>Sausagelike swelling of R VA</td>
<td>Definite Wallenberg’s syndrome 2 days later but recovered well, persistent R VA fusiform aneurysm 1</td>
</tr>
<tr>
<td>5</td>
<td>33/M</td>
<td>L VA</td>
<td>HA, diplopia</td>
<td>Pearl and string sign of L VA</td>
<td>Uneventful recovery, angiographic resolution 1</td>
</tr>
</tbody>
</table>

VA Indicates vertebral artery; BA, basilar artery; HA, headache; L, left; and R, right.

and inappropriate slice position. We judged an MR finding to be a definite dissection when the abnormal finding was located along the vertebrobasilar artery as expected from the angiographic findings and when the intensity of the abnormality and its size were sufficient to avoid confusion with normal surrounding structures or artifacts. We judged an MR finding to be a possible dissection when the abnormal finding was located along the vertebrobasilar artery as expected from the angiographic findings but when the intensity of the abnormality or its size was not sufficient to distinguish it clearly from normal surrounding structures or artifacts. The dissection on MR imaging was judged to be nondetectable when no abnormalities of the vertebrobasilar artery were observed, although the sectional planes included the dissected portion of the artery. An MR finding was classified as inappropriate slice position when the sectional planes failed to include the dissected portion with reference to the angiographic findings.

The signal intensity of the intramural hematoma in definite dissection and possible dissection was classified into five categories and was described by number: hyperintense, +2; slightly hyperintense, +1; isointense, 0; slightly hypointense, −1; and hypointense, −2.

We examined these data from three aspects: (1) characteristic MR appearances of dissection, (2) chronological change of signal intensity of the intramural hematoma, and (3) detectability of dissection by MR imaging in each stage.

Results

The Shape of Intramural Hematoma on Magnetic Resonance Imaging

The shapes of the dissection (ie, intramural hematoma) varied, depending on the relation between the axis of the affected vessel and the imaging plane. They were curvilinear, crescentic (circumferential), “bamboo-cut,” “bandlike,” and spotty (Fig 1, Table 2). “Bamboo-cut” depicts the appearance of bamboo that has been cut obliquely to the longitudinal axis. This image was observed when the axis of the affected vessel was oblique to the imaging plane.

Chronological Change of Signal Intensity of Intramural Hematoma

The changes of signal intensity on T1-weighted and proton density images were parallel. On both pulse sequences, the signal intensity of intramural hematoma was mostly isointense or slightly hyperintense in the acute stage. Signal intensity gradually increased and became brighter (hyperintense) during the subacute to early chronic stage. Thereafter, signal intensity decreased and returned to isointensity or slight hyperintense.
TABLE 2. Summary of Serial Magnetic Resonance Findings in Five Patients With Vertebrobasilar Dissections

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>MR Appearance of Intramural Hematoma</th>
<th>First MRI</th>
<th>Follow-up MRI</th>
<th>Detectability</th>
<th>MR Follow-up Period, mo</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>First MRI</td>
<td>AS</td>
<td>SS</td>
<td>CS</td>
</tr>
<tr>
<td>1</td>
<td>Circumferential, spotty (Ax); curvilinear (Co); curvilinear (Sa)</td>
<td>+1</td>
<td>...</td>
<td>+2</td>
<td>ND</td>
</tr>
<tr>
<td>2</td>
<td>Curvilinear, bamboo-cut (Ax); curvilinear, bandlike (Co)</td>
<td>+1</td>
<td>+2</td>
<td>+2</td>
<td>+1</td>
</tr>
<tr>
<td>3</td>
<td>Curvilinear, crescent, bandlike, bamboo-cut (Ax); curvilinear, bamboo-cut (Co); curvilinear, bamboo-cut (Sa)</td>
<td>+1</td>
<td>+1</td>
<td>+2</td>
<td>+1</td>
</tr>
<tr>
<td>4</td>
<td>Curvilinear (Ax)</td>
<td>+2</td>
<td>+2</td>
<td>+2</td>
<td>ND</td>
</tr>
<tr>
<td>5</td>
<td>Crescent (Ax)</td>
<td>ISP</td>
<td>...</td>
<td>+1</td>
<td>ND</td>
</tr>
</tbody>
</table>

MR indicates magnetic resonance; MRI, magnetic resonance image; AS, acute stage (day 0-3); SS, subacute to early chronic stage (day 4-60); CS, chronic stage (day >60); Ax, axial projection; Co, coronal projection; Sa, sagittal projection; +1, slightly hyperintense; ... examination not done; +2, hyperintense; ND, nondetectable; P, possible dissection; D, definite dissection; and ISP, inappropriate slice position.

*The highest of the signal intensities observed on the T1-weighted and proton density images on the first MR examination and in each stage is given.
†The best of the detectabilities observed on the T1-weighted and proton density images on the first MR examination and in each stage is given.

tensity (Figs 2 and 3). The larger hematomas (patients 2 and 3) remained hyperintense for a longer period, while the smaller hematomas (patients 1, 4, and 5) disappeared earlier. No definite conclusion could be drawn from the signal intensity change on T2-weighted image because intramural hematoma was difficult to differentiate from the surroundings on this pulse sequence. To be concise, the highest of the signal intensities observed on the T1-weighted and proton images was determined for the first MR examination and for each stage; the results are given in Table 2.

Detectability of Dissections by Magnetic Resonance Imaging

On the first MR examination preceding definite diagnosis by angiography, dissection of the vertebrobasilar artery was suspected (possible or definite dissection) in four of the five patients. In the single case (patient 5) in which MR imaging failed to demonstrate the dissection, the sectional planes of the axial scan had been inappropriately selected. If the sectional planes had been correctly selected, the MR imaging might have revealed the pathology. Furthermore, in all five patients dissection was demonstrated on the MR image in the subacute to early chronic stage and was definitely hyperintense in four patients. However, after 2 months the intramural hematoma could not be seen on MR images in three patients. The intramural hematomas were visualized mostly on T1-weighted and proton density images and only in one patient on the T2-weighted image. Thus, in this study detection of dissection virtually depended on T1-weighted and proton density images. To be concise here as well, instead of showing all the detectabilities given to each projection of each pulse sequence, we summarized these data and determined, for the first MR examination and for each stage, the best of the detectabilities observed on the T1-weighted and proton density images. The results are given in Table 2.

Discussion

The Appearance of Dissection on Magnetic Resonance Imaging

Diagnosis of arterial dissection in general depends mainly on demonstration of intramural hematoma, as it did in this study as well as in previous reports (including those of internal carotid dissection and extracranial
vertebral artery dissection). The shape of intramural hematomas visualized on the MR images varies according to the relation between the axis of the affected vessel and the imaging plane. In previous reports the shapes of intramural hematomas were described as crescentic, crescent-shaped or oval, semilunar, or circumferential. The various shapes of intramural hematomas in this study are described in “Results” (Table 2).

The signal intensity of an intramural hematoma also varies. The age of the intramural hematoma and the pulse sequence selected for imaging are the major determinants of signal intensity. Aside from the chronological alteration of its signal intensity, an intramural hematoma usually appears hypointense both on T1- and T2-weighted images when it is best visualized on MR imaging. This was true in this study as well as in previous reports. In some reports all or part of the intramural hematoma appeared hypointense on T1-weighted images. These hypointensities most likely represent acute clot deoxyhemoglobin or hemosiderin in the chronic stage, both of which cause signal loss at high field strength (1.5 T).

Chronological Intensity Changes of Intramural Hematoma

The chronological change of signal intensity of the intramural hematoma observed in this study seemed to follow that of an intracerebral hematoma. On the T1-weighted image, the intramural hematoma appeared iso- to slightly hyperintense on the first day. Three days after onset, most of the intramural hematomas became slightly to definitely hyperintense. Thereafter, the hematomas further increased in their intensity and remained hyperintense for approximately 2 months. However, the intramural hematomas seemed to begin diminishing in their intensity, and most of the hematomas became isointense or unrecognizable (possibly absorbed) within 6 months. The signal intensity change on the proton density image paralleled that of the T1-weighted image. We could obtain little information on the appearance of an intramural hematoma on the T2-weighted image for reasons described below.

Only in patient 3 of the present series was the intramural hematoma demonstrated on the T2-weighted image, which appeared hyperintense from the first day. The intramural hematoma remained hyperintense until day 44, but it could not be detected on day 152. A hyperacute hematoma is known to appear hypointense at a high field strength (>1.0 T) because preferential T2 proton relaxation enhancement (PT2PRE) is exaggerated under such a condition, but the intramural hematoma in patient 3 seemed to be hyperintense on the T1-weighted image even in the hyperacute stage (the first T2-weighted image of patient 3 was obtained within 4 hours of onset). This is probably because the PT2PRE effect was less prominent in this MR study, which was performed using a 0.5-T unit.

The time required for resolution of abnormal intensity of the intramural hematoma was almost the same as in previous reports. In the series of Gelbert et al, abnormal signal intensities observed in three extracranial vertebral artery dissections and in two carotid artery dissections could not be recognized at 4 to 6 months. In case 2 of the series of Rothrock et al, the intramural hematoma in a carotid artery dissection had disappeared within 4 months. In case 1 of the series of Goldberg et al, resolution of the intramural hematoma was observed at 7 weeks. However, in the report by Chen et al, the intramural hematoma appeared bright on the T1-weighted image even 3 months after the onset.

The Detectability of Dissection by Magnetic Resonance Imaging

In this study the detectability of arterial dissection on the first MR examination performed before angiography was 80%. In other words, in four of the five patients examined, the MR images provided sufficient findings to arouse suspicion of the presence of dissection. During the entire course, we confirmed arterial dissection at least once by MR imaging in all five patients. Iwama et al performed MR examinations in three patients with angiographically confirmed intracranial vertebrobasilar artery dissection at 5 years, 7 days, and 3 months after
onset. They recognized some abnormality on the MR images of each dissecting aneurysm. Their results together with ours indicate that MR imaging may be regarded as a sensitive tool in detecting intracranial vertebral artery dissection. However, it should be emphasized at the same time that intramural hematoma in the acute stage is less prominent than in the subacute to early chronic stage and hence may require careful observation for diagnosis. When evidence of arterial dissection cannot be found on the first MR examination in the acute stage, repeat examination should be performed in the subacute stage. Potential of MR detectability of intracranial vertebrobasilar artery dissection in the chronic stage should be mentioned with reservations, because intramural hematoma may have been absorbed by that time. We can only say that visualization of intramural hematoma with MR in the chronic stage would be difficult because of decreased signal intensity.

We consider that detectability of dissection depends on the size, shape, intensity of the intramural hematoma, resolution of the MR imaging, pulse sequence, imaging plane used, thickness of the section plane, and appearance of the surrounding structures on the MR image. On T1-weighted and proton density images, the cerebrospinal fluid surrounding the vertebrobasilar artery appears hypointense, as does the flow void of the artery. Isointensity or hyperintensity of an intramural hematoma contrasts sharply with its surroundings and is easy to recognize on these pulse sequences. However, the affected basilar artery overlaps the isointensity of the pons on coronal scans, and the affected vertebral artery sometimes overlaps the isointensity of the medulla oblongata on sagittal scans. These factors make an intramural hematoma less conspicuous unless the hematoma is hyperintense. Conversely, an intramural hematoma of the basilar artery can be easily recognized on sagittal scans. On axial scans, the cranial nerves surrounding the vertebrobasilar artery may cause confusion in interpreting curvilinear isointensity of the intramural hematoma. On the T2-weighted image, cerebrospinal fluid appears hyperintense. Under this condition, a hyperintense intramural hematoma merges with hyperintense cerebrospinal fluid on the T2-weighted image, whereas a hypointense intramural hematoma would merge with the flow void of the affected artery on this sequence. This seemed to be the major factor that made the T2-weighted image have less diagnostic value.

In interpreting MR findings, it should be taken into consideration that the wall of a normal vertebrobasilar artery may occasionally appear as an isointense signal that is circumferential on axial scans or curvilinear along the artery on coronal and sagittal scans, mimicking an intramural hematoma. In such cases, differentiation from intramural hematoma is not difficult because the wall of a normal artery is usually regular in its thickness and is never hyperintense. However, atherosomatous thickening of the wall may appear similar to an isointense dissection as well as slow flow in the vertebral and basilar arteries. Also, we should be aware that a mural thrombus within aneurysms or occluded vessels may resemble an intramural hematoma on MR images. In this sense, MR imaging is not a specific examination. MR angiography and Doppler sonography, which are now increasingly used, might be helpful in such situations, although their sensitivity and specificity in detecting intracranial vertebrobasilar dissection also need to be determined in the future.

The Role of Magnetic Resonance Imaging

This study as well as those in previous reports has shown that magnetic resonance imaging, even at intermediate field strength, is a sensitive method for diagnosing intracranial vertebrobasilar artery dissection, particularly in the subacute to early chronic stage. MR imaging can directly demonstrate an intramural hematoma whose signal intensity varies with the age of the hematoma. MR imaging and angiography are complementary to each other, and we consider both to be necessary for accurate diagnosis. Because MR imaging is less invasive than angiography, MR examinations should be performed before angiography for screening whenever possible.

References

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