Magnetic resonance imaging–documented extravasation as an indicator of acute hypertensive intracerebral hemorrhage

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Object. The aim of this study was to determine the usefulness of magnetic resonance (MR) imaging–documented extravasation as an indicator of continued hemorrhage in patients with acute hypertensive intracerebral hemorrhage (ICH).

Methods. The authors studied 108 patients with acute hyperintensive ICH. Imaging modalities included noncontrast-enhanced computerized tomography (CT) scanning, gadolinium-enhanced MR imaging, and conventional cerebral angiography obtained within 6 hours after the onset of hemorrhage. A repeated CT scan was obtained within 48 hours to evaluate enlargement of the hematoma.

Findings on MR imaging indicating extravasation, including any high-intensity signals on T₁-weighted postcontrast images, were observed in 39 patients, and 17 of these also showed evidence of extravasation on cerebral angiography. The presence of extravasation on MR imaging was closely correlated with evidence of hematoma enlargement on follow-up CT scans (p < 0.001).

Conclusions. Evidence of extravasation documented on MR imaging indicates persistent hemorrhage and correlates with enlargement of the hematoma.

KEY WORDS • intracerebral hemorrhage • magnetic resonance imaging • extravasation

HYPERTENSIVE intracerebral hemorrhage (ICH) is believed to be primarily a monophasic event, but bleeding can persist for up to 6 hours postictus. Several authors have also reported that some patients with ICH continued to bleed even after hospitalization. Continued bleeding in hypertensive ICH may lead to enlargement of the hematoma and may result in progressive neurological deterioration.

We have previously described six patients with spontaneous hypertensive ICH in whom evidence of extravasation on magnetic resonance (MR) imaging indicated active bleeding (H Yokota, et al., unpublished data). We sought to determine whether evidence of extravasation on MR imaging, that is, leakage of contrast medium from the hemorrhage site in the hyperacute stage and the presence of high-intensity areas on T₁-weighted images, would be useful in identifying active bleeding in hypertensive ICH. As a corollary, we evaluated the absence of extravasation as an indication of hemostasis and used this as a basis for decisions concerning surgery and intensive care. Finally, we correlated the presence of extravasation on MR imaging with that on conventional cerebral angiography (Fig. 1).

Clinical Material and Methods

Patient Population

Two hundred eighty-eight consecutive patients treated for hypertensive ICH between 1990 and 1996 were assessed retrospectively. Patients who met the following criteria for hypertension were included in the study: the presence of hypertension based on history, prescribed medications, and/or evidence of ventricular hypertrophy on electrocardiography. Patients in whom cerebral angiography, computerized tomography (CT) scanning, or MR imaging showed an underlying intracranial lesion such as an aneurysm, vascular malformation, moyamoya disease, or brain tumor were excluded. We also excluded patients with a history of malignancy and those receiving anticoagulant medications. Patients who met the following criteria were included in this study: 1) presence of ICH on neuroimaging studies; 2) initial CT scan within 6 hours postonset and repeated CT scan within 48 hours of onset; 3) MR imaging obtained with and without gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA) enhancement within 6 hours of onset; and 4) cerebral angiography performed within 6 hours of onset. Of the 288 patients with hypertensive ICH, 180 were excluded: 92 had been admitted more than 6 hours postictus, 34 did not undergo a repeated CT scan because of emergency surgery or hemodynamic instability, and 54 did not undergo MR imaging and/or angiography because of emergency surgery, hemodynamic instability, or our inability to obtain consent. Of the remaining 108 patients, 66 (61%) were men; the mean age was 59.3 years.

On admission, a neurological examination was performed and vital signs were recorded. The presence of ICH was confirmed on CT scans, and a calcium channel antagonist was administered to reduce the systolic blood pressure (BP) to 80% of the initial value or to keep...
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the systolic BP below 150 mm Hg. The BP was continuously monitored for 24 hours after the onset of ICH. Venous blood was collected, and the white and red blood cell counts, platelet counts, glutamic-oxaloacetic transaminase, glutamic-pyruvic transaminase, and γ-glutamyltransferase concentrations, albumin/globulin ratio, activated partial thromboplastin and prothrombin times, and fibrinogen level were determined. Patients or their family members were interviewed concerning the patient’s smoking history (cigarettes/day), alcohol consumption (g/day), and medical history. The participants were divided into groups with and without MR imaging evidence of contrast medium extravasation and were compared with respect to blood test results, alcohol consumption, smoking, and the systolic and diastolic BPs at admission and at 6 hours after onset. The study was approved by the ethics committee at our hospital and informed consent was obtained from all patients or their families.

Neuroradiological Studies

Computerized Tomography. The hematoma volume (in milliliters) on CT scans was estimated based on an ellipsoid model as follows: \( \frac{\pi}{6} \times \text{long diameter} \times \text{short diameter} \times \text{number of slices} \) (slice thickness was 1 cm). When intraventricular hemorrhage (IVH) was present, accurate measurement of the parenchymal hemorrhage was considered to be impossible because of the spread of blood in the cerebrospinal fluid, and the hematoma volume was not calculated. When the hematoma was irregular in shape, the volume of each portion was estimated separately. The initial CT scan was obtained at 0.5 to 3 hours (mean 1.6 hours) after onset. Repeated CT scanning was performed at the following times: 1) before surgery when surgery was clinically indicated; 2) several hours after the initial CT scan when surgery was not clinically indicated and extravasation of contrast material was detected on MR imaging; and 3) within 48 hours postictus when surgery was not clinically indicated and extravasation of contrast material was not detected on MR imaging. When expansion of the size or shape of the hematoma was detected on CT scanning, an increase in volume of at least 20 ml was defined as significant.

Magnetic Resonance Imaging. When ICH was detected on the initial CT scan, T₁-weighted MR images with and without Gd-DTPA contrast enhancement were obtained. When extravasation of contrast medium was seen on the initial contrast-enhanced image, MR studies were repeated at approximately 12 hours postonset (range 5–24 hours, mean 10.9 hours).

The MR imaging apparatus was a 0.2-tesla superconducting unit (MRP-20; Hitachi Corp., Tokyo, Japan) with saturation recovery pulses (TR 600 msec, TE 25 msec). The contrast medium (Magnevist; Schering AG, Osaka, Japan) was injected intravenously at a rate of 10 ml per 30 seconds at doses of 0.3 ml/kg.

Cerebral Angiography. Conventional cerebral angiography was performed following CT and MR studies to confirm the presence of extravasation and also to exclude causes of hemorrhage other than hypertension (for example, vascular anomalies, cerebral aneurysms, moyamoya disease, and brain tumors). Findings on conventional angiography were correlated with the various patterns of contrast enhancement on MR imaging. Using the standard Seldinger method, the right common femoral artery was punctured, and a one- or two-vessel cerebral angiogram was obtained by means of the cut-film technique.

Selection of Treatment

Surgery for putaminal hemorrhage was indicated when the hematoma volume exceeded 30 ml and/or the patient suffered impairment of consciousness and was able to tolerate an operation. We have performed surgery in some patients harboring ICHs less than 30 ml in volume and centered in the putamen, thalamus, cerebellum, or subcortical region. We changed our criteria in 1990, after publication of the fifth Japanese cooperative study reporting on its conclusions regarding the surgical indications for patients with putaminal hemorrhage. Prior to that report, we often performed surgery in patients harboring putaminal hemorrhages with volumes less than 30 ml; we treated patients with pontine or caudate nucleus hemorrhages conservatively. Patients with thalamic, cerebellar, or subcortical hemorrhages underwent surgery if we observed impairment of consciousness and provided that the patient was able to tolerate an operation.

After hemostasis was confirmed on MR imaging, CT-monitored stereotactically guided aspiration of the hematoma was performed. In patients with concomitant massive IVH, ventricular drainage was also performed. Patients harboring hematomas too large or too irregular for stereotactically guided aspiration underwent cranioto-
my. Emergency craniotomy was performed when the MR findings indicated persistent hemorrhage and CT-monitored stereotactically guided aspiration was not possible.

Statistical Analysis

Data are expressed as the mean ± standard deviation (SD). Comparisons between groups were analyzed using the chi-square test.

The frequency of hematoma expansion or extravasation as seen on cerebral angiography was also compared for each pattern of contrast enhancement on MR imaging. Furthermore, the site of the hemorrhage, the actual hematoma size, and the MR findings were compared for the putamen and the thalamus. Student’s t-test was used for analysis of differences between groups.

Results

There was no significant difference in the time to initial CT scan between patients with evidence of extravasation on MR imaging and those without (Table 1). The time interval between the pre- and postcontrast CT studies was 7.2 to 16.7 minutes (mean 9.4 minutes). Because of the need for emergency surgery in 16 of the 58 surgically treated patients, the repeated CT scan was obtained within 8 hours; no evidence of extravasation could be demonstrated on cerebral angiography or MR imaging in 11 patients. Enlargement of the hematoma was not detected on the presurgery repeated CT scans in these patients. Repeated CT scans showed enlargement of the hematoma in three of the five remaining patients, and MR imaging in the other two showed evidence of contrast medium extravasation but no enlargement of the hematoma was seen on the repeated CT scan (obtained approximately 6.5 hours postictus). These patients were considered to have no evidence of enlargement of the hematoma.

There was evidence of contrast material extravasation on MR imaging in 39 patients, and cerebral angiography showed extravasation in 17 (Table 1). There was a significant correlation between extravasation on cerebral angiography and evidence of contrast medium extravasation on MR imaging (p < 0.001). Enlargement of the hematoma was detected on follow-up CT scans in 19 patients (Table 1) and showed a statistically significant correlation with MR imaging evidence of extravasation (p < 0.001). However, extravasation was found on cerebral angiography in two patients who did not show evidence of this condition on MR imaging. Three patients with no evidence on MR imaging of contrast medium extravasation were also found to have enlargement of the hematoma. Table 2 shows the correlation between the MR findings and hematoma enlargement and with cerebral angiography findings. The mode of contrast enhancement was classified (Fig. 2) into one of four patterns: punctate (Type A), linear (Type B), both punctate and linear (Type C), or absent (Type D). Enlargement of the hematoma was significantly more common in patients with Type A (58.3%) and Type C (30%) MR findings. Extravasation was detected on cerebral angiography in many of the patients with Type A (54.2%) and Type C (40%) MR imaging findings. Extravasation was not seen in patients with Type B or Type D.

TABLE 1

<table>
<thead>
<tr>
<th>Extravasation on MR Imaging</th>
<th>Time of Initial MR Image (hrs)†</th>
<th>Enlargement of Hematoma</th>
<th>Time of Initial CT (hrs)</th>
<th>Extravasation on Angiography</th>
<th>Time of Initial CT (hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>present in 39</td>
<td>3.08 ± 1.28</td>
<td>present in 19</td>
<td>1.39 ± 0.92</td>
<td>present in 17</td>
<td>1.37 ± 0.81</td>
</tr>
<tr>
<td>absent in 69</td>
<td>3.50 ± 1.40</td>
<td>present in 20</td>
<td>1.61 ± 0.98</td>
<td>absent in 22</td>
<td>1.61 ± 1.04</td>
</tr>
</tbody>
</table>

† Data are presented as the mean ± SD.

† Not significant.

TABLE 2

<table>
<thead>
<tr>
<th>Bleeding Pattern</th>
<th>No. of Patients</th>
<th>Extravasation on Angiography (%)</th>
<th>Enlargement of Hematoma (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>24</td>
<td>13 (54.2)</td>
<td>14 (58.3)</td>
</tr>
<tr>
<td>B</td>
<td>5</td>
<td>0 (0)</td>
<td>1 (20)</td>
</tr>
<tr>
<td>C</td>
<td>10</td>
<td>4 (40)</td>
<td>4 (40)</td>
</tr>
<tr>
<td>D</td>
<td>69</td>
<td>2 (2.9)</td>
<td>3 (4.3)</td>
</tr>
</tbody>
</table>
Extravasation on MR imaging in hypertensive intracerebral hemorrhage

Fig. 2. Classification of contrast enhancement patterns on MR imaging illustrated with schematic drawings and angiographic findings. A: Type A: punctate area of high-intensity signal within the hematoma. Left: Precontrast T₁-weighted MR image showing a mass in the left thalamus that is isointense to gray matter. Center Left: Postcontrast T₁-weighted MR image showing three areas of intense enhancement within the hematoma. Center Right: Schematic drawing showing the relationship between the contrast enhancement and hematoma. Right: Right vertebral angiogram showing active extravasation of contrast medium (arrow). B: Type B: area of linear high-intensity signal surrounding the hematoma. Left: Precontrast T₁-weighted MR image showing a mass in the left putamen isointense to gray matter. Center Left: Postcontrast T₁-weighted MR image showing a linear area of intense enhancement surrounding the hematoma. Center Right: Schematic drawing showing the relationship between the contrast enhancement and hematoma. Right: Left external carotid angiogram showing no extravasation of contrast medium. C: Type C: punctate and linear area of high-intensity signal within the hematoma. Left: Precontrast T₁-weighted MR image showing a mass in the right putamen isointense to gray matter. Center Left: Postcontrast T₁-weighted MR image showing linear intense enhancement surrounding the hematoma and an area of intense enhancement within the hematoma. Center Right: Schematic drawing showing the relationship between the contrast enhancement and hematoma. Right: Right internal carotid angiogram showing active extravasation of contrast medium (arrow). D: Type D: no enhancement within the hematoma. Left: Precontrast T₁-weighted MR image showing a mass in the right putamen isointense to gray matter. Center Left: Postcontrast T₁-weighted MR image showing no area of focal enhancement within the hematoma. Center Right: Schematic drawing showing no contrast enhancement in the hematoma. Right: Right internal carotid angiogram showing no extravasation of contrast medium.
time from onset of ictus to surgical intervention ranged from 4.5 to 31 hours (mean 10.1 hours). In 50 of these 58 patients, CT-monitored stereotactically guided aspiration was performed, and five of them also underwent ventricular drainage. Craniotomy was performed in five patients. Two patients (3.4%) showed postoperative enlargement of the hematoma. We have performed surgery in 16 patients with the Type A pattern, three patients with Type B, and five patients with Type C. We have encountered only two patients with active bleeding at surgery (Type A pattern); both of these patients had suffered putaminal hemorrhages.

### Discussion

Our results indicate that evidence of contrast material extravasation on MR imaging was well correlated with hematoma enlargement and with evidence of extravasation on cerebral angiography. Therefore, such MR findings seem to indicate persistent hemorrhage and increase in the size of hematoma. Among the 39 patients with evidence of contrast material extravasation on MR imaging, 22 underwent repeated MR imaging between 5 and 24 hours after onset (mean 10.9 hours) to confirm hemostasis, and no evidence of extravasation was seen on the sec-

### Laboratory values in patients with hypertensive ICH and with and without evidence of extravasation on MR imaging

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Range</th>
<th>Extravasation</th>
<th>No Extravasation</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC (10^12/L)</td>
<td>400–530</td>
<td>472.9 ± 63.9</td>
<td>453.7 ± 63.2</td>
</tr>
<tr>
<td>WBC (10^9/L)</td>
<td>4–9</td>
<td>8.7 ± 3.4</td>
<td>9.3 ± 3.8</td>
</tr>
<tr>
<td>platelets (10^12/L)</td>
<td>13–40</td>
<td>25.1 ± 7.3</td>
<td>23.3 ± 6.8</td>
</tr>
<tr>
<td>GOT (IU/L)</td>
<td>10–35</td>
<td>30.9 ± 29.7</td>
<td>28.5 ± 29.6</td>
</tr>
<tr>
<td>GPT (IU/L)</td>
<td>5–40</td>
<td>18.3 ± 11.7</td>
<td>16.0 ± 12.1</td>
</tr>
<tr>
<td>γGTP (IU/L)</td>
<td>4–63</td>
<td>118.2 ± 234.5</td>
<td>81.1 ± 186.8</td>
</tr>
<tr>
<td>albumin/globulin ratio</td>
<td>1.2–2.0</td>
<td>1.66 ± 0.29</td>
<td>1.63 ± 0.39</td>
</tr>
<tr>
<td>APTT (sec)</td>
<td>25–40</td>
<td>30.8 ± 9.0</td>
<td>29.6 ± 4.1</td>
</tr>
<tr>
<td>PTT (sec)</td>
<td>10.4–11.6</td>
<td>12.1 ± 5.5</td>
<td>11.5 ± 0.7</td>
</tr>
<tr>
<td>fibrinogen (mg/dl)</td>
<td>185–500</td>
<td>282 ± 72.0</td>
<td>297 ± 98.8</td>
</tr>
</tbody>
</table>

* Data are presented as the mean ± SD. There was no significant difference in any of the parameters when patients with and without extravasation were compared. Abbreviations: APTT = activated partial thromboplastin time; γGTP = gamma-glutamyltranspeptidase; GOT = glutamic-oxaloacetate transaminase; GPT = glutamic-pyruvic transaminase; PTT = partial thromboplastin time; RBC = red blood cell; WBC = white blood cell.

### Blood pressure parameters in patients with and without evidence of extravasation on MR imaging

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Extravasation</th>
<th>No Extravasation</th>
</tr>
</thead>
<tbody>
<tr>
<td>initial systolic BP</td>
<td>185.5 ± 29.5</td>
<td>192.2 ± 34.2</td>
</tr>
<tr>
<td>initial diastolic BP</td>
<td>107.9 ± 26.0</td>
<td>107.0 ± 21.7</td>
</tr>
<tr>
<td>systolic BP at 6 hrs</td>
<td>160.3 ± 25.8†</td>
<td>151.8 ± 20.2</td>
</tr>
<tr>
<td>diastolic BP at 6 hrs</td>
<td>87.7 ± 23.6</td>
<td>87.0 ± 16.0</td>
</tr>
</tbody>
</table>

† Significant at p < 0.05. Other parameters showed no significant differences between groups.

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**TABLE 3**

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of Patients</th>
<th>Extravasation on MR Imaging</th>
<th>Extravasation on Angiography</th>
<th>Enlargement of Hematoma (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>putamen</td>
<td>57</td>
<td>23</td>
<td>11</td>
<td>14 (24.6)</td>
</tr>
<tr>
<td>thalamus</td>
<td>34</td>
<td>12</td>
<td>6</td>
<td>6 (17.6)</td>
</tr>
<tr>
<td>subcortex</td>
<td>6</td>
<td>2</td>
<td>2</td>
<td>1 (16.6)</td>
</tr>
<tr>
<td>pons</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0 (0)</td>
</tr>
<tr>
<td>cerebellum</td>
<td>8</td>
<td>1</td>
<td>0</td>
<td>1 (12.5)</td>
</tr>
<tr>
<td>caudate nucleus</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

**TABLE 4**

**TABLE 5**

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Magnetic resonance imaging is noninvasive, an advantage over angiography, although not many institutions have the resources to perform emergency MR studies 24 hours a day.

**Significance of MR Imaging Findings**

When the MR findings were classified as Type A (that is, punctate contrast enhancement within the hematoma and at its margin), enlargement of the hematoma was seen in 14 of 23 patients and extravasation on cerebral angiography was found in 12. These findings were the strongest indicators of persistent hemorrhage. Yamamoto, et al.,26 have classified MR imaging findings into Type A (no contrast enhancement), Type B (punctate enhancement within the hematoma or within 5 mm of its margin), and Type C (zonal or macular contrast enhancement). As defined by Yamamoto, et al., Type C enhancement indicated continued hemorrhage, which largely corresponds to our Types A and C.

In some reports6–19 it has been demonstrated that the site of hemorrhage most susceptible to hematoma enlargement is the thalamus adjacent to the third ventricle. In the present study, there were no significant differences in MR findings for hemorrhage in the putamen as compared with the thalamus, although actual expansion of the hematoma was more common in the putamen. The apparent difference between our results and those of other studies may be because we excluded patients with IVH from the calculation of hematoma size. In addition, no pathological examination was performed, and the possibility exists that hemorrhage in some of our patients was caused by factors other than hypertension, such as amyloid angiopathy with subcortical hemorrhage.

**Relationship With Clinical Parameters**

In the hyperacute stage of hypertensive ICH, the risk factors for hematoma expansion include liver dysfunction,5,10,12 excessive alcohol consumption,8,10,16,24 anticoagulant therapy,7,11,16 thrombocytopenia,7,22 poorly controlled hypertension,6,7,16,19 shape of the hematoma on initial CT scans,11 and extravasation on cerebral angiography.20,22 In the present study, MR findings of extravasation showed significant correlation only with systolic BP at 6 hours after onset. These results may be related to the fact that we compared the MR imaging contrast-enhancement findings rather than the actual expansion of the hematoma.

**References**

5. Caille JM, Lemanceau B, Bonnemain B: Gadolinium as a contrast agent for NMR. *AJNR* 4:1041–1042, 1983
17. Kanaya H: [Results of conservative and surgical treatment in hypertensive intracerebral hemorrhage—co-operative study in Japan.] *Jpn J Stroke* 12:509–524, 1990 (Jpn)

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