Original Article

Hyperacute intraventricular hemorrhage: detection and characterization, a comparison between 5 MRI sequences


Department of Neuroradiology, cliniques universitaires Saint-Luc, 1200 Brussels, Belgium
Department of Neuroradiology, centre hospitalier Sainte-Anne, université Paris-V, Paris, France

Keywords
Intraventricular hemorrhage; MR imaging; FLAIR sequence; Echo-planar imaging; Diffusion weighted imaging

Abstract We aimed to evaluate the diagnostic accuracy of MRI for detecting early intraventricular hemorrhage (IVH) (within 6 hours after hemorrhage and to describe the MR features that allow diagnosis. For this purpose, MR data of 22 patients with hyperacute intraparenchymal hemorrhage were independently rated as negative or positive for IVH by two observers, in a blind, retrospective study taking computed tomography (CT) as providing the correct diagnosis of IVH. Sensitivity, specificity, intra- and interobserver agreement were assessed. On FSE-FLAIR, EPI-GRE-T2* and DWI images, all cases of IVH were correctly rated (sensitivity of 100%). For b0 EPI images, obtained from diffusion-weighted echo planar sequences, one case of IVH was missed by one reader (sensitivity of 88%). For T1 images, one patient was incorrectly rated negative for IVH by the two readers (sensitivity of 90%). Three forms of IVH were described, including clotted hematoma, layered hemorrhage and red blood cell deposit. When CT images were obtained within a time span of less than 3 hours after MRI, volume was assessed. Volume of hemorrhage on CT correlated best with DWI images but was underestimated on EPI-GRE T2* images.

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Introduction

During the last decade, MR imaging has been increasingly used as the primary imaging tool for patients with suspicion of acute stroke. Although CT is still the usual means for detecting early hemorrhage, several studies support the use of early MR imaging for this purpose [1,8,12,14,16-18] owing to its high sensitivity for identifying early blood degradation products. Despite the fact that appearance and progression of intraparenchymal hemorrhage (IPH) on MR images have received extensive study over the past several years [2,3,6,7,9-11,15], the appearance of intraventricular blood, within the first few hours, has rarely come to clinical attention. In an early report, Bakshi et al. was the first group to describe two forms of intraventricular hemorrhage (IVH) by MRI, a fixed clot or a free-flowing mixture layering with cerebrospinal fluid (CSF), with or without blood-CSF level [4]. Later, they showed that FLAIR MR imaging was very sensitive to acute and subacute intraventricular hemorrhage [5]. The present study was conducted:

- to evaluate the sensitivity of MRI in the detection of hyperacute IVH, associated with deep, large hematoma, among 5 different MR sequences, using CT as the gold standard;
- and to investigate the appearance of intraventricular blood.

Material and methods

Study population

After a retrospective review of the stroke unit reports of our hospital between November 1997 and April 2004, two-hundred and five consecutive patients (N = 205) with acute stroke were identified. Only those with IPH who were referred for MR examination in the setting of acute stroke within 6 hours after symptoms onset and who had a CT-scan within the first 7 days confirming the diagnosis of IVH, without any clinical change or brain surgery, were included. Among the 205 patients considered, only 22 patients with IPH remained and by coincidence exactly half of them had secondary IVH. The patients without IVH were considered as control subjects.

Imaging protocols

All patients underwent MRI followed by CT. The mean time between MR examination and symptom onset was 3 hours (range, 15 minutes -6 hours). The mean interval between MRI and CT was approximately 36 hours (range, 30 minutes-7 days), and 5 CT were acquired less than 3 hours after MRI. MRI imaging was performed on a 1.5T system (GE unit, Milwaukee, WI) with echo planar imaging capability (EPI), using 5-mm sagittal T1- (TR/TE 300/2,9ms; matrix 256 × 160; imaging time, 50 s), 5-mm axial fast spin-echo fluid-attenuated inversion recovery (FSE-FLAIR)(TR/TI/TE 4200/140/50 ms; matrix 256 × 160; imaging time 3 min 1 s), echo-planar imaging gradient-recalled echo (EPI GRE T2*)- (TR/TE/T1 10002/140/2200 ms; matrix 256 × 160; imaging time 3 min 1 s), echo-planar imaging gradient-recalled echo (EPI GRE T2*)- (TR/TE 4200/50 ms; matrix 128 × 128; imaging time: 59 s) and axial diffusion weighted imaging (DWI) using spin-echo EPI, and two b values, including b = 0 s/mm2 and b = 1000s/mm2 (TR/TE 4500/94 ms; matrix 96 × 64; imaging time: 36 s).

Most of the time, this whole standardized MR protocol for hyperacute stroke was not performed because the diagnosis of IPH was obvious. Overall, 20 patients had sagittal GRE-T1, 11 axial FLAIR, 21 GRE EPI-T2* and 13 DWI sequences were obtained. Of the 11 patients with IVH, 10 T1, 3 FLAIR, 11 GRE EPI-T2* and 8 DWI sequences were obtained.

To qualify for enrollment, at least two sequences had to be completed.

Computed tomographic scans were performed on 1 of the following scanners: MX 8000 (Marconi), DUAL and Bril-
lance (Philips). Images were acquired in the orbito-meatal plane with 5 mm thickness.

Data collection and Processing

The studies were reviewed retrospectively by two neuroradiologists (D.H., reader 1, T.D. reader 2) blinded to the clinical information. All MR sequences from each subject were randomly ordered and interpretations were performed on different days to avoid reader recognition. They were independently rated on a 6-level scale: [1] no IVH (with high certitude), [2] no IVH (with medium certitude), [3] no IVH (with low certitude), [4] IVH (with low certitude), [4] IVH (with medium certitude), [6] IVH (with high certitude). For sensibility and specificity analysis, a binary decision was extracted from this 6-level scale: no for levels 1-4, yes for levels 5-6. The sensitivity and specificity of each MR sequence was calculated, taking the CT diagnosis as the gold standard. Inter- and intra-observer agreement were calculated for each sequence by kappa-analysis on the 6-level scale. IVH was further evaluated for pattern and signal intensity for each lateral ventricle (2 descriptions/patient) by a consensus reading of two others radiologists (A.D., G.C). Third and fourth ventricles were not observed because their involvement was less common. Pattern of intraventricular blood was assessed according to the forms described by Bakshi [4], to which we added a new pattern, called red blood cell deposit (RBC deposit), corresponding to sedimentation of agglutinated red blood cells in the posterior aspect of the ventricles. Signal intensity of IVH was compared with those of the intraparenchymal hematoma of origin and to CSF. When a CT acquired less than 3 hours after the MRI (5 patients) was available, a visual comparison was done between CT and MRI to assess the volume of IVH, and quoted as similar, underestimated or overestimated on MR.

Results

Twenty-two patients with IPH, eleven of them with IVH, were retrospectively studied. There were 18 males and 4 females patients ranging in age from 51 and 89 years (mean age, 69 years). Sixteen cases involved arterial hypertension; one, arteriovenous malformation; one, low platelets; and four, unknown etiology.

The accuracy of MR for identifying hyperacute IVH is shown in Tables 1, 2. The results of imaging findings of IVH, analyzing pattern and signal intensity, are summarized in Tables 3, 4. FLAIR detected all cases of IVH (3 of 3), and none of the FLAIR images of the control subjects were falsely rated (100% sensitivity, 100% specificity). T1 detected 9 of the 10 cases of IVH, but for reader 2, two of the T1 images of the control subjects falsely depicted IVH (90% sensitivity, 100% specificity). T1 detected 9 of the 10 cases of IVH, but for reader 2, two of the T1 images of the control subjects falsely depicted IVH (90% sensitivity, 100% specificity). T1 detected 9 of the 10 cases of IVH, but for reader 2, two of the T1 images of the control subjects falsely depicted IVH (90% sensitivity, 100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity). EG EPI-T2* detected all cases of IVH (11 of 11), and none of the EG EPI-T2* images of the control subjects were falsely detected IVH (100% specificity).

Table 1 Diagnostic performance figures (with 95% confidence intervals) of both readers for detection of IVH on 5 MRI sequences

<table>
<thead>
<tr>
<th>Pulse sequence</th>
<th>Reader 1 Sensitivity</th>
<th>Reader 1 Specificity</th>
<th>Reader 2 Sensitivity</th>
<th>Reader 2 Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>90% (9/10)</td>
<td>100% (10/10)</td>
<td>90% (9/10)</td>
<td>80% (8/10)</td>
</tr>
<tr>
<td>FLAIR</td>
<td>100% (3/3)</td>
<td>100% (8/8)</td>
<td>100% (3/3)</td>
<td>100% (8/8)</td>
</tr>
<tr>
<td>EPI T2*</td>
<td>100% (11/11)</td>
<td>100% (8/10)</td>
<td>80% (8/10)</td>
<td>100% (11/11)</td>
</tr>
<tr>
<td>B0</td>
<td>100% (8/8)</td>
<td>100% (5/5)</td>
<td>100% (7/8)</td>
<td>100% (8/8)</td>
</tr>
<tr>
<td>B1000</td>
<td>100% (8/8)</td>
<td>100% (5/5)</td>
<td>100% (8/8)</td>
<td>100% (5/5)</td>
</tr>
</tbody>
</table>

Table 2 Intra- and interobserver agreement for each MRI sequence

<table>
<thead>
<tr>
<th>Pulse sequence</th>
<th>Intraobserver agreement (k)</th>
<th>Interobserver agreement (k)</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>0,662</td>
<td>0,723</td>
</tr>
<tr>
<td>FLAIR</td>
<td>0,891</td>
<td>1</td>
</tr>
<tr>
<td>EPI T2*</td>
<td>0,779</td>
<td>0,804</td>
</tr>
<tr>
<td>B0</td>
<td>0,922</td>
<td>0,922</td>
</tr>
<tr>
<td>B1000</td>
<td>0,838</td>
<td>0,917</td>
</tr>
</tbody>
</table>

Table 3 Number and patterns of intraventricular hemorrhage identified on sequence considering two ventricles per patient (N = 11, 22 ventricles). RBC = red blood cell

<table>
<thead>
<tr>
<th>Pulse sequence</th>
<th>Clotted</th>
<th>Layered</th>
<th>RBC deposit</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>9</td>
<td>5</td>
<td>0</td>
<td>14</td>
</tr>
<tr>
<td>FLAIR</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>EPI T2*</td>
<td>12</td>
<td>1</td>
<td>18</td>
<td>31</td>
</tr>
<tr>
<td>B0</td>
<td>6</td>
<td>1</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>B1000</td>
<td>8</td>
<td>5</td>
<td>0</td>
<td>13</td>
</tr>
</tbody>
</table>
Table 4 Signal intensity of IVH (versus intraparenchymal hematoma of origin and versus CSF) identified in each sequence

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>FLAIR</th>
<th>EPI T2*</th>
<th>B0</th>
<th>B1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clotted</td>
<td>Iso/hyper</td>
<td>Iso/hyper</td>
<td>Iso/Iso CSF, with a hypo rim</td>
<td>Iso/hypo vs CSF, with a hypo rim</td>
<td>Iso/hyper vs CSF</td>
</tr>
<tr>
<td>Layered</td>
<td>Slightly hypo/slightly hyper vs CSF, with a gradient</td>
<td>Slightly hypo/hyper vs CSF</td>
<td>Slightly hypo/hyper vs CSF</td>
<td>Slightly hypo/hyper vs CSF</td>
<td></td>
</tr>
<tr>
<td>RBC</td>
<td>Very hypo</td>
<td>Very hypo</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1  Hypertensive parenchymal hematoma in the left basal ganglia with intraventricular extension of clotted pattern. a. Axial CT scan obtained after MR images. b. Axial b1000 diffusion-weighted image. c. Axial GRE EPI-T2* weighted image at the same level. d. Sagittal T1 image. The clotted hematoma appears as a lesion of high signal intensity on DWI (arrowheads), of lower signal on GRE EPI-T2*, surrounded by a rim of low intensity (arrows), and of mildly high signal relative to CSF on T1.

reade 1 but 7 of 8 for reade 2. None of the b0 images of the control subjects were falsely rated (100 and 88% sensitivity, 100% specificity). b1000 detected all cases of IVH (8 of 8) but one of the b1000 images of the control subjects was falsely rated by rd 2 (100% sensitivity, 100 and 80% specificity). Ranges for interobserver agreement based on the $\kappa$.
statistic for paired observers were from 0.662 to 0.922 for MRI. Intraobserver agreement varied from 0.723 to 1.

Three patterns of IVH in lateral ventricles were observed: clotted hematoma, layered hemorrhage and red blood cell deposit. These patterns co-existed frequently but had different signal characteristics. They were almost always in the lateral ventricles, usually bilateral. **Clotted hematoma**, well limited, appeared as a lesion of similar appearance as parenchymal hematoma, of mildly high signal relative to CSF on T1 (Fig. 1d), of high intensity on FLAIR (Fig. 2) and DWI images (Fig. 1b, Fig. 5c). On b0 images (Fig. 5b) and EG EPI-T2* (Fig. 1c), however, it was of lower intensity than CSF, surrounded by a characteristic peripheral rim of low intensity. **Layered hemorrhage** appeared more as a homogenous lesion of less high intensity on T1 and FLAIR (Fig. 3) than coexisting IPH because of dilution. On EG EPI-T2* and b0 images (Fig. 5b) layered hemorrhage was difficult to detect. **Red blood cell deposit** appeared as a small lesion limited to the bottom of the occipital horns (Fig. 4), of deep hypointense signal and only detected by EPI EG-GRE T2*.

For evaluating the volume of hemorrhage, DWI images were the most similar to CT (Fig. 1a,b and Fig. 5a,c).

**Discussion**

In analogy to subarachnoid hemorrhage (SAH), fresh intraventricular blood degrades more slowly than coexisting infraparenchymal hematoma because dilution by CSF and higher local oxygen content [6]. Therefore, recognition of IVH has prognostic value and may help to select patients for interventional therapy to avoid obstructive hydrocephalus. There are only a few studies on MR imaging of hyperacute intraventricular hemorrhage. Bakshi was the first to analyze the MR features of IVH, to describe two major forms, clotted hematoma and layered hemorrhage [4] and later to demonstrate the high sensitivity of FLAIR for acute and subacute IVH [5]. In our study, a third form of IVH was
shown, called red blood cell (RBC) deposit, corresponding to little hemorrhage limited to the bottom the ventricular horns, detected by more advanced techniques, including GRE EPI-T2* and SE EPI imaging. We observed that the 5 studied MRI sequences were able to assess the presence of intraventricular blood with high sensitivity and specificity. As expected, FLAIR and diffusion weighted imaging had the best accuracy for detecting clotted and layered hemorrhage. The usefulness of FLAIR in the diagnosis of IVH and SAH is well known [13] and relates to the hyperintense appearance of these hemorrhage, caused by a higher protein concentration, against a dark CSF background allowing blood to be seen in ventricles and sulci. On the diffusion-weighted images, IVH also appears as a hyperintense lesion against a dark CSF background, but corresponds to an area of decreased diffusion. On these sequences, the volume of hemorrhage was the most similar to CT. b0 EPI and EPI-GRE T2* sequences are sensitive to the susceptibility effects of magnetic field inhomogeneity of hyperacute intracranial hemorrhage [18]. For detecting IVH however, CSF dilutes blood and therefore lowers the concentration of paramagnetic deoxyhemoglobin necessary to cause hypointensity on

Figure 5  Clotted hematoma in the left lateral ventricle secondary to a deep hematoma, associated with a layered hemorrhage in the right ventricle. a. axial CT scan obtained after MR images; b. b0 SE EPI images; c. b1000 images. Clotted hematoma appears as a lesion of lower intensity than CSF on the b0 images, but of high intensity on the b1000 images. Layered hemorrhage is of less high intensity on b1000 and not detectable on b0 images.

Figure 5  Hématome cailloté dans le ventricule latéral gauche, secondaire à un hématome intracérébral profond, associé à une hémorragie déclive dans le ventricule latéral droit. a. Coupe axiale en CT obtenue après l’IRM. b. Coupe axiale pondérée b0 en séquence de diffusion. c. Coupe axiale pondérée b1000 en séquence de diffusion. L’hématome cailloté a une intensité moindre que celle du LCR sur l’image b0, moins plus élevée à b1000. L’hémorragie déclive est de moindre intensité à b1000 et n’est pas détectable à b0.
these sequences. For this reason, together with the high signal of surrounding CSF, clotted and layered IVH were more difficult to see. Detection of clotted hematoma was possible because of its mildly lower signal intensity than CSF and the presence of a characteristic rim of low intensity, due to susceptibility artifacts at the interface clot-CSF. However, RBC deposit was very well seen, probably explained by a higher concentration of aggregated red blood cells and the consecutive addition of paramagnetic effect of each RBC. Greater contrast resolution was achieved by GRE EPI-T2*, allowing the detection of subtle hemorrhagic lesions that may not be apparent on SE EPI T2. The weaknesses in this study include small size, failure to obtain all MR sequences and delays between CT and MR imaging. Indeed, because this study was retrospective, we did not perform a complete MR imaging protocol for every patient, which would have allowed to have a sufficient number of each sequence. Nevertheless, false negative or false positive diagnosis were certainly possible, and concerned often intraventricular hemorrhage of small size. In such cases, the diagnosis was not missed on the other sequences.

Conclusion

MRI seems to be diagnostic in the evaluation of hyperacute intraventricular hemorrhage as a complication of a deep intraparenchymal hematoma and may be of use as a primary diagnostic tool in stroke patient. FSE-FLAIR best performed for detection of clotted and layered hemorrhage and b1000 SE-DWI for quantification. EPI-GRE-T2* significantly underestimated the extent of bleeding when compared to CT. b0 images could not replace GRE images for the detection of acute blood products.

References