Diffusion-Weighted Imaging of Traumatic Subdural Hematoma in the Subacute Stage
—Five Case Reports—

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Abstract
Five cases of traumatic subdural hematomas in the subacute stage (from 7 to 20 days after head injury) were treated in one male and four females, aged from 63 to 82 years, with evacuation via craniotomy in three and aspiration via burr hole surgery in two. All hematomas were evaluated by T1-, T2-, and diffusion-weighted magnetic resonance imaging, and measurement of the apparent diffusion coefficient (ADC). Diffusion-weighted imaging showed the hematoma as a crescent high intensity area with a low intensity rim close to the brain surface (two-layered structure) in four cases and as high intensity with low intensity components in one case. The high intensity areas under the dura mater on diffusion-weighted imaging appeared as homogeneous high intensity on T1- and T2-weighted imaging in four cases, and inhomogeneous high intensity on T1- and isointensity on T2-weighted imaging in one case. The mean ADC value of the high intensity areas was 0.58 ± 0.23 (mean ± standard deviation) × 10^{-3} mm²/sec. The operative findings revealed the high intensity areas as solid clots. The low intensity areas on diffusion-weighted imaging appeared as homogeneous high intensity in four cases and inhomogeneous isointensity with high intensity components in one case on T1- and T2-weighted imaging. The mean ADC value of the low intensity areas was 2.03 ± 0.27 × 10^{-3} mm²/sec. The operative findings revealed the low intensity areas as mixtures of resolved clot and cerebrospinal fluid. Diffusion-weighted imaging showed the characteristic two-layered structure in traumatic subdural hematomas in the subacute stage, and analysis of the ADC values was useful for differentiating solid from liquid hematoma and for selection of the surgical procedure.

Key words: traumatic subdural hematoma, chronic subdural hematoma, diffusion-weighted magnetic resonance imaging, apparent diffusion coefficient, subacute stage

Introduction
Diffusion-weighted magnetic resonance (MR) imaging shows characteristic findings in cases of organized subdural hematoma and chronic subdural hematoma, and the apparent diffusion coefficient (ADC) values may be useful for differentiating solid from liquid hematoma. However, the diffusion-weighted MR imaging characteristics of traumatic subacute subdural hematomas have been rarely reported, and the potential of diffusion-weighted MR imaging for the evaluation of traumatic subacute subdural hematoma has not been established. Here we describe five cases of traumatic subdural hematoma in the subacute stage and evaluate diffusion-weighted imaging and the ADC values of the hematoma as indicators for the operative procedure.
Fig. 1 Case 1. A: Computed tomography scan showing the subdural hematoma as isodense including high density components with remarkable midline shift. B: T1-weighted magnetic resonance (MR) image showing the hematoma as high intensity with isointense components. C: T2-weighted MR image showing the hematoma as high intensity with isointense components. D: Diffusion-weighted MR image showing the hematoma as a crescent high intensity area with a low intensity rim close to the brain surface (two-layered structure).

Case Reports

Case 1: An 82-year-old female with a thin acute subdural hematoma suddenly became comatose with left hemiparesis 15 days after head injury. Computed tomography (CT) showed an isodense subdural hematoma with remarkable midline shift (Fig. 1A). T1-weighted (repetition time/echo time [TR/TE] = 500/15 msec) and T2-weighted (TR/TE = 3630/110 msec) MR imaging showed the hematoma as high intensity with isointense components. Echo-planar diffusion-weighted imaging (TR/TE = 3000/110 msec, field of view [FOV] 300 × 150 mm, slice thickness 7.0 mm with a 1.5 mm gap, matrix 128 × 64 mm, motion probing gradient [MPG] of 12 mT/m; and maximum b-factor of 993 sec/cm² [MAGNEX EPIOS 15; Shimadzu, Kyoto]) showed the hematoma as a crescent high intensity area with a low intensity rim close to the brain surface (Fig. 1D). The ADC value was calculated based on the Stejskal and Tanner equation, ADC = ln(SI₀/SI₁)/(b₀ – b₁), where SI₀ and SI₁ are the pixel signal intensities acquired from the diffusion-weighted imaging with b₀ of 0 sec/mm² and b₁-factor of 993 sec/mm². ADC maps were created on a pixel-by-pixel basis.

Regions of interest (ROIs) (mean 30 mm² per area) were drawn in the high and low intensity areas in the subdural hematoma, and in the central white matter and lateral ventricle on the diffusion-weighted MR images, and the ADC values of the ROIs were measured. The ADC values of the high and low intensity areas, and the brain tissue were 0.55 × 10⁻³, 2.1 × 10⁻³, and 0.75 × 10⁻³ mm²/sec, respectively. The ADC value of the high intensity component was lower than that of normal brain tissue, indicating that the hematoma was solid. The high intensity hematoma occupied a large part of the subdural space on diffusion-weighted imaging and the patient was comatose. Therefore, urgent craniotomy and evacuation of the hematoma was performed.

Intraoperatively, the hematoma consisted of black-brown outer membrane, red-brown solid or jelly clot, and some yellowish liquid. The solid clot was located underneath the dura mater and the yellowish liquid was located close to the brain. During removal of the hematoma, bleeding from a cortical artery was observed through the torn arachnoid membrane on the brain surface around the right sylvian fissure, and the inner membrane could not be confirmed after evacuation of the hematoma. The postoperative course was uneventful and the patient fully recovered.

Case 2: A 75-year-old female with a history of osteoporosis complained of severe headache and presented with disorientation of time and person 7 days after head injury. Brain CT showed a low density hematoma with isodense components in the right subdural space (Fig. 2A). T1-weighted (TR/TE = 500/10 msec) and T2-weighted (TR/TE = 3000/99.7 msec) MR imaging showed the hematoma as homogeneous high intensity with isointense components (Fig. 2B, C). Echo-planar diffusion-weighted imaging (TR/TE = 4800/83.3 msec, FOV 200 × 200 mm, slice thickness 8.0 mm with a 2.0 mm gap, matrix 128 × 128 mm, three orthogonal MPGs of 14 mT/m; and maximum b-factor of 1000 sec/cm² [Signa Horizon Infinity EXCITE; General Electric Medical Systems, Tokyo]) showed the
hematoma as high intensity with low intensity components (Fig. 2D). The ADC value was calculated based on the Stejskal and Tanner equation, \[^{13}\] with \(b_0\) of 0 sec/mm\(^2\) and \(b_1\)-factor of 1000 sec/mm\(^2\), and ROIs as in Case 1. The ADC value of the high intensity component was \(0.25 \times 10^{-3}\) mm\(^2\)/sec, lower than that of normal brain tissue (\(0.78 \times 10^{-3}\) mm\(^2\)/sec). The high intensity hematoma was considered to be solid and occupied a large part of the subdural space on the diffusion-weighted imaging. Craniotomy and evacuation of the hematoma was performed because of her headache and gradually developing disorientation.

Intraoperatively, the subdural hematoma consisted of hemorrhagic outer membrane, dark-brown solid or jelly clot, and yellowish fluid. No inner membrane was found under the subdural hematoma, and a bleeding cortical artery was confirmed through a small torn arachnoid membrane on the brain surface of the right frontal lobe. The postoperative course was uneventful.

**Case 3:** A 63-year-old male with a thin acute subdural hematoma caused by head trauma complained of persistent headache for 20 days after the injury. CT showed the hematoma as isodense and low density areas including high density components (Fig. 3A). T\(_1\)- and T\(_2\)-weighted MR imaging with similar parameters to Case 2 showed the hematoma as homogeneous high intensity (Fig. 3B, C). Diffusion-weighted imaging showed the hematoma as a crescent high intensity area with a low intensity rim close to the brain surface (Fig. 3D). The ADC value of the high intensity component was \(0.56 \times 10^{-3}\) mm\(^2\)/sec, lower than that of normal brain tissue...
(0.82 × 10^{-3} \text{ mm}^2/\text{sec}) \text{ (Fig. 3E). The high intensity hematoma was considered to be solid and occupied a large part of the subdural space on diffusion-weighted imaging. Craniotomy and evacuation of the hematoma were performed because his headache had gradually developed.}

Intraoperatively, the subdural hematoma consisted of hemorrhagic outer membrane, dark-brown solid or jelly clot, and yellowish fluid. The solid or jelly clot was located underneath the dura mater, and the yellowish fluid was located close to the brain. No inner membrane was found, and the brain surface was intact. The postoperative course was uneventful.

**Case 4:** An 81-year-old female who suffered a head injury about 16 days prior to admission complained of persistent headache. T1- and T2-weighted MR imaging with parameters similar to Case 2 showed the left subdural hematoma as homogeneous high intensity (Fig. 4A, B). Diffusion-weighted imaging showed the hematoma as a crescent high intensity area with a low intensity rim close to the brain surface (Fig. 4C). The ADC value of the high intensity area was 0.90 × 10^{-3} \text{ mm}^2/\text{sec}, lower than that of the low intensity area (2.04 × 10^{-3} \text{ mm}^2/\text{sec}), indicating that the hematoma was solid.

Standard burr hole surgery was performed, but only about 20 ml of xanthochromic fluid was aspirated containing a piece of solid clot. Relatively fresh clot was confirmed underneath the outer membrane, and cerebrospinal fluid (CSP) leaked through the burr hole. The intraoperative findings showed subdural hematoma with outer membrane in the subacute stage, not typical chronic subdural hematoma. The postoperative course was uneventful.

**Case 5:** A 72-year-old female who suffered a head injury 19 days prior to admission complained of persistent headache without neurological deficits. T1-weighted MR imaging with parameters similar to Case 2 showed the hematoma as inhomogeneous slightly high intensity associated with isointense components (Fig. 5A). T2-weighted imaging showed the hematoma as inhomogeneous high intensity with isointense components (Fig. 5B). Diffusion-weighted imaging showed the hematoma as a crescent high intensity area with a low intensity rim close to the brain (Fig. 5C). The ADC value of the high intensity area was 0.65 × 10^{-3} \text{ mm}^2/\text{sec}, lower than that of the low intensity area (2.42 × 10^{-3} \text{ mm}^2/\text{sec}), indicating that the hematoma was solid.
Table 1  Summary of five cases of traumatic subdural hematoma in the subacute stage

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Interval from the head injury (days)</th>
<th>Symptoms</th>
<th>ADC values (×10⁻³ mm²/sec)</th>
<th>Comparison of size of HIA and LIA</th>
<th>Surgical procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>82/F</td>
<td>15</td>
<td>semicoma, lt hemiparesis</td>
<td>0.55 2.10 0.75 3.08</td>
<td>HIA &gt; LIA</td>
<td>craniotomy</td>
</tr>
<tr>
<td>2</td>
<td>75/F</td>
<td>7</td>
<td>headache, disorientation</td>
<td>0.25 1.67 0.78 2.73</td>
<td>HIA &gt; LIA</td>
<td>craniotomy</td>
</tr>
<tr>
<td>3*</td>
<td>63/M</td>
<td>20</td>
<td>headache</td>
<td>0.56 1.93 0.82 2.87</td>
<td>HIA &gt; LIA</td>
<td>craniotomy</td>
</tr>
<tr>
<td>4*</td>
<td>81/F</td>
<td>16</td>
<td>headache</td>
<td>0.90 2.04 0.74 3.47</td>
<td>HIA &lt; LIA</td>
<td>burr hole</td>
</tr>
<tr>
<td>5*</td>
<td>72/F</td>
<td>19</td>
<td>headache</td>
<td>0.65 2.42 0.81 2.88</td>
<td>HIA &lt; LIA</td>
<td>burr hole</td>
</tr>
</tbody>
</table>

*Diffusion-weighted imaging showed the two-layered structure. ADC: apparent diffusion coefficient, CSF: cerebrospinal fluid, HIA: high intensity area, LIA: low intensity area.

(Fig. 5D, E).

Standard burr hole surgery was performed, and about 50 ml of xanthochromic fluid was aspirated containing a piece of solid clot. The solid clot was confirmed underneath the thick outer membrane, and CSF leaked through the burr hole. Intraoperative findings showed subdural hematoma with outer membrane in the subacute stage, not typical chronic subdural hematoma. The postoperative course was uneventful.

**Discussion**

Traumatic subdural hematomas can be divided into three groups based on the timing of head injury symptoms as follows: acute (within 3 days), subacute (4 to 20 days), and chronic (over 21 days). Subdural hematoma in the subacute stage includes so-called traumatic subacute subdural hematoma and chronic subdural hematoma-like hematoma. However, the diagnostic criteria, pathophysiology, and therapeutic procedures of these traumatic subdural hematomas are still not established.

Diagnostic criteria for traumatic subacute subdural hematoma have been proposed as follows: headache, consciousness disturbance, and focal neurological deficits within 4 days to 3 weeks after head and/or facial trauma; subdural hematoma on CT and/or MR imaging without cerebral contusion, traumatic intracerebral hematoma, or epidural hematoma; and no evidence of subdural hematoma except in cerebrovascular accidents, hemorrhagic tendency causing hematological disorder and drug abuse, or pathophysiology resulting in consciousness disturbance except for subdural disturbance. Cases 1–3 fulfilled the above-mentioned criteria. Cases 4 and 5 were considered to be subdural hematoma with outer membrane in the subacute stage based on the intraoperative findings. These subdural hematomas may be the same as the chronic subdural hematoma-like hematoma previously proposed.

Table 1 summarizes the clinical manifestations and ADC values of the present cases. Neurological symptoms and signs appeared over 2 weeks after injury in Cases 1, 3, 4, and 5. Moreover, diffusion-weighted imaging showed the hematoma as a two-layered structure consisting of a crescent high intensity area with a low intensity rim close to the brain surface in these four patients. The mean ADC values of the high and low intensity areas in the subdural hematoma, brain, and CSF were 0.58 ± 0.23 × 10⁻³, 2.03 ± 0.27 × 10⁻³, 0.77 ± 0.03 × 10⁻³, and 3.01 ± 0.29 × 10⁻³ mm²/sec, respectively. The mean ADC value of the high intensity areas was similar to that of the brain. The mean ADC value of the low intensity areas was considerably higher than those of the high intensity areas and brain tissue. The high intensity areas on the diffusion-weighted imaging corresponded to the high intensity areas on both T₁- and T₂-weighted imaging. In general, late subacute intracerebral hematoma appears as high intensity on both T₁- and T₂-weighted imaging, reflecting lysis of the erythrocytes and predominantly extracellular methemoglobin. The mean ADC value of the extracellular methemoglobin is 0.58 ± 0.10 × 10⁻³ mm²/sec. The high intensity areas in the subacute hematoma on diffusion-weighted imaging were considered to mainly consist of extracellular methemoglobin.

The low intensity areas on diffusion-weighted imaging also corresponded to high intensity areas on both T₁- and T₂-weighted imaging. The mean ADC value (2.03 ± 0.27 × 10⁻³ mm²/sec) was smaller than that of CSF (3.01 ± 0.29 × 10⁻³ mm²/sec), but similar to that of chronic subdural hematoma (1.81 ± 0.79 × 10⁻³ mm²/sec). Therefore, the low intensity areas were considered to

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be fluid containing extracellular methemoglobin influenced by water molecular diffusion.

The mechanism of the increase in the hematoma volume may be as follows: rebleeding into the hematoma cavity,\textsuperscript{11}) influx of CSF into the hematoma cavity according to the osmotic gradient,\textsuperscript{11}) and development of subdural effusion derived from the torn arachnoid membrane associated with lysis of the subdural hematoma.\textsuperscript{5,10}) The operative exploration of Cases 1–3 revealed that the subdural hematomas consisted of hemorrhagic outer membrane, dark-brown solid or jelly clot, and yellowish fluid. The yellowish fluid was located under the solid clot as if the fluid was enclosed between the solid clot and the arachnoid membrane. The inner membranes were not formed, and the arachnoid membrane was torn in these three cases. The cortical arteries bled through the torn arachnoid membrane after the solid clot was removed in Cases 1 and 2. The subdural contents were aspirated (about 20 and 50 ml of xanthochromic fluid) confirming the solid clot underneath the thick outer membrane and CSF leakage through the burr hole in Cases 4 and 5. The operative findings supported the origin of subdural effusion from the torn arachnoid membrane as the solid subdural hematoma resolved.\textsuperscript{5,10}) The ADC values of the low intensity areas on the diffusion-weighted imaging reflected the mixture of CSF and extracellular methemoglobin derived from the solid clot.

Craniotomy is recommended for the treatment of traumatic subdural hematoma containing more solid clots, in which diffusion-weighted imaging shows a larger high intensity area than low intensity area, as in our Cases 1–3. Direct observation of the wide operative field allows evacuation of the entire hematoma and control of the bleeding point. On the other hand, burr hole surgery can easily decrease the hematoma volume, so is indicated for hematoma containing more liquid components, in which diffusion-weighted imaging shows a larger low intensity area than high intensity area, as in our Cases 4 and 5.

Diffusion-weighted imaging showed the characteristic finding of a two-layered structure in traumatic subdural hematoma in the subacute stage, and analysis of the ADC values was useful for differentiating solid from liquid hematoma and for selection of the surgical procedure.

References


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