Subdural Hyperintense Band on Diffusion-Weighted Imaging of Chronic Subdural Hematoma Indicates Bleeding From the Outer Membrane

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Abstract

The diffusion-weighted magnetic resonance (MR) imaging characteristics of chronic subdural hematoma and the correlation between hematoma liquidity and apparent diffusion coefficient (ADC) were investigated in 26 consecutive patients, 16 males and 10 females aged 42 to 92 years (mean ± SD 73.3 ± 13.1 years), with 31 chronic subdural hematomas. The chronic subdural hematomas were divided into homogeneous, separate, and trabecular types based on diffusion-weighted MR imaging findings. Almost all hematomas were low intensity on diffusion-weighted imaging, and the mean ADC value was 1.81 ± 0.79 × 10⁻³ mm²/sec. The high intensity areas in the subdural hematomas consisted of several types: high intensity line along the dura mater (subdural hyperintense band), high intensity along the intrahematoma septum, and laminar shape along the inner membrane. The subdural hyperintense bands accounted for almost all high intensity areas in the subdural hematomas. The mean ADC value of the high intensity areas was 0.76 ± 0.24 × 10⁻³ mm²/sec, close to that of the normal brain. The subdural hyperintense bands were considered to be intracellular and/or extracellular methemoglobin based on the T₁- and T₂-weighted imaging and intraoperative findings. The subdural hyperintense band is an important finding indicating relatively fresh bleeding from the outer membrane. Diffusion-weighted imaging shows liquid subdural hematoma as low intensity, and measurement of the ADC values can differentiate between liquid and solid components of the chronic subdural hematoma.

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

Introduction

Diffusion-weighted magnetic resonance (MR) imaging is extremely sensitive to changes in the microscopic motion of water protons. 12,13) Diffusion-weighted MR imaging has proved useful in a study of the natural history of ischemic stroke, 15) and is now a promising technique for early detection of cerebral infarction in routine clinical practice. 2,14,19,22) We recently confirmed that diffusion-weighted imaging showed solid clots as high intensity areas in patients with traumatic subacute subdural hematoma 10) and organized subdural hematoma, 11) and measurement of the apparent diffusion coefficient (ADC) values was useful for differentiating solid from liquid components. 11) However, the diffusion-weighted imaging appearance of chronic subdural hematomas has been rarely reported, 9) and the potential of diffusion-weighted imaging for the evaluation of chronic subdural hematoma has not yet been established.

The present study investigated the diffusion-weighted imaging appearance of chronic subdural hematoma and tried to predict the hematoma liquidity based on the ADC values.

Patients and Methods

Twenty-six consecutive patients, 16 males and 10 females aged 42 to 92 years (mean ± SD 73.3 ± 13.1 years), were treated for 21 unilateral (12 on the left and 9 on the right) and five bilateral chronic
subdural hematomas from April 2003 to March 2004. Chronic subdural hematoma was characterized by: typical neomembrane; typical liquefied blood within the hematoma cavity; if following acute subdural hematoma, at least 3 weeks had passed. All diagnoses of chronic subdural hematomas were confirmed during evacuation of the hematoma with closed subdural drainage for 1 or 2 days. Patients with hygromas, immature chronic subdural hematomas, and calcified chronic subdural hematomas were excluded from this study.

All patients were preoperatively evaluated by T₁-weighted (repetition time/echo time [TR/TE] = 500/10 msec), T₂-weighted (TR/TE = 3000/99.7 msec), and diffusion-weighted MR imaging using a 1.5-T superconducting system (Signa Horizon Infinity EXCITE; General Electric Medical Systems, Tokyo). Single-shot echo-planar diffusion-weighted MR imaging was obtained in three orthogonal motion probing gradients (MPGs) (TR/TE = 4800/83.3 msec, field of view 200 × 200 mm, slice thickness 8.0 mm with a 2.0 mm gap, matrix 128 × 128 mm, MPG of 14 mT/m; and maximum b-factor of 1000 sec/cm²). The ADC value was calculated based on the Stejskal and Tanner equation,20)

\[
\text{ADC} = \frac{\ln\left(\frac{S_1}{S_0}\right)}{b_1 - b_0},
\]

where \(S_1\) and \(S_0\) are the pixel signal intensities acquired from the echo-planar diffusion-weighted imaging with b₁-factor of 1000 sec/mm² and \(b_0\) of 0 sec/mm², respectively. The ADC maps were created using this calculation on a pixel-by-pixel basis. Regions of interest (ROIs) (30 mm² on the average per area) were drawn in high, iso-, and low intensity areas in the hematoma cavity and in the central white matter and lateral ventricle on the diffusion-weighted MR images, and the ADC values of the ROIs were measured.

Statistical analysis was performed using analysis of variance and Scheffe’s F test. Significance was assumed if the probability value was less than 0.05. All statistical analyses were performed using commercially available statistical software (StatView, Version 4.5; SAS Institute, Lin, Cary, N.C., U.S.A.).

Results

The appearances of the subdural hematomas on diffusion-weighted MR imaging were divided into three groups based on their internal architecture: homogeneous, separate, and trabecular types. Isointensity was defined as equal or near-equal intensity to that of the brain (ranging from the gray matter to the white matter). The homogeneous type was defined as homogeneous low or high intensity (Fig. 1A). The separate type was defined as two components of different intensities with or without a clear boundary between them (Fig. 1B). The trabecular type was defined as a hematoma with multiple cavities divided by septa running between the inner and outer membranes on the background (Fig. 1C).

The 26 patients harbored 31 subdural hematomas, of which 16 were homogeneous, seven were separate, and eight were trabecular on diffusion-weighted MR imaging. Fourteen of the 16 homogeneous chronic subdural hematomas were low intensity and two were isointensity. All trabecular chronic subdural hematomas were iso- and/or high intensity on low intensity backgrounds. Twenty-

Fig. 1 Diffusion-weighted magnetic resonance images showing homogeneous (A), separate (B), and trabecular type (C) subdural hematomas mainly as low intensity areas. A crescent hyperintense area along or underneath the dura mater (subdural hyperintense band) was prominent compared to the normal dura mater (B, arrowhead).
Fig. 2 Case 1. A 72-year-old diabetic male with motor weakness of the bilateral lower extremities. (A) T₁-weighted magnetic resonance (MR) image showing bilateral subdural hematomas as isointensity including inhomogeneous high intensity components. (B) T₂-weighted MR image showing bilateral subdural hematomas as high intensity including inhomogeneous low intensity components. (C) Diffusion-weighted MR image showing homogeneous type subdural hematoma associated with subdural hyperintense bands (arrowheads) considered to be intracellular methemoglobin based on the T₁- and T₂-weighted imaging findings. (D, E) The ADC value of the high intensity area in the left subdural hematoma was $0.96 \times 10^{-3}$ mm²/sec, and those of the low intensity areas in the bilateral subdural hematomas were $2.89 \times 10^{-3}$ and $2.60 \times 10^{-3}$ mm²/sec.

Nine of the 31 chronic subdural hematomas contained low intensity components and 12 contained a crescent hyperintense area along or underneath the dura mater (Fig. 1B), which we named the subdural hyperintense band in this paper. The 12 subdural hyperintense bands were seen in seven homogeneous, two separate, and three trabecular chronic subdural hematomas. The eight subdural hyperintense bands appearing as high intensity on T₁-weighted imaging and low intensity on T₂-weighted imaging were interpreted as intracellular methemoglobin. The four subdural hyperintense bands appearing as high intensity on both T₁- and T₂-weighted imaging were interpreted as extracellular methemoglobin.

The ADC values of the subdural hematoma, brain, and cerebrospinal fluid (CSF) were measured in the ROIs in the low, iso-, and high intensity areas in the subdural hematoma, internal capsule and corona radiata, and lateral ventricle. The ADC values were $1.81 \pm 0.79 \times 10^{-3}$ (mean ± SD), $1.36 \pm 0.60 \times 10^{-3}$, and $0.76 \pm 0.24 \times 10^{-3}$ mm²/sec in the low, iso-, and high intensity areas in the subdural hematoma, respectively. $0.80 \pm 0.11 \times 10^{-3}$ mm²/sec in the brain, and $3.1 \pm 0.30 \times 10^{-3}$ mm²/sec in the CSF. The ADC value in the high intensity areas was close to that of the brain. The ADC of the low intensity areas was significantly higher than that of the high.

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Fig. 3  Case 2. A 75-year-old male presenting with gradually decreasing activity in daily life. (A) T₁- and (B) T₂-weighted magnetic resonance (MR) images showing bilateral subdural hematomas as high intensity including irregular low intensity components. (C) Diffusion-weighted MR image showing the bilateral trabecular type subdural hematomas associated with a left subdural hyperintense band (arrowhead) considered to be extracellular methemoglobin. (D, E) The ADC values of the low, iso-, and high intensity areas in the left subdural hematoma were $1.11 \times 10^{-3}$, $1.08 \times 10^{-3}$, and $0.81 \times 10^{-3}$ mm²/sec, respectively. The ADC value of the high intensity area in the right subdural hematoma was $0.84 \times 10^{-3}$ mm²/sec.

Illustrative Cases

Case 1: A 72-year-old diabetic male suffered a head injury about 2 months prior to admission and presented with bilateral lower extremity motor paresis. T₁-weighted MR imaging showed bilateral subdural hematomas as isointensity including high intensity components (Fig. 2A). T₂-weighted MR imaging showed the bilateral subdural hematomas as high intensity including low intensity components (Fig. 2B). Diffusion-weighted MR imaging showed homogeneous low intensity areas with subdural hyperintense bands (Fig. 2C). The subdural hyperintense bands were considered to be intracellular methemoglobin based on the T₁- and T₂-weighted imaging findings (Fig. 2C). The ADC values of the low intensity areas in the bilateral subdural hematomas were $2.89 \times 10^{-3}$ and $2.60 \times 10^{-3}$ mm²/sec, and that of high intensity area in the left subdural hematoma was $0.96 \times 10^{-3}$ mm²/sec (Fig. 2D, E). Standard burr-hole surgery was performed for the right subdural hematoma, and about 120 ml of chocolate-like fluid containing solid clots was evacuated, confirming the presence of relatively high intensity areas and brain tissue ($p < 0.05$). Therefore, the ADC values indicated liquid as low intensity areas and solid clot as high intensity areas.
fresh clot underneath the outer membrane. Closed drainage of the subdural space was continued for 1 day. The postoperative course was uneventful.

**Case 2**: A 75-year-old male with a history of hypertension presented with gradually decreasing activity in daily life. He had suffered a traffic accident about 3 months prior to admission. He presented with mild right hemiparesis predominantly in the upper extremities. T₁- and T₂-weighted MR imaging showed high intensity including irregular low intensity components in the bilateral subdural spaces (Fig. 3A, B). The left subdural hematoma was low intensity including irregular isointensity components associated with an iso- to high intensity subdural band on diffusion-weighted imaging (Fig. 3C). The subdural hyperintense band in the left subdural hematoma was considered to be extracellular methemoglobin based on the T₁- and T₂-weighted imaging findings. The ADC values of the low, iso-, and high intensity areas in the left subdural space were 1.11 × 10⁻³, 1.08 × 10⁻³, and 0.81 × 10⁻³ mm²/sec, respectively (Fig. 3D, E). The ADC value of the high intensity area in the right subdural hematoma was 0.84 × 10⁻³ mm²/sec. The left subdural hematoma containing some solid clots was evacuated, confirming the presence of fresh clot underneath a thick outer membrane during the standard burr-hole surgery. The postoperative course was uneventful.

**Discussion**

Chronic subdural hematoma is a clearly delineated and encapsulated collection of fluid and blood clots of various ages between the dura mater and arachnoid. The MR imaging appearance of chronic subdural hematoma is usually described as a short T₁ and long T₂ pattern, since extracellular methemoglobin induces marked shortening of the T₁ relaxation time and prolongation of the T₂ relaxation time. If the concentration of the extracellular methemoglobin is decreased by dilution, absorption, or degradation, the subdural hematoma becomes usually slightly hypointense to isointense on T₁-weighted imaging resulting from the decrease in the T₁ shortening effect.

In this study, 29 of the 31 chronic subdural hematomas showed low intensity components on diffusion-weighted imaging due to the free water molecular movement rather than the paramagnetic effect of the extracellular methemoglobin, because the ADC value (1.81 ± 0.79 × 10⁻³ mm²/sec) of the low intensity areas was larger than the reported ADC value of extracellular methemoglobin (0.58 ± 0.10 × 10⁻³ mm²/sec).

The high intensity areas in the present subdural hematomas consisted of several types: high intensity line along the dura mater (subdural hyperintense band), high intensity along the intrahematoma septum, and laminar shape along the inner membrane. The subdural hyperintense bands accounted for almost all high intensity areas in the subdural hematomas. Therefore, the ADC value of the high intensity areas in the subdural hematoma corresponded to that of the subdural hyperintense band on diffusion-weighted imaging. The high intensity bands may reflect extracellular and/or intracellular methemoglobins without the influence of water molecular diffusion because the ADC value (0.76 ± 0.24 × 10⁻³ mm²/sec) of the high intensity areas in the chronic subdural hematomas was similar to the reported ADC values of the extracellular (0.58 ± 0.10 × 10⁻³ mm²/sec) and/or intracellular (0.55 ± 0.23 × 10⁻³ mm²/sec) methemoglobins.

Repeated hemorrhaging from the outer membrane has been considered to be a causative factor of the chronic subdural hematoma. Intravenous infusion of ⁵¹Cr-labeled red blood cells in patients with chronic subdural hematoma resulted in the subsequent presence of labeled cells in the subdural fluid. The presence of ⁵¹Cr-labeled erythrocytes in the chronic subdural hematoma fluid confirms rebleeding from capillaries in the outer membrane as a major source of expansion in the chronic subdural hematoma. Thrombomodulin is expressed on the sinusoidal vessels in the outer membrane, and the blood coagulation system is inhibited in subdural hematoma. These findings indicate that these vessels are continuously injured and fail to heal, resulting in persistence of hemorrhage from the sinusoidal vessels.

In general, subacute intracerebral hematomas predominantly contain intracellular methemoglobin, appearing as high intensity on T₁-weighted imaging and low intensity on T₂-weighted imaging. Late subacute hemorrhage appears as high intensity on both T₁- and T₂-weighted imaging, reflecting lysis of the erythrocytes and predominantly extracellular methemoglobin. The subdural hyperintense band is considered to consist of relatively fresh hemorrhage derived from the outer membrane, because we intraoperatively confirmed thick outer membranes and solid clot underneath the dura mater, and the ADC value of the hyperintense band was close to the reported value of the intracellular and/or extracellular methemoglobin. The subdural hyperintense band on diffusion-weighted imaging is conspicuous in contrast to the adjacent normal tissues because the liquid components in the chronic subdural hematomas appear as low intensity.
whereas the dura mater appears as iso- and/or low intensity. T1- and T2-weighted imaging show the areas corresponding to the subdural hyperintense band less clearly than diffusion-weighted imaging. T1-weighted imaging shows the signal intensity of the areas as higher than the surrounding hematoma. T2-weighted imaging also shows the areas with slightly higher intensity (extracellular methemoglobin) or low intensity (intracellular methemoglobin). Therefore, relatively fresh bleeding is difficult to distinguish from the surrounding fluid clot on conventional T1- and T2-weighted imaging.

In conclusion, diffusion-weighted imaging shows liquid component of subdural hematoma as low intensity, with a higher ADC value (1.81 ± 0.79 × 10⁻² mm²/sec) than the solid component and the brain tissue appearing as high intensity. Therefore, diffusion-weighted imaging and measurement of the ADC values are useful for differentiation between liquid and solid chronic subdural hematoma. The subdural hyperintense band observed on diffusion-weighted imaging is an important finding reflecting relatively fresh bleeding from the outer membrane and may be a predictive factor of enlargement of the hematoma or transformation from subdural effusion. Moreover, the subdural hyperintense band may be a useful preoperative indicator for determining the site or number of burr holes for satisfactory evacuation of the hematoma.

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Commentary

The authors investigated the diffusion-weighted imaging appearance of chronic subdural hematoma and tried to predict the hematoma liquidity based on the ADC values. They found that diffusion-weighted imaging showed the liquid component of subdural hematoma as low intensity with a higher ADC value, and solid clot as high intensity with a lower ADC. Furthermore, the authors pointed out the importance of the subdural hyperintense band observed on diffusion-weighted image as a finding reflecting relatively fresh bleeding from the outer membrane.

A basic question here is, should we routinely do preoperative MR imaging in every patient with chronic subdural hematoma to get the information of diffusion-weighted imaging? Further studies are necessary to prove the clinical significance of the authors’ observation. For instance, we wish to know how the existence of the subdural hyperintense band relates to surgical results in patients with chronic subdural hematoma.

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Kuwahara et al. report on magnetic resonance (MR) imaging of chronic subdural hematomas. T1-, T2- and diffusion-weighted images in 26 patients and 31 hematomas appear to be correlated with the consistency of the hematoma. It is assumed that the hematoma is fed from the outer membrane.

As the dynamic evolution of chronic subdural hematomas still holds many secrets, and even production of erythrocytes has been identified in the outer membrane,1) this creative report on MR imaging is an important contribution to our understanding. Why do these hematomas enlarge over weeks and why is that progress in most cases but not in all cases reversed by a simple drainage for one or two days? As the signal intensities may give us a clue as to the age and consistency, MR imaging may be of practical value for planning surgery. A liquid hematoma may be drained through a small hole but a firm clot may better be evacuated via a reasonable size craniotomy. The authors are to be congratulated upon their findings.

Reference


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