

# Chronic Subdural Hematoma With Vasogenic Edema in the Cerebral Hemisphere

## —Case Report—

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### Abstract

An 80-year-old male with a history of hypertension presented with chronic subdural hematoma manifesting as progressive consciousness disturbance and left hemiparesis. T<sub>1</sub>-weighted and fluid attenuation inversion recovery (FLAIR) magnetic resonance imaging showed a fresh hematoma in the right subdural space with a midline shift of 15 mm. FLAIR and diffusion-weighted imaging showed a hyperintense area in the right paraventricular white matter compressed by the hematoma. Apparent diffusion coefficients (ADCs) corresponding to the hyperintense area in the central area of the affected cerebral hemisphere on FLAIR images were measured before and one month after the operation. The motion probing gradient was applied in the right-left direction to the body axis. Since the central area in the cerebrum includes nerve fibers perpendicular to the direction of the gradient, the measured ADC appeared to be anisotropic. Preoperative ADC in the right paraventricular white matter was anisotropic and greater than in age-matched normal subjects, so the edema was identified as the vasogenic type. The edema in the right paraventricular white matter resolved promptly with improvement of the midline shift and normalization of the ADC.

Key words: chronic subdural hematoma, brain edema, diffusion-weighted imaging, apparent diffusion coefficient

### Introduction

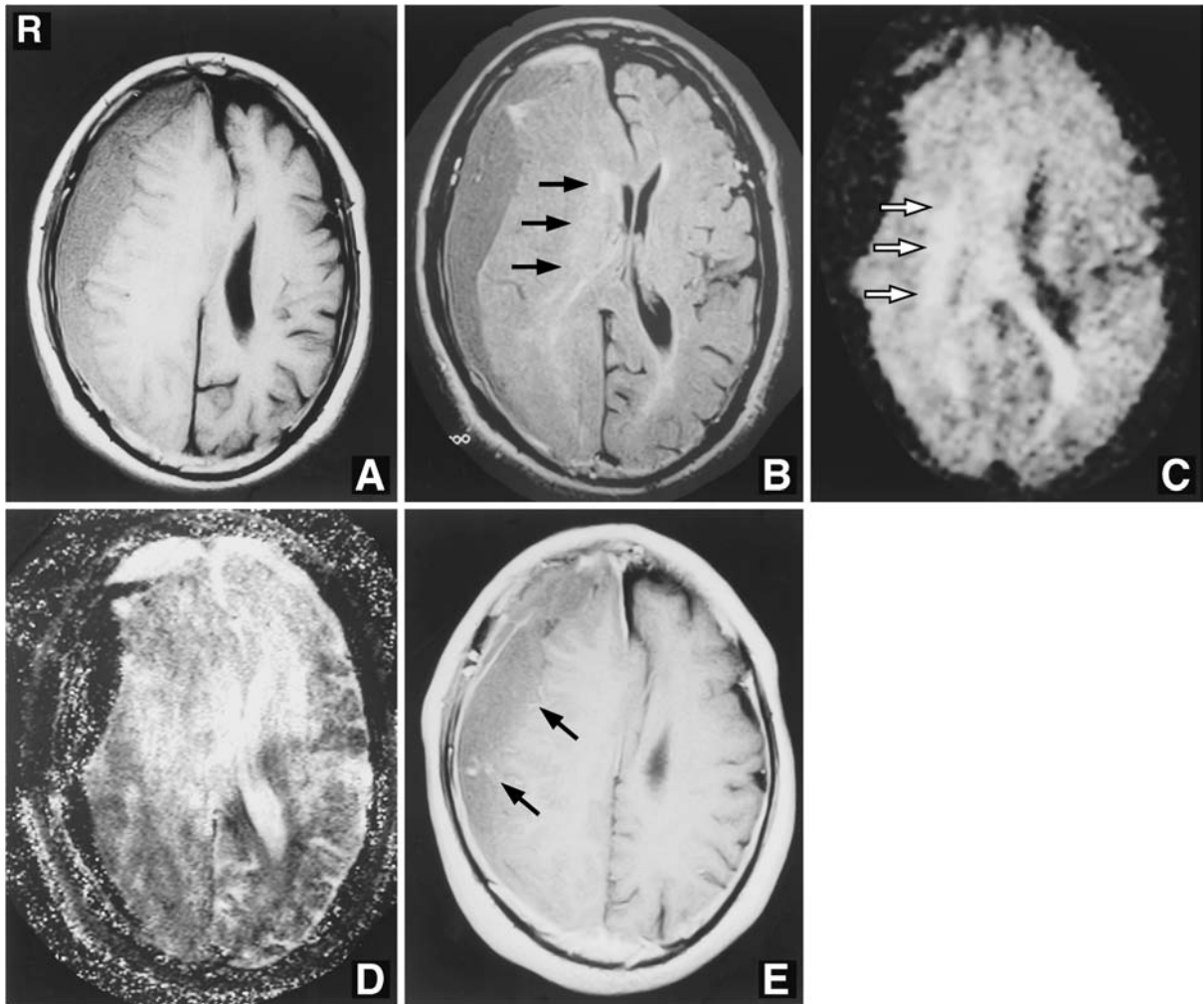
Recent advances in diffusion-weighted magnetic resonance (MR) imaging techniques allow the identification of different types of brain edema.<sup>3,4,9</sup> We describe a case of chronic subdural hematoma associated with vasogenic edema in the ipsilateral cerebral hemisphere identified by diffusion-weighted imaging and measurements of apparent diffusion coefficient (ADC) in the cerebral hemispheres.

### Case Report

An 80-year-old male with a history of hypertension fell down and struck his head on February 1, 2000. He was referred to our clinic with complaints of headache on April 6, 2000. Neurological examination on admission revealed clear consciousness and mild left hemiparesis predominantly in the lower ex-

tremities. His disorientation and left hemiparesis developed progressively and he became somnolent and could not walk independently on the day after admission.

Conventional T<sub>1</sub>-weighted (repetition time/echo time [TR/TE] = 500/15 msec) and fluid attenuation inversion recovery (FLAIR) MR imaging (TR/TE/inversion time = 6882/110/1700 msec) were performed using a 1.5-T superconducting system (MAGNEX EPIOS15; Shimazu, Kyoto), and both showed a hypointense area in the right subdural space indicating fresh blood (Fig. 1A, B). The right cerebral hemisphere was compressed to the left by the right subdural hematoma with a midline shift of 15 mm. Axial FLAIR MR imaging showed swelling of the right cerebral hemisphere and a hyperintense area in the white matter around the right lateral ventricle suggesting brain edema due to the compression (Fig. 1B). Therefore, two burrholes were made urgently under local anesthesia and about 200 ml of liquid hematoma was evacuated. The hematoma cavity encapsulated with a membrane was thor-



**Fig. 1** Preoperative magnetic resonance images. **A:** Axial  $T_1$ -weighted image (repetition time/echo time [TR/TE] = 500/15 msec) showing a hypointense area in the right subdural space indicating fresh blood. **B:** Axial fluid attenuation inversion recovery (FLAIR) image (TR/TE/inversion time = 6882/110/1700 msec) also showing a hypointense area in the right subdural space and remarkable compression of the right cerebral hemisphere with a midline shift of 15 mm. The hyperintense area in the white matter around the right lateral ventricle suggests brain edema due to the compression (arrows). **C:** Axial echo-planar diffusion-weighted image (TR/TE = 3000/155.7 msec) showing a more extensive and conspicuous hyperintense area in the right paraventricular white matter (arrows) compared to the FLAIR image. **D:** Apparent diffusion coefficient image showing more extensive hyperintense areas in the right cerebral hemisphere than the echo-planar diffusion-weighted image and hyperintense areas in both the right and left paraventricular white matter. **E:**  $T_1$ -weighted image with contrast medium showing dilated cortical blood vessels (arrows) underneath the subdural hematoma indicating increased intracranial pressure and venous congestion.

oughly irrigated with saline. His postoperative course was uneventful and his disturbed consciousness and left hemiparesis recovered completely within one week after the operation. Subdural hematoma did not recur for approximately 3 months after the operation.

To evaluate the brain edema in the right cerebral

hemisphere, the ADC value in the affected cerebral hemisphere was measured pre- and postoperatively using the diffusion-weighted images. Axial diffusion-weighted imaging using a single-shot, spin-echo, echo-planer sequence was obtained with the following parameters: TR/TE = 3000/155.7 msec; field of view (FOV)  $300 \times 150$  mm; slice thick-

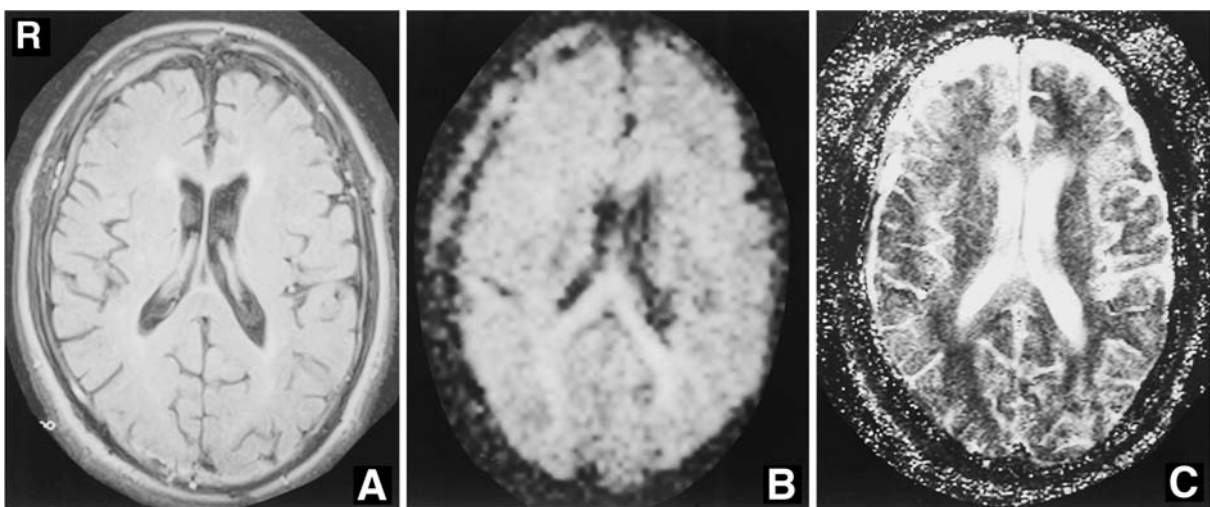
ness 7 mm with a 1.5 mm gap; matrix  $128 \times 64$  mm; motion probing gradient 12 mT/m; and diffusion-sensitizing gradient factor (b-factor), dependent on the motion probing gradient strength, gradient duration, and diffusion time, of maximum b-factor ( $b_1$ ) of  $993.8 \text{ sec/mm}^2$  and minimum b-factor ( $b_0$ ) of  $0 \text{ sec/mm}^2$ . The motion probing gradient of the echo-planar diffusion-weighted imaging was applied along the slice-selection direction, which was parallel to the body axis. Axial diffusion-weighted imaging was also conducted using a cardiac gated spin-echo sequence and the following parameters: TR/TE = 2 RR intervals on electrocardiograms/140 msec; FOV  $230 \times 200$  mm; slice thickness 7 mm with a 1.5 mm gap; matrix  $256 \times 160$  mm; motion probing gradient 10 mT/m; and  $b_1$  of  $884.8 \text{ sec/mm}^2$  and  $b_0$  of  $0 \text{ sec/mm}^2$ . The motion probing gradient of the spin-echo diffusion-weighted imaging was applied along the phase-encoding axis, which was oriented in the right-left direction on the slice. The ADC was calculated based on the Stejskal and Tanner equation,<sup>10)</sup>  $\text{ADC} = \ln(SI_1/SI_0)/(b_0 - b_1)$ , where  $SI_0$  and  $SI_1$  are the pixel signal intensities acquired from the diffusion-weighted images using a cardiac gated spin-echo sequence with  $b_0$  and  $b_1$ , respectively. ADC maps were created using this calculation on a pixel-by-pixel basis.

Preoperative axial echo-planar diffusion-weighted imaging showed a more extensive and conspicuous hyperintense area in the right paraventricular white matter compared to FLAIR imaging (Fig. 1C). Axial ADC mapping showed more extensive hyperintense

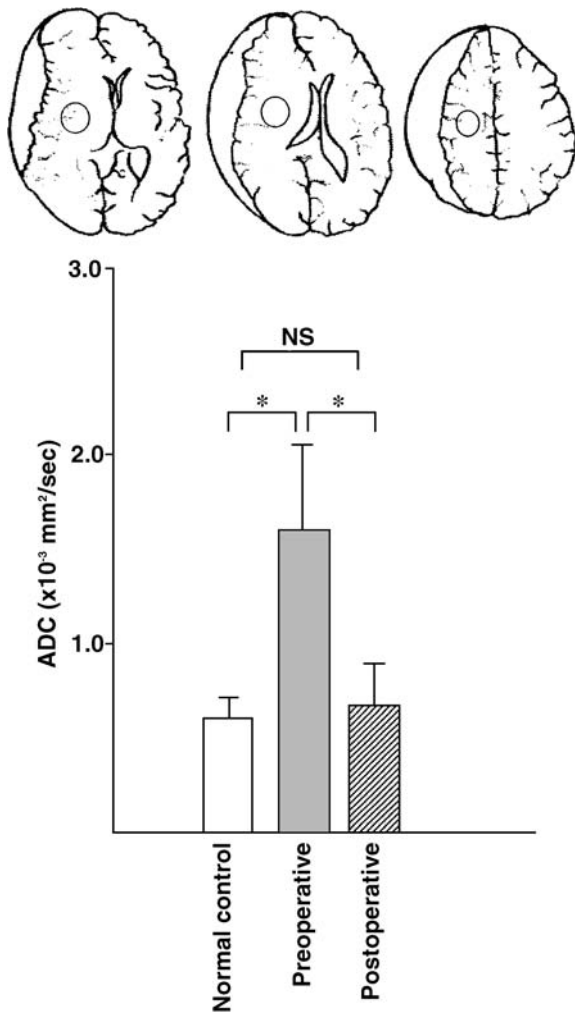
areas than the echo-planar diffusion-weighted imaging, and abnormal hyperintense areas in both the right and left paraventricular white matter (Fig. 1D). Approximately one month after the operation, the hyperintense area in the right cerebral hemisphere disappeared in both the axial FLAIR and echo-planar diffusion-weighted images (Fig. 2A, B), and axial ADC mapping did not show any abnormal hyperintense areas (Fig. 2C). The ADCs in the central area of the affected cerebrum, including the internal capsule and corona radiata corresponding to the hyperintense area on the FLAIR imaging, were measured before and one month after the operation (Fig. 3 upper). The normal ADC in the central area of the cerebral hemisphere of age-matched healthy subjects without intracranial lesions ( $n = 3$ ) was  $0.60 \pm 0.10 \times 10^{-3} \text{ mm}^2/\text{sec}$  (mean  $\pm$  standard deviation). The pre- and postoperative ADC values in the central area of the affected cerebral hemisphere were  $1.61 \pm 0.49 \times 10^{-3}$  and  $0.63 \pm 0.24 \times 10^{-3} \text{ mm}^2/\text{sec}$ , respectively. The preoperative ADC value was significantly higher than those of normal subjects ( $p < 0.05$ , Student's t test). The postoperative ADC value improved to a normal level (Fig. 3 lower).

## Discussion

Chronic subdural hematomas are slowly expanding intracranial mass lesions that can cause reversible reduction of cerebral function by compressing and displacing the brain. The clinical presentation of chronic subdural hematoma can be modified by



**Fig. 2** Postoperative magnetic resonance images. Fluid attenuation inversion recovery image (A) showing disappearance of the right subdural hematoma and hyperintense area in the white matter around the right lateral ventricle. Axial echo-planer diffusion-weighted image (B) and apparent diffusion coefficient image (C) showing a normal appearance.



**Fig. 3** upper: Illustrations showing the central regions in the right cerebral hemisphere where the apparent diffusion coefficient (ADC) was measured. The circles include the internal capsule and corona radiata corresponding to the hyperintense area on the fluid attenuation inversion recovery image, and the nerve fibers were perpendicular to the applied motion probing gradient, indicating that the measured ADC was anisotropic. lower: Graph showing the pre- and postoperative ADC values. The preoperative ADC was significantly higher than that of the normal control and the anisotropic ADC as shown in the upper figure, suggesting vasogenic edema. The postoperative ADC is normal. \* $p < 0.05$ . NS: not significant.

many factors including the volume, enlargement rate, and site of the hematoma, and the anatomical variation of individual patients. Clinical manifestations may not appear until critical levels are

reached. Subdural hematoma compressing the cerebral hemisphere often provokes certain immediate and delayed responses within the subjacent brain tissues. Focal cerebral edema just beneath the compressed brain was observed pathologically in patients with subdural hematoma who died without undergoing surgery. Regional swelling and edema are the probable pathophysiological basis for the clinical features of patients with chronic subdural hematoma.<sup>2)</sup>

Symptomatic swelling of the white matter subjacent to chronic subdural hematoma is undoubtedly rare and was recognized in only 6% of a series of patients, all of whom were comatose at the time of diagnosis.<sup>1)</sup> Swelling of the white matter found only at autopsy and in severe cases might be the final outcome of the disease process of chronic subdural hematoma rather than the actual pathogenesis of clinical symptoms.

Brain edema is an abnormal accumulation of fluid within the brain parenchyma resulting in volumetric enlargement of the brain tissue, and can be generally classified into three categories: vasogenic, cytotoxic, and interstitial edema.<sup>5)</sup> Diffusion-weighted imaging and measurements of ADC can discriminate between these different types of brain edema.<sup>4)</sup> Cytotoxic edema originates from an interruption of the energy supply leading to ionic pump failure and osmoregulation, resulting in intracellular increases in water and sodium levels. The ADC of water in cytotoxic brain edema is less anisotropic and lower than that in normal brain.<sup>4,9)</sup> In contrast to cytotoxic edema, the ADC of water in vasogenic and interstitial edema is anisotropic and greater than that in normal brain tissue.<sup>4)</sup> The edematous fluid is mainly present in the extracellular space, in which the water molecules can diffuse freely. The increased ADC in such edema is attributed to an increase in the volume fraction of the extracellular space. Furthermore, an increase in the extracellular space enlarges the intermyelin distance, leading to a reduction in the restrictive effect of myelin on the diffusion properties of extracellular water. The major difference between interstitial and vasogenic edema is that the volume of free water increases greatly in the extracellular space of the periventricular white matter in interstitial edema. The combination of the greatly decreased fraction of bound water and the increased fraction of free water results in a greater ADC in interstitial than in vasogenic edema.<sup>4)</sup>

In our case, echo-planar diffusion-weighted and FLAIR MR imaging showed a hyperintense area in the paraventricular white matter of the cerebral hemisphere ipsilateral to the subdural hematoma, and the ADC value in this area was significantly

greater than that in age-matched normal subjects. The motion probing gradient was applied in right-left direction to the body axis, so the gradient direction was perpendicular to the nerve fibers in the central area of the affected cerebral hemisphere and the measured ADC was anisotropic. There was no hydrocephalic influence on the occurrence of edema, so this brain edema with a large and anisotropic ADC was identified as vasogenic edema.

Changes in cerebral venous hemodynamics due to persistent compression on the bridging veins<sup>7)</sup> or cerebral cortical veins<sup>1)</sup> is suspected to the cause of brain edema in cases of traumatic subacute subdural hematoma associated with cerebral hemispheric swelling. Our case was also thought to have cerebral venous circulatory disturbance, because T<sub>1</sub>-weighted imaging with contrast enhancement showed dilated cortical veins beneath the chronic subdural hematoma (Fig. 1E), which might have resulted from delayed capillary and venous flow due to increased intracranial pressure and venous compression.<sup>6)</sup>

Regional cerebral blood flow (CBF) appears to be correlated with neurological deficits in cases of chronic subdural hematoma. Reduction of CBF in the putamen and thalamus on the hematoma side was observed in patients presenting with hemiparesis and reduction of CBF in the whole brain was demonstrated in patients with consciousness disturbance.<sup>8)</sup> The preoperative hyperintense areas in the bilateral cerebral hemispheres on the ADC maps in our case might be related to the reduction of CBF in the whole brain. Further investigation is necessary to clarify the mechanism of occurrence of brain edema and the abnormal diffusion of water molecules associated with chronic subdural hematoma.

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