Background: Cerebral venous thrombosis (CVT) is a cause of stroke with obscure pathophysiologic properties that differ from arterial stroke. Its main mechanisms of pathophysiology are the breakdown of the blood-brain barrier and the coexistence of cytotoxic and vasogenic edema. However, conventional magnetic resonance imaging (MRI) cannot differentiate between vasogenic and cytotoxic edema.

Objectives: To describe the diffusion-weighted imaging (DWI) findings and characterize the clinical applications of DWI in CVT.

Setting: A tertiary referral center, neurology department.

Design and Methods: From November 1998 to March 2001, 14 patients (5 men, 9 women; mean age, 43±10 years) with CVT underwent DWI, conventional MRI, MR venography, or conventional cerebral angiography. Abnormal findings on DWI and conventional MRI indicated the necessity of MR venography and conventional angiography to confirm the diagnosis of CVT. Apparent diffusion coefficient (ADC) values were measured in all of the abnormal lesions with visual inspection of DWI and T2-weighted echo planar imaging.

Results: Findings on DWI were grouped according to 3 patterns: (1) Heterogeneous signal intensity (SI) (10 patients) showed mixed bright high SI and low SI and the corresponding ADC values were inversely correlated to the DWI SI. The areas of prominent low SI on DWI were reversed with adequate treatment on follow-up MRI in 1 patient. (2) Multifocal high SI (3 patients) was similar to that observed in acute arterial stroke. The corresponding ADC values were decreased and DWI was performed in the acute stages. (3) Intravascular clot with high SI was found with (1 patient, also in heterogenous SI group) or without (1 patient) parenchymal lesions. In 1 patient, DWI demonstrated T2-negative and fluid attenuated inversion recovery–negative lesions without correlative symptoms.

Conclusions: These data suggest that DWI with ADC maps can be used to discriminate between types of edema for tissue viability and to provide information about stages and diagnostic clues in CVT.

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Diffusion-weighted imaging (DWI), first developed by Le Bihan et al., can detect changes in water diffusion associated with cellular dysfunction and can be used to detect ischemic lesions of the brain within the first few hours of stroke onset. The application of DWI in diagnosing arterial stroke is well established and has been demonstrated by numerous experimental and clinical studies to show an early decrease and late increase, or normalization, of the apparent diffusion coefficient (ADC). It has been well documented that cytotoxic edema, related to acute infarction, is characterized by markedly decreased diffusion and that increased interstitial water in vasogenic edema demonstrates increased diffusion. Conventional magnetic resonance imaging (MRI) cannot differentiate between vasogenic and cytotoxic edema.

For editorial comment see page 1538

Cerebral venous thrombosis (CVT) is a cause of stroke with obscure pathophysiologic properties that differ from arterial occlusion. Rother et al. summarized the mechanisms: (1) Increased pressure in the superior sagittal sinus results in reduced capillary perfusion pressure and increased cerebral blood volume. (2) Obstruction to venous flow leads to increased intracranial pressure and blood-brain barrier disruption, resulting in decreased cerebral blood flow. (3) The net capillary filtration increases, leading to progressive cerebral edema with intracerebral and subarachnoid hemorrhage additionally compromising the brain tissue.
METHODS

From November 1998 to March 2001, 14 consecutive patients (5 men, 9 women; mean age, 43 ± 10 years) with CVT underwent DWI, conventional MRI, MR venography (MRV), or conventional cerebral angiography. Magnetic resonance venography and conventional angiography were performed after DWI and conventional MRI findings strongly suggested CVT. Cerebral venous thrombosis was diagnosed by abnormal findings on conventional MRI (empty delta, high signal intensity [SI] in venous sinuses, and parenchymal lesions), MRV (nonvisualization of venous sinus and cerebral veins other than the asymmetry of transverse sinuses), or conventional angiography. The time taken from onset of stroke to DWI was variable (1-30 days). Detailed characteristics of patients and MRI findings are presented in Tables 1 and 2. The patients were examined using a 1.5-T MRI unit (Signa Horizon, Echospeed; General Electric Medical Systems, Milwaukee, Wis) with echo planar imaging capability. Fast-spin echo T2-weighted images (T2WI; TR/TE, 4200/112 milliseconds; field of view, 21 × 21 cm; matrix, 256 × 192; and slice thickness, 5 mm with 1.5-mm gap) were obtained. A DWI was obtained in the transverse plane using single-shot echo planar imaging (TR/TE, 6500/125 milliseconds; field of view, 24 × 24 cm; matrix, 128 × 128; slice thickness, 5 mm with 2.5-mm gap; and 2 b values, 0 and 1000 mm²/s). The diffusion gradi ents were applied along 3 axes (x, y, z) simultaneously. The ADC was calculated based on the Stejskal-Tanner equation11 as the negative slope of the linear regression line best fitting the points for b vs ln SI—the SI from the region of interest within the images acquired at each b value. Apparent diffusion coefficient maps were created by performing this calculation on a pixel-by-pixel basis. The respective ADC values are described (Figures 1, 2, 3, and 4, and Table 2). Normal ADC values of the parenchyma and white matter ranged from 0.78 to 0.91 × 10⁻³ mm²/s (Chu et al, unpublished data, 2000). Regions of interest were carefully drawn in the abnormal areas on calculated average ADC maps as well as in normal-appearing areas with variable sizes. The selection of regions of interest was made with the help of T2-weighted echo planar images obtained by the same method of acquisition as the diffusion images (ie, images generated from the diffusion sequence with diffusion sensitivity, b=0); this was to avoid errors in regions of interest selection caused by spatial distortions leading to discrepancies between diffusion images and conventional MRIs. The analyses of images and ADC values were performed by expert neuroradiologists (Kee-Hyun Chang, MD, PhD, Seoul, Korea) and neurologists (K.C. and D.-W.K.). Perfusion-weighted MRI was not performed.

There have been a few case reports on the application of DWI in patients with CVT.16,18 These reports revealed that the most striking feature of DWI findings was that reversible ADC changes (decrease16,17 or increase18) were evident during the acute period of CVT. More recently, various DWI results have been reported,19,20 such as heterogenous findings and the possibility that cytotoxic edema is a feature of CVT.

We report various DWI findings in CVT and evaluate the prognostic value of DWI. Furthermore, the mechanisms of venous stroke were characterized by analyzing ADC maps.

RESULTS

Based on the patterns of SI on DWI and ADC maps, we classified the DWI findings into 3 groups: heterogeneous SI, multifocal high SI, and high SI in clots. The characteristics of the groups are described.

HETEROGENEOUS SI GROUP (PATIENTS 1-10)

Heterogeneous SI indicated nonhemorrhagic venous infarcts (patients 1-8). Apparent diffusion coefficient maps showed normal ADC values (0.75-0.86 × 10⁻³ mm²/s) in the areas of very bright SI and elevated ADC values (1.64-1.78 × 10⁻³ mm²/s) in mildly bright SI areas on DWI (eg, patient 1, Figure 1B). Anticoagulation therapy was initiated and the patient’s symptoms and signs were completely normalized. A follow-up MRI of patient 1 was obtained 1 month later. The heterogeneous SI on DWI disappeared completely and the ADC values had returned to normal (0.72-0.78 × 10⁻³ mm²/s). A follow-up T2-weighted image depicted high SI remaining in a previously very bright lesion on the initial DWI (Figure 1D). Diffusion-weighted imaging results and ADC maps of the rest of the group (patients 2-8) showed similar findings.

In patient 9, the area of heterogeneous SI had a dark, thin rim surrounded by low SI (Figure 2). The very bright SI with a dark rim indicated a hemorrhagic signal. Apparent diffusion coefficient maps depicted decreased ADC values (0.51-0.52 × 10⁻³ mm²/s) in the very bright SI region (hemorrhagic cavity) on DWI and high ADC values (1.45 × 10⁻³ mm²/s) in the surrounding low SI region on DWI. The findings of DWI and ADC maps were similar in patient 10.

MULTIFOCAL HIGH SI GROUP (PATIENTS 11-13)

This type of DWI finding (Figure 3) would be expected for an acute arterial stroke (patient 11). The corresponding ADC values were decreased (0.53-0.59 × 10⁻³ mm²/s) on ADC maps. The values were low in all of the multiple lesions on DWI and similar to those observed for an acute arterial stroke. The diagnosis of CVT in this patient was confirmed by the visualization of high SI of the thrombus in the cerebral venous sinus on T1-weighted MRI and nonvisualization of the right transverse sinus and superior sagittal sinus on MRV. The MRI findings of patients 12 and 13 were similar. The multiple MRI lesions did not correlate with clinical symptoms and signs, such as focal neurologic deficits. After commencing anticoagulation therapy, the symptoms disappeared within a week and no follow-up MRI was obtained.

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High SIs in clots were found in the cerebral veins and sinuses (Figure 4) with (patient 7) or without (patient 14) parenchymal involvement. The corresponding ADC values of the clots were very low (0.43–0.55 × 10⁻³ mm²/s) compared with those of cerebrospinal fluid (3.05–4.75 × 10⁻³ mm²/s) in patient 14. The ADC values of the clot in patient 7 (Figure 2A and B) were also very low (0.32 × 10⁻³ mm²/s).

Table 1. Summary of Patient Characteristics

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Symptoms</th>
<th>Cause</th>
<th>Outcome</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/30</td>
<td>Headache, papilledema, confusion</td>
<td>Eclampsia</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>2/F/51</td>
<td>Right hemiparesis, global aphasia, confusion</td>
<td>Not found</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>3/F/47</td>
<td>Headache, papilledema, blurred vision, diplopia</td>
<td>Androgen therapy for aplastic anemia</td>
<td>No disability</td>
<td>None</td>
</tr>
<tr>
<td>4/M/26</td>
<td>Headache, altered mentality, seizure</td>
<td>Antithrombin III deficiency</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>5/F/52</td>
<td>Headache, altered mentality, seizure</td>
<td>Postoperative site infection (acoustic neutoma)</td>
<td>Severe disability</td>
<td>AC</td>
</tr>
<tr>
<td>6/M/42</td>
<td>Headache, altered mentality, seizure</td>
<td>Chronic alcoholism</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>7/M/30</td>
<td>Headache, seizure</td>
<td>Left mastoiditis</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>8/F/29</td>
<td>Headache, papilledema</td>
<td>Postpartum state</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>9/F/34</td>
<td>Headache, seizure, papilledema</td>
<td>Oral contraceptive</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>10/M/47</td>
<td>Headache, seizure, left hemiparesis</td>
<td>Lupus anticoagulant (+)</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>11/M/53</td>
<td>Fever, neck stiffness, altered mentality, papilledema</td>
<td>Lupus anticoagulant (+), decreased antithrombin III, decreased protein C</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>12/F/62</td>
<td>Cognitive decline, confusion, seizure</td>
<td>Protein S deficiency</td>
<td>No disability</td>
<td>AC</td>
</tr>
<tr>
<td>13/F/58</td>
<td>Quadripareisia, confusion, headache, papilledema</td>
<td>None</td>
<td>Mild disability</td>
<td>AC</td>
</tr>
<tr>
<td>14/F/45</td>
<td>Headache, papilledema</td>
<td>Myelodysplastic syndrome</td>
<td>No disability</td>
<td>AC</td>
</tr>
</tbody>
</table>

*AC indicates anticoagulation.

Table 2. Summary of MRI Findings

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Time to DWI, d</th>
<th>Site of Occlusion</th>
<th>Conventional</th>
<th>DWI</th>
<th>ADC Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/30</td>
<td>10</td>
<td>Internal cerebral veins</td>
<td>HSI in left BG, brainstem, cerebellum (T2)</td>
<td>Heterogenous SI in both BG, brainstem, cerebellum</td>
<td>↑ or →</td>
</tr>
<tr>
<td>2/F/51</td>
<td>27</td>
<td>SSS, left TS, left SS</td>
<td>HSI in left frontotemporal lobe with gyral swelling (T2); nonvisualization of SSS, left LS (T1, T2)</td>
<td>Heterogenous SI in left frontotemporal lobe</td>
<td>↑ or →</td>
</tr>
<tr>
<td>3/F/47</td>
<td>30</td>
<td>SSS, left TS</td>
<td>Left tentorial SDH, HSI in left SS, left superior sylvian vein (T1, T2)</td>
<td>Heterogenous SI in bilateral frontal lobes</td>
<td>↑</td>
</tr>
<tr>
<td>4/M/26</td>
<td>5</td>
<td>SSS</td>
<td>HSI in left parietal lobe (T2)</td>
<td>Heterogenous SI in left parietal lobe</td>
<td>↑</td>
</tr>
<tr>
<td>5/F/52</td>
<td>6</td>
<td>SSS, right TS, LS</td>
<td>HSI in left parieto-occipital lobes (T2), SSS and bilateral TS signal void (T1, T2)</td>
<td>Heterogenous SI in left parieto-occipital lobes</td>
<td>↑ or →</td>
</tr>
<tr>
<td>6/M/42</td>
<td>3</td>
<td>SSS</td>
<td>HSI in right frontal lobe (T2)</td>
<td>Heterogenous SI in right frontal lobe</td>
<td>↑ or →</td>
</tr>
<tr>
<td>7/M/30</td>
<td>15</td>
<td>SSS, both TS</td>
<td>HSI in both BG, thalamus, caudate nucleus, frontal white matter (T2), LME</td>
<td>Heterogenous SI in both BG, thalamus, caudate nucleus, frontal white matter</td>
<td>↑ or →</td>
</tr>
<tr>
<td>8/F/29</td>
<td>4</td>
<td>SSS, both TS</td>
<td>HSI in right frontal lobe and frontal white matter (T2)</td>
<td>Heterogenous SI in right frontal lobe and frontal white matter</td>
<td>↑ or →</td>
</tr>
<tr>
<td>9/F/34</td>
<td>5</td>
<td>SSS, both TS</td>
<td>Hemorrhagic infarct in right frontal lobe, empty delta, LME, HSI clot in cerebral veins (T1, T2)</td>
<td>Heterogenous SI with dark, thin rim and surrounding low SI and HSI in clot</td>
<td>↑</td>
</tr>
<tr>
<td>10/M/47</td>
<td>7</td>
<td>SSS, both TS</td>
<td>Hemorrhagic infarct in right frontal lobe</td>
<td>Heterogenous SI with dark, thin rim and surrounding low SI</td>
<td>↑</td>
</tr>
<tr>
<td>11/M/53</td>
<td>5</td>
<td>Right TS, SSS</td>
<td>Multiple HSI in left parieto-occipital lobe (T2), empty delta, HSI in right TS (T1, T2)</td>
<td>Multifocal HSI in left parieto-occipital lobe</td>
<td>↓</td>
</tr>
<tr>
<td>12/F/62</td>
<td>2</td>
<td>Left TS, SSS</td>
<td>Multiple HSI in left temporo-occipital lobe (T2) with gyral swelling, left TS signal void (T1, T2)</td>
<td>Multifocal HSI in left temporo-occipital lobe</td>
<td>↓</td>
</tr>
<tr>
<td>13/F/58</td>
<td>1</td>
<td>SSS</td>
<td>Multiple HSI in both frontoparietal lobes (T2) with gyral swelling</td>
<td>Multifocal HSI in both frontoparietal lobes</td>
<td>↓</td>
</tr>
<tr>
<td>14/F/45</td>
<td>20</td>
<td>Right TS, SS</td>
<td>HSI in right TS, SS (T1, T2), LME, gyral swelling in right parieto-occipital lobe</td>
<td>HSI in clot only</td>
<td>↓</td>
</tr>
</tbody>
</table>

*MRI indicates magnetic resonance imaging; DWI, diffusion-weighted imaging; ADC, apparent diffusion coefficient; HSI, high signal intensity; BG, basal ganglia; ↑, increased; →, normal; ↓, decreased; SSS, superior sagittal sinus; TS, transverse sinus; SS, sigmoid sinus; LS, lateral sinus; SDH, subdural hemorrhage; and LME, leptomeningeal enhancement.

HIGH SI IN CLOTS GROUP (PATIENTS 7 AND 14)

High SIs in clots were found in the cerebral veins and sinuses (Figure 4) with (patient 7) or without (patient 14) parenchymal involvement. The corresponding ADC values of the clots were very low (0.43-0.55 × 10⁻³ mm²/s) compared with those of cerebrospinal fluid (3.05-4.75 × 10⁻³ mm²/s) in patient 14. The ADC values of the clot in patient 7 (Figure 2A and B) were also very low (0.32 × 10⁻³ mm²/s).
Findings of CVT on DWI have several distinctive aspects compared with those of arterial stroke. We observed 3 patterns in the DWI findings: heterogeneous SI, multifocal high SI, and high SI in clots. Conventional MRI images (T2 and fluid-attenuated inversion recovery) depicted similarly high SIs for the areas of venous congestion and infarct and cannot be used to differentiate between the types of edema.

HETEROGENOUS SI IN NONHEMORRHAGIC VENOUS INFARCT

The heterogenous SI group included nonhemorrhagic and hemorrhagic venous infarctions. Nonhemorrhagic venous infarctions (patients 1-8) were indicated by the combination of cytotoxic and vasogenic edema on DWI, and the SI was inversely correlated with the correspond-

ing ADC value. The experimental CVT study reported an early decrease (<48 h), and late increase (>48 h) in ADC values, demonstrating that early cytotoxic edema preceded late vasogenic edema. Corvol et al16 and Manzione et al17 reported that rapid MRI scanning during the acute period (within 24 hours) revealed decreased ADC, which was later reversed by suitable treatment. Keller et al18 reported that DWI findings, obtained 1 day after the onset of symptoms, revealed high ADC values, which suggested the existence of a predominantly vasogenic edema. Their DWI findings correlated well with a previous experimental study.15 Diffusion-weighted image results and ADC maps in this group, performed at the early to late subacute stages (3-30 days), showed normal to increased ADC values.

Recently, heterogenous SI on DWI was reported.19,20 It was observed that ADC values decreased up to 16% in the acute stage and increased or were normalized within 4 days. In 1 patient in the series,19 the ADC values were

Figure 1. Patient 1. A, Initial diffusion-weighted image (DWI), performed 10 days after onset of stroke, shows heterogenous signal intensity (SI) in left basal ganglia and high SI in the right basal ganglia. B, Apparent diffusion coefficient map indicates normal values (0.85-0.86 × 10−3 mm²/s) in the area of very bright SI on DWI (solid arrows), and elevated values (1.74-1.78 × 10−3 mm²/s) in the area of mildly bright SI on DWI (dotted arrows). C, Initial T2-weighted image shows high SI in the bilateral basal ganglia and left caudate intracerebral hemorrhage and intraventricular hemorrhage. D, Follow-up T2-weighted image, performed 1 month later, demonstrates remnant high SI in the previously very bright area on DWI and otherwise normal findings.
were very low (0.3-0.4 \(\times 10^{-3}\text{mm}^2/\text{s}\)) and the abnormal region turned out to be hemorrhagic. Most of the high ADC region disappeared on follow-up images.

In our study, DWI in the heterogeneous SI group depicted variable and high SI, very bright, high SI in the normal ADC regions, and high SI in the high ADC regions. The high SI despite the normal to high ADC values was probably owing to a T2 shine-through effect. As with T1- and T2-weighted images, DWI is not a pure map of ADC but contains mixed contributions from spin density and T2 effects. Because of these shine-through effects, the DWI should be interpreted with reference to ADC maps. The diagnostic information provided by DWI and ADC maps is not identical.

Figure 2. Patient 9. A, Diffusion-weighted image (DWI), performed 15 days after onset, indicates high signal intensity (SI) in the right frontal lobe (dotted arrow) with surrounding low SI (solid arrow). High SI of the clot in superior sagittal sinus is also seen (dashed arrow). B, Apparent diffusion coefficient (ADC) map indicates decreased ADC values (0.51-0.52 \(\times 10^{-3}\text{mm}^2/\text{s}\)) in very bright SI region on DWI and high ADC values (1.45 \(\times 10^{-3}\text{mm}^2/\text{s}\)) in the surrounding low SI region on DWI. The ADC values of the clot are decreased (0.32 \(\times 10^{-3}\text{mm}^2/\text{s}\)). C, T1-weighted sagittal image shows high SI in the superior sagittal sinus. D, T2-weighted magnetic resonance image shows high SI in right frontal lobe.

The ability to differentiate between the different types of edema is important because of their relationship with tissue viability. Follow-up DWI and conventional MRI results in patient 1 depicted an almost complete disappearance of the multiple lesions in the bilateral basal ganglia, brainstem, and cerebellum, which had previously shown high ADC values at the onset of clinical improvement. The multiple high ADC lesions were caused by disruption of the blood-brain barrier and were not necessarily associated with cellular damage. Preservation of neuronal tissue was also documented by Hsu et al using proton MR spectroscopy in a case of deep CVT. The authors suggested that although the tissue was impaired, it was still viable.
HETEROGENOUS SI OF HEMORRHAGIC VENOUS INFARCT

The heterogenous SI group had an additional factor: SI attributed to hemorrhage (patients 7, 8). The bright SI of the hemorrhagic clot on DWI was owing to the paramagnetic effect of the intracellular methemoglobin, and the surrounding low SI with high ADC values was probably caused by vasogenic edema. Between these, a thin rim of low SI was observed, suggesting the occurrence of hemosiderin. Diffusion-weighted imaging and ADC measurement of intracranial hematoma were recently reported by Atlas et al. The authors suggested that the determining factors of ADC values in hematoma may be the paramagnetic effects of the methemoglobin rather than restriction of water movement. Apparent diffusion coefficient values suddenly rise in the late subacute stage during which the red blood cells start to lyse and the intracellular methemoglobin switches to the extracellular state. Our DWI findings and ADC values of patients 9 and 10 reflected those findings.

MULTIFOCAL HIGH SI OF VENOUS INFARCT

The second type of DWI abnormality was manifested as multifocal high SI (patients 11-13). The ADC values of the lesions were as low as those observed for arterial stroke. Cerebral venous thrombosis was diagnosed by the abnormalities observed with conventional MRI and MRV. Time interval from onset to DWI was rather short (5 days in patient 11, 2 days in patient 12, 1 day in patient 13); DWI findings of this group may represent the acute stages. Forbes et al reported the initial ADC decrease in patients with CVT and the ADC decrease returned to normal or increased within 4 days. Anticoagulation was immediately initiated after the MRI results were obtained.

Figure 3. Patient 11. A and B, Diffusion-weighted images show multiple high signal intensity (SI) lesions in the left hemisphere and their corresponding apparent diffusion coefficient values are 0.53-0.59 × 10^-3mm^2/s (arrows). C, Initial fluid-attenuated inversion recovery image shows high SI in the left parieto-occipital lobes. D, Initial magnetic resonance venograph, performed 12 days after onset, shows filling defects in superior sagittal sinus (arrow).
The clinical symptoms disappeared completely after the anticoagulation therapy.

The mechanisms of multifocal high SI on DWI are not well known. However, Manzione et al\textsuperscript{17} reported a similar reversible ADC decrease in multiple high SI lesions on DWI. They suggested that a variety of different mechanisms in diffusion abnormalities in CVT probably occur, resulting in a local increase in transepidermal and interstitial pressure rather than failure of the tissue energy state.\textsuperscript{17} The high SIs on DWI and low ADC values do not always indicate irreversibility but rather the presence of tissue at risk. Dardzinski et al\textsuperscript{28} reported progressive ADC changes during a period after permanent middle cerebral artery occlusion and suggested the ranges of ADC values as follows: (1) \(<0.45 \times 10^{-3} \text{mm}^2/\text{s}\), severe ischemia and irreversible damage will occur; (2) \(>0.55 \times 10^{-3} \text{mm}^2/\text{s}\), infarction will not occur; (3) \(0.45-0.55 \times 10^{-3} \text{mm}^2/\text{s}\), potentially reversible. Our results (ADC values, \(0.53-0.59 \times 10^{-3} \text{mm}^2/\text{s}\)) correspond well to the previous reports. With adequate treatment, DWI abnormalities with low ADC values may be reversible in CVT as with the cells in ischemic penumbra.

### SI OF CLOTS IN SINUSES

Diffusion-weighted imaging also showed high SI of the intravascular clots, which has not previously been reported (patients 7 and 14). The corresponding ADC values of the intravascular clots were observed to decrease (0.43-0.55, \(10^{-3} \text{mm}^2/\text{s}\)), which means that the high SI of the clot could not be attributed to T2 shine-through effects. Our report is the first to our knowledge to describe the DWI findings of an intravascular clot at the early subacute stage with an analysis of corresponding ADC values. We suggest that the SI of the clot in DWI was owing to the paramagnetic effect of the clot (intracellular methemoglobin). Furthermore, the DWI of patient 11 depicted abnormality in the clot SI alone without any visible parenchymal lesion.

The appearances of clots on conventional MRIs and the variable stages associated therewith have been well described by Bianchi et al.\textsuperscript{26} The T1, T2, and fluid-attenuated inversion recovery SIs were also high and our findings are similar to the early subacute stage thrombus described by Bianchi et al.\textsuperscript{26} Intraluminal clots evolve at stages similar to parenchymatous hematomas depending on the paramagnetic effects of hemoglobin degradation products.\textsuperscript{29,30} The evolution of the MRI signal from parenchymal hematomas does not differ markedly from venous thrombosis. However, eventually hemorrhage will show a peripheral rim of low SI on T1-weighted, T2-weighted, and DWI results, which is characteristic of hemosiderin. This feature is absent in the venous sinuses, which are not able to accumulate macrophages containing hemosiderin.\textsuperscript{25,26} At the subacute stage, the clots first become hyperintense on T1 with persisting low SI on T2 images and then appear hyperintense on both T1 and T2 images.\textsuperscript{26} Atlas et al\textsuperscript{27} reported the ADC changes during the evolution of the hematoma. They suggested that the potential mechanisms of the ADC decrease in early hematoma may be summarized as follows: (1) a shrinkage of extracellular space with clot retraction; (2) a change in osmotic environment; (3) conformational changes of the hemoglobin macromolecule within the RBC; and (4) contraction of intact red blood cells.\textsuperscript{27}

### DETECTION OF THE SUBCLINICAL ABNORMALITIES

Diffusion-weighted imaging findings in patient 3 showed T2-negative mixed SI in the right high frontal lobe. The location of the lesion also correlated well with the thrombosed cortical veins and sinuses (high SI on T1- and T2-weighted MRIs). However, the patient exhibited no specific symptoms. The DWI findings were thus able to resolve subtle or preclinical venous congestion before visible lesions appeared on conventional MRI and clinical symptoms became apparent. This case was reported previously elsewhere\textsuperscript{29} and Manzione et al\textsuperscript{17} also reported similar findings.

There were some limitations to our study. First, the number of patients was too small from which to extrapolate our findings, especially for the grouping attempt. Second, the time from the onset of disease to DWI was variable and not homogenous. This can be attributed to the diverse clinical manifestations of CVT. It is well known...
that the course of the disease in humans can progress over days and weeks.\textsuperscript{31-33} We were therefore unable to perform DWI in all of the patients immediately after the onset of disease as in the previous experimental study.\textsuperscript{10} This may reflect the more gradual stepwise progression of CVT that occurs naturally in humans compared with the complete and rapid thrombosis induced in animal studies. Third, the relative lack of experimental evidence and the previous experience with DWI in CVT may not fully support our findings.

Despite these limitations, this article is the first to describe the various DWI findings in patients with CVT in various stages. It may be difficult to diagnose CVT by conventional MRI findings alone. To confirm the diagnosis, further tests, such as MRV or conventional angiography, may be necessary. Diffusion-weighted imaging can be used to discriminate between different types of edema, assess tissue viability, detect subclinical abnormalities, deliver time-saving information for early diagnosis, and facilitate basic imaging research of the pathophysiology of CVT.

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REFERENCES