

Case Reports

Diffusion-Weighted Magnetic Resonance Imaging in a Case of Cerebral Venous Thrombosis

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Background—Diffusion-weighted imaging (DWI) is the most sensitive MR sequence in acute arterial ischemic stroke but has not yet been evaluated in venous cerebral ischemia. We describe a patient with DWI performed at the acute phase of a venous ischemic stroke.

Case Description—A rapid cerebral MRI including DWI and fast fluid-attenuated inversion recovery (FLAIR) sequences was performed at the acute phase of a venous stroke confirmed by conventional angiography. DWI showed a slight decrease in apparent diffusion coefficient values 3 hours after onset ($0.53 \pm 0.07 \times 10^{-3} \text{ mm}^2/\text{s}$) and was normal 48 hours later ($0.064 \pm 0.15 \times 10^{-3} \text{ mm}^2/\text{s}$). Fast FLAIR sequences showed large left frontoparietal hyperintensities. The lack of a clear decrease in apparent diffusion coefficient values associated with marked FLAIR abnormalities may suggest prominent or early associated vasogenic edema. Physiopathological differences between arterial and venous ischemia may explain the different type of DWI FLAIR abnormalities during the acute phase as well as the better recovery of neurological deficit in venous stroke than in arterial ischemic stroke.

Conclusions—In the context of an acute stroke, the contrast between marked FLAIR and subtle DWI abnormalities on MRI may reflect the venous mechanism of cerebral ischemia. (*Stroke*. 1998;29:2649-2652.)

Key Words: cerebral veins ■ magnetic resonance imaging ■ magnetic resonance imaging, diffusion-weighted

The arterial origin of an ischemic stroke must be confirmed at the acute phase because thrombolysis indication depends on an early diagnosis and is proposed within 3 hours after onset.¹ Although progress in neuroimaging makes the diagnosis of cerebral venous thrombosis (CVT) easier,²⁻⁴ the acute phase of a venous stroke may be difficult to differentiate from arterial ischemic stroke (AIS) because of the polymorphism of clinical presentation,^{2,5} which sometimes mimics an AIS. Diffusion-weighted imaging (DWI) is a new MR technique that shows early abnormalities within minutes after stroke onset, related to early cytotoxic edema in AIS.⁶⁻¹⁰ DWI performed at the acute phase of CVT has not yet been evaluated.

We describe a patient with CVT whose initial clinical presentation suggested AIS. The early DWI pattern was unusual for AIS, and the venous mechanism of ischemia was confirmed by angiography. The MR data are suggestive of prominent vasogenic edema, which may be the basis for the pathophysiology of venous ischemia.

Case Report

A 64-year-old woman was admitted to the hospital 2 hours and 30 minutes after the sudden onset of right hemiplegia and nonfluent aphasia. On admission, this right-handed woman

had right hemiplegia and hypoesthesia and nonfluent aphasia. She was afebrile, and blood pressure was 140/80 mm Hg with regular pulse of 70/min. CT scan performed 2 hours and 50 minutes after onset was normal. Three hours after deficit onset, a rapid MRI was obtained with a total acquisition time of only 5 minutes. Two sequences were performed on 1.5-T MR equipment (GE Signa Horizon Echospeed). The first sequence featured multislice single-shot, spin-echo diffusion, echo-planar imaging with a pair of diffusion gradients centered around the 180° pulse with 6-mm axial slice thickness, 1.5-mm gap, 96×64 matrix, 28×21-cm field of view, repetition time of 4000 ms, and effective echo time of 120 ms. Five sets of 17 slices were acquired with 5 values of b ($\delta=25$ ms, $\Delta=28$ ms), from $b=0$ to 800 s/mm², with diffusion gradients applied simultaneously in 3 orthogonal spatial directions. The DW raw images were transferred to an independent workstation. Dedicated software (Functool, GE) allowed the quantification of the diffusion changes. Calculation of the apparent diffusion coefficient (ADC) was based on the Stejskal and Tanner equation¹¹ as the negative slope of the linear regression best fitting the point for b versus $\ln(SI)$, where SI is the signal intensity from a region of interest of the images acquired at each b value. Performing this calculation on a pixel-by-pixel basis created maps of ADC. The second

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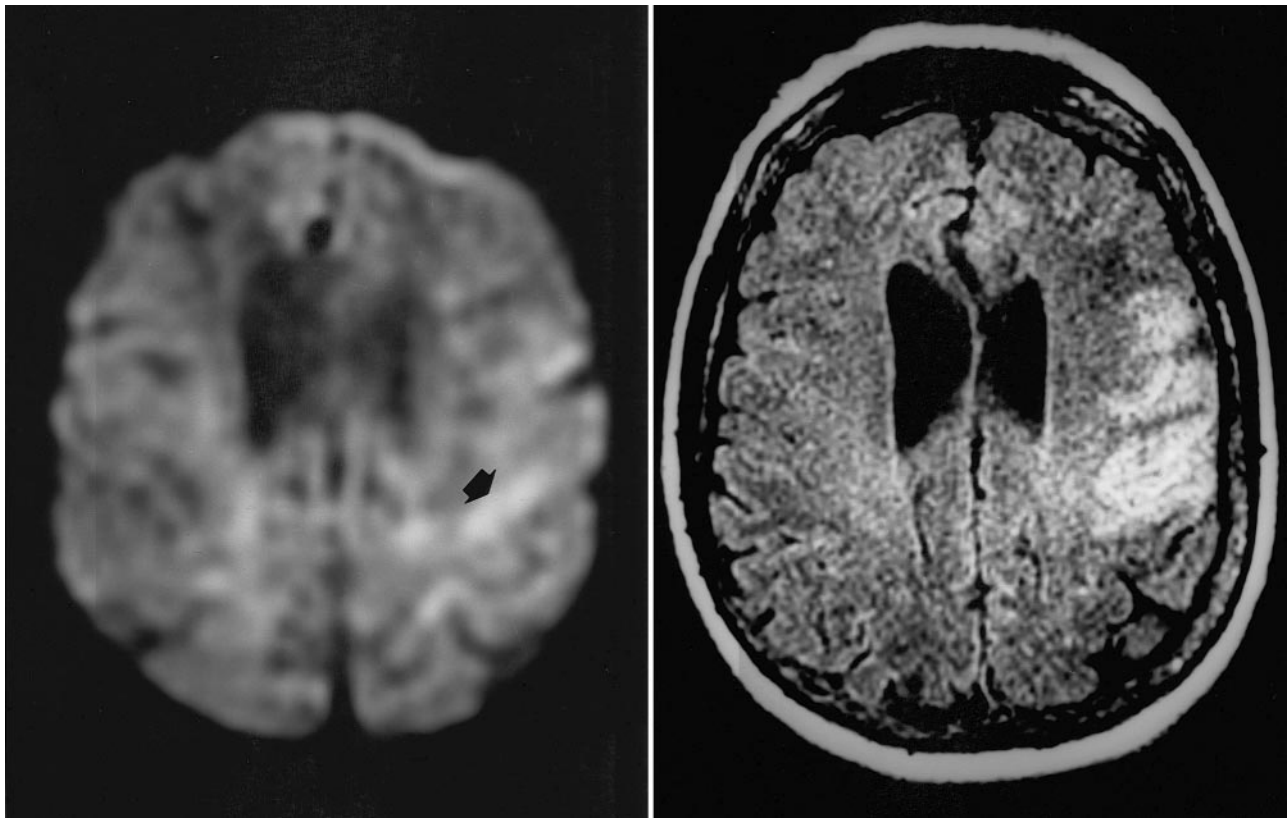


Figure 1. Left, Axial cerebral DWI ($b=800$ s/mm²) obtained 3 hours after onset shows a barely visible hypersignal limited to the left rolandic area (arrow). There is a corresponding slight decrease of ADC values ($0.53 \pm 0.07 \times 10^{-3}$ mm²/s) on the ADC maps compared with a matching location in the uninvolved contralateral hemisphere ($0.61 \pm 0.09 \times 10^{-3}$ mm²/s). Right, Axial cerebral fast FLAIR sequences obtained on the same date at the same level show large left frontoparietal hyperintensities. Note the contrast between subtle abnormalities in DWI and marked fast FLAIR abnormalities.

sequence featured fast fluid-attenuated inversion recovery (FLAIR) acquisition with 5-mm axial interleaved slices, 256×256 matrix, 24×24-cm field of view, repetition time of 10 002 ms, effective echo time of 148 ms, inversion time of 2200 ms, and 32-kHz bandwidth; direction of the frequency encoding gradient was superior to inferior.

DWI showed a discrete hypersignal limited to the left rolandic area in only 1 slice (5 mm). There was a corresponding very slight decrease of ADC values on the ADC maps ($0.53 \pm 0.07 \times 10^{-3}$ mm²/s) compared with a matching location in the uninvolved contralateral hemisphere ($0.61 \pm 0.09 \times 10^{-3}$ mm²/s) (Figure 1, left panel). This slight decrease of ADC values contrasted with the clear ADC drop usually observed at the acute phase of AIS. Conversely, the fast FLAIR sequences showed large left frontoparietal hyperintensities visible on 7 slices (35 mm) (Figure 1, right panel).

The patient had a generalized seizure just after the MRI was performed. She was therefore rejected for thrombolysis, and a second MRI was performed 48 hours later. The hypersignal on DWI completely disappeared, and ADC values returned to normal ($0.64 \pm 0.15 \times 10^{-3}$ mm²/s) compared with the uninvolved contralateral hemisphere ($0.63 \pm 0.19 \times 10^{-3}$ mm²/s) (Figure 2). Signal abnormalities on FLAIR sequences were unchanged.

These results, further supported by the generalized seizure, were unusual for AIS and led us to suspect a venous stroke. This was confirmed by MRI angiography and conventional

angiography, which showed extensive thrombosis of the superior sagittal and left lateral sinuses (Figure 3). D-dimer values were >1000 ng/mL (enzyme-linked immunosorbent assay method). The patient had a hemoglobin concentration of 61 g/L, with anemia related to a previously unknown gastric ulcer. Platelets and leukocytes were normal; coagulation tests, including normal levels of protein C, protein S, and antithrombin III, were normal; and antinuclear and antiphospholipid antibodies were negative. The patient's family later reported a history of 8 days of continuous headaches preceding the stroke. She was treated with intravenous heparin therapy (adjusted according to partial thromboplastin time 2 to 3 times the partial thromboplastin time before administration of heparin), phenytoin 300 mg/d per os, omeprazole 20 mg/d, and eradication of *Helicobacter pylori*. The patient's outcome was favorable, with marked regression of neurological deficit within 3 days. She was discharged 3 weeks later with only a moderate right ataxia.

Discussion

The context of a sudden neurological deficit with a normal initial CT scan was compatible, in this patient, with AIS in the left superficial middle cerebral artery territory. Initially, this patient was considered to have AIS and was admitted to our stroke center for possible inclusion in a thrombolysis protocol (European Cooperative Acute Stroke Study II).

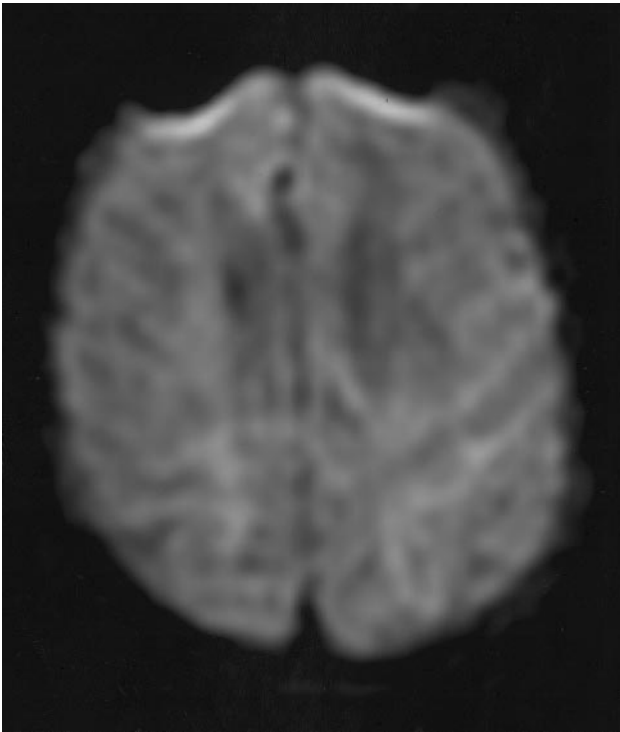


Figure 2. Axial cerebral DWI ($b=800$ s/mm²) obtained 48 hours after onset shows the disappearance of the left rolandic hyper-signal. ADC value returned to normal ($0.64 \pm 0.15 \times 10^{-3}$ mm²/s) on the ADC maps compared with a matching location in the uninvolved hemisphere ($0.63 \pm 0.19 \times 10^{-3}$ mm²/s).

A 5-minute MRI including DWI and a fast FLAIR sequence was obtained. DWI, first developed by Le Bihan et al,⁶ is a new MR technique based on the molecular motion of water. It is extremely sensitive at the acute phase of AIS since abnormalities appear within minutes after onset in animals,¹² related to slower diffusion motion in intracellular cytotoxic edema induced by ischemia, resulting in a drop of ADC values.^{8–10} In our patient, the slight decrease of ADC values observed on DWI 3 hours after onset (Figure 1, left panel)



Figure 3. Cerebral digitalized cerebral angiography. Lateral projection shows thrombosis of the superior sagittal sinus (arrows) with flow inversion in cortical veins (arrowheads).

was inconsistent with the clear ADC drop usually observed in AIS.¹³ Moreover, ADC values returned to normal at 48 hours after onset (Figure 2), while they should reach minimum values in AIS.⁹

FLAIR sequences, developed by Rydberg et al¹⁴ allow the acquisition of heavily T2-weighted, cerebrospinal fluid-nulled images. These sequences are highly sensitive to ischemic strokes,^{15,16} but, as shown for conventional T2 sequence,¹⁷ FLAIR abnormalities may be delayed compared with DWI abnormalities in AIS. However, the hyperintensities were marked and larger than on DWI only 3 hours after the sudden clinical deficit, suggesting preexisting but clinically asymptomatic focal abnormalities. Overall, these data were compatible with a venous ischemic stroke developed on a preexisting cortical venous and superior sagittal sinuses thrombosis, as confirmed later by angiography.

The venous strokes share some common features with other types of focal ischemia, such as a reduced capillary perfusion pressure,^{18,19} increased cerebral blood volume,²⁰ and decreased cerebral blood flow.²¹ However, venous ischemia also has specific characteristics, such as an early blood-brain barrier disruption and an increase of the net capillary filtration, that may result in early vasogenic edema.^{19,24} In our patient, the contrast between slight DWI abnormalities and marked FLAIR abnormalities may reflect prominent and early vasogenic edema associated with mild cytotoxic edema. This contrast may explain the excellent and rapid recovery of neurological deficit of most cases of venous stroke that is treated early.²² A relative preservation of neuronal tissue has also been documented recently by Hsu et al,²³ using proton MR spectroscopy in a case of deep CVT. The authors suggested an impaired but viable tissue in that case.

Another argument for an important vasogenic rather than a pure cytotoxic component of the edema in venous ischemia is derived from the experimental rat model of CVT described by Röther et al.²⁴ Although a strict comparison is difficult to make, the authors, who also used DWI, showed an early marked decrease of ADC values, followed by an increase during 48 hours. They concluded that early cytotoxic edema was closely followed by vasogenic edema.

To our knowledge, this is the first reported case of early DWI after onset of venous stroke in humans. Although the diversity of CVT patterns of presentation precludes definitive conclusions from a single case, the present findings clearly show that a 5-minute MRI protocol, which can easily be performed within the therapeutic window of cerebral ischemia, may greatly improve the accuracy of diagnosis in acute stroke. Moreover, our results suggest that new MR techniques may improve knowledge of the pathophysiology of cerebrovascular disorders.

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