Diffusion- and Perfusion-Weighted Magnetic Resonance Imaging in Deep Cerebral Venous Thrombosis

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Background—Diffusion-weighted (DWI) and perfusion-weighted (PI) MRI are highly sensitive techniques for early diagnosis of arterial infarction, but little data on venous cerebral ischemia are available. We describe a case in which DWI, PI, and fast T2–weighted sequences were performed in the acute phase of deep cerebral venous thrombosis (CVT).

Case Description—An 11-year-old girl with Crohn’s disease developed deep CVT in which extensive edema was shown in the deep gray matter on T2-weighted sequence images. Isotropic echo-planar DWI demonstrated a local augmentation of the apparent diffusion coefficient (1.1 to 1.6 $\times 10^{-3}$ mm$^2$/s), consistent with vasogenic edema. In dynamic contrast–enhanced PI, the regional cerebral blood volume was increased and the passage time of the contrast bolus was markedly prolonged. Clinically, the patient recovered totally after intravenous full-dose heparinization. T2 abnormalities, apparent diffusion coefficient values (0.8 to 0.92 $\times 10^{-3}$ mm$^2$/s), and brain perfusion alterations resolved without damage to brain tissue.

Conclusions—Unlike arterial infarction, DWI demonstrated vasogenic edema in a patient with deep CVT, which proved to be reversible in follow-up magnetic resonance imaging. PI showed areas with extensive venous congestion, but perfusion deficits were missing. Therefore, we believe that DWI and PI may play a role in detecting venous congestion in CVT and in prospective differentiation of vasogenic edema and venous infarction. (Stroke. 1999;30:1144-1146.)

Key Words: cerebral veins ■ diagnosis ■ diffusion ■ magnetic resonance imaging ■ perfusion ■ thrombosis

Thrombosis of the deep cerebral venous system (CVT) is a rare and sometimes critical disease, especially in untreated patients.1,2 Routine MRI and MR angiography are able to establish the diagnosis by demonstrating a lack of flow in the cerebral veins and typical T2 hyperintense signal abnormalities in the deep gray and adjacent white matter.3 However, conventional MRI sequences cannot differentiate between cytotoxic and vasogenic edema or determine the degree of venous congestion of the affected brain tissue. The purpose of this report was to evaluate the prognostic value of echo-planar diffusion-weighted (DWI) and perfusion-weighted (PI) MRI in a patient with deep CVT and extensive brain edema.

Case Report
An 11-year-old girl with Crohn’s disease was admitted to hospital, suffering from anemia and with a level of hemoglobin of 5.2 g/dL. She was successfully treated with erythrocyte transfusions, with no complications. The anemia was caused by sanguineous stools 10 times daily. The agent Clostridium difficile was found in stool samples, which led to treatment of the patient with intravenous antibiotics. At the end of the therapy, the patient suddenly developed headache and confusion. After the patient experienced further deterioration of consciousness over the course of 1 day, a CT scan was administered.

The initial CT examination (Figure, panel A) showed hypodense areas in the left basal ganglia and both thalami, with obscuration of the borders between lenticular nucleus, thalami, and internal capsules. High density was present in the internal cerebral veins and the straight sinus, which indicated possible deep CVT.

Immediately after the CT examination was completed, the diagnosis was confirmed by use of MRI and MR angiography. Additionally, DWI showed an augmentation of apparent diffusion coefficient (ADC) values, consistent with vasogenic edema. In PI, the regional cerebral blood volume (rCBV) was elevated in the affected areas and the timing of the bolus passage was prolonged, which reflected venous congestion. No deficits were visible in the rCBV maps.

After the diagnostic procedures, the patient was treated with intravenous full-dose heparinization by doubling the activated partial thromboplastin time. Total clinical recovery occurred within 2 weeks. MR follow-up examination after 2 weeks and 7 months demonstrated venous recanalization and no parenchymal defects.

Examination Technique
The MR examination was performed with a 1.5-T system (Gyrosan ACS-NT, compact plus; maximal gradient strength, 23 mT/m; rise time, 0.2 ms; maximal slew rate, 115 T/s, Philips Medical Systems) capable of echo-planar imag-
ing (EPI). A fast T2-weighted gradient and spin-echo sequence combining turbo spin-echo technique and echo-planar imaging was used, with the following parameters: TR, 3520 ms; TE, 90 ms; echo-train length, 10; EPI factor, 3; field of view (FOV), 20×22 cm; matrix, 226×256; slice thickness, 5 mm with 0.5-mm gap; and 2 NSA; 56 s (Figure, panel B). Then, phase-contrast MR angiography (3D PCA, coronal; TR, 11 ms; TE, 5.8 ms; flip angle, 20°; VS, 15 cm/s; FOV, 20×23 cm; matrix, 245×256; slice thickness, 4 mm with 2-mm overlap; and 1 NSA; sagittal maximum intensity projection) (Figure, panel C) showed the missing signal in the deep cerebral veins and the straight sinus.

DWI was performed with a finger pulse–triggered multishot EPI sequence (TR, 828 ms; TE, 105 ms; EPI factor, 15; FOV, 16×23 cm; 12 slices with 6-mm slice thickness; matrix, 100×128; and 4 NSA) with integrated navigator echo in the phase-encoding direction for motion corrections (Figure, panel D). The diffusion gradients were applied in 3 orthogonal directions with 3 increasing b values (0.250 100 0 s/mm²). The diffusion coefficients could be calculated on a pixel-by-pixel basis with the implemented software and displayed as ADC maps (Figure, panel E). The ADC values were determined from the ADC maps by 4 measurements in regions of interest containing at least 50 pixels. In the affected tissue, mean ADCs ranged from 1.1 to 1.6×10⁻³ mm²/s, and in the surrounding subcortical white matter, from 0.68 to 0.8×10⁻³ mm²/s. In the follow-up examination, ADC in the deep gray matter reached normal values (0.8 to 0.92×10⁻³ mm²/s).

For susceptibility-based PI, the transitory signal loss during the bolus passage was detected with a T2*-weighted FFE-EPI sequence (TR, 267 ms; TE, 30 ms; 6 slices with 6-mm slice
DWI and PI in Deep Cerebral Venous Thrombosis

Thrombosis of the deep venous system has a variable and sometimes poor prognosis. A retrospective review in the literature of 49 patients demonstrated a mortality rate of 13% for patients treated with either intravenous heparin or local thrombolysis, compared with 48% in untreated patients. The reason for this critical situation is that the Galenic system, which drains the midbrain, thalamus, basal ganglia, and adjacent white matter, has only 1 vein as its venous outflow and no substantial collateral drainage. Pathological findings in CVT vary from blood-brain barrier disruption that results in vasogenic edema to obstructive hydrocephalus, perivascular hemorrhage, and ischemic venous infarction with reduced capillary perfusion pressure, which leads to irreversible cytotoxic edema.

In our case of deep CVT with rapidly declining state of consciousness, early diagnosis was established by CT, MRI, and MR angiography. The intraluminal thrombus was best visible in CT, whereas the hyperacute thrombus could not be detected on MRI because of susceptibility effects. Extensive brain edema in the deep gray matter resulted in hyperintense T2 abnormalities, but conventional MRI does not allow a prospective differentiation between vasogenic and cytotoxic edema.

As a recent technique, DWI has shown the potential to detect an extensive diminution of the ADC that is based on a marked restriction of free proton diffusion in cytotoxic edema of acute arterial infarction. Cytotoxic edema occurs after energy failure of the cell with depolarization of the cell membrane (loss of Na+/K+ pump activity) and influx of ions and water into the cell, which quickly leads to irreversibly damaged brain tissue (infarction). In contrast, vasogenic edema develops with disruption of the blood-brain barrier (capillary tight junctions) and is not primarily associated with cellular damage. When observed with DWI, the ADC values in vasogenic edema are increased, as in our patient. A similar case was recently published by Corvol et al, who found slight, reversible DWI and marked fluid-attenuated inversion recovery abnormalities in a patient with CVT of the superior sagittal sinus and flow reversal in the cortical veins. The findings were judged to be vasogenic edema; the patient recovered rapidly after treatment with intravenous heparin and had a complete normalization of ADC values. A preservation of neuronal tissue was also documented by Hsu et al, using proton MR spectroscopy in a case of deep CVT.

In the case presented in this article, PI provided additional information, because perfusion patterns differed from arterial infarction. We found an increase in the rCBV and a marked delay of the bolus passage, which reflects venous congestion, a characteristic feature of venous stroke. Perfusion deficits in the rCBV maps, as are seen in arterial ischemia, were not detected.

In an experimental study, Röther et al used DWI and PI to characterize the pathophysiology of CVT in the rat. In this study, the superior sagittal sinus was completely occluded by ligation and injection of thrombogenic material. The induced venous infarction was characterized by perfusion deficits and early cytotoxic edema (ADC declined to 56±7% closely followed by vasogenic edema after blood-brain barrier disruption (ADC, 84±8% by 48 hours). This animal study proves that venous infarction with damage of neuronal tissue can be predicted by typical patterns in DWI and PI, which are known from arterial stroke and are more severe than in the case presented in this article and the case of Corvol et al.

In conclusion, we believe that in addition to their use for evaluation of ischemic stroke after arterial vessel occlusion, DWI and PI can play a role in diagnosis of cerebral venous ischemia. The MR features presented in this case reflect the underlying pathophysiology of venous congestion with impaired but viable neuronal tissue. Typical patterns of venous stroke with favorable outcome seem to be missing cytotoxic edema and rCBV decrease.

References

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