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 1.6×10⁻³ mm²/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 0.92×10⁻³ mm²/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.

Key Words: cerebral veins ■ diagnosis ■ diffusion ■ magnetic resonance imaging ■ perfusion ■ thrombosis
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, 243×256; 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 multi-shot 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
Discussion

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, Rother 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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