Serial T2*WI Studies in the Acute Phase of Cerebral Venous Thrombosis

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Abstract

We report a case of a 49-year-old woman with headache who was diagnosed with cerebral venous thrombosis (CVT) of the superior sagittal and right transverse sinuses. Serial gradient recalled-echo T2*-weighted imaging (T2*WI) studies demonstrated dynamic changes of thrombosed segments. T2*WI is useful not only as a diagnostic tool for CVT, but also in evaluating changes to thrombus.

Key words: cerebral venous thrombosis, neuroimaging, T2*WI

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Introduction

Cerebral venous thrombosis (CVT) is often misdiagnosed because: 1) clinical presentation is highly variable without focal symptoms; and 2) conventional magnetic resonance (MR) imaging (T1- and T2-weighted imaging) is insensitive in the acute phase (1). The utility of gradient recalled-echo T2*-weighted imaging (T2*WI) as a diagnostic technique has recently been reported (2, 3). We herein report that serial T2*WI demonstrated dynamic changes in signal intensity of thrombus in the acute phase of CVT.

Case Report

A 49-year-old woman awoke one morning with a dull headache and nausea. These symptoms had persisted all day. The following day she complained of a severe headache on awakening together with left hemiparesis and was admitted to our hospital. She had no previous history of illness. On admission, her height was 160 cm and body weight was 71 kg. Body temperature was 36.8°C. Blood pressure of right arm was 154/84 mmHg. There was no significant difference between both arms. Heart rate was 70 beats/min with regular sinus rhythm. No abnormal cardiac murmurs or carotid or orbital bruits were audible. Neurological examination revealed disturbance of consciousness (Glasgow Coma Scale was 13), left unilateral spatial neglect, facial palsy, hemiparesis and sensory disturbance. Extensor plantar response was present on the left foot. National Institute of Health stroke scale score was 11 on admission. Laboratory data identified: leukocyte count, 12,380/mm³; erythrocyte count, 497/mm³; hemoglobin, 10.3 mg/dL; platelets, 43.5/mm³; C-reactive protein, 0.52 mg/dL. D-dimer was 4.5 μg/mL (normal, <0.5 μg/mL) and thrombin-ant thrombin (TAT) was 16.1 ng/mL (normal, <3.0 ng/mL). Tests for anti-nuclear antibody, anti-DNA antibody, lupus anticoagulant, anticardiolipin antibody and anti-β2GPI antibody yielded negative results. Serum levels of International normalized ratio, activated partial thromboplastin time, protein C and S and antithrombin were normal.

She was examined using a commercially available echo planar operation on a 1.5-T MR unit (Signa EchoSpeed Horizon; GE Medical Systems, Milwaukee, WI). Diffusion-weighted imaging (DWI) on admission showed a hyperintense lesion in the right middle cerebral artery (MCA) territory. Apparent diffusion coefficient (ADC) value of the lesion was elevated. MR angiography revealed no abnormalities. MR venography (MRV) showed defects of the superior sagittal sinus. T2*WI showed diminished signal and enlargement of the right transverse sinus, superior sagittal sinus and cortical veins. Therefore, CVT was suspected and the patient was taken to the angiography suite. After cerebral angiography revealed defects of the superior sagittal and right trans-
verse sinuses, we diagnosed her as having CVT. Anticoagulation treatment with heparin was initiated on day 1 and adjusted to achieve an activated partial thromboplastin time (APTT) ratio of 1.5 to 2.0-fold baseline. Heparin treatment was changed to warfarin treatment on day 15 after admission.

Serial T2*WI studies were performed on days 1, 4, 8, 14 and 29 after admission and demonstrated dramatic changes in signal intensities (Fig. 1). Enlargement of the superior sagittal sinus and cortical veins gradually reduced and disappeared by day 14 (Fig. 1E). Signal intensity of the superior sagittal sinus also gradually changed from hypointense to hyperintense as follows. A small hyperintense lesion appeared on day 8, it was enlarged by day 14 and became obvious by day 29 (Fig. 1D, E, G). Follow-up cerebral angiography was performed on day 21, revealing recanalization of the thrombosed segments (Fig. 1F). On day 29, no enlarged lesion of the sinus was apparent and hyperintense signal was seen on T2*WI (Fig. 1G). Although other serial imaging sequences including T1-weighted imaging (T1WI), T2-weighted imaging (T2WI), fluid-attenuated inversion recovery (FLAIR), DWI, time-of-flight MRV, and contrast-enhanced MRV were also performed several times on the same day with T2*WI and compared, no sequences provided more useful information than T2*WI.

Although focal seizures occurred during the first few days, clinical symptoms gradually improved. Five weeks later, the patient was transferred to a rehabilitation facility with moderate hemiparesis.

**Discussion**

The present case showed three major findings. First, T2*WI was initially very useful in diagnosing the acute phase of CVT. Second, serial T2*WI studies were able to assess dynamic changes in thrombosed segments. Finally, signal changes of T2*WI studies can allow detection of recanalization in CVT.

In the present case of CVT, T2*WI showed diminished signals and enlargements of venous sinuses and cortical veins. These signal changes were compatible with previous reports by Selim et al (3). Before the T2*WI sequence was developed, the combination of T1WI, T2WI and MRV was the most common method of diagnosing CVT (1, 4). However, it is difficult to diagnose the acute phase of CVT using these sequences, as both T1WI and T2WI show isointensity in the thrombosed segment, and MRV is also unable to distinguish venous flow reductions from hypoplastic sinus. Additionally, although DWI detects signal changes in the cortical and subcortical lesions caused by cytotoxic or vasogenic edema, the sensitivity of the thrombosed segments is insufficient (5). Indeed, in the present case, DWI could demonstrate hyperintense lesions that seemed to be come from vasogenic edema based on ADC value, but it was unable to show any signal changes of the thrombosed segments. Conversely, Idbaih et al reported that T2*WI showed diminished signal and enlargement of the thrombosed segment in >90% of CVT patients within the first 3 days after symptom onset.
CVT in the present case demonstrated serial signal changes on T2*WI studies. These results are partly in line with a previous report by Leach et al (7) that analyzed 18 patients with CVT and reported that 91% of cases within the first 7 days of onset demonstrated enlarged and diminished signal of the thrombosed segment on T2*WI sequence, compared with 23% at ≥ 8 days after onset. However, in the report by Leach et al, serial T2*WI studies were not performed in the acute phase in each case. In the present case, serial T2*WI studies were able to detect dynamic changes in thrombosed segments of cerebral veins.

In terms of the meaning of the signal changes, paramagnetic compounds (deoxyhemoglobin, intracellular methemoglobin and hemosiderin) are generally known to produce signal loss on T2*WI. In cases of CVT, deoxyhemoglobin within trapped erythrocytes in the thrombus induces T2*WI signal loss in the acute stage and thrombus organization due to fibroblasts, macrophages and capillaries induces signal changes in the chronic stage (7, 8). However, no reports have described associations between T2*WI signal change and recanalization of the thrombosed segments; we then compared serial changes on T2*WI with cerebral angiography. Indeed, we serially identified signal changes on T2*WI and recanalization of the venous sinus on follow-up cerebral angiography. Serial signal changes on T2*WI corresponded with improvements in cerebral angiography. We suspect that T2*WI signal changes occur not only with organization, but also with recanalization of the thrombus.

References


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