

T2*-Weighted MRI Detected Dilated Cerebral Veins in a Patient with Acute-Phase Cerebral Venous Sinus Thrombosis—A Case Report

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We describe a 45-year-old man who presented with nausea, vomiting, and strong occipital headache on the right side. Although no abnormalities on neurological examination or computed tomography imaging were found on admission, peripheral blood cell counts showed polycythemia (hemoglobin 20.6 g/dL) and electrocardiography demonstrated atrial fibrillation. Therefore, anticoagulant treatment with heparin was started immediately. On the following day, the occipital headache continued. Brain T2*-weighted (T2*WI) magnetic resonance imaging (MRI) and, to a lesser extent, susceptibility-weighted imaging showed dilation of numerous cortical veins, suggesting the possibility of cerebral venous thrombosis (CVT). MR venography (MRV) showed a deficit of the right transverse sinus. Contrast-enhanced MRI revealed partial defects of the right transverse sinus, and led to the definite diagnosis of CVT, and the anticoagulation therapy was continued. On day 7 the headache disappeared, and MRV on day 16 showed the recanalization of the right transverse sinus. There were no complications subsequent to the CVT. On day 25, the patient was discharged with no after-effect. We speculate that the dilation of cortical veins on T2*WI is a helpful sign in detecting acute-phase CVT.

Key Words: Acute cerebral venous thrombosis—dilation—T2*-weighted MRI—diagnosis—anticoagulant

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Introduction

Cerebral venous thrombosis (CVT) is a relatively rare disease in which the cerebral vein is obstructed, resulting in cerebral edema and hemorrhagic infarction.¹ It is important that the diagnosis of CVT be confirmed immediately, because any delay in diagnosis could lead to stroke, which

can entail severe sequelae and in some cases death.² Although CVT is often diagnosed by angiography, magnetic resonance venography (MRV) and/or contrast enhanced computed tomography (CT)/magnetic resonance imaging (MRI), it has been reported that obstructed sinusoids exhibit an abnormally low signal on MRI-T2*-weighted imaging (T2*WI)³ and susceptibility-weighted imaging (SWI).⁴

We herein report a case whose CVT was diagnosed in the acute phase by detection of cerebral vein dilation on T2*WI, and who had a good clinical course with anticoagulant therapy.

Case Report

A 46-year-old man visited our hospital with nausea, vomiting, and strong occipital headache on the right side that had persisted for 2 days. He had diagnoses of hypertension and gout, and he used medicine regularly. No

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abnormalities on either neurological examination or CT imaging were found on admission. His height was 177 cm, body weight 85 kg, blood pressure 131/90 mm Hg, pulse rate 98 beats/min and irregular, and body temperature 36.9°C. Peripheral blood cell counts showed polycythemia (hemoglobin 20.6 g/dL). Coagulation studies were almost normal except for a mild increase in D-dimer (1.2 $\mu\text{g}/\text{mL}$). Electrocardiography demonstrated atrial fibrillation; therefore, anticoagulant treatment with heparin was started immediately. On the following day, nausea and vomiting improved, but the occipital headache continued. Brain T2-weighted (T2WI), T1-weighted (T1WI) MRI, diffusion-weighted imaging (DWI), T2*WI and SWI showed no abnormal signals and fluid-attenuated inversion recovery showed no edematous lesions, except for slightly high-intensity signals, suggesting the existence of venous congestion, in the right transverse sinus (Fig 1, A-F). Although we did not initially interpret these findings as abnormal, T2*WI and SWI showed many dilated cerebral veins in the bilateral cortical area (Fig 1, G1-G3,H); these were indicated more clearly on T2*WI than on SWI. The dilation of cerebral veins spread to the parietal and temporal lobe cortex on both sides, mainly on the right hemisphere. We suspected that the dilatation of the cerebral veins was associated with venous congestion due to CVT, and MRV revealed

the deficit of right transverse sinus (Fig 1, I). Contrast-enhanced T1WI MRI showed a partial defect of the right transverse sinus, suggestive of a thrombus, confirming the diagnosis of CVT (Fig 1, J). No other diseases such as dural arteriovenous fistula have been found to dilatation of the cerebral veins in this case.

Anticoagulant treatment with heparin was continued. On day 16, MRV demonstrated the recanalization of the right transverse sinus and showed the disappearance of the left transverse sinus, suggesting the hypoplasia (Fig 1, M), while T2*WI and SWI showed resolution of the cerebral vein dilation (Fig 1, K1-K3,L) and T2WI revealed no high-intensity signals in the right transverse sinus (Fig 1, N). Visualization of cortical veins and superior sagittal sinus MRV on day 16 (Fig 1, M) is better than that at initial MRV (Fig 1, I), it was considered due to the mechanism that elevated venous and capillary pressure caused by venous sinus thrombosis was improved. After switching the anticoagulant treatment to a direct oral anticoagulant, he was discharged from the hospital with no after-effects on day 25.

Discussion

We describe a case of CVT diagnosed in the acute phase by the appearance of dilated cerebral veins on T2*WI,

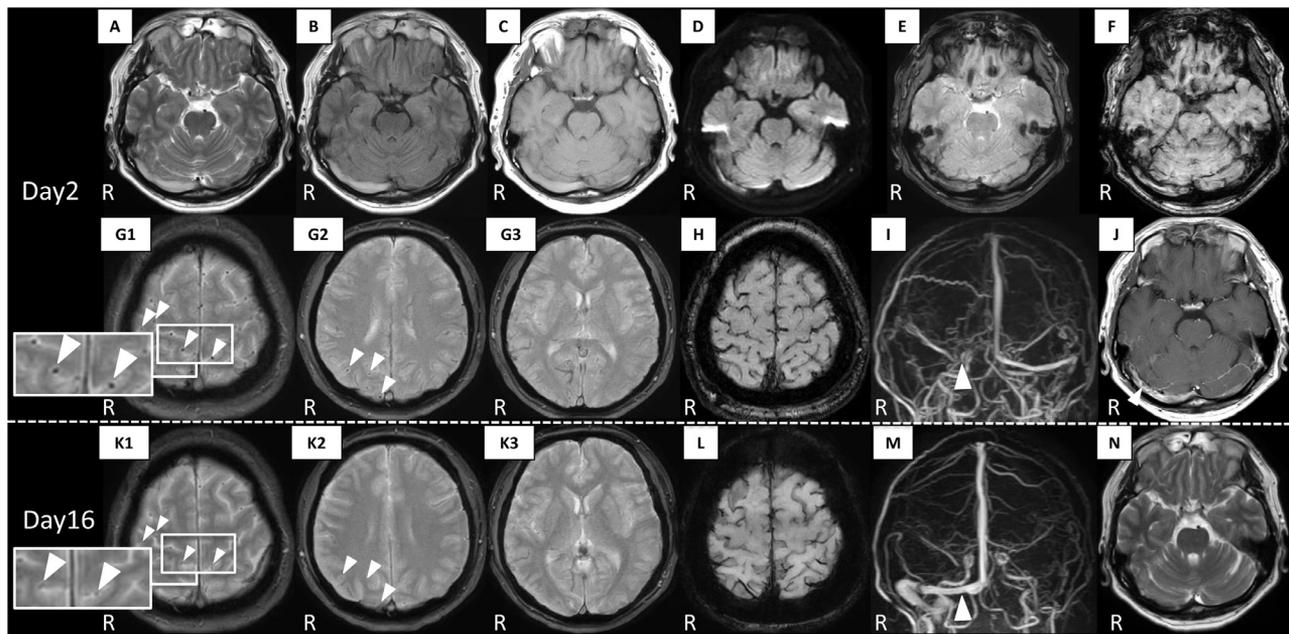


Figure 1. MRI and MRV on day 2 and day 16. (A) T2WI, (B) FLAIR, (C) T1WI and (D) DWI on day 2 demonstrated no abnormal signals except for slightly high intensity signals in the right transverse sinus. (E) T2*WI and (F) SWI on day 2 did not clearly demonstrate thrombus in the right transverse sinus. (G1-3) T2*WI and (H) SWI on day 2 showed dilation of many cerebral veins in the bilateral cortical area (arrowheads). (I) MRV on day 2 revealed the deficit of the right transverse sinus (arrowhead). (J) Contrast-enhanced T1WI MRI showed a partial defect of the right transverse sinus (arrowhead). (K1-3) T2*WI on day 16 clearly showed no dilation of the cerebral veins (arrowheads) in the bilateral cortical area, even when compared with T2*WI on day 2 (G1-3). (L) SWI on day 16 similarly showed no dilation of cerebral veins in the bilateral cortical area. (M) MRV on day 16 revealed the recanalization of the right transverse sinus (arrowhead) and the disappearance of the left transverse sinus. (N) T2WI on day 16 revealed no high-intensity signals in the right transverse sinus.

who had a good clinical course thanks to prompt anticoagulant treatment.

Some kind of thrombotic risk factor is identified in about 80 percent of patients with CVT, such as congenital coagulation abnormalities, autoimmune diseases, infectious diseases, blood diseases, use of contraceptives, trauma, malignant disease, etc.⁵ In this case, CVT was secondary to polycythemia, which may have caused the CVT.

The most sensitive examination techniques have been reported to be a combination of MRI and phase-contrast MRA⁶ or MRV.¹ However, it is difficult to diagnose acute-stage CVT using conventional T1WI, T2WI, and fluid-attenuated inversion recovery, which will show a hyperintense signal from the thrombosed sinuses. We could not diagnose CVT by those sequences alone. In addition, it is difficult to distinguish between cerebral venous sinus thrombosis and hypoplastic sinus on MRV. We initially interpreted the left transverse sinus as intact on MRV, despite its being a hypoplasia (Fig 1, I,M).

Recently, several reports have shown that T2*WI and/or SWI are more useful MR sequences than conventional MR sequences in detecting CVT.^{4,7,8} Moreover, T2*WI and/or SWI are more useful diagnostic imaging tools for direct clot detection in the acute phase of cerebral venous thrombosis, resulting from the susceptibility effects of deoxyhemoglobin within the blood clots.^{9,10} However, in this case, we could not detect the thrombus clearly in the cerebral venous sinus using T2*WI and SWI: the thrombus had not yet become large because it was in the hyperacute phase of CVT on the third day after symptom onset and the venous outflow had been maintained through the hypoplastic cerebral venous sinus on the contralateral side. Despite being in the hyperacute phase, T2*WI showed many dilated cortical veins, probably due to venous congestion from the CVT.

Progressive thrombosis of the cerebral vein could obstruct cerebral venous outflow, causing of dilation of the superficial and deep medullary veins.^{11,12} Kawabori et al reported that SWI revealed dilated cortical veins due to a sagittal sinus thrombosis.⁴ In this case, the dilated cortical veins were demonstrated more clearly on T2*WI than on SWI, and this visualization helped us to diagnose CVT (Fig 1, G1,H). We considered that originally T2*WI is less sensitive than SWI to detect cerebral veins, so T2*WI hardly shows cortical veins in normal cases. In this case,

by detecting the originally invisible veins, it may be easy to notice the change of the dilated veins.

We were fortunate in this case that the patient's atrial fibrillation had incidentally prompted the early start of anticoagulation therapy, likely contributing to the good clinical course.

In conclusion, this case suggests that signs of dilation of cortical veins on T2*WI is useful for early detection and diagnosis of CVT.

Conflict of Interest

The authors state that they have no conflicts of interest.

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