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Clinical Observations

The Use of Susceptibility-Weighted Imaging for Epileptic Focus Localization in Acute-Stage Pediatric Encephalopathy: A Case Report

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ABSTRACT

BACKGROUND: Susceptibility-weighted imaging is a novel high-spatial-resolution three-dimensional gradient-echo magnetic resonance imaging technique with phase postprocessing that accentuates the paramagnetic properties of blood products. The use of susceptibility-weighted imaging for epileptic focus localization in the acute stage of encephalopathy in a child has not been documented. **PATIENTS:** We report three pediatric patients with status epilepticus in the setting of fever, in whom susceptibility-weighted imaging showed transient prominence of the focal venous vasculature. **RESULTS:** Conventional cranial T1- and T2-weighted images and diffusion-weighted images showed no abnormalities. The prominence of the focal venous vasculature in these patients, as demonstrated by susceptibility-weighted imaging, was consistent with the epileptic foci suggested by both clinical symptoms and electroencephalograph findings and resolved completely without neurological sequelae in all patients. **CONCLUSIONS:** Susceptibility-weighted imaging may facilitate assessing epileptic focus localization in the acute stage of encephalopathy in children.

Keywords: susceptibility-weighted imaging, child, status epilepticus, encephalopathy, febrile seizure

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Introduction

Susceptibility-weighted imaging (SWI) is a novel magnetic resonance (MR) technique that uses both magnitude and phase images to evaluate the magnetic properties of tissues.^{1,2} Paramagnetic substances such as deoxyhemoglobin and ferritin are known sources of magnetic susceptibility in tissues. Deoxygenated venous blood with deoxyhemoglobin causes magnetic field inhomogeneity because of a reduction in T_2^* as well as a phase difference between the vessels and surrounding parenchyma.³ Thus, SWI is exquisitely sensitive for depicting the venous vasculature because it detects deoxygenated blood in small

veins without contrast medium, even when conventional MR imaging shows no definite structural changes.⁴ Recently, the diagnostic role for SWI during sporadic hemiplegic migraine has been reported, in which three pediatric patients presented with reversible transient focal cerebral venous prominence that resolved when the motor aura ended.⁵ In addition, two patients, a 37-year-old man and a 9-year-old boy, both suffering from migraine with aura and presenting with reversible transient prominence of the focal cerebral venous vasculature on SWI but no abnormalities on conventional MR imaging, have been reported.⁶ However, the use of SWI for epileptic focus localization in the acute stage of encephalopathy in a child has not been documented.

We describe three children with acute epileptic encephalopathy showing reversible transient focal SWI abnormalities. None of these individuals exhibited abnormalities on conventional T1- and T2-weighted or diffusion-weighted images (DWI).

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Case 1

A 10-month-old boy presented with status epilepticus and fever. The patient's development had been normal and there was no family history of convulsive disorders. Two days before admission, he developed a high fever. On the day of admission, he started vomiting, then his eyes deviated to the right and he experienced his first seizure—a generalized tonic-clonic seizure—that progressed to status epilepticus lasting 40 minutes. During anticonvulsant treatment, clonic seizure of the fingers of his right hand persisted until the seizure stopped completely. Venous blood gas analysis showed acute respiratory failure (pH 6.951, partial pressure of carbon dioxide [pCO₂] 95.6 mm Hg, bicarbonate [HCO₃] 20.6 mmol/L, base excess [BE] -12.8 mmol/L, lactate 4.81 mmol/L). Routine cerebrospinal fluid workup revealed no abnormalities, though human herpes virus-6 DNA was later detected by polymerase

chain reaction assay of his cerebrospinal fluid. Cranial MR images, including DWI and SWI, were obtained 80 minutes after seizure onset. Routine T1- and T2-weighted images (not shown), T2-weighted images (Fig 1A), and DWI (Fig 1B) showed no abnormal findings. SWI showed prominence of the venous vasculature in the left cerebral hemisphere (Fig 1C). Electroencephalography (EEG) was performed 3 hours after the seizure onset and showed immature spindles in the patient's right central area but no spindles in his left central area, suggesting impaired left cerebral hemisphere function, the so-called lazy phenomenon on EEG.

His consciousness had normalized 30 hours after the episode, and the final diagnosis was human herpes virus-6 encephalopathy. Cranial MR images were reevaluated 4 days after admission, and SWI (Fig 1D) and other conventional forms of imaging showed no abnormalities at this time.

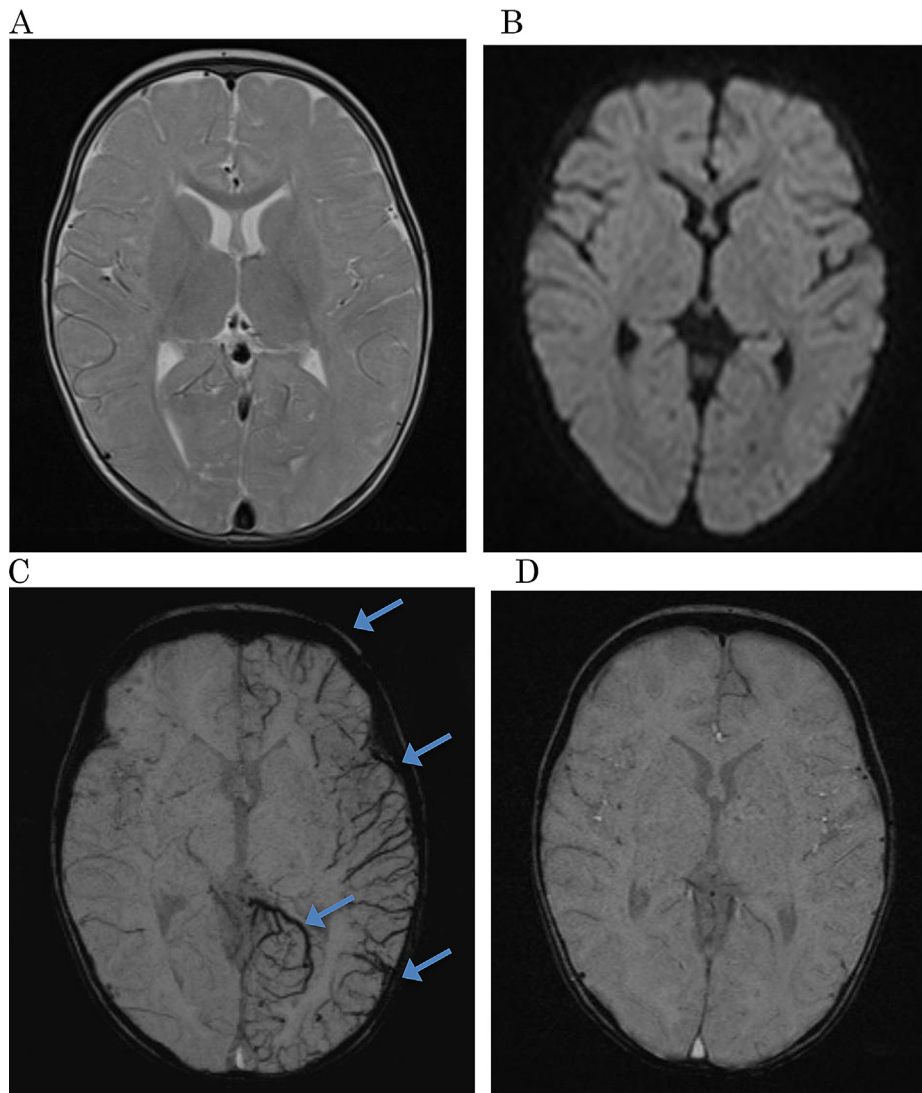
**FIGURE 1.**

Image from a 10-month-old boy with human herpes virus-6 encephalopathy obtained 80 minutes after seizure onset. Axial T1 (not shown), T2 (A) (repetition time [TR]/echo time [TE] = 3500/90 ms), and diffusion-weighted images (B) (TR/TE = 3034/95 ms) at the basal ganglia level were normal. Susceptibility-weighted imaging (C) (minimum intensity projection image; TR/TE = 49/40 ms) delineates prominent veins in the left cerebral hemisphere (arrows). Follow-up susceptibility-weighted imaging (minimum intensity projection image, TR/TE = 49/40 ms) obtained 4 days after the seizure onset (D) was normal.

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