Reversible Signal Changes in MR Diffusion-Weighted Imaging in a Patient with Status Epilepticus

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Abstract-

Background: Abnormality in diffusion-weighted magnetic resonance imaging representing early changes of acute ischemic lesions in human and animal models of focal status epilepticus has been reported to correlate with clinical outcome.

Case Report: We reported a 35 year-old woman with initial status epilepticus, probably related to previous head injury with traumatic intracerebral hemorrhage. The presenting MRI showed reversible hyperintensity lesions on DWI, which is probably corresponding to the epileptogenic lesion. Similar abnormalities in the splenium as a remote effect were demonstrated in this case.

Conclusion: The atrophic changes in the splenium and right parietal lobe in the follow-up MRI scans were supposed to correlate with the following neurological sequelae.

Key Words: status epilepticus, diffusion magnetic resonance imaging, corpus callosum

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INTRODUCTION

Diffusion-weighted imaging (DWI) is a recently introduced magnetic resonance imaging (MRI) technique that allows relative measurement of water diffusion in brain parenchyma. With this technique it is possible to measure the apparent diffusion coefficient (ADC) of water. DWI becomes a noninvasive tool for the early detection of acute ischemic lesions in human and animal models of focal status epilepticus. In rat models of kainate-induced status epilepticus, the ADC mapping decreased by 7-30% about 5-24 hours postictally. Similar changes may last for 1-3 days and completely resolve 9 days later[1-3]. Some data on humans with periictal changes on DWI suggest that there are significant correlations among the areas of diffusion abnormalities, increased perfusion, and electro-corticographic abnormalities[4-6]. We presented a patient with reversible signal changes in DWI after status epilepticus.

CASE REPORT

A 35-year-old woman was brought to the Emergency Department in Landseed Hospital for acute sudden of unconsciousness and long-lasting convulsion.

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Based on the history reported by her family, she presented initially with tonic-clonic convulsion of the left limbs, followed by generalized convulsion for more than 30 minutes. When she was aged 20, she had a traumatic intracerebral hemorrhage with sequelae of mild clumsiness of the left limbs. She could perform daily activities independently and had a fair work performance despite the neurologic deficits. There was no past history of seizure prior to this admission.

The physical examination in the ER revealed that the patient was stuporous, dyspneic and cyanotic in lips with generalized convulsion. She was intubated immediately for airway protection and oxygen supply. Post-ictal EEG showed frequent focal paroxysmal sharp and slow waves with maximal activity over right anterior temporal areas and intermittent delta waves over right frontotemporal regions. Those findings on EEG indicated a probable epileptogenicity in the right anterior temporal area (Fig. 1). Her convulsions subsided after intravenous injection of midazolam 5 mg on arrival, and followed by intravenous infusion of phenytoin 500 mg within 30 minutes. Regular use of phenytoin 100 mg q8h was prescribed afterwards. She regained consciousness and was extubated on the next day.

MRI performed 3 days after the episode on a 0.5 Tesla system (GE medical system) with fast-spin echo T2 -weighted images demonstrated extensive high signal intensity changes in the gray and subcortical white matter, mainly in the right frontoparietal lobes (Fig. 2A). Isotropic DWI showed high signals in the same regions and also in the splenium (Fig. 2B).

Left hemiparesis (muscle power of left upper limb: grade 2/5 by Medical Research Council (MRC) Scale, left lower limb: MRC grade 3/5) was noted after the cessation of the convulsions. Follow-up EEG 4 days later showed focal slowing over the right hemisphere without epileptiform discharges. The patient was discharged 2 weeks later with residual weakness of the left limbs (MRC grades 3/5, 4/5 in the upper and lower limbs, respectively). She returned for follow-up 1, 3, 5, 7 and 9 months after discharge with an uneventful course. A brain MRI (1.5-T, GE medical system) performed 4

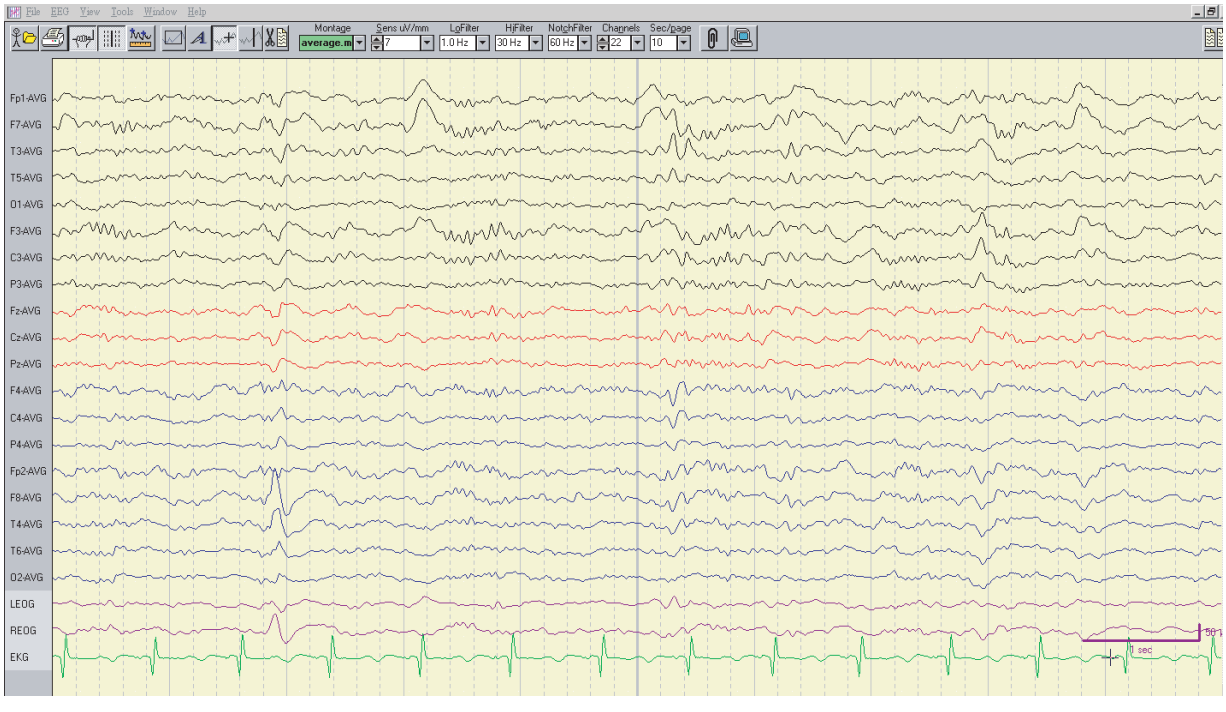

Figure 1. Post-ictal EEG showed focal paroxysmal epileptiform sharp and slow waves with maximum activity over right anterior temporal area.

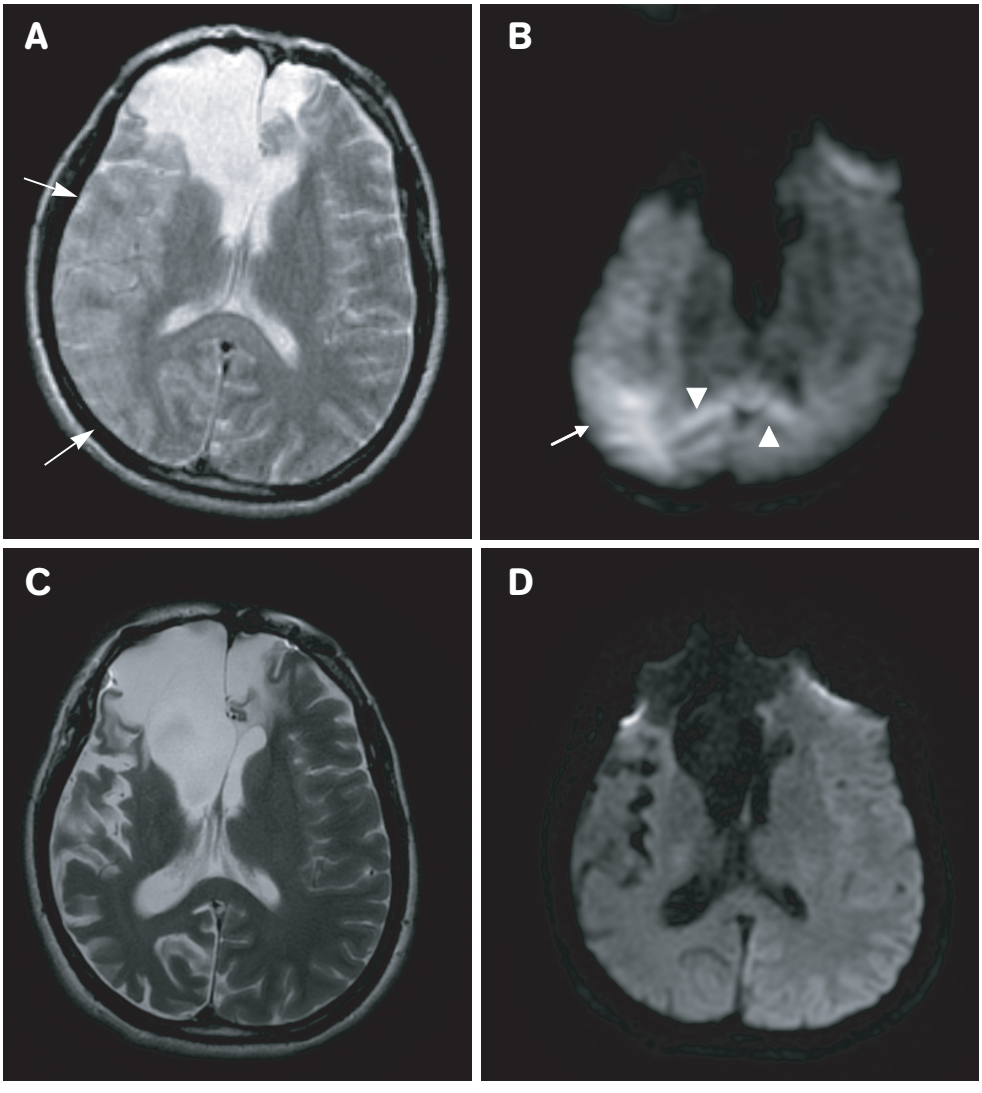

Figure 2. Figure (A) A T2-weighted MRI 3 days after seizure episode showed high signal intensity lesions over the right fronto-parietal lobe (arrows). (B) A diffusion-weighted MRI sequence showed a high signal intensity in splenium (arrowheads) and in the corresponding edematous areas as shown in Figure 2A (arrow). (C) A T2-weighted MRI 4 months after the onset of seizure showed resolved signals of cerebral edematous changes with atrophy in the right frontoparietal areas. (D) A diffusion-weight MRI 4 months after the onset of seizure showed resolved signals in the right fronto-parietal area and spleniu.

months after onset of epilepsy demonstrated regression of the signal abnormalities (Fig. 2C, 2D). Prominent sulci in the right frontoparietal areas are suggestive of atrophic changes. No more episodes of seizures had been reported after discharge with the medications of phenytoin 300 mg qd. However, she did have residual psychiatric changes, such as impatience and indifference, and she was unable to return to work due to the neurologic and psychiatric deficits. During the follow-up period, there was one episode of phenytoin overdose 7 months after discharge. Phenytoin overdose was noted because she was afraid of further attack and she took nigher dose of phenytoin (400~500 mg qd). Levetiracetam 500mg bid was added for epilepsy prevention afterwards.

Seizure had never occurred after discharge.

DISCUSSION

Diffusion-weighted magnetic resonance imaging may provide valuable information in the evaluation of an individual with epilepsy. Seizure-associated changes in DWI are dynamic. Early clinical studies assessing DWI in patients with status epilepticus showed restricted diffusion (bright on DWI and dark on ADC mapping), increased T2 signal intensity, and local hyperperfusion. These abnormalities normalized in most patients by day 14[1-3,6,7]. This phenomenon seems to be a consequence of increased neuronal energy demands in the epileptogenic area, which is not adequately matched by the compensatory enhanced blood flow. It leads to an anaerobic glycolysis in neurons, Na^+/K^+ ATP-ase pump failure, and thereby to regional cytotoxic and vasogenic edema[8]. Compared with acute ischemia, in which DWI images reveal restricted diffusion before any abnormality of the T2 signal becomes apparent, DWI and T2 signal changes appear to occur synchronously in status epilepticus[8].

Interestingly, we found a reversible high DWI signal in the whole splenium of the corpus callosum in the follow-up imaging, which is not responsible for epileptogenicity. Some studies have reported that periictal MRI changes may occur in the region of the epileptic discharge or in distant structures[7-9]. Andrew et al. demonstrated periictal imaging abnormalities in both local and remote areas[8]. The local signals alteration correlates with electro-clinical activity well in partial status epilepticus[10]. The findings consist of cortical lesions with focal mass effect, hippocampal swelling, abnormal enhancement, suggesting blood-brain barrier breakdown, and increased vessel caliber with enhanced flow[8]. The areas of DWI changes, with electroencephalography, could help define the focus of epileptogenicity and involvement of maximal activity.

The findings in remote areas included posterior leukoencephalopathy, abnormalities in unilateral or bilateral diencephalon, splenium[8], thalamus[11], and cerebellar diaschisis[9]. However, the pathophysiology of these remote lesions is not understood. Reversible lesions of the splenium may also reflect abnormal activity in white matter tracts driven by the epileptic focus, and several patients with splenial lesions were reported to have independent bitemporal epileptic foci and associated psychiatric disease[8]. However, the mechanism and the precise nature of the white matter changes responsible for the increased T2 signal and restricted diffusion in the splenium are poorly understood. This hypothesis correlating specific abnormal MRI findings in the splenium of corpus callosum and subsequent psychiatric or behavioral problems should be examined prospectively by systematic imaging and serial psychiatric examinations[8].

The longitudinal follow-up of the findings of MRI in status epilepticus could reveal long-term changes of abnormal signals. The signal changes in DWI were reversible in this patient; nevertheless the follow-up imaging did show the changes of decreasing volume in the areas of previous T2 and DWI changes, which could explain her and neurological sequelae. The permanent atrophic changes were demonstrated by the follow-up MRI study[12]. Patients with prolonged and severe epilepsy carry higher risk for subsequent neurological deficit. To avoid residual effects, patient education, early recognition and treatment of status epilepticus should be emphasized.

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