Clinical Neurology and Neuroscience
2017; 1(1): 20-23
http://www.sciencepublishinggroup.com/j/cnn
doi: 10.11648/j.cnn.20170101.15

Case Report

Differential Changes on Diffusion-Weighted MRI in a Patient with Partial Status Epilepticus: Case Report

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To cite this article:
Jang Sang Hyun. Differential Changes on Diffusion-Weighted MRI in a Patient with Partial Status Epilepticus: Case Report. *Clinical Neurology and Neuroscience.* Vol. 1, No. 1, 2017, pp. 20-23. doi: 10.11648/j.cnn.20170101.15

Received: October 17, 2016; **Accepted**: December 27, 2016; **Published**: March 27, 2017

Abstract: We report a patient who showed mixed cytotoxic and vasogenic edema on Magnetic Resonance Imaging (MRI) in association with partial status epilepticus. The patient experienced clonic movements of the left arm and leg. As the clonic movements continued, resulting eventually in epilepsia partialis continua, MRI showed characteristic cytotoxic edema in the epileptogenic focus and vasogenic edema in the region remote from the epileptogenic focus. The aim of this report is to support the evidence that partial seizure spreads through efferent projections from the cerebral cortex to the cerebellum, and findings of serially followed MRI suggest that cytotoxic edema persisted for a long time in contrast to vasogenic edema.

Keywords: Partial Status Epilepticus, MRI, Cytotoxic Brain Edema, Vasogenic Brain Edema

1. Introduction

In relation to partial status epilepticus (PSE), MRI has generally shown high signal on T2-weighted, diffusion-weighted MRI (DWI) and decreased apparent diffusion coefficient (ADC), which suggest cytotoxic edema [1]. MRI changes suggesting vasogenic edema have been reported in only a few patients, following seizures. The mechanism of vasogenic edema is still uncertain, but may be related to disruption of the blood-brain barrier (BBB) and relatively lower seizure activity. We report a 50-year old man with partial status epilepticus and vasogenic edema in the remote areas from epileptogenic focus on MRI.

2. Case Report

A 50-year-old, right-handed man was admitted for sudden aggravation of dysarthria. Four months ago, he had undergone surgical removal of a large hematoma transformed from atherosclerotic infarction in the right parietal region. Since then, he had had sequelae of dysarthria, left-sided hemiparesis, sensory dysfunction, and hemianopsia. Bain MRI showed acute atherosclerotic infarction of the left thalamus and encephalomalatic change in the right fronto- parieto-occipital lobes. Six days later, he suddenly began to suffer recurrent clonic jerks in whole extremities, more prominent in the left leg with maintenance of consciousness. After intravenous benzodiazepine injection, the frequency and amplitude of clonic jerks were diminished and discontinuous, but repeated clonic jerks of the left leg persisted, resulting eventually in epilepsia partialis continua. Fluid attenuated inversion recovery (FLAIR) MR image on a 3.0 tesla system, performed 3 hours after the seizure onset, demonstrated newly developed extensive high signal in paracentral region of the right frontal lobe adjacent previous infarction, left splenium of corpus callosum, left occipital cortex and left cerebellum. DWI showed fluid restriction in the identical region in the right frontal lobes without any signal changes in the other regions. On ADC map, relatively decreased intensity in the paracentral region of bilateral frontal lobe and increased intensity in the other regions (right parieto-occipital lobe, left corpus callosum, left occipital lobe and bilateral cerebellums) were showed in comparison with those of unaffected regions (Figure 1). EEG combined with electromyography performed 4 hours after the seizure onset showed periodic lateralized epileptiform discharges (PLEDs) in the right fronto-central region from F4/C4 leads with time-locked by clonic jerks of his leg (Figure 2).

The patient's seizures disappeared gradually during 5 days

after treatment of antiepileptic drugs. 10 days after the seizure onset, MRI still showed high signal in the right frontal lobe on DWI. However, previously decreased ADC value was somewhat increased and previously increased ADC value in the other regions regressed to slightly higher than the baseline value. 25 days after seizure onset, MRI results of high signal on DWI and decreased ADC value in the right frontal lobe were remained. But ADC values were normalized in the other regions.

3 months after seizure onset, MRI showed resolution of signal abnormalities on DWI which were seen in the right frontal lobe. Patient was seizure-free for 1 year of follow-up with antiepileptic drug treatment.

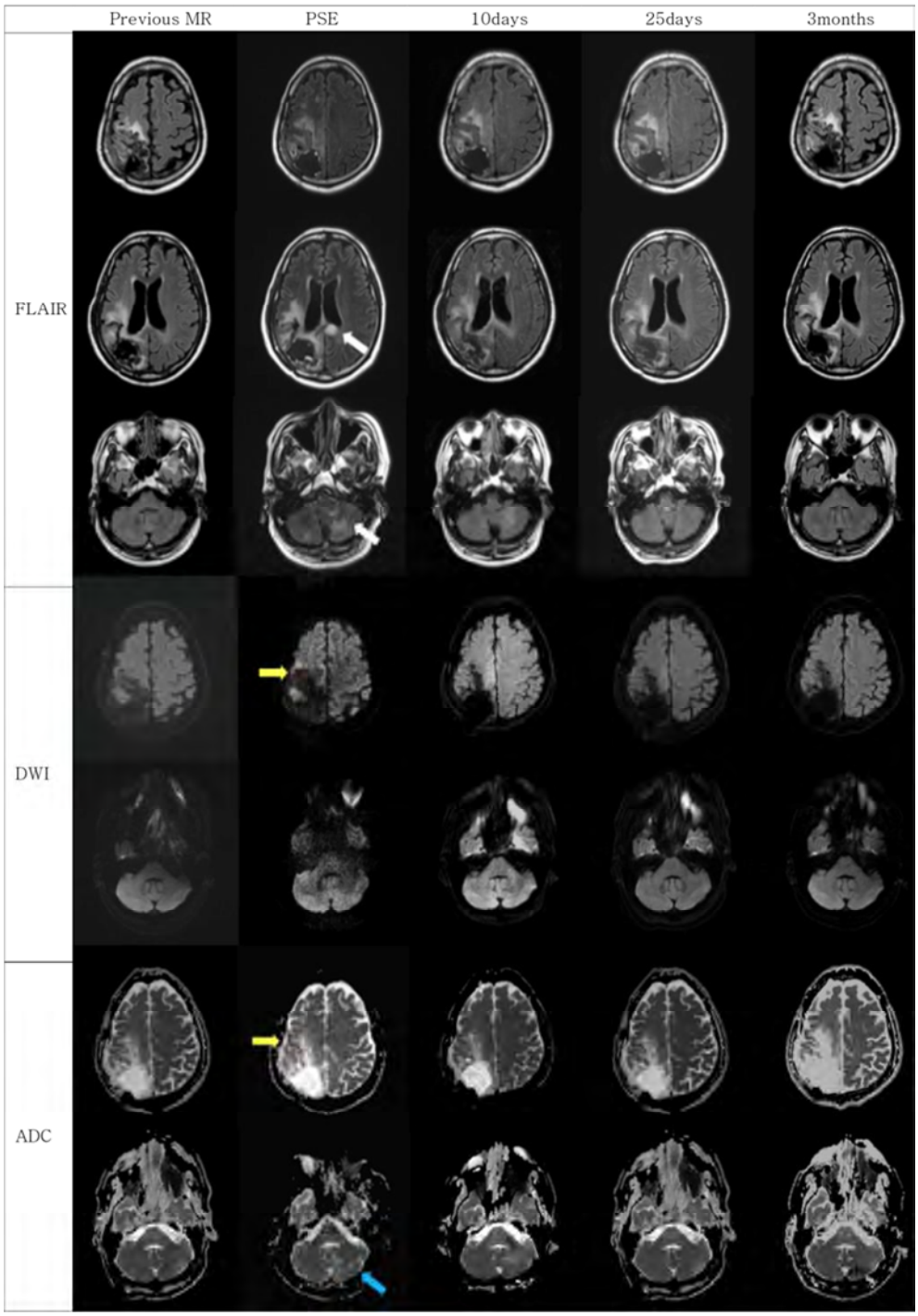

Figure 1. *Serially followed FLAIR, diffusion-weighted MRI (DWI) and apparent diffusion coefficient maps (ADC) from patient before, during and after partial status epilepticus (PSE). During PSE, FLAIR MR images show newly developed high signal in the right frontal, parieto-occipital lobe, corpus callosum and left cerebellum (white arrows). DWI shows high signal in the right frontal lobe and low ADC in the corresponding area (yellow arrows). ADC map shows high ADC in the corpus callosum, left occipital lobe and left cerebellum (blue arrow). Follow-up MRI performed 10 days, 25 days and 3 months later showed that previous high signal in the cerebellum and corpus callosum disappeared in FLAIR MRI. High signal on DWI and decreased ADC on ADC map in the right frontal region disappeared gradually.*

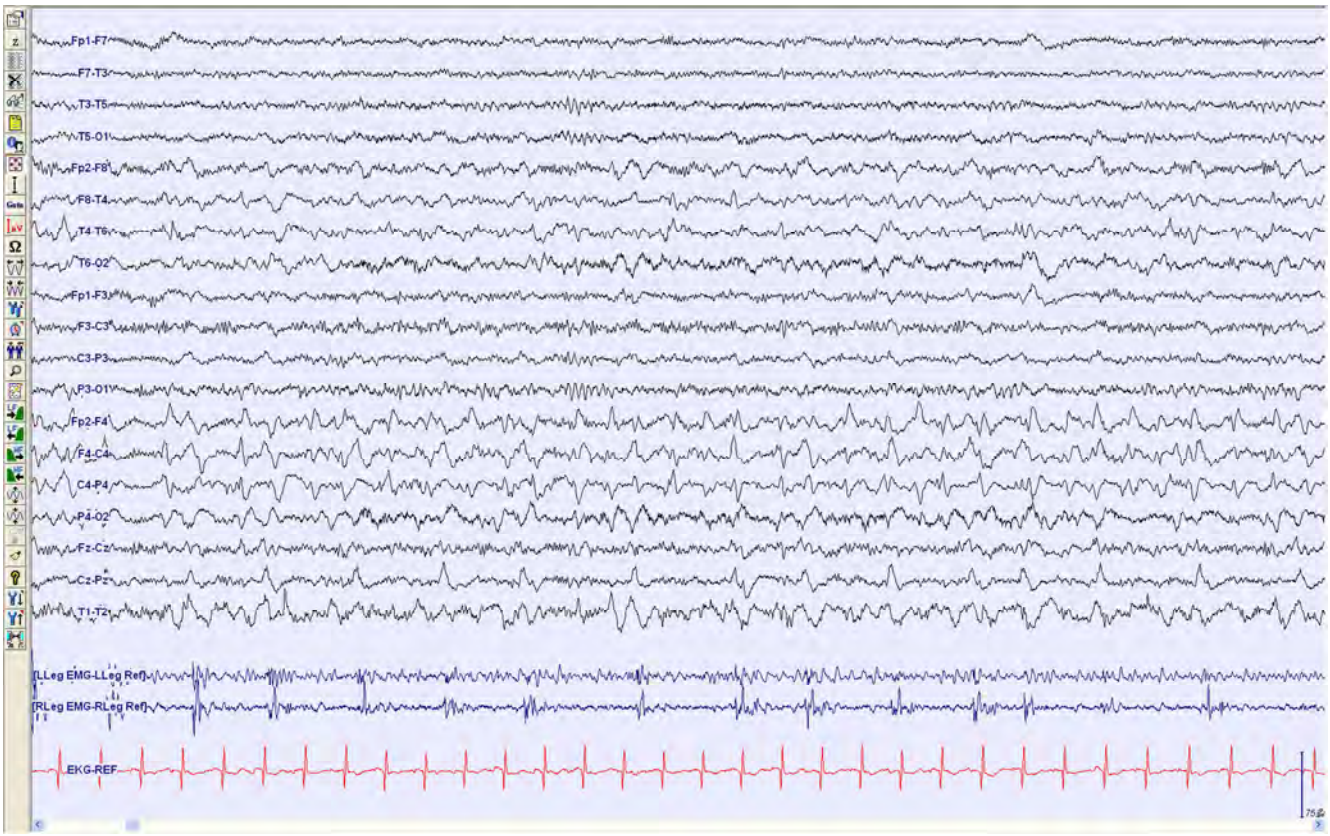

Figure 2. *EEG during partial status epilepticus. Persistent periodic sharp waves in the right frontal and central electrodes.*

3. Magnetic Resonance Imaging Technique

All MRI studies were performed on a 3.0 T MR unit (Siemens, Erlangen, Germany) with an echoplanar imaging (EPI) capability. FLAIR (repetition time (TR)/echo time (TE), 9000/134 ms; field of view, 23 cm × 23 cm; matrix, 320 × 320; and slice thickness, 4mm) was obtained. DWI was obtained in the transverse plane using a single-shot EPI (TR/TE, 4039/81 ms; field of view, 23 cm × 23 cm; matrix, 128 × 128; slice thickness, 4mm; and b values, 1000 s/mm^2). The diffusion-gradients were applied along the three axes (x, y, z) simultaneously.

4. Discussion

In humans, it is common that MRI performed during and in an early stage after PSE demonstrate cytotoxic edema in the epileptogenic focus or remote from focuses either. This signal changes are seen not only in the areas primarily involved in seizure activity but also in those remote from but functionally connected to the epileptogenic area [1]. Irreversible focal atrophic change may be produced [2]. The failure of Na^+/K^+-ATPase, increased membrane permeability, and excessive release of excitatory amino acids are the proposed mechanisms of cytotoxic edema [3], [4].

MRI changes suggesting vasogenic edema have been reported in only a few patients following seizures [5], [6]. Vasogenic edema has been explained by several mechanisms, such as disruption of the blood-brain barrier (BBB) due to hyperperfusion and altered cerebrovascular endothelium related to seizure activity, although delayed timing of DWI which can result in an increase in ADC due to the loss of diffusion barriers, following cell death [7]. Therefore, vasogenic edema is usually considered reversible in comparison with cytotoxic edema.

In our patient, brain MRI demonstrated cytotoxic edema in the paracentral lobule presumed to be the epileptogenic focus and adjacent region, and vasogenic edema in remote areas functionally connected to the epileptogenic area, such as the splenium and the cerebellum. The splenium and the cerebellum (especially in the contralateral side) have been reported to be involving regions of cytotoxic edema in association with PSE, which is explained by seizure activity propagated from the epileptogenic area through corticopontocerebellar tract [3], [8]. Acute DWI abnormalities of thalamus during PSE were reported in the case with epileptogenic focus in the posterior quadrants [9], [10]. These remote areas could receive less burden of seizure activity and less insult than the epileptogenic area. In our patient, follow-up brain MRIs showed sequential recovery of these changes, first in the cerebellum, splenium, and then the paracentral lobule.

Vasogenic edema following cytotoxic edema may be chronologically induced in the epileptic focus, as suggested by Hattori et al., who have reported vasogenic edema

associated with frequent simple partial and complex partial seizures [11]. Because the threshold of seizure intensity during PSE for maintaining vasogenic edema may be too narrow, vasogenic edema formation by BBB disruption may be easily overwhelmed by cytotoxic edema formation induced by intense seizure activity. The seizure intensity which does not progress to the threshold during PSE (presumed to occur more frequently) may not show any abnormality related to PSE.

Comprehensively considering previous reports and studies, it is not surprising that focal seizure activity from frontal lobe spread to cerebellum via corticopontocerebellar tract and induce the vasogenic edema along this tract. But, to the best of our knowledge, diffusion MR changes suggesting cytotoxic edema in the epileptogenic focus and vasogenic edema in the region remote from focus during PSE have not been reported.

5. Conclusion

In conclusion, it is report of PSE in which DWI studies shows differential changes of seizure activity along spreading pathway.

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