

# Diffusion-Weighted Imaging Findings in Patients with Status Epilepticus: Report of Two Cases

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We present MR diffusion-weighted imaging (DWI) findings of status epilepticus in two patients. DWI showed a focal or diffuse hyperintensity with decreased apparent diffusion coefficient (ADC) value, indicating cytotoxic edema in the cerebral hemispheric cortices. The hyperintensities were located in the bilateral temporoparietooccipital areas and insular cortex in one patient, and unilaterally in the temporal lobe in the other patient.

**Index words :** Status epilepticus, Diffusion, MR

Status epilepticus is an unvarying and enduring epileptic condition characterized by epileptic seizure, which is either sufficiently prolonged or is repeated at sufficiently brief intervals. Briefly, it is defined as more than 30 minutes of continuous seizure activity or two or more sequential seizure without full recovery of consciousness between seizures (1). As evidenced by neuropathologic studies both in human and experimental animal studies (2), status epilepticus is consistently associated with widespread neuronal necrosis in vulnerable regions of the brain, probably because of a complex interaction between excitatory and inhibitory mechanisms. Evaluations of status epilepticus should include clinical assessment, electroencephalography (EEG), laboratory investigations, and MR imaging. Even though MR imaging is indispensable for the evaluation of epileptic patients, only a small number of reports have focused on the diffusion weighted imaging (DWI) of status epilepticus. Here we report upon the MR DWI and

apparent diffusion coefficient (ADC) map findings of status epilepticus in two cases.

## Case 1

A 56-year-old man was admitted for altered consciousness. The patient was afebrile and acyanotic but the altered consciousness, eye signs including eye blinking and nystagmoid movements, and a chewing movement were persistent. The cerebrospinal fluid examination was normal. The EEG showed continuous, semirhythmic, bifrontal sharp waves of 2Hz. Continuous EEG monitoring showed the sudden development of 8-Hz rhythmic waves on the left temporal area.

Brain MR imaging was performed during seizure activity lasting more than 30 minutes, using a 1.5T system (Signa Horizon, Echospeed; GE). Conventional MR imagings revealed hyperintensity on T2-weighted images and hypointensity on T1-weighted images, without

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a focal enhancing lesion in the bilateral cerebral cortices. DWI ( $b = 1000 \text{ s/mm}^2$ ) showed bilateral diffuse, gyriform cortical hyperintensity in the frontal, temporal, occipital and parietal lobes and insular cortices bilaterally (Fig. 1A). The ADC map showed hypointensity in the same areas (Fig. 1B). Compared with the ADC values of normal appearing cortices, which measured  $1,474 \pm 47 \times 10^{-6} \text{ mm}^2/\text{s}$ , the ADC values of T2-hyperintense areas were reduced and ranged from  $341 \pm 37 \times 10^{-6} \text{ mm}^2/\text{s}$  to  $570 \pm 36 \times 10^{-6} \text{ mm}^2/\text{s}$ . Midazolam administration was continued for 2 days and then tapered. EEG performed on hospital day 6 showed a generalized, diffuse, and mixed slowing of background activity. Afterwards, clinical symptoms gradually improved.

### Case 2

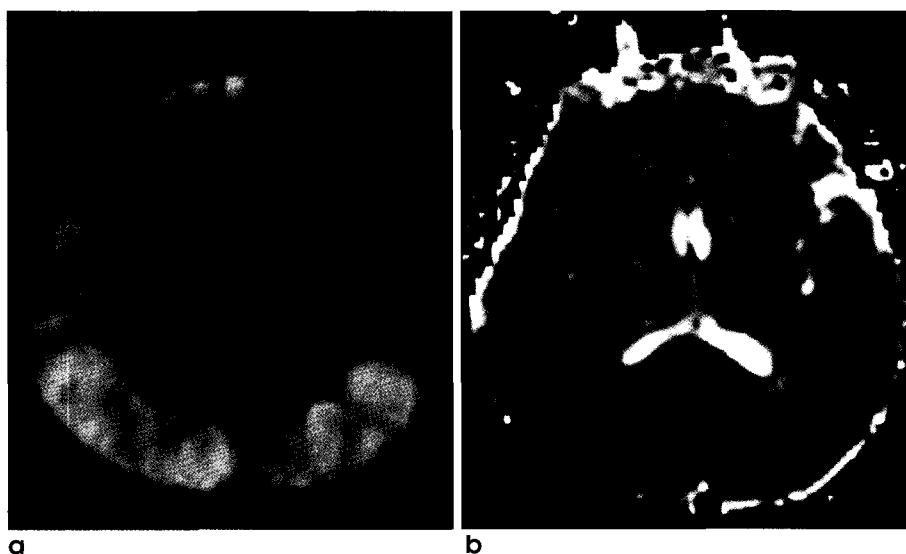
A 59-year-old man presented with headache and brief loss of consciousness. His medical history showed that he had experienced head trauma about 40 years earlier. He suffered from generalized tonic clonic seizures around 3–4 times per year over the 10 years period prior to presentation. Occasionally he has experienced gross convulsive movement without tongue bite, free voiding, and defecation for 1–2 minutes with postictal headache and fatigue. Before admission, he complained of a headache, vomiting, right eyeball deviation and intermittent loss of consciousness over a period of 1 week. After admission, his clinical symptoms continued and their duration extended. There was no evidence of fever, leukocytosis, or an abnormal CSF profile. During

an interval of loss of consciousness lasting some 3–5 minutes and without full recovery, brain MR imaging was performed on the same 1.5 T MR system as case 1.

Conventional MR images showed hyperintensity on T2-weighted images and hypointensity on T1-weighted images in the left posterior parietal lobe (Fig. 2A, B). DWI ( $b = 1000 \text{ s/mm}^2$ ) showed cortical hyperintensity in the left posterior temporal lobe (Fig. 2C). On the ADC map his lesion appeared hypointense (Fig. 2D). The ADC value of the lesion was measured  $756.7 \pm 203 \times 10^{-6} \text{ mm}^2/\text{s}$ , while that of the contralateral normal parietal lobe was  $1064.7 \pm 379 \times 10^{-6} \text{ mm}^2/\text{s}$ . At the same time, waking EEG showed several episodes of ictal rhythm with low voltage, and fast beta activities in the left parietooccipital lobe. The administration of diphenylhydantoin and valproic acid was continued. The patient's symptoms subsequently improved. Follow-up MR imaging 2 months later showed a normalized signal intensity on T2- and T1-weighted images in the same lesion (Fig. 2E, F).

### Discussion

Our two cases revealed decreased ADC values in the cerebral cortices during status epilepticus. These findings were in agreement with those previously reported (3–6). In the rat, ADC decreases of 14–49% were observed during kainite- or flurothyl-induced status epilepticus (3, 4). In human, gyriform hyperintensity was noted in the discharging cortex on DWI, without respect to vascular territories (5). The ADC values in the cortex

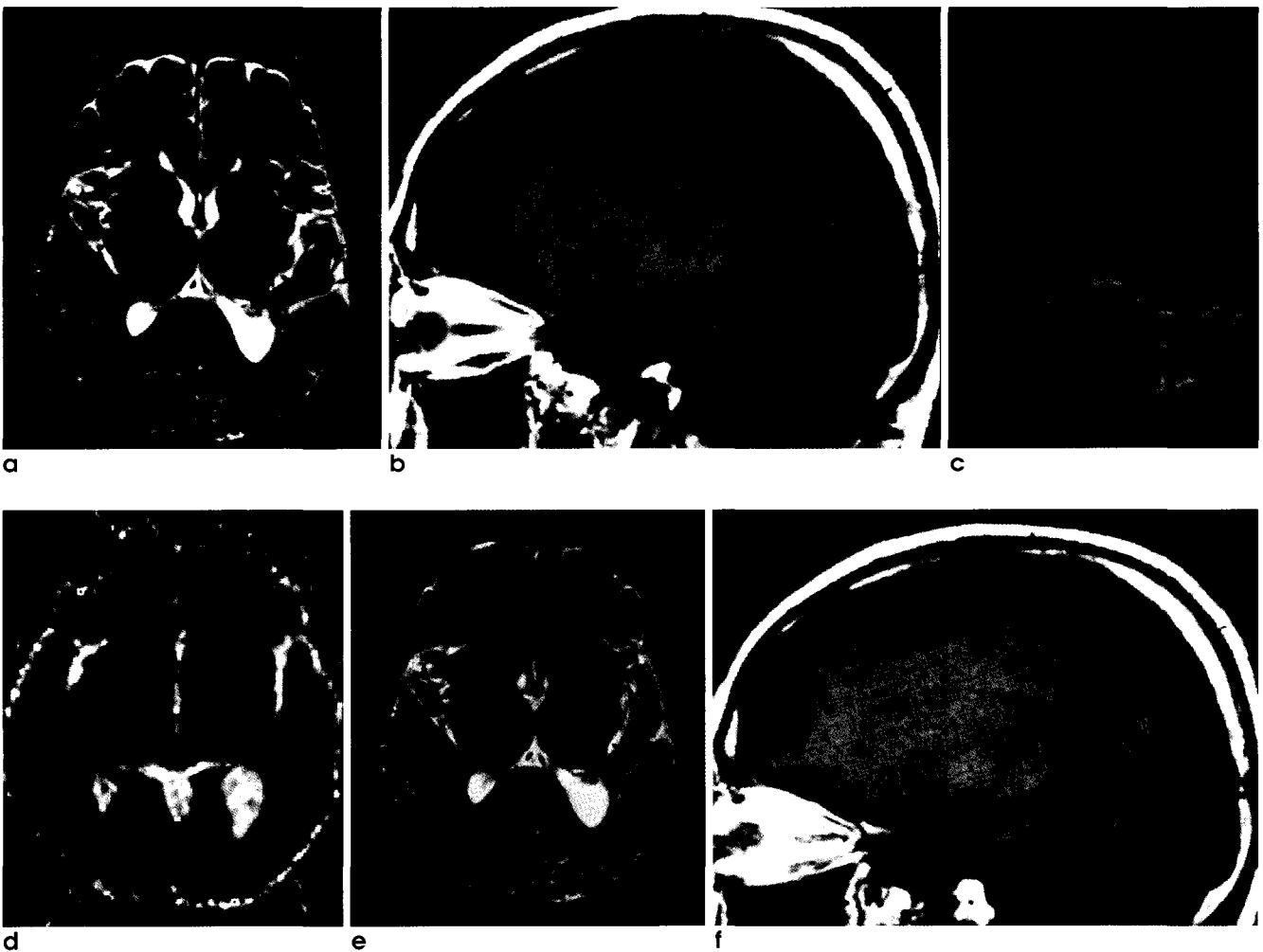


**Fig. 1.** **a.** DWI ( $b = 1000 \text{ s/mm}^2$ ) shows diffuse cortical hyperintensity in the frontal, temporal, parietal and occipital lobe and insular cortices bilaterally. The lesions of the left frontal and temporal lobes appear less hyperintense than those of the right side. **b.** ADC map image shows hypointensity of the lesions corresponding to the hyperintense areas on DWI.

showed a decrease, ranging from 18 to 36%, in versus the normal side (5). In our two cases, the ADC values of the hyperintense areas on DWI showed 23% and 29% decreases, respectively, compared with normal cortices.

The exact mechanism of decreased ADC in status epilepticus is uncertain. A hypothesis has been put forward, that it is due to primary membrane permeability changes, with extracellular potassium accumulation and influx of  $\text{Na}^+$  and  $\text{Ca}^{2+}$  ions, accompanied by water shift to the intracellular space, leading to swelling of glial cells and neurons (7, 8). The swelling of dendrites and astrocytes has been demonstrated histologically in an experimental study (10). In detail, with status epilepticus glucose and oxygen metabolism in the brain are el-

evated, which causes increased cerebral blood flow. If the compensatory increase of blood flow is insufficient, anaerobic metabolisms may take over, resulting in excess production of lactic acid and decreased phosphocreatine levels. In particular, in status epilepticus, the metabolism is markedly increased, leading to depletion of adenosine triphosphate and energy reserves at the later stage. This will cause subsequent impaired ion exchange pump function and will increase cellular membrane ion permeability, eventually resulting in an increase of extracellular potassium ion concentration and an accumulation of intracellular calcium ion together with a swelling of neuronal and glial cells, consistent with the findings observed in cytotoxic edema (9). The



**Fig. 2.** **a.** T2-weighted axial image shows slight hyperintensity in the cortex of the left posterior temporal lobe. **b.** T1-weighted sagittal image shows slight hypointensity with poor corticomedullary differentiation in the cortex of the left posterior temporal lobe. **c.** DWI ( $b = 1000 \text{ s/mm}^2$ ) shows focal cortical hyperintensity in the left posterior temporal lobe. **d.** ADC map shows hypointensity in the lesion of left posterior temporal lobe. **e.** Focal hyperintense lesion in the left posterior temporal lobe is normalized on follow-up T2-weighted axial image. **f.** Slightly hypointense lesion in the left posterior temporal is normalized on follow-up T1-weighted sagittal image.

increase of T2 signal intensity and the decrease of T1 signal intensity may be attributed to the increase in total amount of water in the epileptogenic brain tissue induced by increased perfusion and vascular permeability because of neuronal hyperactivity (1).

DWI and ADC map findings of status epilepticus with decreased water diffusion are similar to those of early cerebral ischemia. In the case of early cerebral ischemia, the cerebral blood flow decreases, resulting in subsequent glucose metabolic imbalance, impaired pump activity with increased permeability of the cell membranes, and cell necrosis (9). Location and extent of the lesions in status epilepticus may be various, and depend upon the duration of the seizure. In our case 1, some cortical areas of the cerebral hemispheres, basal ganglia and hippocampus were spared, while in our case 2 only the left posterior temporal area was involved. In case 1, the ADC value measured in the occipital lobes was much lower than those of other areas, even though the ADC maps showed a similar degree of hypointensity. The reason why decreased water diffusion varies in degree, extent, and location is not understood.

From the radiological point of view, differentiation of status epilepticus from some other neurologic disorders remains a challenge. Acute ischemic infarct and some acute encephalitis, in which water diffusion is decreased, may show similar findings on DWI and ADC maps. However, correct diagnosis can be made by correlating clinical manifestation and decreased water diffusion in the cerebral cortices irrespective of the vascular territory. In conclusion, MR DWI findings of status epilepticus are a focal or diffuse hyperintensity with decreased apparent diffusion coefficient (ADC) value in the cerebral hemispheric cortices because of cytotoxic edema with

cell membrane permeability changes.

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## 경련 증첩증 환자의 확산 강조 영상 소견: 2 증례 보고

서울대학교 의과대학 방사선과학교실, 서울대학교의학연구원 방사선의학연구소, 서울대학교병원 임상의학연구소

정성일 · 권배주 · 김건하 · 한문희 · 장기현

저자들은 2명의 경련 증첩증 환자의 확산 강조 자기공명영상을 보고하고자 한다. 확산 강조 영상은 대뇌 반구에서 세포독성 부종을 시사하는 현성확산계수의 감소가 동반된 국소적 또는 미만성 고신호강도를 보였다. 이러한 고신호강도는 한 환자에서는 양측 측두두정후두엽과 도피질, 다른 환자에서는 편측성 측두엽에서 나타났다.

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