Transient Occipitotemporal Subcortical Diffusion-Weighted Magnetic Resonance Imaging Abnormalities Associated With Status Epilepticus
—Case Report—

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Abstract
A 30-year-old man presented with a generalized seizure manifesting as decreased consciousness. Diffusion-weighted magnetic resonance imaging showed transient areas of high intensity in the gray and subcortical white matter of the left occipital and temporal lobes. The lesions did not reflect the vascular territories. After a period of over 2 weeks, his consciousness level improved associated with reduced intensity of the abnormal areas. These findings suggest that seizure induced reversible cytotoxic and vasogenic edema. Transient diffusion-weighted magnetic resonance imaging abnormalities may be associated with generalized seizures and the intensity may reflect the clinical condition.

Key words: diffusion-weighted magnetic resonance imaging, status epilepticus, reversible edema

Introduction
Magnetic resonance (MR) imaging has frequently been used as the primary imaging modality for identifying the anatomy related to the epileptogenic foci in the preoperative planning for surgical candidates with medically intractable extratemporal epilepsy. In particular, T₂-weighted or fluid-attenuated inversion recovery MR images have been useful for clarifying the target of resection. In fact, the clarity of the lesion on MR imaging has been correlated with the prognosis for postoperative seizure control, with more diffuse lesions associated with poorer prognosis.2,4,7,11,12) Furthermore, more recent methods of functional imaging such as positron emission tomography, single photon emission computed tomography, magnetic source imaging, and MR spectroscopy have all shown promise for use in the planning of epilepsy surgery as well as evaluation of patients with medically refractory seizures.13) Diffusion-weighted MR imaging shows the random movement of water as low intensity due to spin dephasing, and restricted movement of water as higher intensity. Recent prolonged seizure causing cytotoxic edema in acutely damaged cells results in the restriction of free water movement by cell membranes and intracellular water accumulation.10) Therefore, diffusion-weighted MR imaging is more likely to detect abnormal lesions with higher intensity in patients with longer episodes of status epilepticus or focal structural lesions than in patients with shorter seizures or without obvious parenchymal lesions.10)

We treated a patient with decreased consciousness due to generalized seizure. Diffusion-weighted MR imaging showed transient high signal intensity changes in the gray and subcortical white matter of the left occipital and temporal lobes which did not reflect the vascular territories. His consciousness level improved over 2 weeks, associated with reduced signal intensity, suggesting the presence of reversible cytotoxic and vasogenic edema induced by the seizure.

Case Report
A 30-year-old man who sustained a generalized seizure with persistent reduction of consciousness
level was transferred to our hospital for evaluation in October 2004. The patient had a history of cerebral palsy and recurrent generalized seizures since early childhood, presumably due to perinatal hypoxic-ischemic encephalopathy. Prior to admission, the patient had been treated with a triple antiepileptic regimen including carbamazepine (200 mg) two times a day, valproic acid (200 mg) two times a day, and phenytoin (200 mg) two times a day. Three months prior to admission, the patient suffered a generalized seizure and third degree burns covering greater than 30% body area. He was discharged on the day prior to the current admission. Intermuscular injection of 5 mg of diazepam and intravenous injection of 10 mg of diazepam were used to prevent status epilepticus during the generalized tonic-clonic seizure which occurred at the referral hospital prior to transfer to our hospital.

On admission, neurological examination revealed that the patient was somnolent and semi-obtunded with no evidence of Todd’s paresis. His consciousness level was 3-10 (Japan Coma Scale), with no response to verbal commands, no spontaneous eye opening, and no verbal production or grimace in response to pain, and the reticular activating-spinal reflex was intact. Laboratory data showed an elevated white blood cell count of 18,100/μl, and increased creatine phosphokinase activity of 1,151 IU/l. Vital signs were consistent with the observed, generalized epileptic seizure, including body temperature of 38.4°C, oxygen saturation of 97% (room air), and blood pressure of 136/77 mmHg.

Diffusion-weighted MR imaging demonstrated extensive areas of high intensity in the gray and subcortical white matter of the left occipital lobe and the left temporal lobe (3 × 12 cm and 5 × 6 cm, respectively) (Fig. 1). There was no evidence of mass effect or narrowing of the adjacent sulci or subarachnoid space. Cranial computed tomography and MR angiography showed no abnormalities. Over the course of 2–3 weeks the patient gradually regained consciousness, but verbal communication was limited to several words. MR imaging with contrast medium showed no sign of blood-brain barrier disruption. Diffusion-weighted MR imaging performed 1 month after admission revealed nearly complete disappearance of the high intensity areas demonstrated on admission (Fig. 2). Electroencephalography performed in the acute post-ictal phase revealed epileptogenic spike waves with high voltage and slow wave background activity in the temporal and occipital areas of the dominant lobe (Fig. 3). These abnormalities correlated well with the findings of diffusion-weighted MR imaging. The patient remained seizure-free throughout the hospital course.

On discharge the patient showed persistent mild difficulty with speech and slightly decreased consciousness level. After discharge, the patient remained seizure-free at follow-up examinations.

Discussion

A seizure (from the Latin sacire, “to take possession of”) has been defined as a paroxysmal, self-limited change in behavior associated with excessive electrical discharge from the central nervous system, whereas epilepsy is a condition of recurrent seizures due to a chronic, underlying process.11,13,16] Approximately 5% to 10% of the general population will have at least one seizure during their lifetime in contrast to a 0.3% to 0.5% incidence of epilepsy.16]
Fig. 3 Electroencephalography performed in the acute post-ictal phase revealing epileptogenic spike waves with high voltage and slow wave background activity in the temporal and occipital areas of the dominant lobe.

A few case reports have documented patients with status epilepticus and transient changes on diffusion-weighted MR imaging.1,9,22) Our patient had a clear history of cerebral palsy, a developmentally regulated presumptive epileptogenic factor, which may have lead to a chronically hyperexcitable condition. Cerebral palsy is associated with a higher incidence of seizure disorders (15–60%), with onset in the neonatal period in most cases.5,14) In our patient, diffusion-weighted MR imaging showed areas of high intensity in the gray and subcortical white matter of the left occipital and temporal lobes which gradually diminished in intensity over a 1 month period. These findings suggest that vasogenic edema might have been the predominant pathology in our patient with a generalized tonic-clonic seizure.

The changes in intensity on diffusion-weighted MR imaging may be related to the histological changes associated with focal status epilepticus, as swelling of astrocytes and dendrites in the affected brain have been observed 2 to 24 hours after systemic injection of kainic acid.3) Similar observations have been made on acute cerebral ischemia, with initial decrease in intensity on diffusion-weighted MR imaging reflecting cytotoxic edema and an increase in intensity after cell fragmentation.15) Similarly, increased intensity and decreased apparent diffusion coefficient values which reflect cytotoxic edema were observed in the epileptogenic area of animal models.10) Increased intensity could also be explained by acidosis and the breakdown in the blood-brain barrier, and subsequent vasogenic edema caused by regional hyperperfusion and increased vascular permeability seen in clinical cases of partial seizures.8,19,21) In humans with partial status epilepticus, vasogenic edema is considered to be the most probable mechanism for reversible changes seen on MR imaging.8,19)

Cytotoxic edema after prolonged seizure is caused by water shifting from the extracellular space to the intracellular compartment, so that diffusion of water is restricted by cell membranes in the acutely damaged cells. Further evidence of blood-brain barrier breakdown and vasogenic edema has been shown in humans with prolonged partial status epilepticus, as angiography showed capillary blush in the focal area and computed tomography demonstrated focal cerebral edema.20) Furthermore, patients with recent episodes of status epilepticus are more likely to show areas of abnormal intensity on diffusion-weighted MR imaging than patients with shorter seizures due to the greater degree of cellular damage at the epileptogenic focus.9) Patients with other structural focal lesions (neoplasm, vascular malformations, etc.) are also more likely to show areas of abnormal high intensity than patients without obvious parenchymal lesions.9)

In our patient, we were unable to perform apparent diffusion coefficient or single photon emission computed tomography studies but the diffusion-weighted MR imaging changes were highly suggestive of vasogenic mechanisms. It is important to note that vasogenic edema is usually considered reversible in comparison to cytotoxic edema which is irreversible. These findings are supported clinically in our case by the fact that the presumptive near resolution of vasogenic changes on diffusion-weighted MR imaging were consistent with the clinical changes in the patient. The minimal irreversible clinical and neuroimaging changes suggested no major associated cytotoxic changes. However, the slight residual persistent abnormalities on diffusion-weighted MR imaging may be explained by prior recurrent episodes of seizures, and the associated clinical signs of persistently reduced consciousness level may be explained by this permanent cytotoxic factor.

The areas of high intensity were mainly located in the gray and subcortical white matter of the left occipital and temporal lobes in our patient. Similar anatomical findings have been reported, notably that MR imaging changes after generalized tonic-
clonic seizure or status epilepticus are usually transient increases in signal intensity and signs of swelling at the cortical gray matter, subcortical white matter, or hippocampus on perictal T2-weighted and diffusion-weighted MR imaging.

In conclusion, vasogenic edema is commonly associated with seizures. In contrast to the cytotoxic changes found in ischemic vascular lesions, the changes caused by seizures are transient and may appear as reversible changes on diffusion-weighted MR imaging. Furthermore, in patients presenting with loss of consciousness and focal neurological signs, extensive areas of high intensity in the gray and subcortical white matter of the left occipital and temporal lobes may indicate seizure and not ischemic infarction. Recognition of transient subcortical changes on diffusion-weighted MR imaging can be important in excluding other ischemic processes and epileptogenic structural lesions.

References


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