



Case Report

Temporary and Permanent Magnetic Resonance Imaging Findings in Status Epilepticus: Case Series

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Abstract

Acute periictal imaging abnormalities caused by status epilepticus (SE) have been reported in recent years. In this study, periictal magnetic resonance imaging (MRI) changes, follow-up MRIs and electroencephalographic (EEG) features (e.g. periodic lateralized epileptiform discharges [PLEDs]) of 4 patients with SE were discussed. Diffusion restriction was the most common periictal MRI change (4/4) and it tended to resolve (3/4). Permanent MRI changes were seen in two patients. Patients with SE and periictal MRI changes mostly had PLEDs in EEG (3/4) and poor outcomes (3/4).

Keywords: Status epilepticus, peri-ictal MRI, diffusion weighted imaging, periodic lateralized epileptiform discharges

Status Epileptikusta Geçici ve Kalıcı Manyetik Rezonans Görüntüleme Bulguları: Olgu Serisi

Özet

Status epileptikusun (SE) sebep olduğu akut periiktal görüntüleme anormallikleri son yıllarda bildirilmiştir. Bu çalışmada, 4 SE tanılı hastanın periiktal manyetik rezonans görüntüleme (MRG) bulguları, takip MRG'leri ve elektroensefalografik (EEG) özellikleri (örneğin periyodik lateralize epileptiform deşarjlar [PLED'ler]) tartışılmıştır. Difüzyon kısıtlaması en sık görülen periiktal MRG değişikliği olmuştur (4/4) ve bunun düzelleme eğiliminde olduğu görülmüştür (3/4). Kalıcı MRG değişiklikleri 2 hastada görülmüştür. Periiktal MRG değişiklikleri görülen SE tanılı hastaların EEG'lerinde PLED'ler sıklıkla (3/4) görülmüştür ve bu hastaların sıklıkla kötü sonlanıma sahip olduğu görülmüştür (3/4).

Anahtar Kelimeler: Status epileptikus, periiktal MRG, difüzyon ağırlıklı görüntüleme, periyodik lateralize epileptiform deşarjlar

INTRODUCTION

Cranial magnetic resonance imaging (MRI) findings can be seen in convulsive and non-convulsive SE (NCSE)^(2,10,11). These findings can be seen both in the area of the epileptic discharges and in non-related localizations, and are believed to be caused by compensatory increased blood

flow to supply the metabolic requirements of the effected neurons. Hyperintensities in diffusion weighted imaging (DWI), fluid attenuated inversion recovery (FLAIR) and T2, and hypointensities in apparent diffusion coefficient (ADC) are the most common abnormal features⁽⁴⁾. Diffusion restrictions in acute phase of SE may transform to atrophy in time and cause

neurologic sequela^(1,2,9). But why these imaging abnormalities occur in some but not all patients and their clinical importance are unknown. In this study, periictal MRI changes, follow-up MRIs and EEG features of 4 patients diagnosed as SE will be reviewed.

CASE PRESENTATION

CASE-1

Eighty-three-year-old female who had herpes encephalitis one year ago admitted to emergency service with confusion and clonic seizures in the left part of her body. Patient was diagnosed as epilepsy partialis continua (EPC) and treated with 20 mg intravenous (IV) diazepam followed by 1000 mg IV phenytoin (PHT). Clonic jerks disappeared partially but her confusion persisted. Right frontotemporal rhythmic unorganized sharp waves in EEG indicated NCSE. On the third day, DWI-ADC MRI showed cortical diffusion restriction in the right temporoparietal lobe. She was somnolent and left hemiparetic in neurological examination. Total dose of

6000 mg IV levetiracetam (LEV) was administered and continued with 2500 mg/day LEV and 400 mg/day carbamazepine (CBZ). On the seventh day, her consciousness improved but she still had left hemiparesis. EEG also changed from SE to right fronto-centro-temporal periodic lateralized epileptiform discharge (PLED) activity. On the tenth day, diffusion restriction improved compared to previous MRI but the right temporoparietal lobe was seen hyperintense in FLAIR sequence. On the 19th day, PLEDs disappeared with little improvement in clinical status and she was discharged. After 7 months, EEG demonstrated mild generalized slowing. DWI-ADC MRI was normal but cortical atrophy predominantly in right temporoparietal area was observed in FLAIR sequenced images. Left hemiparesis persisted and mental functions were worsened with time. She died due to systemic infection 1 year later. EEG and cranial MRI samples were shown in figure-1

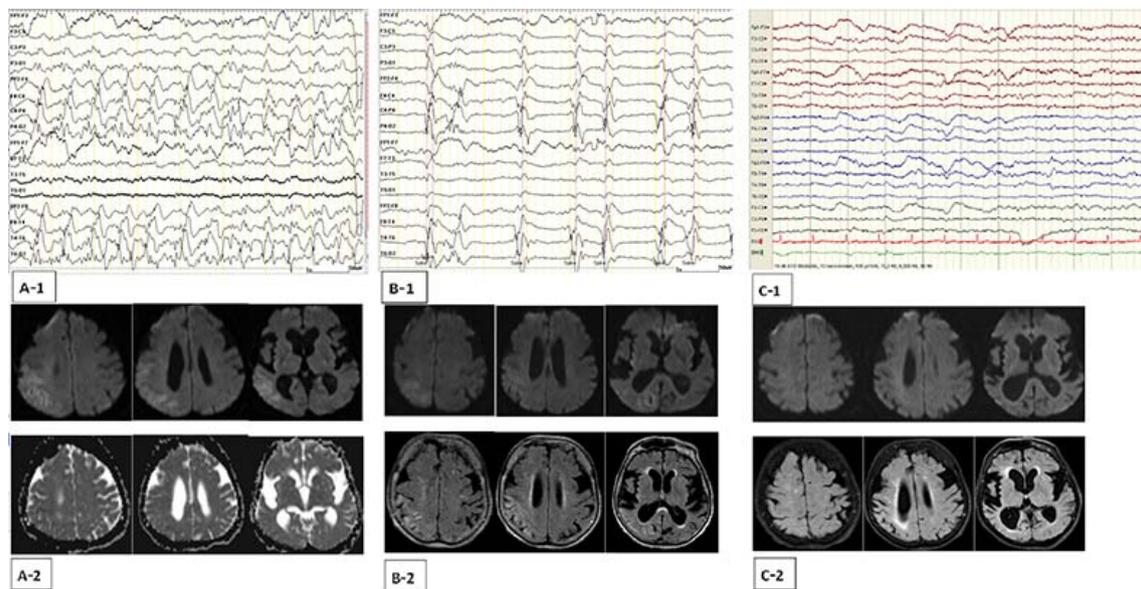


Figure 1: (A-1) EEG shows right hemispheric electrophysiological SE pattern. (A-2) Cranial MRI which was performed simultaneously with EEG, shows right temporoparietal diffusion restriction. (B-1) EEG which was performed 1 week after the first, shows diffused slowing more over the right hemisphere and PLED activity in the right hemisphere (B-2). Iv diazepam was not administrated during PLED activity on EEG. Cranial MRI shows improvement of diffusion restriction but this part now seems hyperintense in FLAIR. (C-1) EEG, which was performed 11 months later, shows mild diffused slowing in the background activity. (C-2) Cranial MRI which was performed 4 months later, shows recovery of diffusion restriction but also increased cortical atrophy with enlarging of right lateral ventricle.

CASE-2

Forty-two-year-old female, who had a diagnosis of Chron's disease in 2004, was hospitalized in the internal medicine service because of diarrhea, abdominal pain and fever, and referred to neurology for seizure which presented with deviation of head and eyes to the right and then secondary generalization. Generalized slowing was observed in EEG during postictal period. In cranial MRI, there were multiple T2 and FLAIR hyperintense lesions without contrast enhancement near lateral ventricles. Glucose and protein levels were normal in cerebrospinal fluid (CSF) and also there were no cells counted. CSF was positive for oligoclonal bands with "pattern 3" which means both intrathecal and systemic IgG production. No antiepileptic drugs were administered and she was discharged from hospital with anti-TNF therapy. Periventricular lesions in MRI and presence of oligoclonal bands suggested an autoimmune demyelinating process. But she refused to be investigated.

One year later, she was again admitted to hospital with infection. On the first day of piperacillin tazobactam therapy continuing jerks on her left arm suggestive of EPC appeared and she became left hemiparetic. She was diagnosed as EPC and was given 30 mg IV diazepam and 1250 mg IV PHT for two days. CBZ was given for maintenance. Cranial MRI showed same periventricular lesions with the addition of right temporooccipital hyperintense lesion on FLAIR which also showed diffusion restriction. EEG showed right temporal highly active epileptic focus. There were no inflammatory cells in lomber puncture with normal glucose and protein levels. Oligoclonal bands were again positive with "pattern 3". One week later, she died due to febrile neutropenia and septic shock. EEG and cranial MRI samples were shown in figure-2

CASE-3

Seventy- three-year-old female patient, who had productive coughing, hallucinations and disorientation for 3 days, was admitted to hospital. She suffered from left hemiparesis and diagnosed with ischemic stroke five months ago. She was confused and had left homonymus hemianopsia and left hemiplegia as sequela in neurological examination. EEG revealed PLED activity with 1-1,5 s frequency in the right occipital hemisphere which disappeared with IV diazepam administration and thought as an ictal activity. Her mental status improved and she started to recognize her relatives but she was still hallucinating. For maintenance, 2000 mg/daily LEV and 100 mg/daily topiramate were given orally. On the fifth day of her hospitalization, she had a continuing seizure with clonic jerks in the middle finger of her left hand, left toe, abdomen and right leg which was thought to be EPC. One thousand five hundred mg IV LEV and 1500 mg IV PHT were administered. DWI-MRI which was performed on the day of EPC onset showed diffusion restriction in the right occipital region accompanied with ADC hypointensity in the left occipital lobe. These defects improved but mild T2 hyperintensity was observed in right occipital region in follow-up MRI, which was done after 12 days of the seizure onset. She was fully oriented and had fewer hallucinations by the time she was discharged. She was reported to have no seizures after that but occasional hallucinations and confusion. She died due to cardiac problems 6 months later. EEG and cranial MRI samples were shown in figure-3

CASE-4

Seventy-one-year-old male was admitted to emergency service with acute loss of vision, monoparesis of the right arm and speech problems. He was on warfarin for previously diagnosed atrial fibrillation and

ischemic stroke. He had Wernicke aphasia, right homonymous hemianopsia, and right hemiparesis in neurological examination. Cranial MRI showed diffusion restriction in the right temporoparietal lobe and chronic infarction on the left MCA area. INR was 2.43. Doppler ultrasonography of carotid and vertebral arteries were normal. EEG showed electroclinical seizure activity originating from left occipitoparietal area and PLED activity

dominantly in the left temporal region. LEV was administrated orally at 1000 mg/daily dose. Follow-up EEG which was performed on the fifth day showed generalized slowing mainly on the left hemisphere. Diffusion restriction was decreased in the control MRI. He had full recovery one month after the onset of symptoms. EEG and cranial MRI samples were shown in figure-4

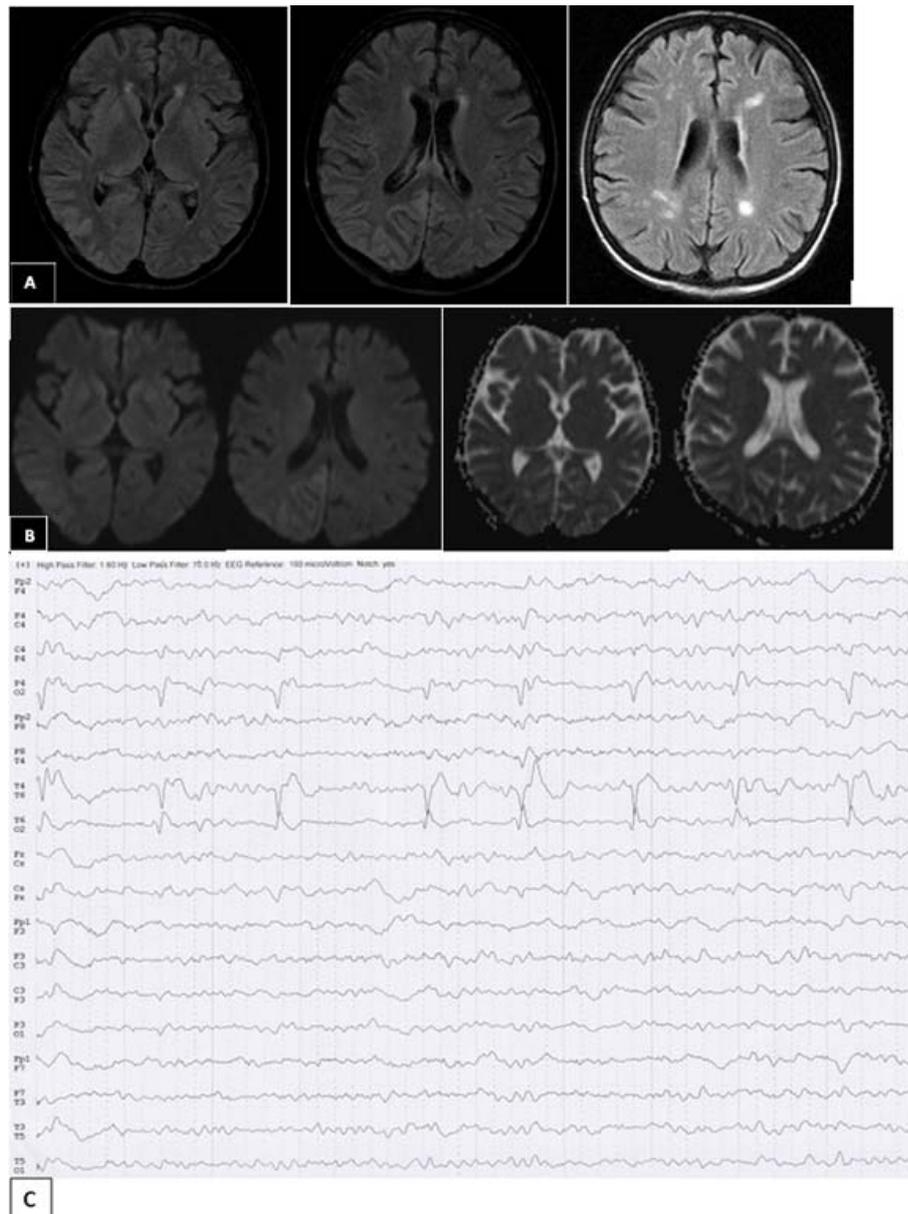


Figure 2: (A) Cranial MRI which was performed 4 days after seizures started, shows right temporooccipital hyperintensity on FLAIR and periventricular lesions. Periventricular lesions were also seen in the MRI which was performed in the previous year. (B) DWI-ADC shows right temporooccipital diffusion restriction. (C) EEG which was performed 2 days after MRI, shows diffused slowing predominantly in the anterior sides of the right hemisphere with highly active epileptic focus in the right temporal region.

DISCUSSION

The period during or just after the seizure is referred to as the peri-ictal period. Peri-ictal imaging findings may be observed in the region of the epileptic discharge or in remote locations. Remote peri-ictal imaging changes including posterior leukoencephalopathy, unilateral/bilateral diencephalic lesions, contralateral cerebellar lesions (diaschisis) and splenic lesions are believed to occur because of the abnormal changes driven by the epileptic activity with no clear pathogenetic mechanisms. Local peri-ictal changes arise due to mass effect, hippocampal swelling, focal cortical lesions, migrating lesions, blood-brain barrier impairment and increased vessel caliber/flow⁽⁴⁾.

Diffusion restriction often indicates irreversible ischemic damage; however, it may be reversible when it is associated with seizures⁽⁷⁾. Acute ischemia presents with diffusion restriction without T2 signal abnormality, while their combined appearance during SE suggests a difference between them with regard to the underlying pathophysiology⁽⁴⁾. During SE, if the compensatory increase in cerebral blood flow can't fulfill the need of epileptic tissue, resultant pathophysiological events lead to cytotoxic edema and reduced extracellular volume (detected by decrease on ADC)⁽⁵⁾. Blood-brain barrier impairment and possible cell death result in vasogenic edema and increased water diffusion⁽¹⁶⁾. In the epileptogenic region, either cytotoxic or vasogenic edema leads to increased⁽¹⁴⁾, decreased⁽¹³⁾ or both increased and decreased⁽⁹⁾ ADC. MR studies in partial SE cases show vasogenic edema as the likely reason behind the reversible nature of the lesion⁽⁸⁾.

Acute peri-ictal imaging changes may surface in days or weeks following the onset of focal seizures. This indicates that duration or severity of the seizure should exceed a certain individual threshold in order to induce acute peri-ictal changes⁽⁴⁾.

There are only a few studies on peri-ictal MRI changes including long-term follow-up assessments. In these studies, first cranial MRI was performed in 10-48 hours after the onset of SE and follow-up MRI was performed 1 week-18 months afterwards in a heterogenous manner. Although diffusion restriction showed improvement, most patients showed permanent MRI changes^(2,10,11).

The first cranial MRI assessments of our patients were performed in 1 to 5 days after the onset of seizures. All showed diffusion restriction, whereas case 2 also manifested increased FLAIR signal synchronously. However, since this patient died, we were unable to perform a follow-up assessment. Diffusion restriction resolved completely in case 1 and 4 and partially in case 3, but case 1 and 3 exhibited permanent MRI changes. Left hemiplegia and cognitive impairment were also observed as sequela in case 1. Three patients displayed consistent MRI and EEG findings; however, interestingly, they were inconsistent in case 4 in whom acute ischemic stroke could not be ruled out. Three patients had poor prognosis while case 4 completely recovered.

Clinical seizures and PLEDs are often observed together and these seizures are generally in the form of EPC or frequently repeating focal seizures^(3,12) as well as occasional NCSEs⁽¹⁵⁾. Since PLEDs are commonly encountered with acute cerebral events, it is not surprising to observe focal neurological deficits in many patients^(3,12). PLEDs are believed to arise from the impaired interaction between the cortical and subcortical structures⁽⁶⁾. Patients with PLEDs can also display MRI changes^(2,10). In a case series all patients with PLEDs and MRI changes except one were comatose or lethargic with regard to outcome⁽¹⁰⁾. In our study, cases 1, 3 and 4 had PLEDs on EEGs. PLEDs were thought to be ictal in case 3 but in case 1 and 4 differential diagnosis could not be made

since IV diazepam was not administered during PLED activity on EEG. While case 1 and 3 had poor prognosis, case 4 completely recovered.

There is need for further investigations focusing on the process starting with the temporary DWI-ADC changes and proceeding until the occurrence of the irreversible neurological deficits, and the correlation between the evolution of the electrophysiological findings in SE and the development of irreversible neurological deficits⁽⁴⁾.

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Received by: 31 March 2016

Accepted: 14 July 2016

The Online Journal of Neurological Sciences (Turkish) 1984-2016

This e-journal is run by Ege University Faculty of Medicine, Dept. of Neurological Surgery, Bornova, Izmir-35100TR

as part of the Ege Neurological Surgery World Wide Web service.

Comments and feedback:

E-mail: editor@jns.dergisi.org

URL: <http://www.jns.dergisi.org>

Journal of Neurological Sciences (Turkish)

Abbr: J. Neurol. Sci.[Turk]

ISSNe 1302-1664

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