COMPLEX PARTIAL SEIZURE-INDUCED TRANSIENT MAGNETIC RESONANCE CHANGE IN SLE AS FIRST MANIFESTATION

Ayhan BÖLÜK, MD *
Kaya SARAÇ, MD **
Sinem KALI, MD *
Cemal ÖZCAN, MD *

We report a case of woman with SLE (systemic lupus erythematosus) woman with transient abnormalities on magnetic resonance imaging (MRI) associated with partial status epilepticus (SE). Her first clinical signs were complex partial seizure, with temporal maximum on ictal EEG. Intensity of the left temporal lobe was increased on MRI T2 during SE, but neuroimaging was normal two months later. Focal cerebral MRI abnormalities consistent with cerebral edema may be due to partial status epilepticus. In a patient with neuropsychiatric SLE, especially with suspicious seizures with clinical and EEG changes should be correlated with MRI.

***Key words**: Magnetic resonance imaging, status epilepticus, partial epilepsy, cerebral edema, systemic lupus erythematosus.*

SLE'de ilk manifestasyon olarak kompleks parsiyel nöbetin ortaya çıkardığı geçici manyetik rezonans değişikliği

Bu yazıda parsiyel status epileptikus (SE) ile ilişkili geçici manyetik rezonans görüntüleme (MRG) anormallikleri olan SLE (Sistemik Lupus Eritrematosis)'li bir kadın olgusunu sunmaktayız. İlk klinik belirtisi iktal EEG'de temporal maksimum ile beraber kompleks parsiyel nöbetti. Sol temporal lobun intensitesi T2 ağırlıklı MR görüntülerinde artmıştı, fakat iki ay sonraki nörogörüntüleme normaldi. Serebral ödemle uyumlu fokal serebral MRG anormallikleri parsiyel status epileptikus yüzünden meydana çıkmış olabilir. Nöropsikiyatrik SLE'si olan, özellikle klinik ve EEG ile şüpheli nöbetleri olan, hastada değişiklikler MRG ile korrele edilmelidir.

***Anahtar kelimeler**: Manyetik rezonans görüntüleme, status epileptikus, parsiyel epilepsi, serebral ödem, sistemik lupus eritrematosis*

* Department of Neurology İnönü University School of Medicine, MALATYA, TURKEY.
** Department of Radiology İnönü University School of Medicine, MALATYA, TURKEY.

Neuropsychiatric (NP) systemic lupus erythematosus occurs in 50% with SLE patients and produces diverse manifestations[1]. Clinical presentations are highly variable, with confusions, disorientation and often decreased consciousness[2]. All SLE patients with encephalopathy should have a complete evaluation for seizures. Cerebral MRI is a method of choice for non-clinical diagnosis of NP-SLE[3]. Focal abnormalities in brain MRI occur frequently in SLE, especially in patients with focal neurologic deficits[4]. We report a patient who developed transient focal hyperintensity on MRI during complex partial seizure.

Correspondce address:
Dr. Ayhan Bölük
İnönü Üniversitesi Tıp Fakültesi, Nöroloji Anabilim Dalı, Malatya, Türkiye

Tel: (422) 3410660-70.
Fax: (422) 3410729, 3254132.
Email:ayhanb@aidata.com.tr

CASE

A 29 year-old female was admitted to our hospital because of the clouding of consciousness and fever. She was confused and agitated, repeating her adress continously as answer to any question. The examination of the cranial nerves was normal. There was no lateralizing signs and other abnormalities in the neurologic examination. She had fever, 38.0°C. The systemic evaluation was normal. She had no neck stiffness. A waxing and waning state alternating between agitated-confusion with lethargy continued during the next twenty days. She developed focal motor seizures in the right side and the subtle face and limb myoclonus lasting several days after admission. She devoleped pericardial effusion and hematologic abnormalities including leukopenia, lymphopenia and thrombocytopenia. She was not taking any drugs known to induce thrombocytopenia.

Sedimentation rate was high (80 mm/h). Antibodies to Sm were positive in her serum. Lupus anticoagulant and anticardiolipin antibodies (IgG) were also detected. The C3 and C4 complement values were low. The LE was positive. Cerebral spinal fluid (CSF) examination revealed elevated protein level, albuminocytological dissociation and decreased glucose level .

Electroencephalography (EEG) showed abnormal activity in the both hemispheres, mainly dominant in the left temporo-fronto-central regions. There was prominent at 7-8 Hz high amplitude sharp wave activity lasting all record (Figure 1). Intravenously diazepam suppressed this activity, together with some improvement in the consciousness.

T2 weighted sections of magnetic resonance imaging (MRI) study showed high signal intensity in the left temporal lobe. The intensity changes was seen both in neocortical and mesial region of the left temporal lobe (Figure 2).

She was considered SLE. Two courses of methyl prednisolon pulse therapy were administered.The clinical signs showed improvement with prednisolon. She had no clinical symptoms and signs in her control examination after two months. Previously observed abnormal findings in the temporal lobe on MRI had also disappeared after two months of the episode.

A B

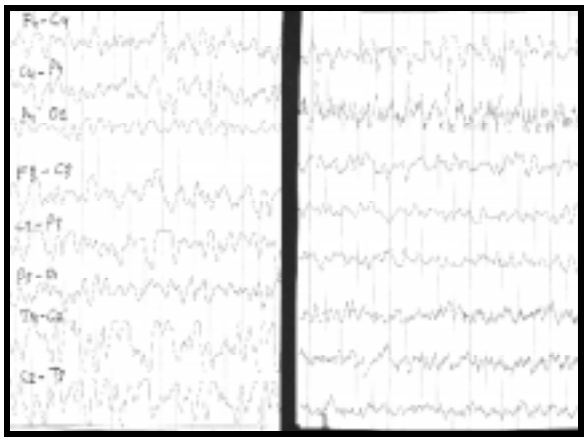

Figure 1. Electroencephalographic recording during complex partial status (A) and after diazepam injection (B).

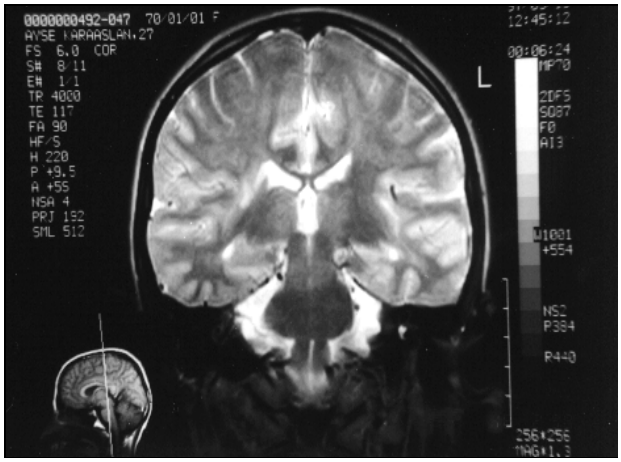

Figure 2. Large focal area as of increased signal intensity at T2 weighted image MRI in the left temporal lobe.

DISCUSSION

In this case peport we present transient changes in magnetic resonance imaging (MRI) scan in an SLE patient with complex partial status epilepticus. Complex status epilepticus was the first and only manifestation of central nervous involvement. MRI showed increased signal intensity in left temporal lobe without

enhancement. The prednisolon therapy caused clinical improvement including complex partial status epilepticus. Also, MRI changes showed resolution after prednisolon therapy.

Systemic lupus erythematosus (SLE) is a multisystemic inflammatory disease, which characterised by highly variable clinical manifestations. In SLE there is a predilection for clinical involvement of the skin, joints, kidneys, nervous system, serosal surfaces, and blood elements[5]. Seizure and psychosis currently considered to be diagnostic of primary SLE-mediated central nervous system (CNS) involvement by American Romatism Association (ARA)[6].

The neurologic involvement including stroke, seizure, dementia, psychosis, coma, encephalopathy, peripheral neuropathy, and myositis has been described in SLE[2].

The pleomorphic clinical presentation of nonconvulsive SE indicates that EEG and a therapeutic trial of antiepileptic drugs afford the best diagnostic measures in acute waxing and waning confusional states associated with agitation, bizarre behaviour, staring, increased tone, mutism, or subtle myoclonus[7]. This disorder may be divided into generalised (absence) or partial (complex partial) forms[8].

The complex partial seizure activity is either focal (usually temporal lobe) or seconder generalised from a focal pacemaker[9].. The ictal discharges have been detected generalised, diffuse with focal predominance, and focal. Demonstration of focal epileptic features in response to intravenous (I.V.) diazepam and presence of interictal focal epileptiform discharges in some cases have been suggested possible focal onset seconder generalized cases [10]. Neuroimaging studies in patients with complex partial seizures demonstrate the epileptogenicity of a more or less localized underlying lesion. Correlation with the topography of the EEG focus permits affirmation of origin of seizure[11] .

MRI findings of SLE has been reported as four groups : 1) large focal areas as of increased signal intensity at T2 weighted image, 2) patchy subcortical foci, 3) cerebral infarcts signs, 4) normal findings. Large focal area of increased signal intensity at T2 weighted images were observed in patients SLE with seizure and with encephalopathy[12].

Stroke is an important cause in the differential diagnosis of focal MRI changes in patients with SLE. Although focal and diffuse neurologic abnormalities in SLE are often attributed to "CNS vasculitis", autopsy suggest that this is rare[13,14] . The cause of stroke in SLE is generally cardiogenic embolism or a hypercoagulable state [15].

The transient focal cerebral abnormalities on computed tomography and magnetic resonance imaging (MRI) has been known in the partial status epilepticus. Most of these reports suggest focal cerebral edema as the basis of these transient neuroimaging abnormalities. Focal cerebral MRI abnormalities consistent with cerebral edema may be due to partial SE but also may indicate underlying glioma[16]. This transient seizure-induced MR changes has been consistent with ictal episod or postictal hyperemia and breakdown of the blood-brain barrier and should be erroneously attributed to mesial temporal sclerosis, encephalitis, tumor, or infarction[17].. Abnormal findings of MRI in SLE patients with seizures has been reported[11] and a 10 years-old girl with status epilepticus[18], which was almost completely resolved after treatment.

The transient MRI changes of our case also probably represented focal cerebral edema, developing during focal status epilepticus. This has been supported by absence of abnormalities MRI on the right, lower amplitude of ictal electrical activity than the right hemisphere and resolution of changes with treatment. In patients with neuropsychiatric SLE, especially suspected seizures clinical signs and electroencephalografic changes should be correlated before interpretation is made of focal lesions seen by magnetic resononces imagings.

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