Nonconvulsive Status Epilepticus

Nonkonvulsif Status Epileptikus

Ayhan AKÖZ,[1] Lütfi ÖZEL,[2] Atıf BAYRAMOĞLU,[1] Murat SARITEMUR,[1] Recep DEMİR,[2] Zeynep GÖKCAN ÇAKIR[1]

[1]Department of Emergency Medicine Atatürk University, Faculty of Medicine, Erzurum;
[2]Department of Neurology, Atatürk University, Faculty of Medicine, Erzurum

SUMMARY

Status epilepticus (SE) can be divided into two subgroups, convulsive status epilepticus (CSE) and nonconvulsive status epilepticus (NCSE). NCSE is clinically characterized by recurrence or persistence of absence or complex partial seizures which unconsciousness persists. Seizures are lasted more than 30 minutes, and accompanied by mental and behavioral changes. Although the main feature is the change of level of consciousness, affective, memory, cognitive, speech, motor systems, behavioral and psychiatric disorders can be also seen. Electroencephalography (EEG) is the single diagnostic method in this situation. NCSE constitutes approximately 25% of all SE, however it is thought that sometimes it is misdiagnosed and the incidence of NCSE may be higher. The causes are disorders of central nervous system (stroke, infection, trauma, tumor), and metabolic factors (hypoxia, renal diseases, drugs, failure to use antiepileptic drug). The treatment contains the standard therapy of SE. Short-acting benzodiazepines are preferred in initial treatment. For more resistant cases, loading of phenytoin is applied. If there is no response to treatment, midazolam and propofol additionally to barbiturates can be used. We aimed to present a case admitted to our ED with altered mental status and diagnosed as NCSE that is under-diagnosed.

Key words: Electroencephalography; emergency department; nonconvulsive status epilepticus; suspicion.

ÖZET

Status epileptikus (SE), konvulsiv status epileptikus (KSE) ve nonkonvulsiv status epileptikus (NKSE) olarak iki grupta ele alınabilir. NKSE, klinik olarak 30 dakikadan fazla süren mental durum ve davranış değişikliği ile şekillenen absans ya da kompleks parsiyel nöbetlerin bilincin açılamayacak kadar sık tekrarlaması veya sürekli devam etmesidir. Bilinç değişikliği temel özellik olmakla birlikte, affektif, bellek, kognitif, konuşma, motor sistem, davranış ve psikiyatrik bozukluklar görülebilmektedir. Elektroensefalogram (EEG) tek tanı koydurucu yöntemdir. Tüm SE'lerin yaklaşık %25'ini oluşturan NKSE'nin gerçek oranının daha yüksek olabileceği, bazen tanınamadığı düşünülmektedir. Nedenleri arasında merkezi sinir sistemi hastalıkları (inme, enfeksiyon, travma, tümör) ve metabolik etkenler (hipoksi, renal hastalıklar, ilaç, antiepileptik ilaç aksatımı) yer almaktadır. Prensip olarak standart SE tedavisi uygulanmaktadır. Akut tedavide kısa süreli benzodiazepinler ilk sırada tercih edilmektedir. Daha dirençli vakalarda fenitoin yükleme uygulanırken, çok dirençli olgularda barbitüratların yanısıra midazolam veya propofol de kullanılabilir. Biz acil servisimize değişken bilinç durumu ile başvuran ve NKSE tanısı koyduğumuz bir vakayı sunmayı amaçladık.

***Anahtar sözcükler**: Elektroensefalografi; acil servis; nonkonvulsif status epileptikus; şüphe.*

Introduction

Status epilepticus (SE) is divided into two groups as convulsive status epilepticus (CSE) and non-convulsive status epilepticus (NCSE).[1,2] NCSE is a state of unconsciousness due to continuation or frequent recurrence of seizures.[3] Clinically, it is associated with altered mental status and behavioral changes lasting more than 30 minutes.[3] NCSE occurs in two ways as generalized absence (ASE) and complex partial (CPSE).[3] NCSE constitutes approximately 25% of all SE.[2] The rate is thought to be higher than the actual number

Submitted *(Geliş tarihi):* 17.09.2012 **Accepted** *(Kabul tarihi):* 12.11.2012 **Published online** *(Online baskı):* 14.02.2013
Correspondence *(İletişim):* Dr. Ayhan Aköz. Atatürk Üniversitesi Tıp Fakültesi Acil Tıp Anabilim Dalı, 25240, Erzurum, Turkey
e-mail *(e-posta):* akozayhan@gmail.com

because of under-diagnosis.[2] The diagnosis is difficult because the patients with NCSE demonstrate a wide variety of clinical manifestations. Initially, the diagnosis must be considered and electroencephalography (EEG), the test for definitive diagnosis, should be applied in all suspected cases in emergency department (ED).[4] We aimed to present a case admitted to our ED with AMS and diagnosed as NCSE that is under-diagnosed.

Case Report

A 41-year-old male patient was brought to our ED with the complaint of AMS without an epileptic attack. His family stated that he has a diagnosis of epilepsy since 11 years old and has been on phenytoin 100 mg tablet (Eptantoin®) twice a day. There was no additional medication. On the admission day, the patient was apathetic, and his level of consciousness decreased progressively. His previous attacks were similar and began with these symptoms: contractions of body, foam at the mouth, and sometimes urinary incontinence. However, there were no contractions and urinary incontinence on this admission. The patient's vital signs, blood tests and computed tomography of brain were in normal range. He was consulted to neurology. The EEG showed bilaterally synchronic multiple paroxysms of spike-spike waveforms which were spreading through both hemispheres (Figure 1). The patient admitted to neurology clinic with diagnosis of NCSE. Diazepam 0.15 mg/kg IV was given and phenytoin 200 mg/day orally was continued. Levetiracetam tablet started with dose of 1000 mg twice a day. After the treatment, the patient was recovered and the new EEG showed no pathologic waves. His treatment was arranged and the patient was discharged 7th day of admission.

Discussion

NCSE is defined as a clinical state consistent with unexplained change in behavior and mental status.[4] Simultaneous EEG reveals seizure activity but not accompanied by convulsions.[4] The incidence may vary 15-60/100000 years.[4] In

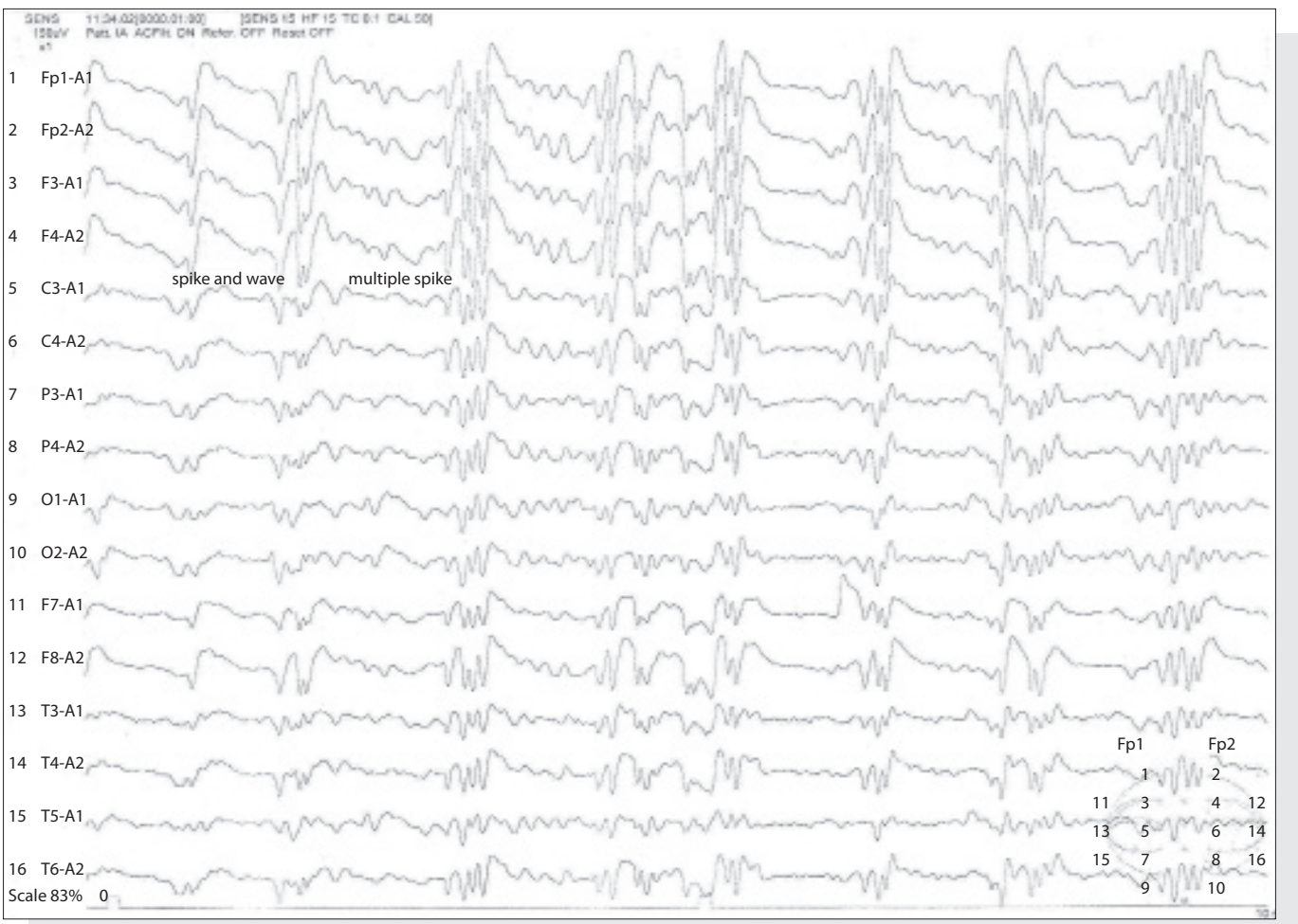

Figure 1. The EEG showed bilaterally synchronic multiple paroxysms of spike-spike waveforms which were spreading through both hemispheres.

a study by Labar et al.,[1] they diagnosed NCSE in 10 of 141 (7.1%) patients between the ages of 65-95 who admitted to the epilepsy center for one year. The first epileptic attack may be a form of NCSE in one third of cases with NCSE.[5]

The main clinical signs of NCSE are changes in mental status such as tendency to sleep, confusion, and especially lack of attention to the place, time and people.[6] Statement of fluctuating confusion or delirium may be accompanied by psychiatric symptoms such as agitation, impulsivity, and aggressiveness, and clinical symptoms such as decreased speech, mutism, echolalia, a blank look, eye blinking, swallowing, complex automatisms, periorbital, facial or laterally myoclonus, new and unexplained various behaviors in daily living, unsuitable laughing , crying or singing, anorexia, severe fatigue and reluctance, nistagmoid eye movements, aphasia, and unexplained somnolence.[4] In our case, there were AMS, somnolence, confusion, and apathic appearance when presented to the ED.

NCSE can be seen in a person who had no history of epilepsy before.[4] NCSE should be suspected in patients with AMS whether there is history of seizures or not before and neurological evaluation with EEG should be performed.[4]EEG findings of NCSE include focal or generalized periodic lateralized epileptiform discharges, bilateral independent periodic lateralized epileptiform discharges, periodic epileptiform discharges and generalized triphasic waves.[3] Our case was admitted to ED with complaints of lethargy and apathy. EEG showed multiple spike and spike-wave paroxysms with frequent intervals.

Causes of NCSE include central nervous system disorders (stroke, infection, trauma, and tumor), metabolic factors (hypoxia, renal diseases, menstruation, drugs) and inadequate using of anti-epileptic drugs.[1] Encephalopathy, toxic and metabolic disorders, various epileptic and psychiatric syndromes have been reported in differential diagnosis. The diagnosis is difficult because of a large number of atypical symptoms.[7] Our patient's family stated that he had not used antiepileptic drugs regularly especially for the last 6 months.

The treatment contains standard SE therapy. Benzodiazepines are first preferred agents in acute treatment.[8] Treatment is continued with the loading of phenytoin in CPSE cases which is considered more resistant and dangerous. Phenytoin may be harmful to patients with ASE. Barbiturate may be applied for patients with resistant CPSE, and intravenous valproic acid treatment can be applied to patients with ASE. Although it is rare, for patients with highly resistant NCSE, midazolam and propofol can be used in addition to barbiturates.[9] Initially 0.15 mg/kg dose of diazepam was administered to our patient. Intravenous 2000 mg loading dose of levetiracetam was given followed by 1000 mg tablet twice a day. Phenytoin was continued. On the second day, the patient began to improve clinically. The patient was completely recovered and discharged on the 7th day.

In conclusion, patients with AMS may admit to ED with different reasons. In such patients, NCSE should be considered in addition to the various reasons of AMS. We recommend rapid EEG assessment in ED for definite diagnosis.

Conflict of Interest

The authors declare that there is no potential conflicts of interest.

References

1. Labar D, Barrera J, Solomon G, Harden C. Nonconvulsive status epilepticus in the elderly: a case series and a review of the literature. J Epilepsy 1998;11:74-8. [CrossRef]
2. Quigg M, Shneker B, Domer P. Current practice in administration and clinical criteria of emergent EEG. J Clin Neurophysiol 2001;18:162-5. [CrossRef]
3. Brenner RP. Is it status? Epilepsia 2002;43:103-13. [CrossRef]
4. Kaplan PW. Nonconvulsive status epilepticus editorial. Neurology 2003;61:1035-6. [CrossRef]
5. Tomson T, Lindbom U, Nilsson BY. Nonconvulsive status epilepticus in adults: thirty-two consecutive patients from a general hospital population. Epilepsia 1992;33:829-35. [CrossRef]
6. Baykal B. NKSE'de klinik bulgular. In: Baykal B, editör. Nonkonvülzif status epileptikus. 1. Baskı. İstanbul: Artpres; 2004. s. 30-3.
7. Afsar N, Kaya D, Aktan S, Aykut Bingol C. Stroke and status epilepticus: stroke type, type of status epilepticus, and prognosis. Seizure 2003;12:23-7. [CrossRef]
8. Litt B, Wityk RJ, Hertz SH, Mullen PD, Weiss H, Ryan DD, et al. Nonconvulsive status epilepticus in the critically ill elderly. Epilepsia 1998;39:1194-202. [CrossRef]
9. Begemann M, Rowan AJ, Tuhrim S. Treatment of refractory complex-partial status epilepticus with propofol: case report. Epilepsia 2000;41:105-9. [CrossRef]