



Nonconvulsive Status Epilepticus Mimicking Hypoactive Delirium in an Elderly Patient

Dear Editor,

An 80-year-old female patient was admitted to our emergency service due to transient consciousness impairment and apathy. On questioning her medical history, it was learned that her symptoms had started 8 h before admission and spontaneously recovered in a gradual manner. Her relatives stated that the patient could not produce a verbal output or communicate during a 2-h period after the onset of symptoms. Other medical history was unremarkable with normal premorbid cognitive functions. On neurological examination, she was alert and cooperative. However, mild poverty in the content of the speech was recognized. She did not speak spontaneously but could respond to questions properly. The Glasgow coma scale (GCS) score was 14 (eye: 4, verbal: 4, motor: 6). Kernig's and Brudzinski's signs were negative. Motor, cerebellar, and sensory evaluations were within normal ranges. Vital sign assessments revealed mild fever (body temperature: 37.1°C, heart rate: 72, respiratory rate: 14, blood pressure: 120/80). Laboratory examinations revealed leukocytosis (white blood cells: 17,200/mL [89% neutrophils, 6% lymphocytes, and 4% monocytes], hemoglobin: 14.2 g/dL, and platelet: 245,000/mL). Urinalysis tests were positive for both nitrite and leukocyte esterase. In addition, microscopic examination of the urine showed 20 leukocytes per μ L of urine. Other examinations were within normal ranges (serum biochemistry, Vitamin B12, and folic acid). After evaluation by an infectious disease specialist, the diagnosis of urinary tract infection was established and sulbactam-ampicillin 4 $\times$ (0.5 g/1 g) was initiated. The clinical presentation was characterized by acute deterioration in the ability to maintain attention and awareness. Remarkably, the dominated symptoms of the clinic were drowsiness and inactivity (as far as it can be learned from her relatives) which suggested an etiology of possible hypoactive delirium in the forefront.¹ However, to exclude a possible predisposing transient ischemic attack or minor stroke, cranial diffusion-weighted imaging (DWI) was performed which resulted in normal ranges. Due to technical problems (the patient was admitted at the weekend and an electroencephalography [EEG] technician was out of office), EEG could not be performed in the early period. At this point, with a preliminary diagnosis of hypoactive delirium (potentially aggravated by urinary tract infection), the patient was hospitalized for the administration of intravenous (IV) antibiotherapy and treatments for delirium. On the 2nd day of hospitalization, the neurological status of

the patient deteriorated progressively within a few hours in a level that the patient was totally noncooperative, and she could make only incomprehensible sounds. The eye opening response was available by only a painful stimulus (GCS score was 9 [eye: 2, verbal: 2, motor: 5]). Cranial magnetic resonance imaging was repeated urgently which also yielded normal findings. At this point, routine EEG was performed (soon after) which showed generalized periodic, slow-wave paroxysms, leading to the diagnosis of nonconvulsive status epilepticus (NCSE) [Figure 1]. After the prompt intervention of IV 10 mg diazepam, a dramatic and rapid improvement was achieved such that she was evaluated as totally orientated and cooperative in the following 30-min time interval. Aphasia examinations were completely normal. EEG, on the next day, showed total resolution of periodic epileptiform discharges [Figure 2]. The diagnosis was established as NCSE, and levetiracetam 2 $\times$ 500 mg was initiated. IV antibiotherapy for the urinary tract infection was continued in this interval. Third EEG, performed on levetiracetam 1000 mg (7 days after admission), showed totally normalization of the background activity [Figure 3].

DISCUSSION

NCSE is a form of status epilepticus which may present with various neurologic deficits, influencing particularly the alertness and cognitive functions. Clinical seizure activity in NCSE is generally very slightly or absent which

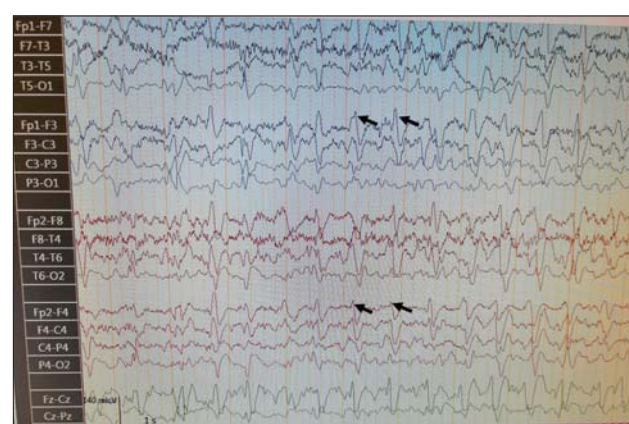


Figure 1: Initial electroencephalography showing bilateral and widespread with high-amplitude rhythmic discharges and slowing of the background activity

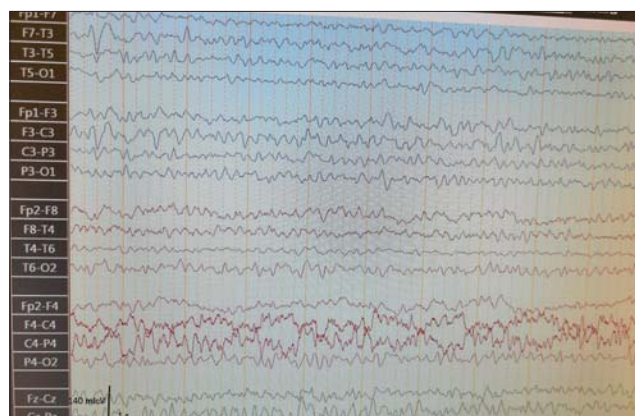


Figure 2: Electroencephalography recorded the next day showing resolution of periodic epileptiform discharges and a moderate recovery of the background activity

may explain that it is one of the most frequently missed diagnoses in patients presenting with altered consciousness.² Remarkably, EEG constitutes a vital diagnostic tool for NCSE. Therefore, demanding EEG is the critical stage for the diagnosis.² Although a wide variety of EEG samples have been associated with a diagnosis of NCSE, there is no consensus on the diagnostic criteria of EEG findings. Classically, NCSE is classified into two subtypes as follows: absence SE – characterized by the generalized spike and slow-wave discharges, and complex partial SE – usually with focal discharges and considered the equivalent of prolonged or repetitive complex partial seizures.² In another crucial report, three features, including epileptiform sharp waves $\pm$ slowing, rhythmicity, and rapid discharges: 1–3.5 Hz, have been remarked as typical features of NCSE.^{2,3} On the other hand, Sutter *et al.* stated that the diagnosis of NCSE can be made with frequencies of >2.5 Hz generalized periodic epileptiform discharges, whereas an additional criterion was mentioned to be required for slower frequencies of periodic discharges.⁴

In our patient, not a clinical risk factor (previous seizure history, convulsions, nystagmus, and eye deviation) was present that had suggested NCSE first. On the other hand, cranial DWI was in normal ranges, and no history of a premorbid history of dementia was obtained removing the suspicion of stroke or hypoactive delirium. Finally, EEG revealed 1.5 Hz generalized periodic epileptiform discharges (PEDs) (which was associated with NCSE previously³) and intervention of 10 mg IV diazepam resulted in a dramatic clinical, as well as electrophysiological improvement, confirming the diagnosis of NCSE (as mentioned above). Recent clinical trials have demonstrated that NCSE constitutes an important cause of prolonged states of confusion in elderly patients.^{5,6} On the other hand, considering that mental status alteration is a

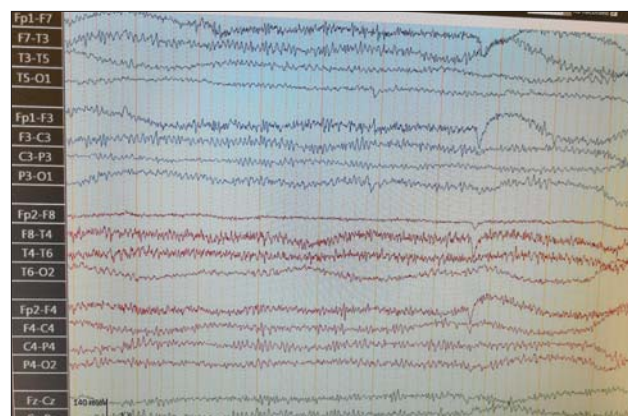


Figure 3: Electroencephalography before discharge (6 days later from the second electroencephalography) showing normal parieto-occipital alpha rhythm (12 Hz)

common, chief complaint among elderly patients, and it is classically associated with delirium, it can be predicted that NCSE might potentially be an underestimated etiological agent in the elderly population. On the other hand, complications due to inappropriate treatments and prolonged seizure activity have been suggested to be as the responsible factors from high mortality rates in these patients.⁵ Remarkably, early recognition and prompt effective intervention generally enable total recovery.² Herein, we present a smart illustration of an elderly patient with NCSE that diagnosis was strictly challenging. Through the presentation of this remarkable patient and related literature, we reemphasize that recognition of this entity as a cause of altered mental status, particularly in the elderly population, is extremely critical for proper clinical outcomes. In presentations of acute consciousness alterations in elderly patients with no demonstrable etiological agent, EEG should certainly be administered to avoid misdiagnosis of this rare, but devastating etiology.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given her consent for her images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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