Clinical Case Report

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Can endotracheal intubation be the first step in management of nonconvulsive status epilepticus?

A case report

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Abstract

Rationale: Nonconvulsive status epilepticus (NCSE) is prolonged seizure activity without motor manifestations. Clinically, there are certain examination findings, in addition to elements of history, that help differentiate it from other encephalopathies. When diagnosing NCSE, the physician faces significant difficulties due to inconsistent clinical presentation and somewhat nonspecific electroencephalogram (EEG) criteria.

Patient concerns: To highlight the problems faced when dealing with such a patient, a case of a 29-year-old male who presented with an altered state of consciousness is put forth for the reader. Only after an extensive laboratory and radiological workup had ruled out other causes, an eventual diagnosis was established when clinical features were correlated with suggestive EEG results.

Diagnoses: The diagnosis that was reached was NCSE.

Interventions: The initial therapeutic interventions generally deployed in such a scenario ultimately failed and consequently the patient had to be sedated and intubated, while being kept on antiepileptic medication.

Outcomes: This measure resulted in satisfactory recovery.

Lessons: Accordingly, we recommend consideration of NCSE in any unconscious patient whose presentation cannot be explained by other disorders. Furthermore, we suggest moving directly to utilizing anesthetic agents and endotracheal intubation, together with anti-epileptic drugs, in the treatment regimen in order to optimize patient outcomes.

Abbreviations: CSF = cerebrospinal fluid, EEG = electroencephalogram, GCS = Glasgow Coma Scale, HSV = Herpes Simplex Virus, ICU = intensive care unit, IV = intravenous, MRI = magnetic resonance imaging, MTS = mesial temporal sclerosis, NCSE = nonconvulsive status epilepticus, SE = status epilepticus, T2/FLAIR = T2-weighted fluid attenuated inversion recovery.

Keywords: electroencephalography, intubation, mesial temporal sclerosis, nonconvulsive status epilepticus

1. Introduction

Status epilepticus (SE) is defined as the occurrence of a continuous seizure of at least 5- minute duration or, alternatively, 2 or more seizures with incomplete recovery in between; it can broadly be categorized into either convulsive or non-convulsive depending on whether it is accompanied by rhythmic jerking movement.[1] It is to be noted that the forms of SE other than generalized convulsive, the duration of continuous seizure activity is defined to be of at least 10-minute duration for focal SE with impaired consciousness and 10 to 15 minutes for the absence of SE.[2] There are certain findings from history and examination that have been reported more likely to be present in non-convulsive status epilepticus (NCSE) such as, remote risk factors for seizures (e.g., previous stroke, tumor, previous neurosurgery, dementia, and meningitis) and ocular movement abnormalities (e.g., nystagmus, hippus, and sustained eye deviation in any direction).[3] The diagnosis of NCSE is heavily dependent on electroencephalography (EEG); however, having mentioned this, an agreed upon unified evidence-based EEG criteria to diagnose NCSE does not exist.[4,5] Recently, the Salzburg EEG criteria have shown to have high diagnostic accuracy, giving it potential for widespread implementation in future clinical practice.[6] NCSE has various subtypes; the International League Against Epilepsy proposed a classification system to facilitate categorization.[2] The minimal incidence of SE in the Caucasian population of industrialized countries is about 20/100,000/year.[7] Since clear definitions of NCSE are not established and the condition is hard to diagnose, epidemiological data on NCSE specifically are sparse.[7] Treatment for NCSE is understandably not well elucidated in literature due to paucity of relevant randomized control trials conducted on the subject.[1] This paper details the case of a young South East Asian gentleman, presenting with an altered state of consciousness, who was ultimately diagnosed with NCSE.

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Patient did not object to publication of this case report.

The authors report no conflicts of interest.

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2. Presenting concerns

A 29-year-old previously healthy Bangladeshi male construction worker was brought to the hospital by police officers in an unconscious state of an unknown duration. The patient was discovered initially by bystanders, who found him lying unresponsive on the side of the road.

3. Clinical findings

No history of vomiting, jerky movements, or urinary incontinence was given. During his assessment in the emergency department, no signs of trauma or toxicity were noted. As per his friends, the patient had displayed reduced activity for the past 3 days. He had no previous relevant medical or surgical history. Although it was reported that he possibly had a prior similar presentation in his home country, no official reports were available.

On examination, the patient had a fluctuating level of consciousness; his Glasgow Coma Scale (GCS) was reaching as low as 3. However, it never remained critically low for a long enough period of time to deem intubation necessary. Further neurological examination revealed considerable loss of power in all 4 limbs with intact tone and reflexes. The examination of other systems was unremarkable. The possibility of malingering could not be disregarded at this point.

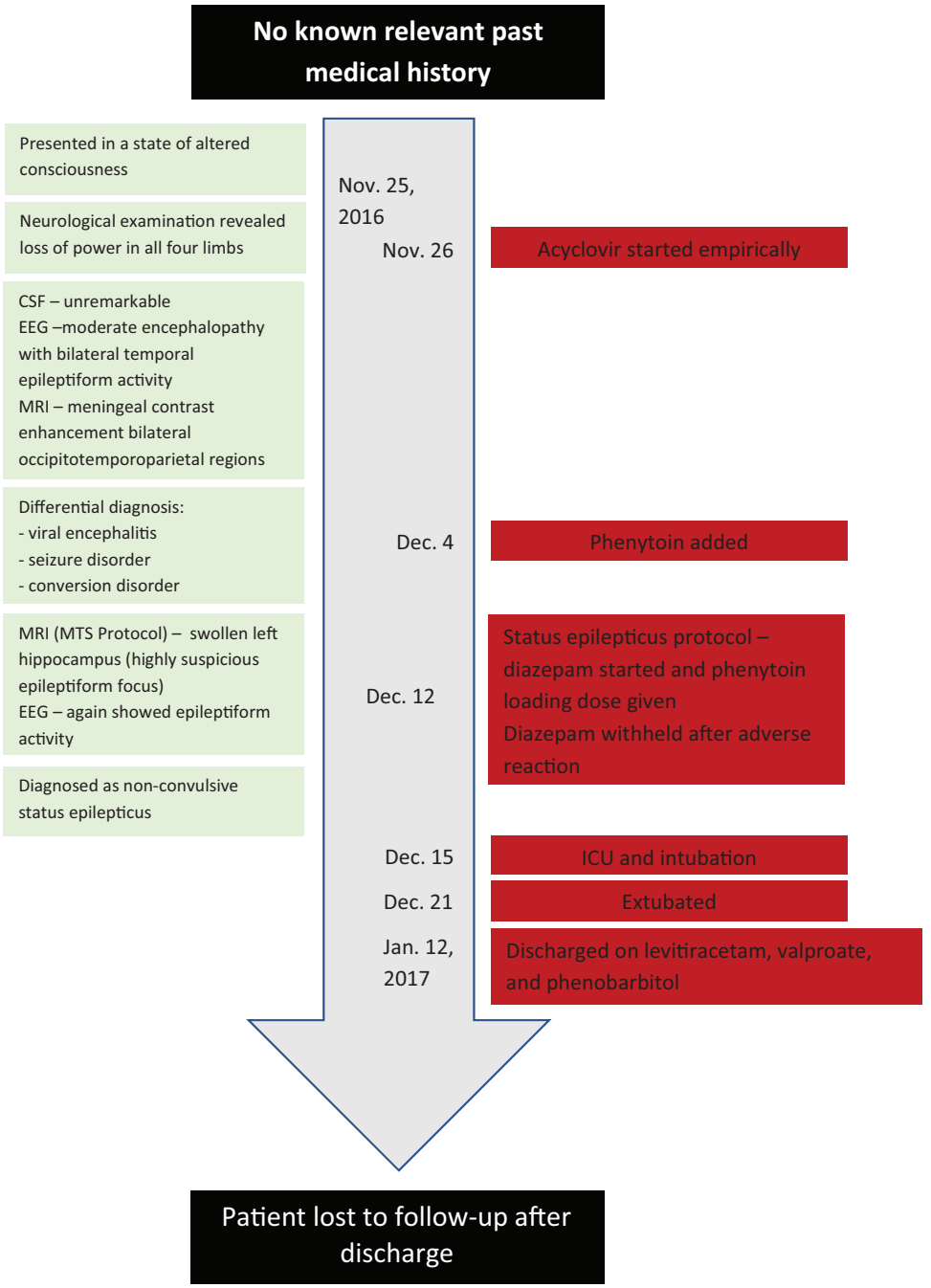

Figure 1. Timeline of events.

4. Timeline

Timeline of events is presented below (Fig. 1).

5. Diagnostic assessment

The patient's initial general laboratory investigations done in the emergency department were within the normal limits. Neurological lab workup was subsequently carried out to rule out infective causes such as meningitis and encephalitis; a cerebrospinal fluid (CSF) sample was taken and analyzed for biochemistry, cytology, and microbiology. His CSF biochemistry revealed slightly elevated glucose concentration of 4.06 mmol/L (normal lab range: 2.2–3.9 mmol/L), and normal protein concentration of 18.94 mg/dL (normal lab range: 15–45 mg/dL). There were no red blood cells or white blood cells seen in the sample and cytology was negative. Moreover, CSF culture showed no growth and was negative for acid-fast bacilli and herpes simplex virus (HSV). CT brain of the patient revealed no significant findings.

Initial EEG was done 1 week after presentation and showed a picture of a moderate encephalopathy with bilateral temporal epileptic form activity, more prominent on the left; additionally, there were some right fronto-central spikes, which were judged as artifacts (Fig. 2). Magnetic resonance imaging (MRI) revealed slightly increased meningeal contrast enhancement in bilateral occipitotemporoparietal regions, raising suspicion of viral meningitis. Therefore, empirical anti-viral therapy was initiated. As a result, the patient did indeed regain consciousness. Unfortunately, he again became unconscious the next day. Subsequently, an MRI with mesial temporal sclerosis (MTS) protocol was carried out and so was a repeat EEG. The MRI showed a mildly swollen left hippocampus, without signal changes on T2-weighted Fluid Attenuated Inversion Recovery (T2/FLAIR) images, with a highly suspicious SE focus (Fig. 3).

Repeat EEG of the patient showed abnormalities in the form of sharp wave discharges in both posterior temporal regions, in addition to slow wave discharges (Fig. 4). Based on the history and EEG findings, along with neuroimaging, a diagnosis of NCSE was finally reached.

Although autoimmune encephalitis could also have been a cause, the clinical course suggested otherwise. The way the patient presented and the nature of progression of his symptoms was not considered indicative enough to pursue this diagnosis.

6. Therapeutic intervention

The patient was started on diazepam and a loading dose of phenytoin was given. Unfortunately, diazepam had to be discontinued because the patient developed severe hypotension after receiving an initial dose of 10 mg intravenously. Likewise, phenytoin (given intravenously 3 times daily at a dose of 200 mg) showed minimal effectiveness. After more than 2 weeks of unsatisfactory improvement in the patient's condition, as a further trial, he was transferred to the intensive care unit (ICU) and intubated after being deeply sedated with propofol (at an infusion rate of 200 mg/h) and while kept on levetiracetam 1 g twice daily. After extubating, the patient regained full recovery from NCSE. He was continued on the same dose of levetiracetam. EEG was repeated and was reported as normal. The patient was kept for observation and thereafter discharged in good condition.

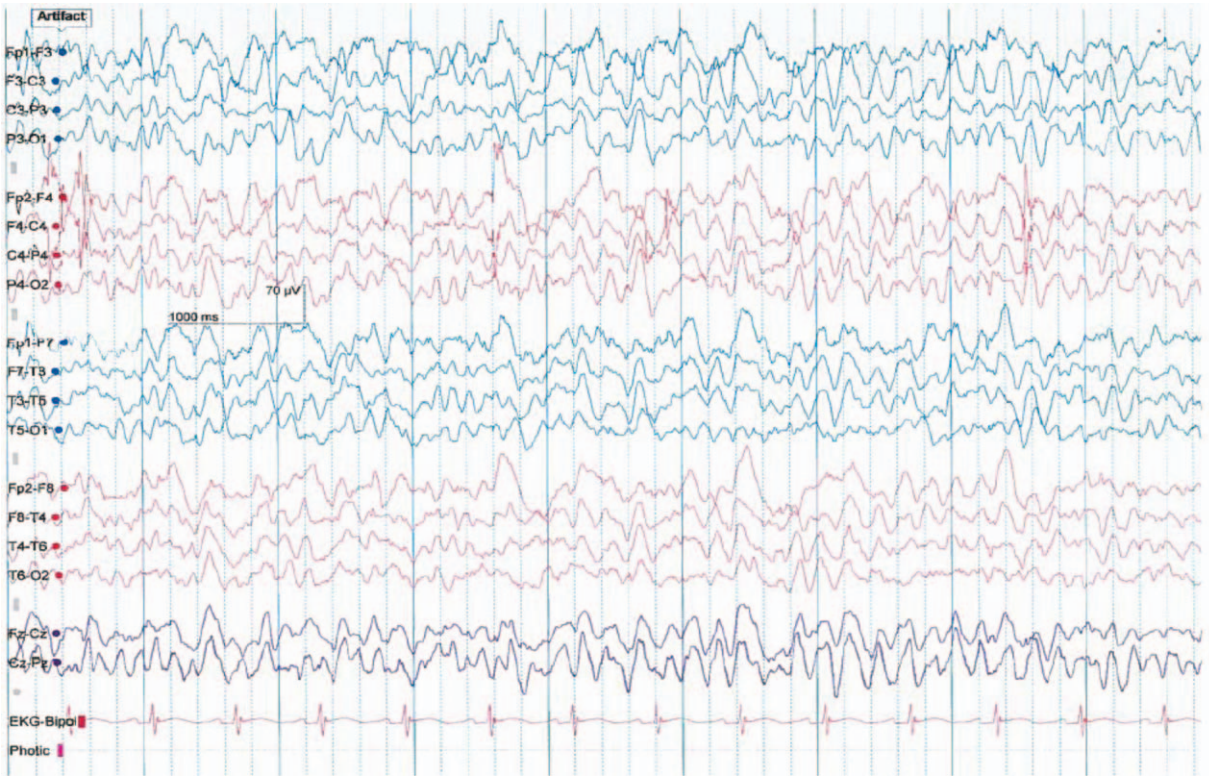

Figure 2. EEG showing a picture of a moderate encephalopathy, with bilateral temporal epileptic form activity more on the left. However, there were some right fronto-central spikes, which were judged as artifacts.

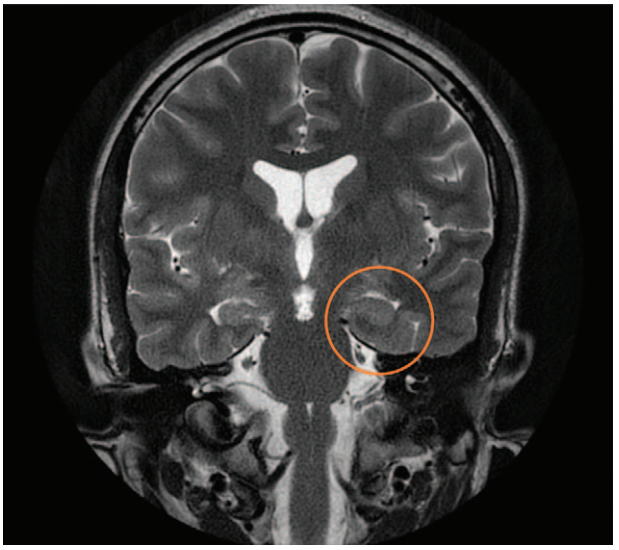

Figure 3. Mild swelling of left hippocampus (circle) without signal changes on T2/FLAIR with highly suspicious status epilepticus focus.

7. Follow-up and outcomes

The patient was not seen after his discharge from the hospital as he relocated to his country of origin.

8. Discussion

The changes in the level of consciousness and mental status often result in mistaking NCSE with other disorders with similar clinical presentation such as hypoxia, and various infectious and metabolic diseases; therefore, limitations exist on establishing diagnosis based on clinical findings alone.[8] In order to confidently diagnose NSCE, correlation between EEG and clinical findings must be made. EEG and MRI brain with MTS protocol can prove to be helpful diagnostic tools.

In our reported case, a good history could not be obtained from the patient's relatives or friends. His only neurological examination finding was diminished power. Furthermore, the patient's lab workup did not yield any additional information. The initial MRI brain was indicative of viral meningitis. In retrospect, this might possibly have been the triggering event; the causative organism might not have been sensitive to the anti-viral therapy used since no improvement was noted after its implementation.

A general overview of the literature has shown that EEG diagnosis criteria for NCSE is categorized depending on patient status; that is, whether the patient is an adult or a child, a known case of epileptic encephalopathy or not, whether he/she is comatose or not, and so on. Table 1 represents the 6 "clear-cut" EEG criteria.[9]

In adjunct to the information presented in the Table 1, Hirsch *et al*. recommended changes in terminology of EEG findings in the critically ill; they suggested that terms such as "periodic

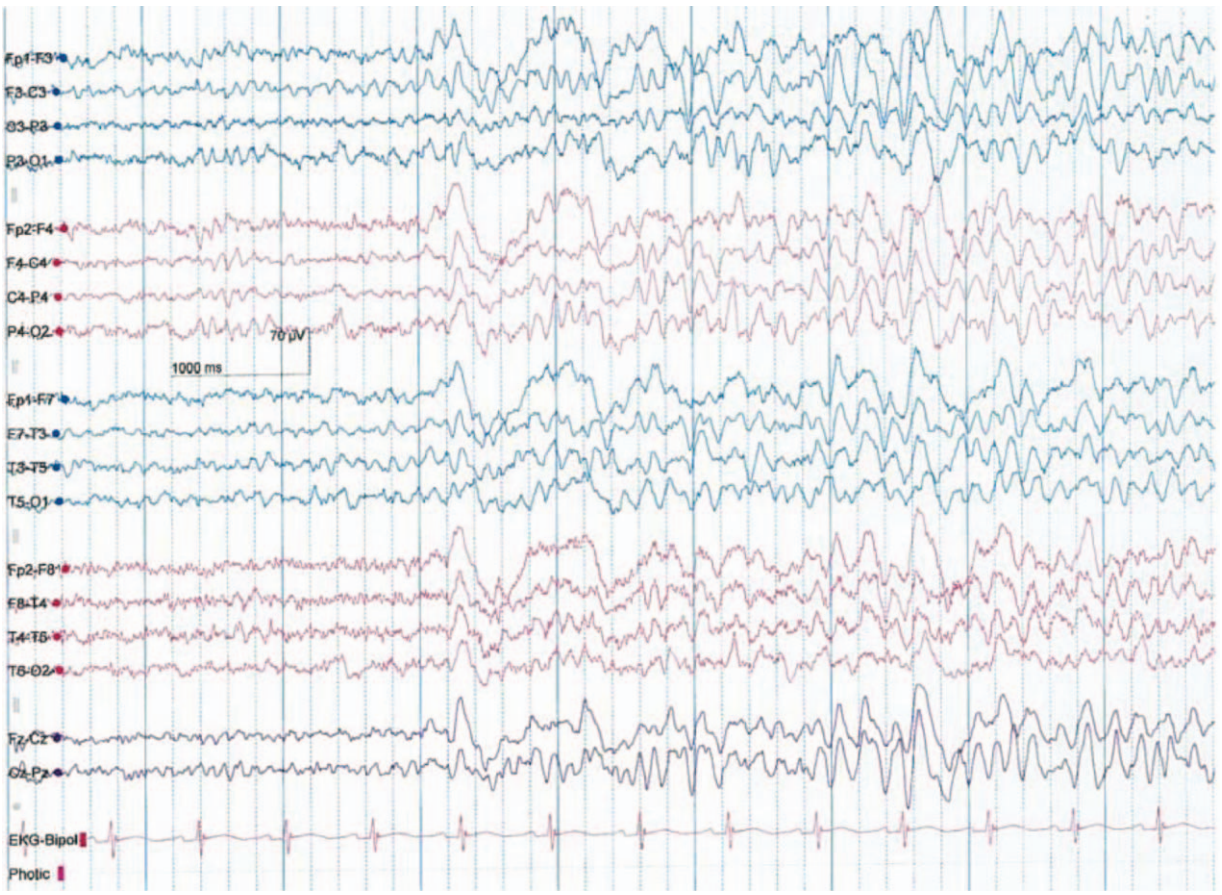

Figure 4. Abnormalities are in the form sharp wave discharges in both posterior temporal region and slow wave discharges.

Table 1

"Clear-cut" EEG criteria for non-convulsive status epilepticus[9].

Clear-cut criteria for nonconvulsive status epilepticus
1. Frequent or continuous focal electrographic seizures, with ictal patterns that wax and wane with change in amplitude, frequency, and/or spatial distribution.
2. Frequent or continuous generalized spike-wave discharges in patients without a previous history of epileptic encephalopathy or epilepsy syndrome.
3. Frequent or continuous generalized spike-wave discharges, which showed significant changes in intensity or frequency (usually a faster frequency) when compared to baseline EEG, in patients with an epileptic encephalopathy or epilepsy syndrome.
4. PLEDs or BIPEDs that occurred in patients in coma in the aftermath of a generalized tonic–clonic status epilepticus (subtle status epilepticus)
5. EEG patterns that were less easy to interpret included: Frequent or continuous EEG abnormalities (spikes, sharp-waves, rhythmic slow activity, PLEDs, BIPEDs, GPEDs, triphasic waves) in patients whose EEGs showed no previous similar abnormalities, in the context of acute cerebral damage (e.g., anoxic brain damage, infection, trauma).
6. Frequent or continuous generalized EEG abnormalities in patients with epileptic encephalopathies in whom similar interictal EEG patterns were seen, but in whom clinical symptoms were suggestive of NCSE.

BiPEDs = bilateral independent periodic epileptiform discharges, EEG = electroencephalogram, GPEDs = generalized periodic epileptiform dischargesPLEDs = periodic lateralizing epileptiform discharges. NCSE = Nonconvulsive status epilepticus

lateralising epileptiform discharges" be replaced by "lateralized periodic discharges."[10] This facilitates matters since EEG patterns that are not believed to be "epileptiform" can still be considered when reaching a diagnosis of NCSE.

The definitive EEG of our patient showed abnormalities in the form of sharp wave discharges in both posterior temporal regions, in addition to slow wave discharges (Fig. 4). When this report was correlated with clinical data, laboratory findings, and the results of neuroimaging, the diagnosis of NCSE was solidified.

The recommended treatment of NCSE is a small intravenous (IV) dose of diazepam or lorazepam. Unfortunately, our patient developed severe hypotension after initial administration of 10 mg diazepam IV. Interestingly, however, he recovered completely after intubation and deep sedation. It is well documented that treatment of unconscious NCSE patients with benzodiazepines may lead to severe hypotension and respiratory depression and accordingly, treatment with anti-epileptics is sometimes suggested as first-line therapy.[11,12]

The prognosis of NCSE is determined by several factors including wrong diagnosis.[13] In our case, even though our patient remained in NCSE for more than 2 weeks, recovery was eventually attained, after endotracheal intubation, to a point where the patient did not require any further inpatient hospital care. To establish the individual prognosis of the different cases and types of NCSE, more well-defined epidemiological studies are required.

This report highlighted the importance of considering NCSE in patients who present to emergency with alteration in the level of consciousness and mental status without an obvious underlying etiology such as hypoxia, infectious or metabolic diseases. Moreover, it introduces the idea of use of anesthetic agents and endotracheal intubation immediately and simultaneously with anti-epileptic medications in an effort to achieve a quick and more complete recovery in such cases. The clinical course of our patient yearns the undertaking of studies further exploring the benefits of this treatment approach to establish quantitative evidence to direct management and ultimately improve patient care.

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