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Analysis of electroclinical features of nonconvulsive status epilepticus: a study of four cases

Dan Wu†, Xiaoyun Liu†, Xingqi Yao, Yanfeng Yang, Jun Zhang, Haoran Yang and Wei Sun*

Abstract

Background: The nonconvulsive status epilepticus (NCSE) is an epileptic condition characterized by little or no obvious symptoms, thus is often easily to be underrecognized, underdiagnosed or even undetected by clinicians. This article is written to advance the recognition and diagnosis of NCSE.

Case presentation: Four cases of NCSE were reported and their semiology, electroencephalogram (EEG) features, etiology, treatment and prognosis were retrospectively analyzed. Most of the 4 cases presented with impaired consciousness (confused, slow reaction and lags in response) and some strange behaviors (being upset and restless or washing hands repeatedly). None of them had any obvious motor symptoms like tonic or clonic movements. EEG of the 4 cases initially manifested with either a focal or a generalized onset, then evolved into spike-and-wave pattern gradually. With a favorable response to antiepileptic drugs, they all had a good outcome without any sequela.

Conclusions: NCSE is much more common than was considered in the past, which is featured by little or no evidence of movement or other symptoms. NCSE can lead to a favorable outcome in most patients.

Keywords: Nonconvulsive status epilepticus (NCSE), Electroencephalogram (EEG), Treatment, Prognosis

Background

Nonconvulsive status epilepticus (NCSE) is an epileptic condition characterized by continuous or recurrent seizure activity, and diverse clinical symptoms such as alterations of mental state, abnormal behavior, perception disturbances or consciousness impairment, accompanied by generalized or focal epileptiform activity on the electroencephalogram (EEG), usually lasting more than 30 min [1, 2]. On the other hand, there is also proposition that the duration be greater than 1 h [3].

The NCSE is much more common than was considered in the past. NCSE constitutes about 25–50% of all status epilepticus (SE) cases, with an incidence of 2–8/100,000 per year [4, 5]. According to previous studies, NCSE is traditionally divided into two subtypes: the generalized NCSE and the focal NCSE. The generalized NCSE includes the absence status epilepticus (ASE) which was first described by Lennox in 1945 [6] and atypical absence SE. The focal NCSE, also referred to as complex partial status epilepticus (CPSE), was initially described by Gastaut in 1956 [7], and is characterized by prolonged or recurrent complex partial seizures. In recent years, some experts put forward a more detailed classification as discussed below.

The diagnosis criteria for NCSE include a period of behavioral change from baseline, EEG evidence of epileptic activity, and a response to antiepileptic drugs (AEDs) [2, 8]. In comparison to clinical signs (if any) which are often subtle and nonspecific, the EEG criterion is indispensable for the diagnosis of NCSE. In most cases, diagnosis of NCSE relies largely on EEG findings, especially

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in comatose patients [9]. In addition, debates still remain on whether a response to AEDs can be used as the diagnostic criterion. Some clinicians believe that although a response to benzodiazepine confirms the diagnosis, an absence of response cannot simply exclude the diagnosis.

In comparison to generalized tonic-clonic status epilepticus (GCSE) which exhibits a state of ongoing convulsions and may cause a significant morbidity and mortality, the NCSE is featured by little or no evidence of movement or other symptoms, and thus is often underrecognized, underdiagnosed or even neglected by clinicians. In this article, we report 4 cases of NCSE and further review the semiology, electroencephalogram features, etiology, treatment and prognosis of this disease, in the aim to help clinicians better recognize and diagnose this subtype of SE.

Case presentation

Case 1

A 47-year-old woman has experienced epileptic seizures for 4 years. During initial seizures, she manifested with complex partial seizures as follows: she first became motionless suddenly, then her eyes and head deviated to the right, with hands fumbling. The event usually lasted no more than 1 min, and there were no signs to predict it. The above seizure type occurred twice a year. The patient did not pay any attention to these events or seek any medical advice until she experienced two generalized tonic-clonic seizures (GTCS) 2 years later. Magnetic resonance imaging (MRI) of the brain showed increased signal on T2-weighted images suggestive of the left mesial temporal sclerosis, and the interictal-EEG showed left frontal and left temporal intermittent sharp waves. Then she began an oxcarbazepine regimen (600 mg/day) and did not experience seizures any more in the next 2 years.

One day at the age of 46, the patient suddenly became confused, upset, slow in reacting to the outside world and gave irrelevant answers to other's questions. This episode lasted through the day and gradually resolved. The patient did not go to the hospital for any treatment. One month later, she followed the doctor's advice to increase the oxcarbazepine dose to 750 mg/day during a routine visit to outpatient service.

At the age of 47, the patient became confused suddenly again, slow to react, and kept doing meaningless movements like washing her hands repeatedly, as noticed by her family. To simple questions, she either failed to respond or gave delayed, often inappropriate responses. She could not execute instructions properly, either. The episode lasted about 20 h without remission, so she was transferred to Xuanwu Hospital. The physical examination showed that the patient was confused, slow in speech and disoriented. She was scored 13 points for MMSE. Video-EEG was applied and demonstrated persistent 2.5- to 3.5-Hz generalized spike-and-wave discharges, with a frontal and central predominance (Fig. 1a). The patient was given intravenous administration of 10 mg diazepam, and almost immediately, the EEG started to recover (Fig. 1b) and the discharge resolved within 2 min (Fig. 1c), but the symptoms still existed. About 30 min later, the video-EEG showed re-occurrence of 2.5- to 3-Hz spike-and-wave, slow waves and spike discharge (Fig. 1d), so the patient was further given an intravenous 10 mg of diazepam. After 3 min, normal background EEG rhythms returned (Fig. 1e), but they only lasted 20 min and again evolved into a spike-and-wave complex (Fig. 1f). However, the clinical symptoms almost all have disappeared by this time. The patient recovered to have clear consciousness, was fluent in speech and capable of responding correctly, and was scored 23 points for MMSE. Given the clinical remission, the patient was given an oral administration of 1000 mg levetiracetam instead of another diazepam dose. Two hours later, her EEG patterns returned to normal, eventually with a total clinical remission (Fig. 1 g). When the patient left hospital, her regimen was adjusted as levetiracetam 1000 mg/day and oxcarbazepine 600 mg/day. In the following 1 month, she never experienced any seizure again, and was scored 29 points for MMSE 1 month after the event.

Case 2

A 62-year-old woman was admitted to Xuanwu Hospital for experiencing episodes of being slow to react, speechless, and answering incorrectly to questions in the past 2 years. Each attack lasted 1 to 2 days. Despite treatment with lamotrigine and carbamazepine (switched to oxcarbazepine later), the seizure still occurred every 3 to 4 days. It is worth mentioning that she never had any GTCS during the disease course. Her previous MRI showed increased signals in the right hippocampus and abnormal signals in the boundary of the right temporal lobe and insula.

During her stay in our hospital, a seizure occurred. The clinical symptoms were almost the same as before: slow reaction, reduced speech and clouding of consciousness. Physical examination revealed normal orientation, decreased calculation, and a slightly lower MMSE (26 points) score than normal. This episode lasted more than 20 h. During the attack, video-EEG monitoring showed 4- to 7-Hz generalized spike-and-wave complex and spikes (Fig. 2a). At first, the patient was given an intravenous 10 mg diazepam and an intramuscular injection of 100 mg phenobarbitone, but no improvement was seen. She did not recover from the seizure and the EEG was basically unchanged. Thirty minutes later, she was given a further intravenous injection of 10 mg diazepam, then the

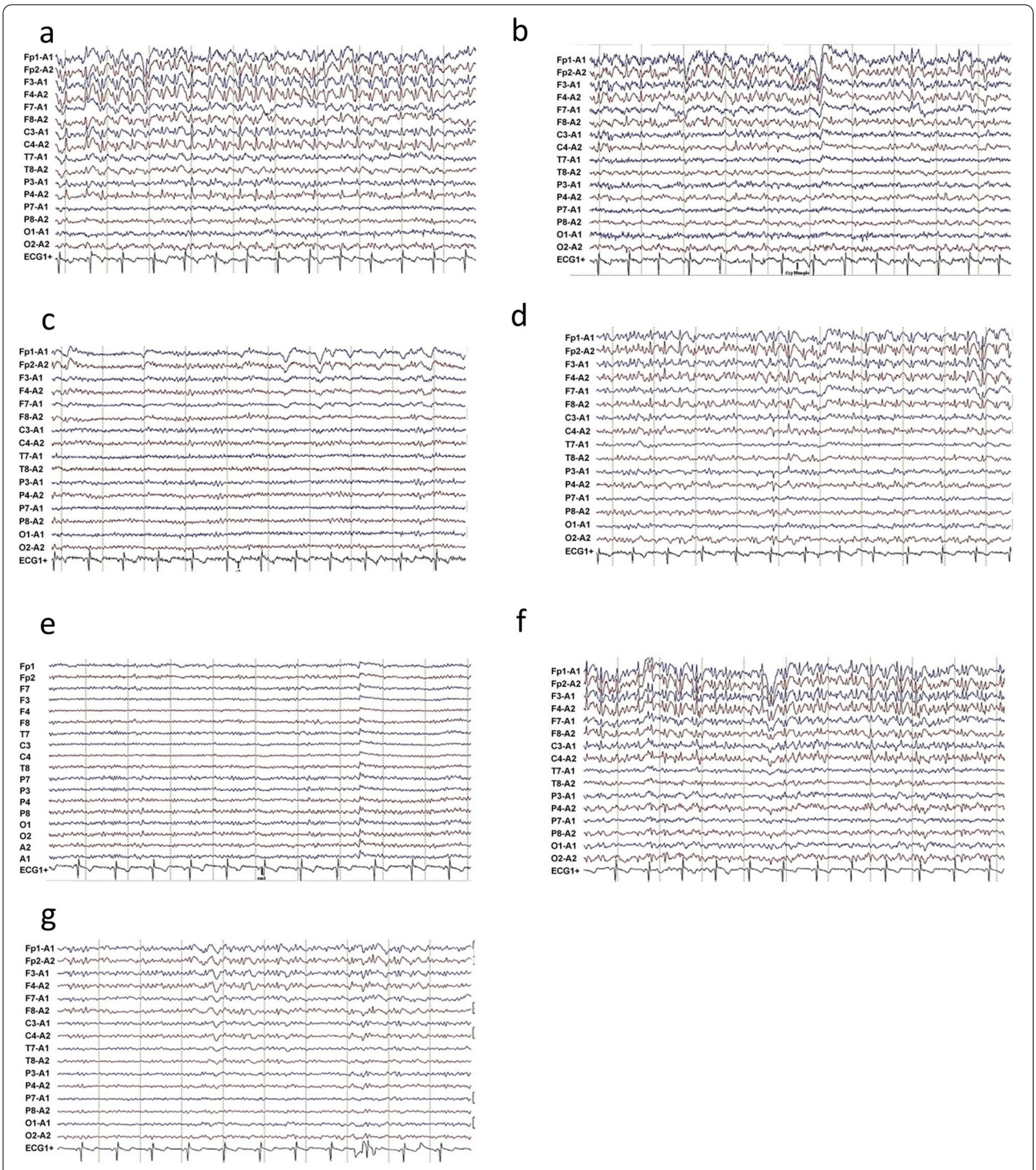

Fig. 1 EEG recordings of case 1. **a** 2.5- to 3.5-Hz generalized spike-and-wave discharges associated with symptoms of being confused, slow in speech and disoriented. **b** The EEG started to return to normal once the patient was given intravenous administration of 10 mg of diazepam. **c** Resolution of generalized spike and-wave pattern within 2 min after intravenous administration of 10 mg of diazepam. **d** Thirty minutes later, EEG showed 2.5- to 3-Hz spike-and-wave, spikes and slow waves with frontal and central predominance again. **e** Within 3 min after another 10 mg of diazepam, normal background EEG rhythms reappeared. **f** Although the EEG evolved into spike-and-wave pattern again, the patient became free from symptoms: being clear in consciousness, fluent in speech and capable of responding correctly. **g** The EEG returned to normal eventually with a total clinical remission

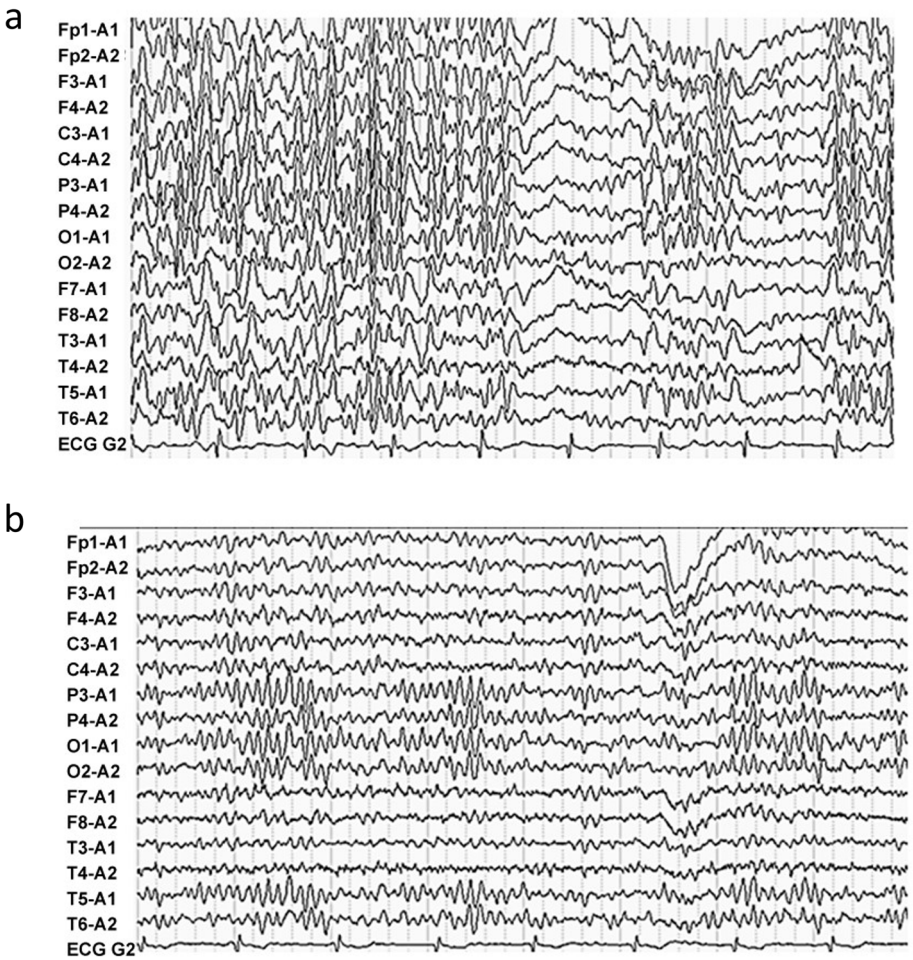

Fig. 2 EEG recordings of case 2. **a** Generalized 4- to 7-Hz spike-and-wave complex and spikes during behaviors associated with slow reaction, reduced speech and clouding of consciousness. **b** The symptoms disappeared and EEG pattern resolved after treatment

symptoms disappeared and EEG resolved as well within 5 min (Fig. 2b).

Case 3

A 48-year-old man was sent to the emergency room of Shunyi District Hospital by his family because they noted that he was a little confused, restless, upset and not fluent in answering questions through the day with no improvement at all. He had been diagnosed as "epilepsy" and "low intelligence" for more than 10 years. With intermittent oral administrations of phenytoin and phenobarbital, the seizures were well controlled. In our emergency room, the video-EEG showed generalized, continuous 2.5- to 4-Hz spike-and-wave patterns (Fig. 3). He was given an intravenous injection of 10 mg diazepam and an infusion of 30 mg diazepam, and several minutes later, he came back to normal state with all symptoms almost gone. But as the postictal EEG was not recorded at that time, whether the spike-and-wave pattern resolved or not after the treatment was unknown.

Case 4

A 59-year-old woman was transferred to Xuanwu Hospital. She was complaining episodes of being confused for 1 day, but could respond to others, though very slow. She could also handle dressing, eat meals and do some housework by herself. It seemed that there was nothing wrong with the patient but a little confused to strangers. She did not have a pre-existing history of epilepsy. Her past history included diabetes mellitus (DM) for several years, but the blood sugar level was well controlled with two kinds of hypoglycemic drugs. Once hospitalized, she received the video-EEG monitoring, which showed sharp-and-wave and sharp waves in the bilateral frontal, central, parietal and sphenoid electrodes (Fig. 4). Given the mild symptoms, she was not given an intravenous therapy, but an oral administration of levetiracetam

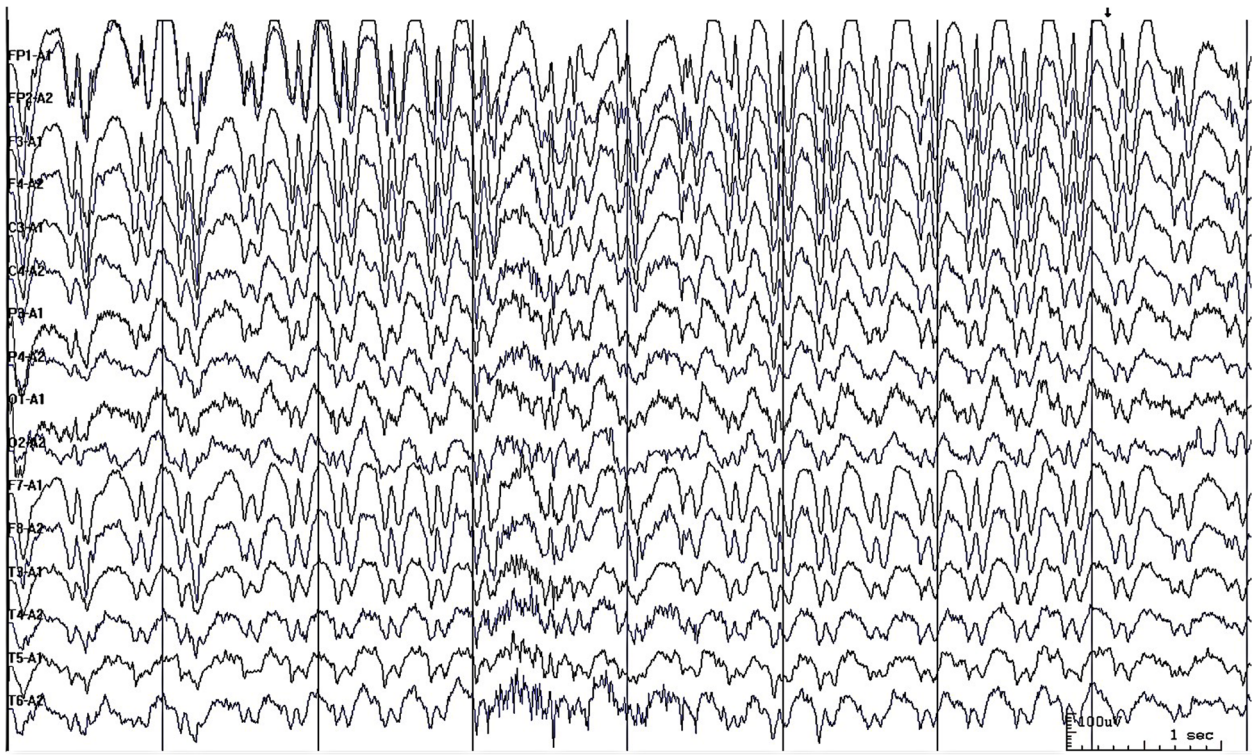

Fig. 3 EEG recordings of case 3. The EEG showed generalized 3.5- to 4.5-Hz spike-and-wave pattern associated with symptoms of being confused, restless, upset and not fluent in answering questions

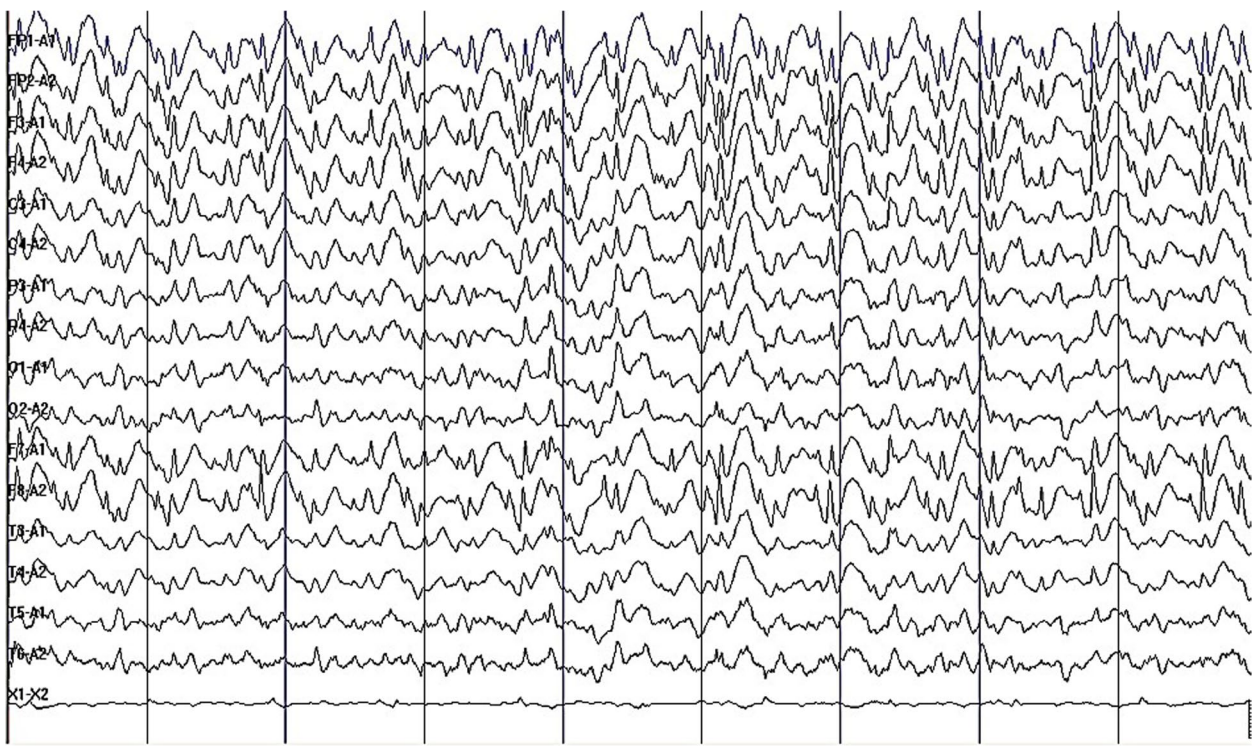

Fig. 4 EEG recordings of case 4. Generalized 3- to 5-Hz spike-and-wave with frontal and temporal predominance

(500 mg/day). Her clinical signs disappeared gradually and were totally gone in the next day. The postictal EEG was also not recorded.

Discussion

In recent years, there have been increasing attempts by physicians to better define and classify NCSE, in order to establish treatment paradigms for different subtypes. Instead of the traditional dichotomy, some clinicians suggest that classification should be more elaborated. First, NCSE can be divided into two categories: the generalized and the focal NCSEs. The generalized NCSE comprises typical absence SE, atypical absence SE and late-onset absence SE. The focal NCSE consists of simple partial SE (SPSE), complex partial SE (CPSE) and subtle SE [10]. Regardless of the type, the classification scheme is mainly based on clinical symptoms and EEG features. However, in fact, it is sometimes quite difficult to distinguish between generalized and focal NSCE, especially when there is no EEG available. Even with the availability of EEG data, it is still hard to differentiate these subtypes because the EEG pattern can be a transient phenomenon. For example, it can be focal initially and transform to be generalized later, or the opposite [11]. Therefore, clinicians should take advantage of all information available to determine the subtypes, with at least diagnosis of NCSE in extreme difficulties.

Unlike convulsive status epilepticus which is easy to diagnose from the clinical manifestations, NCSE is often misdiagnosed, sometimes even undetected because of its protean symptoms. Therefore, NCSE was used to be considered as a rare condition. In this report, the 4 cases were diagnosed as NCSE according to the 2017 ILAE classification [12], and most of them presented with impaired consciousness (confused, slow reaction and lags in response) and some strange behaviors (being upset and restless or washing hands repeatedly). None of them had any obvious motor symptoms like tonic or clonic movements. Notably, the clinical signs of case 4 were so mild that spectators may overlook these abnormities and come to a conclusion of "Nothing wrong with her. Maybe she is just a little tired". Indeed, the semiology of NCSE is diverse and daedal. Some patients present with typical absence or complex partial SE, whereas some may display other unusual alterations of consciousness (varying from mildly inattentive, confused, somnolent to unresponsive), affect (euphoric, anxious, amused, etc.), behavior (agitated, bizarre and inappropriate; Fugue states), speech and language (slow or decreased speech and volume; dysarthria, speech arrest), motor (staring, blinking, bradykinesia; automatisms like chewing, grimacing, licking, kissing, picking, and ambulation; subtle facial, perioral, and limb myoclonus, tremor, apraxia, clumsiness; head deviation) and autonomic/vegetative symptoms [2, 8]. Even comatose patients without overt seizure activity may meet the diagnosis criteria of NCSE [9, 13]. Most symptoms of NCSE are so inconspicuous that they can be easily neglected by others, even the family members. Moreover, it is not uncommon for some clinicians to mistake NCSE for postictal confusion after a generalized tonic-clonic seizure [14], transient global amnesia, hysterical fugue states, acute psychosis, migraine aura, posttraumatic amnesia, and severe depression, which may result in the underrecognition and underdiagnosis of NCSE [3, 10].

Apart from the above clinical manifestations, EEG also plays an important role in the diagnosis of NCSE. Under some circumstances where clinical signs are subtle or even absent, EEG is becoming especially valuable. However, we have to note that EEG interpretation is a subjective "art", and diagnosis of NCSE based on it may not come to consistency among interpreters. Furthermore, EEG of NCSE can have various forms, which makes it more difficult to interpret. Some clinicians suggest that the typical EEG features of NCSE are typical spike-and-wave, atypical spike and wave, multiple spike-and-wave, and rhythmic delta with intermittent spikes. These discharges may be continuous or persistent with brief pauses of a few seconds, or intermittent [15]. Some have also mentioned that different subtypes may show different EEG patterns. ASE usually manifests with continuous or frequently recurring generalized spike and wave discharges during ictal period, and the number of spikes per wave is >1 [16]. CPSE manifests with continuous or persistent sharp wave and spike-and-wave discharges, which can have a generalized onset or a focal onset which frequently progresses into the generalized pattern [17]. In this report, EEG of the 4 cases initially manifested with either a focal or a generalized onset, then evolved into spike-and-wave pattern gradually. Three cases, except for case 2, all presented with focal predominance. To facilitate clinicians to recognize and diagnose NCSE, the following EEG diagnostic criteria have been suggested: frequent or continuous focal electrographic seizures; the amplitude, frequency and spatial distribution can be changing with time; patients without a pre-existing epilepsy history manifest with frequent or continuous generalized spike wave discharge; in patients with an epileptic encephalopathy/syndrome, EEG presents with frequent or continuous generalized spike wave discharges which are significantly different in intensity or frequency (usually a faster frequency) from baseline EEG; patients who are in coma after a generalized tonic-clonic SE show periodic lateralized epileptiform discharges or bilateral periodic epileptiform discharges [18].

According previous studies, the underlying causes and medical conditions of NCSE may include pre-existing epilepsy, metabolic disorders, alcohol withdrawal, the use of some neuroleptic/psychotropic drugs, cerebral infarction or hemorrhage, infection like meningitis and encephalitis, sepsis, carbon monoxide and toxic [10, 19]. There are even some case reports of NCSE associated with AEDs, like tiagabine [20]. Among the 4 cases, three had a history of complex partial epilepsy but none of them experienced any other medical conditions, so the pre-existing epilepsy may be the possible cause for the NCSE. Case 4 did not have a pre-existing history of epilepsy, but she was diagnosed as diabetes mellitus several years ago. Metabolic disorders have been reported as the underlying causes of NCSE in previous studies, so we inferred diabetes mellitus contributed to NCSE of this patient.

As far as is concerned, the most challenging work for clinicians is to diagnose NCSE rather than to treat it. Nevertheless, there is still debate over how aggressive the treatment should be. The most widely-accepted opinion is that the treatment should be individualized, due to the diverse causes and types. Typical ASE is usually treated by intravenous administration of 10 mg of diazepam or 4 mg of lorazepam, which can be repeated if the seizures continue 10 min after the treatment [10]. Atypical ASE may not have a favorable response to benzodiazepines. Valproic acid and phenobarbital are reasonable alternatives. In patients with pre-existing epilepsy, SPSE and CPSE may respond to benzodiazepines rapidly, sometimes even spontaneously terminate without any medical therapy. In this report, the first three patients with pre-existing partial epilepsy all responded well to benzodiazepines (diazepam) in both clinical and EEG aspects. As for those without a history of epilepsy but with other underlying causes and medical conditions, SPSE and CPSE are usually refractory to the first-line treatments. In that case, subsequent intravenous phenobarbital or valproic acid should be added [21]. However, here we found that in the case 4 patient who did not have a previous history of epilepsy, the clinical signs gradually disappeared after an oral administration of levetiracetam, without intravenous medicine like benzodiazepines, phenobarbital or valproic acid. Therefore, it remains unknown whether the NCSE terminated spontaneously or because of the medicine, though levetiracetam has also been proved to be an effective treatment in recent years [22, 23]. Although medical treatment has been proved to be helpful, in some occasions aggressive treatment can have a greater risk on morbidity and mortality [24, 25]. For example, comatose NCSE patients treated with benzodiazepines may worsen [26], so caution should be taken with drug administration.

The outcome assessment of NCSE is challenging for clinicians because it is difficult to separate the effects of ongoing seizure activity from those of an underlying course and complications which occur in the clinical course. The prognosis of NCSE remains controversial. Some case series have reported high mortality and morbidity rates. Shneker et al. found that 18 NCSE patients in their series died (18%), and suggested that the mortality is significantly associated with the underlying etiology, severe mental status impairment, and development of acute complications [27]. Kjersti and his colleagues reported a poor outcome in 48 NCSE patients: 3 died (6.3%), 4 had severe sequelae (8.5%) and 7 had cognitive sequelae (14.9%). They concluded that the absence of previous seizures is a predictor for a worse outcome than the patients with epilepsy before NCSE [28]. Also, some clinicians have emphasized that NCSE, especially CPSE, can lead to a poor outcome: death, persistent or permanent cognitive or memory loss, and motor and sensory dysfunction [29]. Furthermore, some researchers have confirmed that serum neuron-specific enolase (s-NSE), a marker for acute neuronal injury, is increased significantly in NCSE patients, indicating that NCSE can cause brain injury [30–32], so these authors insist that the aggressive therapy is indeed necessary and worthy. On the contrary, some clinicians have suggested that NCSE is a kind of "benign" condition and the outcome is quite good, especially for ASE. They suggest that even inadequate treatment can lead to good favorable prognosis [33]. Some researchers believe that NCSE would not cause damage to the brain, and the high morbidity in some case series of NCSE may be due to the underlying disease of the patients rather than the NCSE per se [34]. In this report, all the 4 patients had a good prognosis without any cognitive and severe sequela. The favorable prognosis may be associated with the pre-existing epilepsy, satisfactory response to medication (the first 3 cases), and the extreme mild clinical signs (case 4).

Conclusions

NCSE is a great burden both for families and in economic concerns. Despite a favorable outcome in most patients, it still can be fatal in some cases. The risk of death will be increased if patients are untreated or receive insufficient treatment. Yet there are no widely accepted definition and criteria of NCSE, which make it difficult to diagnose this disease and administer corresponding treatment. What is more, it remains unclear how aggressive the treatment should be. Further work should be focused on these aspects, in the aim to establish and improve the diagnosis of and treatment patterns for NCSE.

Abbreviations
NCSE: Nonconvulsive status epilepticus; GCSE: Generalized tonic-clonic status epilepticus; SE: Status epilepticus; EEG: Electroencephalogram; ASE: Absence status epilepticus; CPSE: Complex partial status epilepticus; GTCS: Generalized tonic-clonic seizures; SPSE: Simple partial status epilepticus; TSW: Typical spike-and-wave; ASW: Atypical spike-and-wave; MSW: Multiple spike-and-wave; RDIS: Rhythmic delta with intermittent spikes; biPEDs: Bilateral periodic epileptiform discharges.

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Authors' contributions
WS provided the ideas and the revision on the article. DW and XL summarized the views and were major contributors in writing the manuscript. XY, FY, JZ and HY collected the clinical data of patients. All authors read and approved the final manuscript.

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Availability of data and materials
Supporting data are available upon request.

Declarations

Ethics approval and consent to participate
The use of sample from the patients was approved by the Institutional Ethics Committee of Xuan Wu hospital Capital Medical University and informed consents have been obtained from patients prior to analysis.

Consent for publication
The informed consent about publication was obtained from the patient.

Competing interests
Author Wei Sun is the member of the Editorial Board for Acta Epileptologica, who was not involved in the journal's review of, or decisions related to this manuscript.

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