

## LETTER TO THE EDITOR

**Non-convulsive status epilepticus as an unrecognized cause of acute confusion in alcoholics**

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Sir,

We report the case of an alcoholic adult who suffered from a prolonged episode of confusion and cognitive changes as consequence of non-convulsive status epilepticus (NCSE) precipitated by alcohol withdrawal.

Our patient is a 57-year-old man who was admitted to our emergency unit because of disorientation and behaviour disturbances lasting 24 h. He had antecedents of severe chronic alcoholism.

There was no previous history of head trauma, encephalitis or seizures. On examination, he was confused and disoriented to time. A mild distal tremor was also observed. Subsequently, he experienced a brief generalized tonic-clonic seizure at the emergency room. Routine laboratory tests including sodium (142 mEq/l), potassium (4.30 mEq/l), creatinine (1.10 mg/dl), blood urea nitrogen (31 mg/dl) and ammonia (23  $\mu$ mol/l) were within normal limits. A computed tomography scan of the brain was unremarkable. Cerebrospinal fluid revealed normal values. Twelve hours after admission, an electroencephalogram (EEG) showed the existence of generalized long-lasting bursts of well-formed rhythmic sharp-and-slow wave complexes at 2.0–2.5 Hz (Fig. 1). The injection of 10 mg of intravenous diazepam diminished markedly the amount of epileptiform discharges and a significant patient's mental state improvement was observed. On the basis of EEG, we made the diagnosis of generalized NCSE. Treatment with intravenous phenytoin (100 mg/8 h) and clonazepam (0.5 mg/8 h) was begun. Twenty-four hours after the diagnosis, a second EEG revealed a normal background and burst of irregular arrhythmic delta waves localized over frontal regions, but without evidence of epileptiform activity. At that moment, patient's mental state was considered normal. Magnetic resonance imaging of the brain disclosed multiple small deep hyperintense lesions suggestive of subcortical chronic ischaemia. The patient recognized a reduction of alcohol ingestion during the days previous to his

hospital admission. Finally, he was discharged on treatment with oral phenytoin (100 mg/8 h), and the advice of complete alcoholic abstinence. He has not suffered from further seizures for the last 3 years.

It seems reasonable to assume that the episode of confusion described here was consequence of a generalized NCSE provoked by the reduction of the alcohol ingestion. Acute episodes of mental alteration in chronic alcoholics are frequently thought to be due to neuropsychiatric conditions such as delirium tremens, alcohol hallucinosis or Wernicke encephalopathy. However, alcohol has been recognized as an increasing subjacent cause of NCSE [1]. Thomas *et al.* [2] observed that a history of alcohol abuse was a frequent antecedent in those patients diagnosed with *de novo* absence status of late onset. This epileptic entity is considered a situation-related generalized NCSE. Although it is often precipitated by an acute withdrawal of psychotropics drugs, others toxic or metabolic causes including an acute or chronic alcohol abuse have been also reported as triggering factors [2,3]. Nevertheless, *de novo* generalized NCSE is not the unique type of alcohol-related NCSE. Thus, complex partial status epilepticus, a type of localization-related NCSE that usually occurs in the setting of pre-existing partial epilepsy or focal acute or chronic cerebral injury, has been also rarely described in association with acute intoxication or non-supervised withdrawal of alcohol [4,5].

It could be argued that vascular lesions were the origin of the non-convulsive status. However, on that case, focal recurrent epileptiform discharges, periodic anomalies or regional asymmetries should be expected.

In conclusion, our case emphasises the importance to have in account that generalized NCSE secondary to alcohol withdrawal may be the cause of acute confusion and behaviour changes in the absence of evident cerebral injury. An EEG should be carried out as soon as possible to confirm the diagnosis, to classify the type of NCSE and to initiate a prompt antiepileptic therapy.

**References**

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**Figure 1** A portable paper electroencephalogram (EEG) showed the presence of frequent generalized burst of well-formed 2.0–2.5 Hz sharp-and-slow wave complexes in keeping with the diagnosis of generalized non-convulsive status epilepticus. Low filter: 0.5 Hz; high filter: 30 Hz; notch filter: 50 Hz. Vertical bar: 100  $\mu$ V; horizontal bar: 1 s.

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