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Case report

Subacute encephalopathy and seizures in alcoholics (SESA) presenting with non-convulsive status epilepticus

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ABSTRACT

Subacute encephalopathy with seizures in chronic alcoholism (SESA) was first described in 1981 by Niedermeyer who reported alcoholic patients presenting with confusion, seizures and focal neurological deficits and is quite distinct from patients presenting with typical alcohol withdrawal seizures. EEG often reveals periodic discharges and spikes, but SESA presenting with non-convulsive status epilepticus has rarely been described.

We report a case of SESA with non-convulsive status epilepticus in a patient who was initially suspected of having a typical alcohol withdrawal seizure.

A 61 year old woman with a history of chronic alcoholism was admitted at an outside hospital for confusion thought to be secondary to an alcohol withdrawal seizure. She had right hemiparesis and later developed right facial twitching that did not respond to intravenous fosphenytoin and levetiracetam. She was transferred for further management. Upon arrival, lorazepam and fosphenytoin were given and right face clonic movements resolved. However, continuous EEG monitoring revealed ongoing non-convulsive status epilepticus (NCSE). Following treatment with IV valproate and lacosamide, there was resolution of NCSE.

SESA is likely an under recognized clinical syndrome that is quite distinct from typical alcohol withdrawal seizures and requires a different diagnostic and management approach. NCSE is likely to account for the encephalopathy and focal neurological deficits seen in patients presenting with the clinical syndrome of SESA. Therefore, a high degree of suspicion is warranted and continuous EEG monitoring is recommended for alcoholic patients with encephalopathy and focal neurological deficits.

1. Case report

A 61 year old woman with a history of chronic alcoholism, alcohol withdrawal seizures and hypertension was admitted at an outside hospital for confusion and right hemiparesis. CT of the head was positive for basal ganglia calcifications, but was otherwise unremarkable. A routine EEG showed diffuse 6 Hz background slowing. A subsequent stroke evaluation including repeat CT head, carotid Dopplers and transthoracic echocardiogram were all negative. Laboratory data was unremarkable including urine drug screen and tests of hepatobilliary function (AST = 40 U/L, ALT = 14 U/L, total billirubin = 0.4 mg/dL and ammonia = 50 μmol/L). Alcohol level was less than 7 mg/dL.

She subsequently developed right facial twitching which did not resolve following intravenous loads of fosphenytoin and levetiracetam. She was transferred to the ICU at our facility for further evaluation including continuous EEG monitoring.

Neurologic exam upon arrival revealed the patient to be lethargic and unable to follow simple commands with a left gaze preference, right facial twitching and right hemiparesis. There was hyperreflexia on the right upper and lower extremity with a right Babinski sign. Total phenytoin level was 11.4 μg/mL.

Initial MRI of the brain showed T2 and FLAIR hyperintensities involving the cortical gray matter in the left frontal, parietal and temporal lobes (Fig. 1). Diffusion weighted imaging demonstrated several areas of restricted diffusion and reduced apparent diffusion coefficients in the same regions of the left hemisphere.

Lorazepam and an additional load of fosphenytoin were given and right facial twitching resolved. However, continuous video EEG monitoring revealed frequent left hemisphere electrographic seizures consisting of evolving, rhythmic spike and slow wave discharges lasting 10–20 s, maximal in the left temporal and parietal regions (Fig. 2). In between electrographic seizures, the EEG revealed left hemisphere periodic lateralized epileptiform discharges (PLEDs). The electrographic seizures and periodic discharges were seen in the same distribution as the imaging abnormalities. The patient was diagnosed with subacute encephalopathy with seizures in alcoholics (SESA) and non-convulsive

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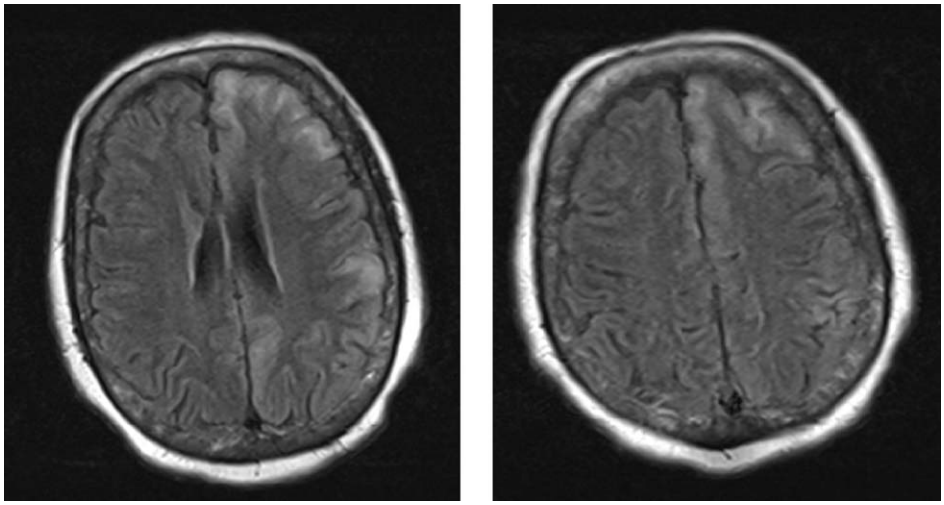

Fig. 1. Axial FLAIR images on initial presentation demonstrating cortical hyperintensities in gyriform pattern in the left frontal, temporal and parietal lobes.

status epilepticus (NCSE). She was loaded with IV valproate followed by lacosamide and had gradual resolution of NCSE followed by clinical improvement in mental status and resolution of right hemiparesis.

One month after discharge this patient was readmitted at an outside hospital for another prolonged episode of confusion with right hemiparesis and right limb focal seizures. It was determined that she had been non-compliant with antiepileptic medications. Lacosamide and valproate were restarted and she returned to baseline. Repeat MRI revealed improvement of the prior T2, FLAIR and DWI hyperintensities (Fig. 3).

2. Discussion

Surprisingly few cases of SESA have been noted in the literature since the original description of 7 cases by Neidermeyer in 1981.[1–8] Although many cases have been associated with periodic lateralized discharges on routine EEG, to our knowledge there have only been two other case reports of SESA and NCSE confirmed by EEG.[5,6] However, this is the first reported case of SESA and NCSE diagnosed by continuous EEG monitoring. As noted, a routine EEG was performed on our patient at the transferring facility which revealed background slowing but no seizures or epileptiform discharges were detected. This finding clearly illustrates the importance of cEEG monitoring in the evaluation of alcoholic patients presenting with the clinical findings of confusion and focal neurological deficits even without report of clinical seizures.

Fernandez-Torre reported the first two cases of patients presenting with the clinical syndrome of SESA and frequent complex partial seizures documented on serial EEG recordings supporting NCSE as integral to the clinical symptoms of SESA.[5,6] In fact, the same authors suggest that SESA should be considered a specific subtype of NCSE given its unique clinical, EEG, imaging and prognostic features.[7] Initial reports describing the neuroimaging

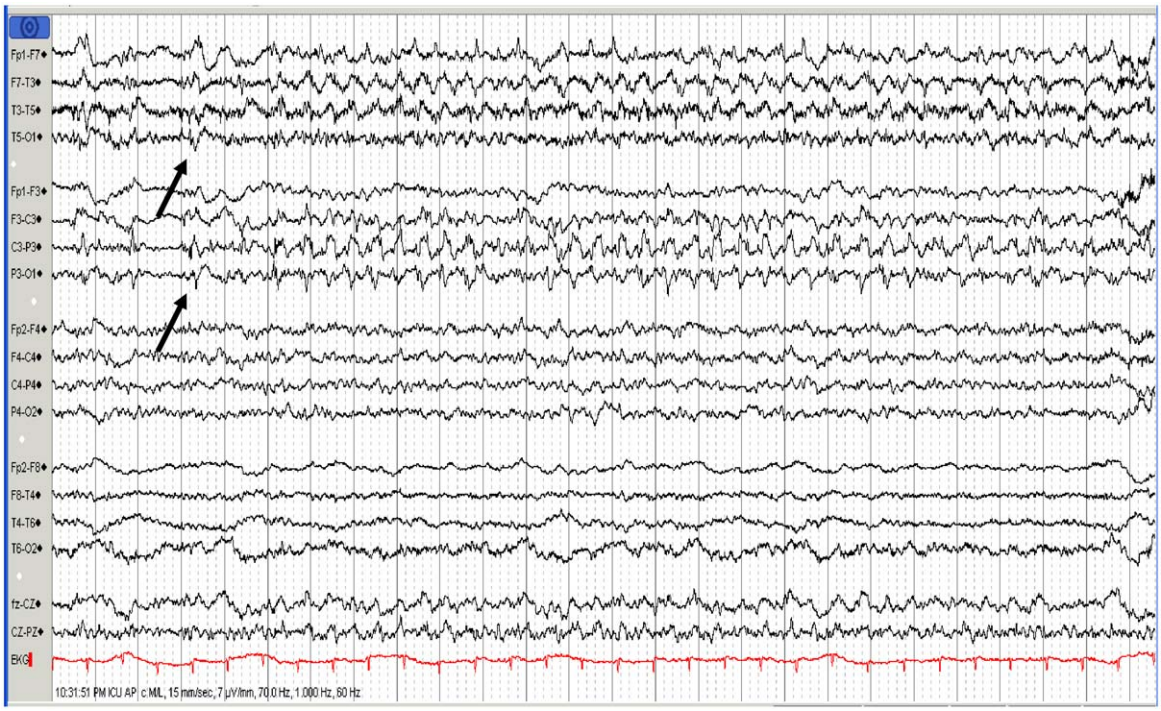

Fig. 2. EEG sample showing typical brief left hemisphere electrographic seizure, maximal in the left temporal and parietal regions.