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Clinical Short Communication

Development of permanent brain damage after subacute encephalopathy with seizures in alcoholics

Coline Duwicquet*, Julien Biberon, Philippe Corcia, Bertrand De Toffol, Nadège Limousin

Unit of Neurology and Clinical Neurophysiology, University hospital Bretonneau, Tours, France

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ABSTRACT

Purpose: To describe additional cases of subacute encephalopathy with seizures in alcoholics (SESA) syndrome, and to question the clinical and radiological course.

Methods: We retrospectively analyzed the clinical characteristics, electroencephalography (EEG), MRI studies at the admission and over the following 6 months of 5 cases of SESA syndrome visited our neurology department between 2010 and 2016.

Results: Five middle-aged males with history of chronic alcohol abuse were admitted for confusion, neurological deficit and seizures. Four patients had recurrent partial seizures requiring 2 or more antiepileptic drugs. EEG showed interictal periodic lateralized discharges in 4 patients and focal rhythmic delta activities in 1. Initial MRI studies revealed unilateral hemispheric cortical–subcortical areas of increased T2/ FLAIR signal and restricted diffusion. Follow up examination after 6 months, revealed persistent focal neurological deficits in 3 patients. Follow-up cerebral MRI at 6 months showed a resolution of the hyperintense lesions, but developing focal atrophic changes in all patients.

Conclusion: SESA syndrome should be included among the alcohol-related encephalopathies as a particular pathophysiological entity. The possibility of permanent brain damage should encourage a better clinical awareness of this syndrome to establish prompt diagnosis, relevant investigation and appropriate treatment of recurrent seizures including, if necessary, intensive care unit treatment.

1. Introduction

Subacute encephalopathy with seizures in alcoholics (SESA) syndrome is an under recognized epileptic entity occurring in a setting of chronic alcoholism first described by Niedermeyer et al. in 1981 [1]. Since then, isolated reports of this rare and controversial disorder appear periodically in the literature although its pathogenesis remains obscure. Fernandez- Torre et al. proposed diagnosis criteria based on clinical and electroencephalographic features [2]. It is characterized by recurrent seizures occurring in alcoholic adult individuals, confusion or lethargy, transient motor deficits, and interictal lateralized periodic discharges (LPDs) on the electroencephalography (EEG). Focal motor and generalized tonic-clonic seizures (GTCSs) were common. Chronic treatment with antiepileptic drugs (AED) is necessary because recurrences are frequent. Radiological features on MRI include transient cortical–subcortical T2-hyperintense areas with restricted diffusion (overlapping the typical findings in status epilepticus (SE)), observed in a patient with atrophy and chronic multifocal vascular lesions [3].

We describe 5 additional cases of SESA syndrome in order to clarify the clinical, neuroimaging characteristics, the prognosis and the physiopathology of this epileptic syndrome.

2. Methods/patients

Five male patients (mean age 61 years (57–76)) with SESA syndrome attended our neurology department between 2010 and 2016. All patients underwent a complete general and neurologic examinations, EEG study and brain MRI on admission and over the following 6 months. The demographic information, clinical details, EEG, neuroimaging data, and prognosis were collated from medical records.

3. Results

Table 1 summarizes the clinical, neuroimaging findings and follow up of our five patients, compared to previous cases in literature.

* Corresponding author at: Neurology, University Hospital of Tours, 2 boulevard Tonnellé, 37044 TOURS Cedex, France.
E-mail address: coline.d@club-internet.fr (C. Duwicquet).

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Table 1
Summary of the clinical and neuroimaging data of our 5 patients in comparison with the previous cases in the literature.

Reference		Age/Sexe	Clinical features	Initial MRI	Seizures duration	Clinical course	Follow up MRI
Rothmeier et al. 2001		60 M	GTCS, confusion, aphasia, L homonymous hemianopsia	Disseminated foci of gliosis, white matter occipital lesions	10 days?	Full neurologic recovery	Occipital changes were reversible but some of the signal enhancements in the white matter persisted.
Otto et al. 2001		66 M	GTCS, confusion, Wernicke aphasia	Cortical and subcortical atrophy. Chronic white matter changes	–	Full neurologic recovery but cognitive impairment	–
Mani et al. 2003		55 M	SPMS, confusion, R hemiparesis	Cortical and subcortical atrophy.	5 days	Mild cognitive impairment	–
Fernandez Torre et al. 2006		65 M	GTCS, CPS, confusion, L hemiparesis	Diffuse cerebral atrophy and deep hyperintense signal areas suggestive of subcortical chronic ischemia. R hemisphere hyperperfusion	4 days	Full neurologic recovery	–
Fernandez Torre et al. 2007		55 M	SPMS and secondarily GTCSs, R hemiparesis	Several hyperintense lesions localized in L frontal and insular cortex	4 days	Full neurologic recovery	–
Laroche et al. 2011		61 F	SPMS, CPS, confusion, R hemiparesis	T2 and FLAIR hyperintensities involving the cortical gray matter in L frontal, parietal and temporal lobes	–	Full neurologic recovery	Improvement of the prior T2, FLAIR and DWI hyperintensities.
Choi et al. 2013	Pat 2	54 F	Secondarily GTCS, aphasia, R hemiparesis	High signal intensity on L medial temporal, parietal and occipital areas on FLAIR, T2-weighted and DWI with high ADC values	4 weeks	Persisting aphasia and motor weakness of the right extremities.	Decreased high signal intensity and atrophic changes at the sites of previous lesion
Fernandez Torre et al. 2014	Pat 1	55 M	GTCS, SPMS, confusion, L hemiparesis	–	–	Full neurologic recovery	–
	Pat 2	58 M	SPMS, confusion, L hemiparesis	Diffuse cortical atrophy, marked ventricular dilatation, R temporo-occipital and L parietal ischemic lesions.	5 days	Full neurologic recovery	–
Drake Perez et al. 2016		69 M	Secondarily GTCS, L hemiparesis	L medial temporal lobe atrophy, R hippocampus, insula, parietal and cingulated cortex, and posteromedial thalamus T2 hyperintensities with restricted diffusion.	–	Gradual neurological recovery	R hippocampal atrophy. Resolution of the T2 hyperintensities.
Current paper	Pat 1	57 M	GTCS, SPMS, R hemiparesis, aphasia	Cortical hyperintensity on diffusion and T2 weighted images on L temporo insular regions and the pulvinar	10 days	Persisting R hemiparesis, aphasia and mild cognitive impairment 4 years later	Disappearance of cortical hyperdensity, atrophy of the underlying cortex and L hippocampic sclerosis
	Pat 2	59 M	SPMS, L hemiparesis, L homonymous hemianopsia	High signal intensity on R temporal, parietal, occipital and limbic areas on T2 and diffusion	5 days	Mild cognitive impairment	Decreased high signal intensities and atrophic changes at the site of the previous lesions
	Pat 3	76 M	SPMS, confusion, R hemiparesis	Unilateral cortical hyperdensity on diffusion and T2 weighted image of the L posterior temporo parietal and occipital regions and pulvinar with variable ADC value	11 days	Persisting R hemiparesis	Atrophic changes at the sites of previous lesion
	Pat 4	58 M	GTCS, SPMS, R hemiparesis, confusion	L medial temporal area, L thalamus hyperintensities on FLAIR, T2-weighted and DWI with high ADC values	1 day	Full neurologic recovery	Resolution of the T2 hyperintensities, L hippocampal atrophy.
	Pat 5	55 M	GTCS, SPMS, L hemiparesis	R temporal parietal and occipital T2 and FLAIR hypertintensities	22 days	Persisting L hemiparesis and anterograde amnesia	Atrophic changes at the sites of previous lesion

CPS: complex partial seizure; GTCS: generalized tonic-clonic seizure; L: left; R; right; SPMS: simple partial motor seizure.

3.1. Clinical cases

Because basically SESA syndrome is a diagnosis of exclusion, all our patients underwent an extensive work up to exclude differential diagnosis. In the setting of chronicle alcoholism, they received intravenous loads of thiamine at the admission. Multiple investigations, including a work-up for infectious (CSF HSV VZV PCR, repeated lumbar punctures), autoimmune and paraneoplastic encephalitis (CSF studies, onconeural antibodies, TAP TDM), were all negatives. Metabolic disorders were excluded by the absence of consistent clinical and laboratory findings (Plasma and CSF lactate were normal). 14.3.3 were performed in all patients.

3.1.1. Case 1

A 57-year-old man with a history of chronic alcohol abuse, mild cognitive impairment (MCI) was admitted to the stroke unit because of sudden right hemiparesis and aphasia. He was in stupor and unable to respond to simple commands. Laboratory data only indicated an increase in hepatic enzymes. His cerebrospinal fluid (CSF) analysis, paraneoplastic, infection and autoimmune screens were normal. Brain MRI showed unilateral cortical hyperintensities on diffusion and T2 weighted images on the left temporo-insular regions and the pulvinar without evidence of infarctions. Routine EEG revealed frequent high amplitude left LPDs. Three days after admission; he presented a GTCS. Treatment with levetiracetam (1000 mg/24 h) was started. Two days later, he had several right side clonic seizures that were controlled after 10 days by add-on lacosamide (400 mg/24 h). Over the ensuing days, there was significant clinical improvement in mental status but his neurological deficit persisted.

Four months later, right hemiparesis and aphasia remained unchanged. Follow-up MRI showed disappearance of cortical hyperintensity associated with an atrophy of the underlying cortex and a left hippocampic sclerosis. Left rhythmic delta activities persisted on EEG. Six months later, in a context of alcohol consumption, focal seizures reappeared requiring the introduction of phenobarbital and topiramate.

3.1.2. Case 2

A 59-year-old man with a chronic alcohol abuse, MCI and alcohol withdrawal seizure history was admitted to the emergency department with a sudden left hemiparesis. He had abstained from drinking alcohol for 2 days. He was confused; he presented a left homonymous hemianopsia (HLH), and a left hemiparesis with permanent left body clonic movement. There were no signs of alcohol withdrawal. Laboratory and CSF examination was unremarkable except for slightly elevated protein content (0.56 g/L). Brain MRI showed hyperintensities in the right temporo-parieto-occipital and limbic areas on FLAIR T2-weighted and diffusion-weighted images (DWI) with restricted diffusion. EEG showed right hemispheric LPDs with background slow activity. Video-EEG monitoring revealed frequent right hemisphere electrographic seizures. Despite extensive work-up that included CSF, infection, paraneoplastic and autoimmune screens no other etiology was determined. He was initially treated by Levetiracetam (1000 mg/24 h) then replaced by Lacosamide (400 mg/24 h). Seizures were controlled 5 days after admission. He was discharged with normal examination.

Six months later, he was seizure free. Follow-up brain MRI showed decreased high signal intensities and progressive atrophic changes at the site of the previous lesions. LPDs persisted on EEG during a month but follow up EEG at 6 months normalized.

3.1.3. Case 3

A 76-year-old man with history of chronic alcoholism and poliomyelitis in childhood developed a focal SE characterized by right arm rhythmic myoclonus with impaired awareness after several months of alcohol abstinence. He was confused and agitated with a right hemiparesis. Initial work up revealed a mild leucocytosis and hyponatremia (123 mmol/L). CSF studies showed slightly elevated protein content (0,58 g/L) and pleocytosis (12 cells/mm). Multiple lab investigations, including a work-up for infectious, autoimmune and paraneoplastic encephalitis were all negatives. Initial brain MRI showed unilateral cortical hyperintensities on diffusion and T2-weighted image of the left posterior temporo-parieto-occipital regions and left pulvinar. EEG showed excess theta-delta activity on the left side with left LPDs.

Treatment with levetiracetam (1000 mg/24 h) was started. After 4 days, mental status worsened and right focal motor seizures persisted, lacosamide (400 mg/24 h) was added. At day 11, focal motor seizures were controlled by add-on valproate (1000 mg/24 h). Recurrence of focal motor SE appeared one month later. Valproate was replaced by Zonesamide (200 mg/24 h), enabling seizure control. He was discharged on Zonesamide and Lacosamide with a right hemiparesis.

At 4 months, he was seizure free but right hemiparesis persisted. Follow-up Brain MRI at 3 months showed a resolution of the hyperintensities but the appearance of a left parieto-occipital atrophy. Subsequent EEGs revealed resolution of the LPDs and persistence of diffuse slowing of the background activity with intermittent theta-delta activity over left temporo-occipital regions.

3.1.4. Case 4

A 58-year-old man was transferred to the Stroke Unit following a GTCS. His past medical history was dominated by chronic alcoholism; alcoholic liver cirrhosis and an acute right subdural hemispheric hematoma treated by surgery a year ago. At admission, he presented a right hemiparesis with left gaze preference and aphasia. He presented a right-sided focal motor seizure. Laboratory data only showed an increase in hepatic enzymes and thrombopenia. CSF studies showed hyperproteinorachia (0.79 g/L). Initial brain MRI showed hyperintensities in the left medial temporal area, and the left thalamus on FLAIR T2-weighted and DWI. His EEG demonstrated bursts of rhythmic delta activities in the left fronto-temporal area. He was treated with levetiracetam (1000 mg/24 h). His mental status became alert and seizures were controlled. At discharge, a month later, aphasia and mild motor weakness of his right upper extremity persisted.

Follow-up brain MRI at one month showed decreased high signal intensity and left slow waves persisted on EEG. At 6 months, clinical examination normalized as well as brain MRI except developing left hippocampal atrophy.

3.1.5. Case 5

A 55-year-old man with a history of chronic alcoholism, chronic renal failure, C and B hepatitis, liver transplant and hypertension was admitted to the emergency room for confusion and left hemiparesis. He had abstained from drinking alcohol for 3 days. He subsequently developed marked eyelid jerking followed by a GTCS, which responded well to intravenous loads of fosphenytoin. He was transferred to the intensive care unit. On examination, he was lethargic, unable to follow simple commands with a right gaze preference and left hemiparesis. His routine blood work, CSF analysis, toxicologic, infection, paraneoplastic and autoimmune screens were normal. Initial brain MRI showed right temporo-parieto-occipital T2 FLAIR hyperintensities. EEG showed right hemispheric LPDs with frequent right hemispheric electrographic seizures associated with subtle left arm jerking. He was first loaded with levetiracetam (2000 mg/24 h). Lacosamide (400 mg/24 h) was added after 2 weeks. Left body SE resolved after 22 days but neither mental status nor left hemiparesis resolved. Due to encephalopathy on routine EEG, levetiracetam was switched with lamotrigine.

After 6 months, the clinical course was unfavourable, mental status impairment and left hemiparesis remained unchanged, however he was seizure free. Follow-up MRI at 2 months showed a cortical atrophy of the underlying cortex.

4. Discussion

The concept of SESA as a distinct syndrome in alcoholics has failed

Table 2
Differential diagnoses to SESA Syndrome.

		Clinical and paraclinical criteria			Clinical and paraclinical criteria
Unihemispheric epileptic syndromes	Stroke	No progression on MRI Vascular systematization	Alcohol-related syndromes	Withdrawal seizure	EEG MRI (+/− cytotoxic edema during the acute and subacute phases and significant volume loss in the temporal regions) Alcohol withdrawal symptoms
	Rasmussen encephalitis	Age at onset MRI (Serial MRIs typically show progression of signal change and atrophy) Clinical and radiological progression		Hepatic encephalopathy	EEG History Liver function tests
	Tumor	MRI (no reversibility)		Delirium Tremens	Alcohol withdrawal symptoms
Inflammatory and infectious diseases	Paraneoplastic syndrome	Tumor search Onconeural antibodies CSF findings		Gayet Wernicke encephalopathy	MRI (medial thalami, mamillary bodies, tectal plate, and periaqueductal gray matter, in a symmetric fashion) Response to IV loads of thiamine
	Herpes virus meningo-encephalitis	MRI (temporal Hyper T2 intensities with restricted DWI, bilateral but asymmetric) PCR HSV CSF		Marchiafava-Bignami Disease	MRI (corpus callosum + +)
	Creutzfeldt-Jakob-disease	14–3–3 protein in CSF (with no absolute specificity) EEG MRI (Bilateral symmetric, hyperintensities of the basal ganglia on T2-weighted MRIs) Clinical course		Osmotic demyelination syndrome	MRI (central pontine changes (although symmetric signal changes can be seen in basal ganglia, thalami, lateral geniculate body, cerebellum, and cerebral cortex)) History Blood test
	PRESS syndrome	MRI (focal regions of symmetric hemispheric edema) History			
Neuro-metabolic disease	MELAS and other mitochondriopathies	MRI (multifocal areas of hyperT2 signal confided to the cortex and adjacent white matter) History, clinical course Biology			

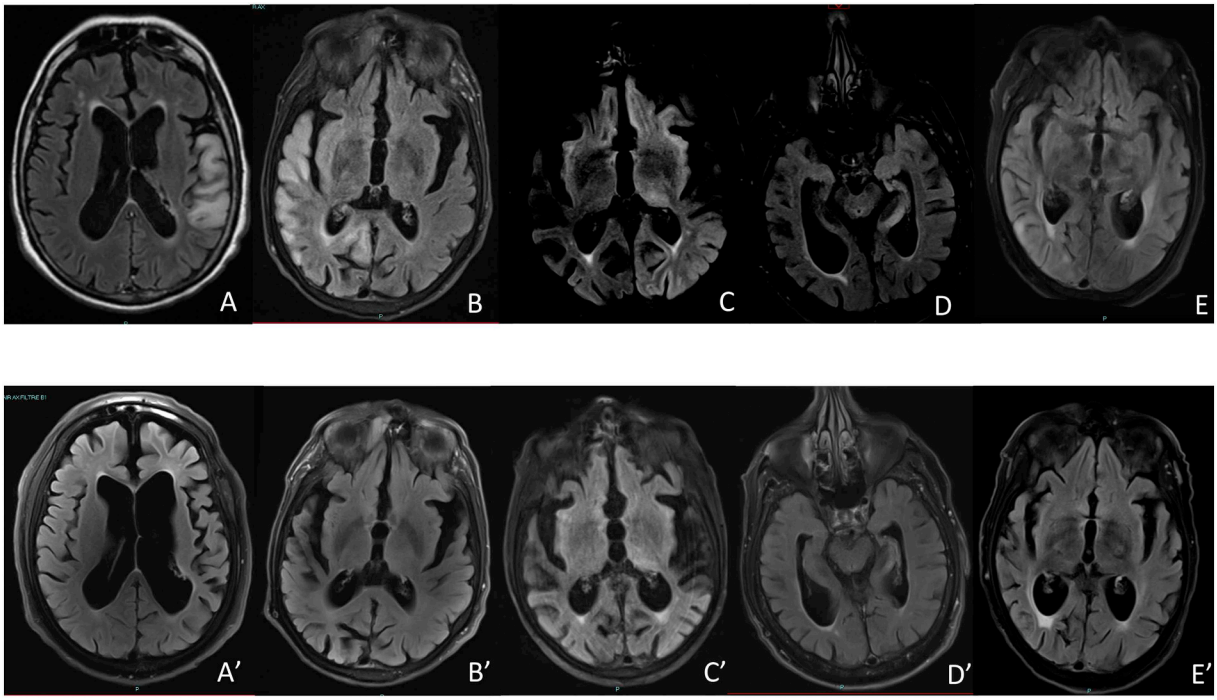

Fig. 1. Initial (ABCDE) and follow up (A'B'C'D'E') brain MRI images from our 5 patients showing the distribution of the signal alterations and their resolution with the development of focal atrophic change (except for patient 4).
Patient 1: Left temporo-insular regions hyperintensities on axial FLAIR images (A), FLAIR MRI acquired 4 months later, showing reversal of the abnormalities with an atrophy of the underlying cortex (A'). Patient 2: Right temporo-parieto-occipital and limbic areas hyperintensities on axial FLAIR image (B), Axial FLAIR images 6 months after initial presentation demonstrating resolving hyperintensities and atrophic changes at the sites of previous lesion (B′). Patient 3: Left temporo parietal and occipital and left pulvinar hyperintensities on axial DWI (C), FLAIR MRI performed 3 months later showing the resolution of the T2 hyperintensities with an atrophy of the underlying cortex (C′). Patient 4: Left medial temporal lobe hyperintensities on axial DWI (D) FLAIR) MRI acquired 4 months later, showing reversal of the abnormalities. (D'). Patient 5: Right temporo–parieto-occipital hypertintensities on axial DWI (E) FLAIR MRI acquired 2 months later, showing reversal of the abnormalities and the development of atrophic changes at the sites of previous lesion. (E').

to gather much traction since Niedermeyer et al.'s 1981 report; indeed, 37 years later we were able to identify only 17 patients other than our own with the purported syndrome (Table 1). The initial proposal considered that the clinical picture described in SESA did not fit any of the known neurological complications of chronic alcoholism. Following this definition, some reported cases were not characteristic for SESA syndrome and had included patients with other neurological processes.

Accurate diagnosis and delineation of acute neurological disorders in patients with chronic ethanol intoxication may be challenging. Various possible alternative causes for seizures and progressive dementia need to be excluded. Differential diagnostic considerations usually need to cover: other alcohol related neurological syndromes; other reasons for focal SE; or other inflammatory or infectious diseases. Potential differential diagnoses and diagnostic steps to exclude these are summarized in Table 2. All those differential diagnosis had been excluded in our patients.

The diagnosis of SESA syndrome rests on clinical, EEG and MRI studies.

As previously described, all of our patients had a previous history of chronic alcohol abuse, and were admitted in a confused state with motor neurological deficits. They all presented focal motor seizures and 3 had secondary GTCS. These recurring seizures lasted on average 9.8 days (ranging from 1 to 22 days). EEG reveal LPDs in all but one patient who presented focal rhythmic delta activities (supplementary material) [2]. These clinical features may represent confounding factors for early diagnosis and therapeutic interventions.

Our cohort with six-months follow-up highlights that patients with SESA may display permanent brain damage, with radiological changes and permanent focal neurological deficits. Three of our patients had a persistent neurological deficit at 6 months, 4 presented a decline in cognitive functions and 1 developed pharmaco-resistant epilepsy. Previously, only 1 case of persistent motor and 2 of cognitive deficits have been reported [4–6] but data on the duration of follow up or the clinical course are not always available. SESA syndrome has been described with a good response to AED. However, most of our patients (4/5) as those reported by Fernandez-Torre (7/9), became seizure free after 2 or more AED [2].

Imaging plays an important role in diagnosis by demonstrating focal or uni-hemispheric involvement and excluding other possible causes. As Drake Perez et al. pointed it out; MRI abnormalities in SESA syndrome differ from other alcohol-related condition [3,7]. Initial brain MRI studies revealed a pre-existing global cortico-sub-cortical atrophy and transient focal cortical hyperintensities on T2 and FLAIR sequences, and DWI in all patients [3]. The location of MRI anomalies correlated with the LPDs on EEG and the clinic. Follow-up MRI at 6 months showed a resolution of the hyperintensies in all patients but they developed focal atrophy of the underlying cortex (Fig. 1). Similar neuroimaging abnormalities have also been reported after NCSE. These findings are not highly specific for SESA and could be found in recent seizures. However, those neuroimaging abnormalities suggest that diffuse brain injury may occur following after prolonged and repeated focal SE with radiological changes. Some authors speculate that SE may create lesions or functional changes, which subsequently caused focal chronic epilepsy [8]. This chronicity is supported by the existence of recurrence whether medication withdrawal and/or alcohol consumption in SESA syndrome and the possibility of a localization-correlated long-term neurological deficits [2].

Given clinical, EEG and imaging data, SESA syndrome could be considered as a characteristic expression of a NCSE in an alcoholic patient with underlying brain damage. Alcoholic patients commonly display cerebral atrophy related to chronic excitotoxicity [9]. Some authors argue that seizures are secondary to such brain atrophy

[10,11]. Moreover, there is a possibility of direct alcohol toxicity in seizure genesis (i.e. permanent lowering of the seizure threshold) [12,13]. Repeated cycles of alcohol exposure and withdrawal progressively intensify the risk for an alcohol-dependent patient to develop seizures [14]. Localized lesions could equally lead to the genesis of a focal epileptic syndrome such as the SESA syndrome. It has been suggested that an underlying chronic vascular lesion could be the cause. Because alcoholic subjects are exposed to a higher risk of cranial trauma we argue that underlying post-traumatic lesions could also have contributed to the epileptogenesis of this syndrome [15].

We believe that SESA syndrome should be included among the alcohol-related encephalopathies as a particular pathophysiological entity. We thought that the existence of underlying brain damage due to chronic alcoholism (cortical atrophy, direct alcohol toxicity, post traumatic localized lesions…) may explain the unique clinical aspect of this syndrome, with its prolonged state of confusion, persistent motor deficiency, the focal or unihemispheric involvement on EEG and brain MRI and the possibility of long-term neurological deficits that correlate with the focal atrophic changes on MRI. However, the possibility of permanent brain damage after focal SE in patients with chronic alcohol abuse, even without alteration of consciousness, should encourage prompt diagnosis and aggressive treatment of recurrent seizures.

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Disclosure

The authors report no disclosures relevant to the manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jns.2018.10.022.

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