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Short communication

Subacute encephalopathy with seizures in alcoholics (SESA syndrome) revisited

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ABSTRACT

Purpose: The aim of this paper is to describe two additional cases of subacute encephalopathy with seizures in alcoholics (SESA syndrome), and to propose that this entity now should be considered as a subtype of nonconvulsive status epilepticus (NCSE).
Methods: We retrospectively analyzed the clinical characteristics, electroencephalography (EEG), neuroimaging data, and prognosis of these two further cases of SESA syndrome. In addition, we compare our findings with the cases previously described in the English literature in order to propose new diagnostic criteria.
Results: Two adults with history of chronic alcohol abuse were admitted because of confusion and seizures. A routine EEG showed frequent periodic lateralized epileptiform discharges (PLEDs) localized over the right temporal regions. In one case, we captured two complex partial seizures (CPSs) arising from the right hemisphere. Neuroimaging studies revealed subjacent chronic vascular pathology. Following transfer to the intensive care unit (ICU), both improved to antiepileptic treatment and were discharged with full recovery.
Conclusion: On the basis of our findings and a review of the literature, we suggest that SESA syndrome represents a subtype of partial or localization-related NCSE given its particular clinical, electroencephalographic, neuroimaging and prognostic characteristics.

1. Background

An unusual picture of a subacute encephalopathy in chronic alcoholics characterized by confusion or lethargy, transient motor deficits, and marked electroencephalography (EEG) abnormalities was initially characterized by Niedermeyer et al.[1] and Freund and Niedermeyer.[2] EEG findings included focal slowing, spikes, and periodic lateralized epileptiform discharges (PLEDs). Partial motor and generalized tonic-clonic seizures (GTCSs) were common. The patients did not fit criteria for other known neurologic complications in alcoholics, and the authors coined the term *subacute encephalopathy with seizures in alcoholics* (SESA syndrome) to denominate this condition. Surprisingly, few cases of SESA syndrome have been reported since.[3–9]

Recently, Fernández-Torre et al.[6,7] described episodes of temporal and extratemporal complex partial status epilepticus (CPSE) with confusion and transient neurologic deficits in this population. The authors hypothesized that CPSE in the setting of SESA syndrome is more frequent than previously suspected, and suggested that recurrent complex partial seizures (CPSs) might explain the encephalopathy observed in this epileptic disorder. We propose a revision and re-characterization of this entity.

The aim of this paper is to describe two additional cases of SESA syndrome that enlarge the electroclinical spectrum of this neurologic disorder. We review the English literature and redefine the characteristics of SESA as a subtype of partial or localization-related nonconvulsive status epilepticus (NCSE) given its particular clinical, electroencephalographic, neuroimaging and prognostic characteristics.

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2. Clinical cases

A summary of the clinical, EEG and neuroimaging data of our two patients in comparison with the previous cases in the English literature is given in Table 1.

2.1. Patient 1

A 55-year-old man with a history of chronic alcohol abuse, chronic hepatic disease and sensorimotor polyneuropathy was admitted to the intensive care unit (ICU) because of a severe change in mental status after a GTCS. He was in stupor and unable to respond to simple commands, with mild weakness of the left arm and leg. Laboratory data only showed an increase in hepatic enzymes. Brain computed tomography (CT) scan showed diffuse cortical atrophy without evidence of infarctions. A routine EEG revealed mild hemispheric asymmetry with an excess of right-sided theta-delta waves, and frequent high amplitude PLEDs over the right posterior temporo-occipital junction (supplementary data). While in stupor, there were two focal seizures consisting of rhythmic high-amplitude spike-wake complexes with superimposed fast low-voltage activity involving the entire right hemisphere associated with tonic posturing and subtle left arm jerking (Fig. 1). Treatment with phenytoin (300 mg/24 h) was started, and a second brain CT disclosed periventricular hypodensities indicative of small vessel disease. The patient then had several partial motor seizures, and levetiracetam (1500 mg/24 h) was added. Over the ensuing days, there was significant clinical improvement in mental status and resolution of the left hemiparesis. He was discharged on oxcarbazepine (600 mg/24 h) and made a full neurologic recovery.

2.2. Patient 2

A 58-year-old man with history of chronic alcoholism and liver disease was brought to the emergency unit with confusion, and a left-sided focal motor seizure after several months of abstinence. Head CT showed cerebral atrophy without focal structural abnormality. He was confused and agitated with a mild left hemiparesis. EEG showed excess theta-delta activity on the right side with frequent right temporal PLEDs. Treatment with phenytoin (300 mg/24 h) was initiated. Laboratory tests only showed a mild leukocytosis with elevation of the hepatic enzymes. Cerebrospinal fluid studies were normal. The next day mental status worsened and there was a further left partial motor seizure after which the patient was transferred to the ICU, and remained for 14 days. Levetiracetam (1500 mg/24 h) and propofol were started, and after three days, propofol was stopped, with gradual improvement in the level of consciousness. Subsequent EEGs revealed resolution of the PLEDs and normalization of background activity. A cranial magnetic resonance imaging (MRI) showed diffuse cortical atrophy, marked ventricular dilatation, right temporo-occipital and left parietal ischemic lesions. He was discharged on levetiracetam with a normal examination.

Table 1
Summary of the clinical, EEG and neuroimaging data of our two patients in comparison with the previous cases in the English literature.

Author/year	Patient number/ age/sex	Precipitating factor	Seizure type	Neurologic deficit	EEG findings	Neuroimaging	ICU	AEDs
Niedermeyer et al.[1]	7/41–61 (25)[a] 4 M, 3F	Alcohol withdrawal independent	SPMS, GTCS	Hemiparesis, hemianopsia, lethargy	Focal slowing, PLEDs	Diffuse cortical atrophy, low density areas	No	PRM, PHT
Otto and Kozian[3]	1/66/M	Acute alcoholic intoxication	GTCS	Wernicke aphasia	L fronto-centro-temporal PLEDs	Cerebral atrophy; subcortical/periventricular hyperintensities	No	DZP, CLB
Rothmeier et al.[4]	1/60/M	Alcohol abstinence, hyponatremia	GTCS	Confusion, L homonymous hemianopsia, aphasia	R parieto-occipital PLEDs	Disseminated foci of gliosis, white matter occipital lesions	No	CBZ, PHT, DZP
Mani et al.[5]	1/55/M	Acute alcoholic intoxication	SPMS, SGTCS	Confusion, R hemiparesis	L parieto-occipital PLEDs	Cerebral atrophy	No	CBZ
Fernández-Torre et al.[6b]	1/65/M	Alcohol withdrawal, hyponatremia	GTCS, CPS NCSE	Confusion, L upper limb paresis	R temporal PLEDs	Cerebral atrophy, R temporal hyperintense lesions, R hemisphere hyperperfusion (SPECT)	No	PHT, CZP
Fernández-Torre et al.[7]	1/55/M	Alcohol withdrawal	SPMS, SGTC, NCSE	Confusion R hemiparesis	L frontal and parasagittal PLEDs Recurrent L frontal Szs	L frontal and insular hyperintense lesions; frontal hyperperfusion (SPECT)	No	PHT, VPA
Bugnicourt et al.[8]	1/63/M	Unknown	CPS[c] NCSE	Confusion L hemiparesis, L hemianopsia	R hemisphere PLEDs	R occipital stroke, R hemisphere transient cortical hyperintensities	No	VPA
LaRoche and Shivdat-Nanhoe[9]	1/61/F	Unknown	SPMS CPS NCSE	Confusion R hemiparesis	L hemisphere PLEDs	Cortical gray matter frontal, parietal and temporal hyperintensities	Yes (cEEG)	LZP, PHT, VPA, LCM
Present paper (patient 1)	55/M	Alcohol withdrawal	GTCS CPS SPMS NCSE	Confusion L hemiparesis	R temporo-occipital PLEDs	Cerebral atrophy; periventricular hypodensities	Yes	PHT, LEV
Present paper (patient 2)	58/M	Alcohol withdrawal	SPMS	Confusion, L hemiparesis	R temporal PLEDs	Cerebral atrophy; R temporo-occipital and L parietal ischemic lesions	Yes	PHT, LEV, PPF

CBZ: carbamazepine; cEEG: continuous EEG monitoring; CLB: clobazam; CPS: complex partial seizure; CZP: clonazepam; GTCS: generalized tonic-clonic seizure; L: left; LCM: lacosamide; LEV: levetiracetam, LZP: lorazepam; NCSE: nonconvulsive status epilepticus; OXC: Oxcarbazepine PHT: phenytoin; PPF: propofol; PRM: primidone; R; right; SGTCS: secondarily generalized tonic-clonic seizure; SPMS: simple partial motor seizure; VPA: valproate.

[a] One patient was 25-year-old.

[b] First paper demonstrating the existence of complex partial seizures in SESA syndrome.

[c] Presumably complex partial seizures.