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Review

Neuroimaging features in subacute encephalopathy with seizures in alcoholics (SESA syndrome)

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ABSTRACT

Purpose: To describe the neuroimaging findings in subacute encephalopathy with seizures in alcoholics (SESA syndrome).
Methods: We reviewed all cases reported previously, as well as 4 patients diagnosed in our center. We included a total of 8 patients. All subjects had clinical and EEG findings compatible with SESA syndrome and at least one MRI study that did not show other underlying condition that could be responsible for the clinical presentation.
Results: Initial MRI studies revealed the following features: cortical–subcortical areas of increased T2/FLAIR signal and restricted diffusion (6 patients), hyperperfusion (3 patients), atrophy (5 patients), chronic microvascular ischemic changes (4 patients). Follow-up MRI was performed in half of the patients, all showing a resolution of the hyperintense lesions, but developing focal atrophic changes in 75%.
Conclusions: SESA syndrome should be included among the alcohol-related encephalopathies. Its radiological features include transient cortical–subcortical T2-hyperintense areas with restricted diffusion (overlapping the typical findings in status epilepticus) observed in a patient with atrophy and chronic multifocal vascular lesions.

1. Introduction

The diagnosis of acute neurological disorders occurring in patients with a history of alcohol abuse may be challenging. An unusual picture of subacute encephalopathy with seizures (SESA syndrome) in chronic alcoholics was initially characterized by Niedermeyer et al. [1] and Freund and Niedermeyer [2] in 1981. SESA syndrome represents a distinct subtype of localization-related non-convulsive status epilepticus (NCSE) in which recurrent complex partial seizures occur in alcoholic adult individuals, with transient neurologic deficits, interictal periodic lateralized discharges (PLDs) on the electroencephalography (EEG), and chronic multifocal vascular cerebral pathology [3]. This syndrome can be precipitated by alcohol abuse or alcohol withdrawal, and chronic treatment with antiepileptic drugs is necessary because recurrences are frequent. Only a few cases have been reported, with the reports mainly focusing on clinical and electroencephalographic features.

The aim of this clinical report is to better characterize the full spectrum of neuroimaging findings in SESA syndrome. A total of 8 cases were retrospectively analyzed. To the best of our knowledge, this is the first radiological investigation of SESA.

2. Case series

We reviewed all cases reported previously with at least one MRI study, as well as 4 patients diagnosed in our center. All subjects met the following criteria: (1) inclusion of patients with clinical and EEG findings compatible with SESA syndrome, having an available description of their MRI study and (2) exclusion of

Abbreviations: SESA, subacute encephalopathy with seizures in alcoholics; EEG, electroencephalography; NCSE, nonconvulsive status epilepticus; PLDs, periodic lateralized discharges; MRI, magnetic resonance imaging; SE, status epilepticus.

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patients with other key pathologic conditions, like tumors, observed on their neuroimaging studies.

We reviewed a total of 10 patients, 4 from our center (3 of them published in previous papers [3–5]) and 6 from other centers [6–10].

The initial proposal by Niedermeyer et al. [1] considered that the clinical picture described in patients with SESA did not fit any of the known neurological complications of chronic alcoholism. Following this definition, we decided to exclude 2 cases from the previous literature that were not characteristic for SESA syndrome, and had included patients with other neurological processes. We have taken this choice in order to reach a more pure group of patients with SESA in this review: (i) the patient described by Bugnicourt et al. [8] had a right hemispheric stroke involving a large vascular territory on the side of PLDs. The resultant encephalomalacia would be highly epileptogenic, a very common cause of focal seizures in adults. The presence of PLDs and resolution of Todd's paralysis would be completely in line with a focal seizure related to an old infarct. Hence, it would be incorrect to diagnose SESA if we assume the initial proposal by Niedermeyer et al. [1]; (ii) the patient 1 described by Choi et al. [10] had hippocampal sclerosis, a potential highly epileptogenic lesion, on the same side as PLDs, so to say that this patient had SESA and not mesial temporal lobe epilepsy would be a misdiagnosis in our opinion.

In Table 1 the main clinical features and MRI findings of the series of 8 patients are summarized.

3. Results

All patients had a previous history of chronic alcohol abuse, and were admitted to the Emergency Unit presenting with a confused state and with motor neurological deficits. Six patients were men and two women with a mean age of 60.3 years (54–69). Two patients had generalized tonic–clonic seizures (GTCSs), 3 secondarily GTCSs and 3 simple partial motor seizures (SPMSs). After clinical and EEG evaluation, 4 subjects were diagnosed with focal NCSE. Other symptoms included Wernicke aphasia, hemianopsia, and fever. An EEG revealed PLDs in all patients (Fig. 1).

Initial MRI studies revealed cortical–subcortical areas of increased T2/FLAIR signal and restricted diffusion in 6 patients. In 5 patients, the affected region included the temporal lobe. The areas of abnormal signal correlated with the origin of the PLDs on the EEG for all 6 cases (Figs. 2 and 3). Hyperperfusion of the region was observed in 3 of the 6 patients (1 patient had increased distal flow on the MRA, and the other 2 had a SPECT revealing hyperperfusion). The other 3 patients did not have a SPECT or MRA to confirm this fact. Atrophy was present in 62.5% of the patients. Among them, 2 patients showed a temporal predominance, while in 3 patients the atrophy was diffuse or not specified. Chronic microvascular ischemic changes were described in half of the patients. Other isolated findings included: hydrocephalus, Chiari I malformation, and choroid fissure cyst.

Follow-up MRI was performed in 50% of the patients, all showing a resolution of the hyperintense lesions (Fig. 4), but

Table 1
Clinical, EEG and imaging features.

Patient	Age Sex	Clinical features	EEG	Brain MRI	Follow-up MRI
Patient 1 of this paper	69 M	Secondarily GTCSs. L hemiparesis	R temporal PLDs	Atrophy (L medial temporal lobe). Chronic white matter changes. R hippocampus, insula, parietal and cingulated cortex, and posteromedial thalamus: T2 hyperintensity with restricted diffusion.	R hippocampal atrophy. Resolution of the T2 hyperintensities.
Fernández-Torre et al. [3]	58 M	Confusion. SPMSs L hemiparesis. Fever	R temporal PLDs Focal NCSE	Atrophy (temporal). R hippocampus and amygdala T2/FLAIR hyperintensities. R temporal hyperperfusion (SPECT). Hydrocephalus. Chiari I	–
Fernández-Torre et al. [4]	65 M	GTCS L hemiparesis. L homonymous hemianopsia	R temporal PLDs Focal NCSE	Atrophy. Chronic white matter changes. R hippocampus and insular T2 hyperintensities. R hemisphere hyperperfusion, especially in temporal lobe.	R temporal atrophy, especially hippocampal. R temporal and splenium of corpus callosum T2 hyperintensities.
Fernández-Torre et al. [5]	55 M	Confusion. SPMSs, secondarily GTCSs R hemiparesis	L frontal and parasagittal PLDs. Focal NCSE	Chronic white matter changes. L frontoinsular T2 hyperintensities with restricted diffusion. Cortical thickening.	–
Otto et al. [6]	66 M	Confusion Wernicke aphasia GTCSs	L fronto-centro-temporal PLDs	Cortical and subcortical atrophy. Chronic white matter changes.	–
Mani et al. [7]	55 M	Confusion SPMSs R hemiparesis	L parieto-occipital PLDs	Cortical atrophy.	–
LaRoche et al. [9]	61 F	Confusion, R hemiparesis SMPSs	L hemisphere PLDs Focal NCSE	L frontal, parietal and temporal cortical T2 hyperintensities with restricted diffusion.	Improvement of the prior T2, FLAIR and DWI hyperintensities.
Choi et al. [10] Patient (2)	54 F	Secondarily GTCS	L parieto-occipital PLDs + rythmic delta activities	L medial temporal, parietal and occipital T2/FLAIR/DWI/ADC hyperintensities. L middle cerebral artery hyperperfusion.	Decreased high signal intensity and atrophic changes of the previous lesions. Normal MRA.

CPS, complex partial seizure; EEG, electroencephalography; F, female; GTCS, generalized tonic–clonic seizure; L, left; M, male; MRA, magnetic resonance angiography; PLDs, periodic lateralized discharges; R, right; RPCA, right posterior cerebral artery; SPMS, simple partial motor seizure; *, presumptive complex partial status epilepticus.