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Letter to the Editor

Development of permanent brain damage after subacute encephalopathy with seizures in alcoholics (SESA Syndrome)

Dear Editor,

Duwicquet et al. [1] retrospectively describe the clinical characteristics, electroencephalography (EEG) and neuroimaging studies of 5 new cases of subacute encephalopathy with seizures in alcoholics (SESA syndrome) contributing to our knowledge of this special epileptic condition. We would like to make some comments.

In SESA syndrome, simple partial motor seizures (SPMSs), primary or secondarily generalized tonic-clonic seizures (GTCSs) and confusion are the most frequent presenting clinical symptoms. Subsequent clinical examination typically reveals diverse types of neurological deficits including hemiparesis, aphasia, neglect, hemianopsia and cortical blindness. However, these symptoms are not usually seen at the onset of the disease. Later, recurrent complex partial seizures (CPSs) and complex partial status epilepticus (CPSE) arising from temporal and extra-temporal regions may occur [2,3]. It is likely that these episodes of CPSE are responsible for the prolonged alteration in mental state [3].

In two of the cases (case 1 and 2) described by Duwicquet and colleagues [1], the onset of symptoms were that of sudden right hemiparesis and aphasia (case 1) and sudden left hemiparesis (case 2). In both patients, evidence of cerebral infarction was absent. The normal neuroimaging findings might be due to the timing of the study, and be a false negative result. In effect, focal neurological deficits are not a typical presentation of SESA syndrome. In case 1, a GTCS and several SPMSs occurred three and five days after admission, respectively. In case 2, SPMSs were seen at hospital admission, with left body clonic movements associated with the neurological deficits (hemiparesis, hemianopsia), suggesting that the focal deficits occurred as a postictal phenomenon. The normalization of the neurological examination at discharge supports this view. In case 1, it could be argued that there could have been lateralized periodic discharges (LPDs), complicated several days later by GTCSs and SPMSs in the context of a patient with chronic alcoholism. In this case, it is difficult to argue that neurological deficits were a negative ictal, or postictal phenomenon. This point is particularly important since in SESA syndrome, epileptic seizures and delirium are the presenting clinical features and it is this clinical course that allows us to distinguish SESA syndrome from other vascular and epileptic complications in alcoholic subjects. We would like to highlight the fact that patients who develop SESA syndrome frequently have pre-existing multifocal chronic cerebrovascular lesions that, in the setting of alcohol withdrawal, acute intoxication, metabolic disturbances or a combination of them, produce LPDs and recurrent focal seizures. However, major cerebral infarctions and hemorrhages are absent [4]. While SPMSs and GCTCs are the cause of the hospital admission, CPSs and CPSE remain underdiagnosed and hence undertreated, possibly responsible for the confusional state.

The article by Duwicquet et al. [1], describe in detail new cases of SESA syndrome contributing to expand the knowledge on this specific epileptic condition. Moreover, they called attention on the potential that this syndrome has to cause permanent neurological damage supporting its chronic course [4]. Although it does not occur frequently and more studies are needed to confirm these findings, is an significant consideration that further emphasizes the importance of knowing this epileptic syndrome in order to initiate an effective treatment and neurophysiological and neuroradiological follow-up approach as soon as possible.

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