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Letter to the editor

Creutzfeldt Jakob disease masquerading as severe depression: a case report

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Dear Editor,

Cognitive disorders and depression are intensely correlated, not only by sharing risk factors, but also by frequently showing similar presentations. This might lead to challenging cases among the elderly population, since, in old age, depression commonly manifests with prominent neurovegetative and cognitive symptoms[1]. Here we report the case of a patient who had been treated, but after clinical evaluation demonstrated to be a case of sporadic Creutzfeldt-Jakob disease (sCJD), a rare and fatal neurodegenerative condition.

A 77 year-old female, without relevant psychiatric or medical history. Six months before admission, she presented with increasingly irritability and sadness. Within days, clinical condition declined, progressing to mutism, lack of interaction, anorexia, immobility and was treated with different combinations of antidepressants and antipsychotics, with the hypothesis of major depression. She was then referred to the Psychiatry inpatient unit of the local University Hospital for diagnostic investigation and clinical support. Mental status examination at admission was compatible with stupor, but three days later she begin to show spontaneous high amplitude myoclonus and intermittent inhibitory paratonia in inferior limbs, leading us to request Neurology consultation. Previous electroencephalography showed no relevant findings, but new analysis of a previous MRI demonstrated hyperintensity in cortex and caudate at DWI, suggestive of sCJD (Figure 1). Cerebrospinal fluid was sent to specific analyses and it was later confirmed presence of 14-3-3 protein. This, altogether with neurological deterioration and MRI images, reinforced the high probability of prion disease. Myoclonus were not evident at admission probably due to continuous use of benzodiazepines before hospitalization. Because it is an invariably fatal pathology, palliative care was instituted and family opted for home-care.

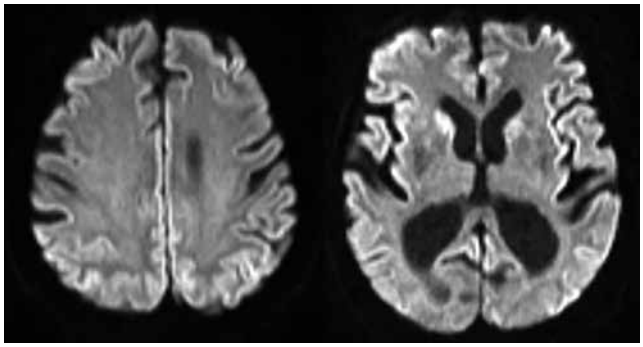

Figure 1. MRI axial cuts showing hyperintensity in areas of the cortex (cortical ribboning) and in caudate nucleus at the DWI sequence.

Prion encephalopathies are caused by an abnormal isoform of a cellular glycoprotein known as a prion, with an infective potential. The most frequent form of prion diseases is sCJD, it affects one in every million people and has no known source of infection[2]. Death usually occurs between 6 and 24 months of illness. About 20% present only with behavioral symptoms at the onset[3], but up to 90% will show psychiatric manifestations at some point of illness evolution, mainly agitation, hallucinations, depression and anxiety[4].

Definite diagnosis of sCJD can only be made through direct analysis of brain tissue. A probable diagnosis is established in the presence of rapidly progressive dementia and two of the following: akinetic mutism, myoclonus, visual/cerebellar signs, pyramidal/extrapyramidal signs; plus one of the tests: EEG with periodic sharp-wave complexes, positive 14-3-3, or MRI with high signal abnormalities in caudate/putamen on DWI or FLAIR; and with routine investigation not suggestive of alternative conditions[5]. Even when a diagnosis of CJD seems probable, the lack of definite noninvasive diagnosis and the irreversibility of the condition, frequently leads clinicians to attempt empirical treatment for other possible etiologies.

This case demonstrates that sCJD is a possible underdiagnosed disease that can be clinically presented masquerade as major depressive disorder. We encourage close collaboration between Neurology and Psychiatry when faced with a patient presenting with a rapid and progressive cognitive deterioration.

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