

Catatonic depression as the presenting manifestation of creutzfeldt-Jakob disease

Sir,

Creutzfeldt-Jakob disease (CJD) is a rare neuro-degenerative prion disease which exists in genetic, acquired (variant and iatrogenic), and spontaneous (sporadic) forms.^[1] The physiopathology of this disease consists of changes to the allosteric conformation of protein in contact with the pathological prion^[2] and neuronal loss is due to deposition in the brain. Typical neuropathology includes spongiosis, neurogliosis, neuronal loss in the absence of inflammation, molecular and genetic alterations.^[3] Clinical symptoms variety with CJD includes anhedonia, anxiety, irritability, depression, insomnia, psychosis, rapid mental deterioration signs and progression of behavioral changes. In the present case, we describe a woman who exhibited a wide range of early psychiatric symptoms initially, and after further systematic diagnostic evaluations eventually turned out to be a probable sporadic CJD (sCJD) case. A 51-year-old woman was referred to psychiatric clinic for markedly reduced attention and concentration, insomnia, weight loss, decreased psychomotor activity and deteriorating memory function. She was diagnosed with depression 20 years ago but she became a full recovery without treatment. The recent history was collected by the patient's sons, who reported that in the last 2 months she exhibited anhedonia, anxiety, a propensity to cry, poor appetite and self-care, talking slowly and in a low tone, short responses to questions, insomnia, frightening face expression and irritability. In detailed neurological examination, she was alert but not oriented in time, toward space and self. Speech was very slow and incomprehensible. Muscle tone was increased in all limbs, with mild-moderate axial rigidity. Muscle strength was normal. Reflexes were brisk. Plantar responses were unresponsive. Because of the different currently psychopathologic history, rapid progression of presenting symptoms and also abnormal neurologic signs; an organic substrate for psychiatric symptoms was hypothesized. Brain magnetic resonance imaging (MRI) showed mild generalized cerebral and cerebellar atrophy and also, diffusion-weighted imaging (DWI) scans demonstrated areas of cortical diffusion restriction. Electroencephalogram (EEG) showed bilateral periodic lateralized epileptiform discharges and

slow wave complexes. There were no myoclonic jerks at first but 2 weeks later, they added to the clinical findings. Cerebrospinal fluid (CSF) studies including paraneoplastic antibody panel, venereal disease, rapid polymerase chain reaction detection for Herpes simplex, Herpes zoster, Epstein-Barr and Cytomegalo virus; fungal antibody survey were unremarkable. However, CSF study was significant for the protein level of 76 mg/dl (reference range, 15-45 mg/dl). Analysis CSF identified elevated level of 14-3-3 protein and tau protein. Final diagnosis was probable sCJD. We initiated treatment with low-dose olanzapine and trazodone (olanzapine up to 15 mg/day and trazodone 50 mg/day) with poor response to treatment. Afterward, the patient's symptoms worsened, with more deterioration in mental status, opposition to feeding and mobilization; neurological symptoms eventually turned evident with akinetic mutism. Finally, she died after 72 days of hospitalization. Consequently, it is very important for psychiatrists to consider CJD among the possible differential diagnoses in elderly patients with particularly negative psychiatric history referring to psychiatrists for recent onset, rapidly progressing symptoms such as cognitive decline, behavioral and personality changes, anxiety, irritability, insomnia, mood changes and non-responsive to treatment psychotic symptoms.

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