A case of catatonia in a patient with bvFTD: insight into a shared physiopathology and treatment

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Catatonia is a psychomotor dysregulation syndrome with inability to make voluntary movements despite full physical capacity
According to DSM-5 criteria, diagnosis of catatonia requires at least 3 of the following 12 clinical features:
mutism, stupor, catalepsy, waxy flexibility, agitation, negativism, posturing, mannerisms, stereotypies, grimacing, echolalia, or echopraxia.
Overlapping symptoms between catatonic syndrome and **behavioural variant of frontotemporal dementia (bvFTD)** have been rarely reported.
Benzodiazepines are first choice of drug treatment.
We aim to to describe a case of retarded catatonia treated with lorazepam and venlafaxine as clinical presentation of bvFTD.

A 63 year-old men was admitted to Medical Department of our hospital for a state of progressive social withdrawal, food refusal with marked weight loss and akinetic mutism. Patient had a 1-year history of mild depression and had an episode of psychomotor retardation 6 months earlier that was treated with sertraline (75 mg/day). At admission patient showed significant weight loss (body weight 42 Kg) and dehydration; he was bedridden with diffuse rigidity, enable to eat, immobile, mute, and with diffuse rigidity. A general medical work-up excluded neoplasm or metabolic/infective conditions. Paraneoplastic or autoimmune encephalitis was ruled out.

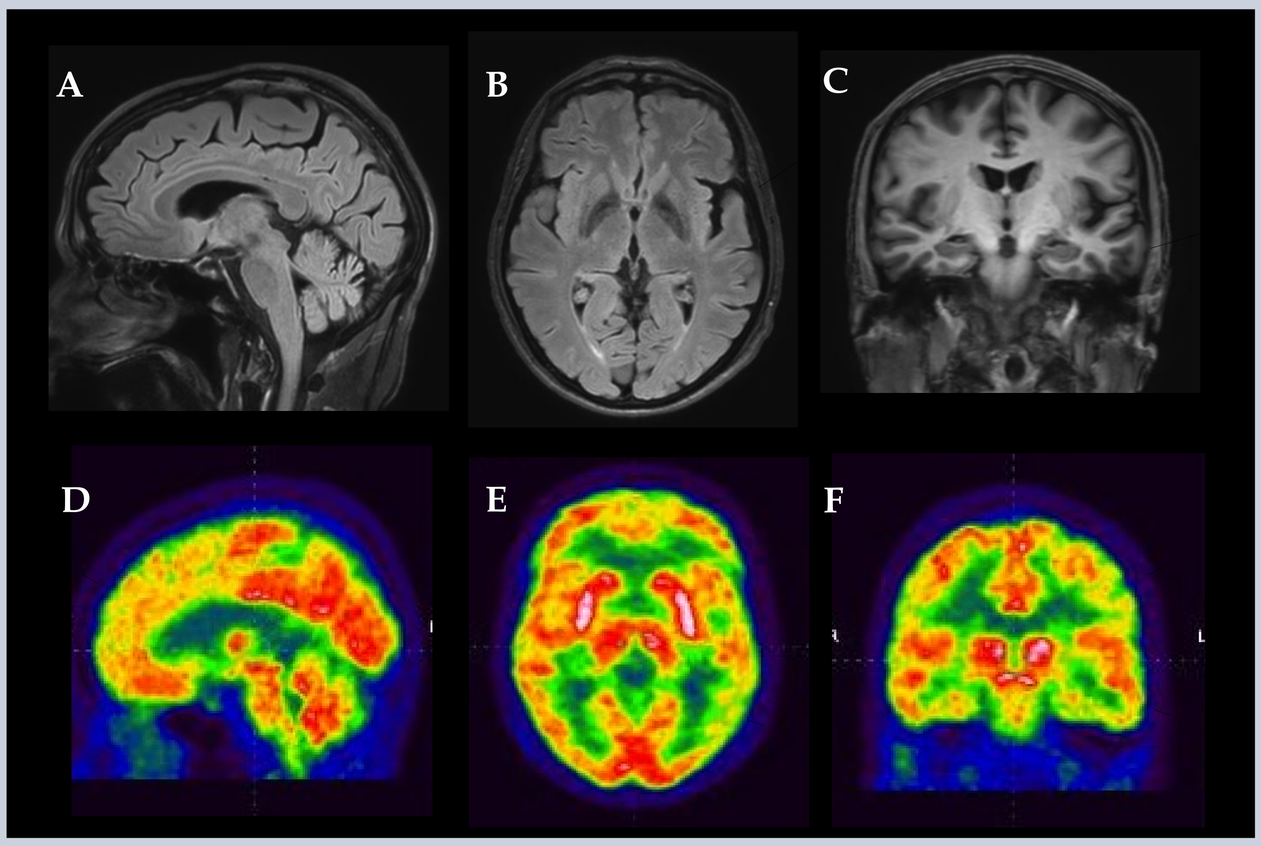

Fig. 1 *MRI brain axial (A), coronal (B), and sagittal (C) scan, with the correspondent PET images (D, E, F). Arrows point to slight left temporal atrophy. Arrowheads point to the left frontal and parieto-temporal hypometabolism.*

Aβ42	978 pg/ml	↑
Total - Tau	118 pg/ml	↓
P - Tau	27 pg/ml	↓

Tab. 1 *Panel of patient's amyloid biomarkers.*

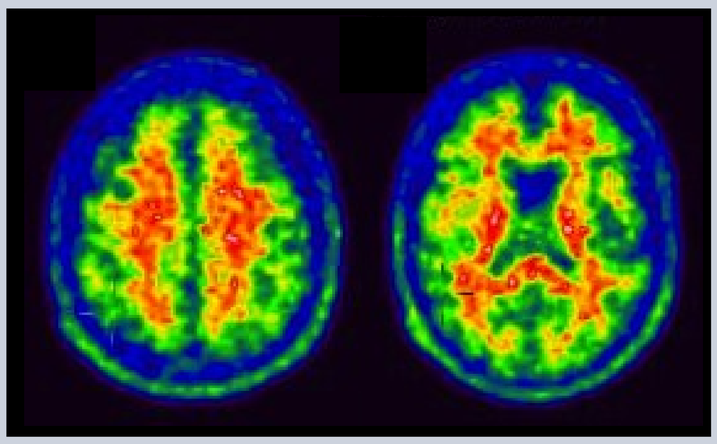

Fig. 2 *^{18}F-florbetaben PET images: note the absence of tracer retention in the cortex.*

CSF analysis showed normal cell count and neurotropic viruses were undetectable on CSF sample using PCR. Brain MRI revealed a very mild atrophy of the left insular cortex; 18F-FDG-PET/MRI scan revealed a hypometabolism on the left parietal-temporal lobe and on left frontal lobe (anterior cingulate cortex and mesial superior frontal gyrus). 18F-Florbetaben PET was amyloid negative. A diagnosis of catatonia was made and **lorazepam** was given orally up to the dose of 5.5 mg daily combined with **venlafaxine** (375 mg/day).

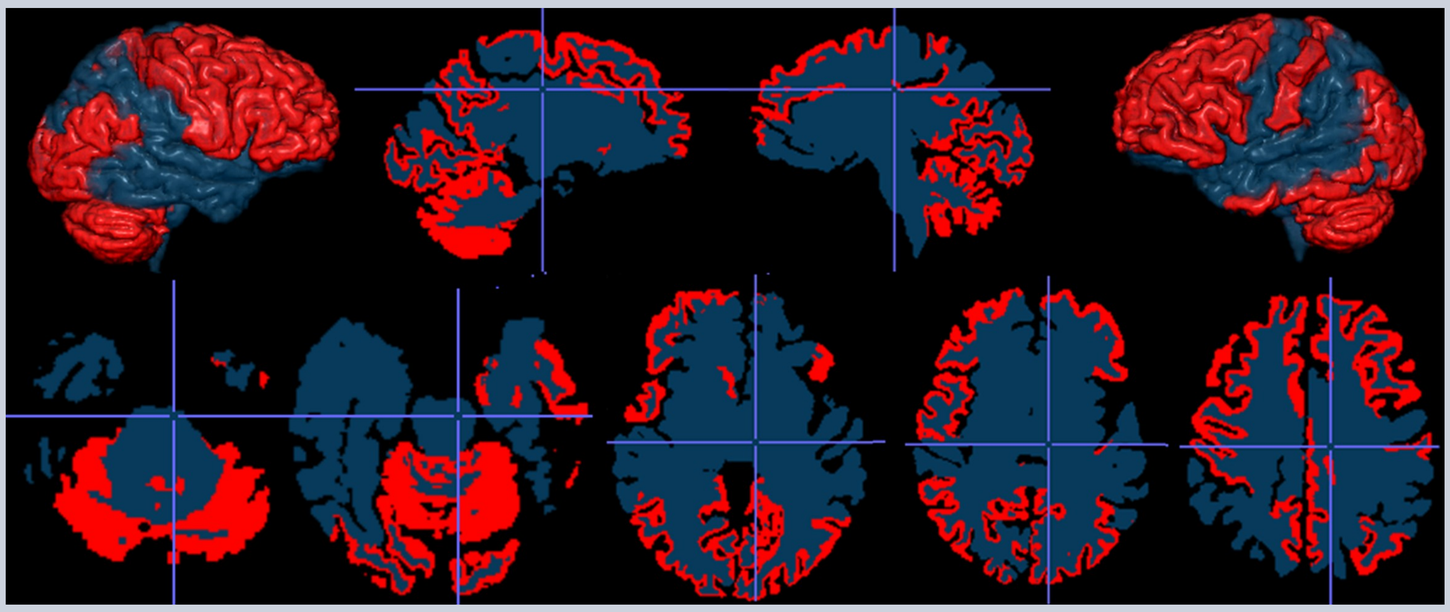

Fig. 3 *Brain MRI skeleton of the patient with superimposed the map of increased metabolism in the second ^{18}FTD-PET-scan respect to the first scan (in red are illustrated the regions with more than 10% of increased metabolism). It could be noted a diffuse increased cortical and cerebellar metabolism (red color), sparing the temporal regions bilaterally and the left insular e parietal cortex (blu color) corresponding to the regions with cortical atrophy (increased silvian fissure on the left).*

A gradual clinical improvement was obtained regarding motor features (patient began walking around hospital) and cognition (MMSE 12/30), his speech became telegraphic and then more fluent; he restarted eating. He was discharged and 6 months later cognitive examination showed mainly executive deficits and difficulties in language comprehension with reduction in phonemic fluency (MMSE 21/30). Repeated ^{18}F-FDG-PET/MRI, 6 months after the first scan, confirmed the pattern of left temporal hypometabolism and revealed a globally increased cortical metabolism in all the other cortical areas and in the cerebellum compared to the previous scan. After 12-month follow-up period a diagnosis of **probable bvFTD**, according to current criteria, was done.

Catatonia is associated with dysfunction of frontal circuits including prefrontal cortex, basal ganglia, and thalamus.
Clinical features of catatonia and bvFTD might overlap, because both syndromes share frontal circuit dysfunction.
Clinicians must keep in mind that, also in the contest of a neurodegenerative disease, catatonia is a potentially reversible life-threatening condition that requires a prompt pharmacological management.

Suzuki K, Takano T, Matsuoka H. A case of catatonia resembling frontotemporal dementia and resolved with electroconvulsive therapy. World J Biol Psychiatry. 2009;10(3):245-7.