Frontotemporal dementia presenting as severe depression

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Frontotemporal dementia (FTD) is a heterogeneous neurodegenerative disease whose early behavioural and emotional symptoms often mimic psychiatric disorders. Here, the authors highlight the challenges of diagnosis with the reassessment of an elderly patient receiving electroconvulsive therapy (ECT) for severe depression.

We met the patient, a retired 76 year old factory worker, for the first time on her readmission to an old age psychiatry unit with recurrent severe depression. She had very recently been discharged following a three to four month admission for psychotic depression earlier in the year, prior to which she had had no contact with psychiatric services or significant medical comorbidities.

Her first admission had been prompted by a period of declining mental state and self-care over several months. She had initially presented to community services with uncharacteristic anxiety (particularly around bills). Later she became increasingly agitated with poor sleep, and finally developing persecutory ideation (‘I know you’re up to something’).

She had begun to refuse antidepressant medication prescribed by her GP, eat minimally, and stopped washing and dressing. Her partner and daughter at this point had assumed most household responsibilities and were providing almost constant supervision. After a period of inpatient treatment with a combination of venlafaxine, mirtazapine, risperidone and eventually ECT (which was prematurely curtailed following a deep vein thrombosis), the patient was discharged home with little progress having been made.

She certainly never returned to the ‘bubbly, talkative, life of the party’ character she was described as having been before her illness. She became passive and unemotional; infrequently initiated conversations, speaking in single words in a tortuously slow manner and engaging in few of the activities she had previously enjoyed. Her partner was shocked to find her uncaring about the birth of her grandson. Significant structure and prompting was required with even simple tasks such as getting out of bed or showering, which would last several hours.

Readmission
Readmission for further ECT was arranged, initially under Section 2 and then converted to Section 3 of the Mental Health Act. At the point where we took over the patient’s care she had received approximately 18 sessions of treatment, during which time it was noted that her mini mental state exam (MMSE) had dropped to 0/30. Assessment revealed a non-reactive affect, severe psychomotor retardation and mild asymmetrical extrapyramidal signs (positive glabellar reflex, right upper limb rigidity and a hesitant fenestrated gait). Significant prompting was required to sustain a conversation, in which she reported ‘feeling fine’ and ‘feeling okay’ after long pauses. She often looked to her partner for reassurance when asked basic questions about her feelings and activities. She seemed entirely unconcerned with her severe difficulties. She generally slept and ate well. No psychotic symptoms were elicited.

Further collateral history
On revisiting the history, the patient’s partner reported previous periods of aimless wandering, and an unusual occasion where she had spontaneously interrupted a restaurant meal to go home – and was later found on a nearby road in a disoriented state.

We were unable to elicit any previous socially inappropriate behaviour, although noted that the patient had been rude to nursing staff (including uncharacteristically swearing). Apathy appeared particularly prominent. Further, she had gained significant weight and was noted to demonstrate a degree of hyperorality in her consumption of sweets and chocolates. There was no family history of dementia or movement disorder (specifically motor neuron disease).

Further investigations
Despite ECT being discontinued, little change was observed in the patient’s presentation. An Addenbrooke’s cognitive examination (ACE) one month post-ECT confirmed continued cognitive impairment (43/100) with prominent fluency and attentional deficits (attention 3/18; fluency 1/14; memory 11/26;
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visuospatial function 11/16). A CT head scan was unremarkable. No abnormalities were detected on a routine battery of blood tests (TSH / B12 / folate / ferritin / syphilis / HIV unremarkable). A SPECT scan (NM HMPAO Ceretec) was requested, a form of functional neuroimaging assessing regional cerebral perfusion and extrapolating energy use / 'activity'. This demonstrated significant symmetrical frontal lobe hypoperfusion – confirming our suspicions of behavioural variant frontotemporal dementia.

In view of the lack of efficacy and extrapyramidal signs, risperidone was withdrawn to no ill effect and the patient was successfully discharged home with social services’ follow up.

Discussion

Frontotemporal dementia encompasses a heterogeneous group of neurodegenerative disorders characterised by progressive behavioural disinhibition, impaired social cognition, and language dysfunction. The complex relationship between genetic-neuropathological abnormalities, patterns of focal atrophy and clinical subtypes continues to be elucidated. Differentiating the insidious onset of emotional blunting, apathy and socially inappropriate behaviour from psychiatric disorders may be more problematic in later-life depression.6 Anecdotally, we have often noticed episodes of uncharacteristic rudeness or swearing in patients who go on to be diagnosed with frontal dementia and note that this is uncommon in the elderly depressed patient. This case led us to consider some interesting questions:

Can ‘hypofrontality’ on SPECT imaging differentiate FTD from severe depression?

Convergent evidence from neuroimaging highlights the role of the prefrontal cortex in depression. However, abnormalities tend to be highly variable in location, nature (eg hyper- or hypoperfusion) and size; only being evident with group-level statistical comparison.7 Positive structural or functional neuroimaging significantly increases the diagnostic certainty of frontotemporal dementia. The behavioural-variant FTD subtype is characterised by an anterior-posterior gradient comprising marked frontotemporal atrophy and hypoperfusion with relative sparing of the parietal lobe.8

Is there any role for genetic testing in FTD in the absence of a strong family history?

In the absence of a family history of FTD or related overlapping neurodegenerative disorder (eg Parkinson’s plus syndrome), the likelihood of detecting identified mutations is low and genetic testing is not currently recommended.9

Can frontal cognitive assessment distinguish FTD from depression?

Clinical tools assessing social cognition and emotional processing have demonstrated significant utility in differentiating the diagnosis early in the disease process, and may have informed the present case.10

Is FTD more common than we imagine in elderly patients with severe depression?

Given the challenges of diagnosis it is very likely that misdiagnosis occurs with greater regularity than is currently recognised.3

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Declaration of interests

The authors declare that they have no competing interests.

References