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CASE REPORT

# It is not depression: A case report of a 37-year-old firefighter with frontotemporal dementia

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## Abstract

A 37-year-old married, right-handed active firefighter presented to the primary care clinic with a self-report of difficulties in memorising and recalling information associated with declining work performance. Collaborative history-taking revealed that he also experienced emotional and social withdrawal, apathy, irritability and distractibility. He was initially diagnosed with major depressive disorder but showed no improvement with an antidepressant. This young man with no history of hereditary neurodegenerative disorder had further deterioration in cognitive function, predominantly executive behaviours, with progressive aphasia. Brain magnetic resonance imaging revealed cerebral atrophy predominant over the frontotemporal lobe. Positron emission tomography showed fluorodeoxyglucose hypometabolism at the bifrontal and left parietal and temporal cortices, consistent with frontotemporal dementia. He required institutionalisation with full nursing care less than 24 months after the onset of the symptoms. This case highlights the need for diagnostic consideration of dementia in young individuals presenting with cognitive impairment among other symptoms. It also emphasises the importance of obtaining collateral information from close relatives to avoid misdiagnosis and addresses the psychosocial impact of dementia at a young age.

## Introduction

Frontotemporal dementia (FTD) is a neurodegenerative disorder characterised by behavioural deterioration, language impairment and cognitive decline. It is the leading cause of young-onset dementia,[1] with a mean age of onset of 58 years.[2] Although occurrences of onset before the age of 40 years are rare, the youngest known case is at the age of 14 years.[3] The diagnosis of FTD in young people is challenging owing to its nonspecific symptoms and findings. Patients are typically unreliable informants for behavioural problems owing to their lack of awareness about this disease. In this case report, the young patient was initially treated for depression before being later discovered clinically to exhibit behavioural changes, progressive cognitive decline and aphasia, accompanied by atrophy over the frontal and temporal lobes, which led to the diagnosis of FTD.

## Case presentation

A 37-year-old man initially visited the primary health clinic with a self-complaint of forgetfulness and difficulty carrying out routine activities at work for a year. He struggled to memorise familiar places and recall friends' names. His wife noticed that the patient had lost interest in daily activities, had less motivation at work and was easily irritable and distractable. He also became emotionally and socially withdrawn. In the past, he was a committed, competent firefighter who loved his job and got along well with his colleague. He lived in a comfortable home with his wife and four children and was a caring husband and father.

The patient was a non-smoker and denied substance misuse or alcohol consumption. Neither history of exposure to toxic substances nor head injury was reported. He had underlying dyslipidaemia treated with simvastatin 40 mg ON. There was no family history of mental illnesses, including dementia. The patient's childhood and adolescence were uneventful.

Physical examination showed normal vital signs, and other systems examinations revealed unremarkable findings. Baseline blood investigation findings were normal, which ruled out underlying thyroid disease or infection. A diagnosis of major depressive disorder (MDD) was initially made, and he was started on sertraline 50 mg daily. However, his response to the antidepressant at the 1-month follow-up was poor. His emotional and behavioural problems persisted, and his memory and language impairments worsened.

The patient was referred to a tertiary centre for further neuropsychological evaluation. A revisit of the history through his wife and colleague disclosed that he had personality and behavioural changes that started around a year ago. He showed disinhibition by taking other people's food without permission and abruptly interrupting other people's conversations. He demonstrated poor judgement in assessing risks by ignoring emergency cases and continued playing on his tablet or watching television while his colleagues attended to them. His wife noted that he had lost compassion, as in a few incidents, he was not bothered by their son's sickness or his colleagues' injuries. His wife also noted that he could not control his diet and gained weight over the past year. More complex compulsive behaviour was also reported as he collected rubber bands and food containers from the trash and brought them back home.

A mental status examination revealed a well-groomed man with poor eye-to-eye contact. He appeared apathetic and laughed when he saw his wife crying. His speech was minimal and irrelevant at times, and his affect was almost mask-like. He smiled only when asked about his mood. The patient denied any hallucinations or delusions. His Mini-Mental State Examination score was 18/30, and other physical examinations showed unremarkable findings.

Extensive investigation findings including complete blood count; electrolyte level; renal, thyroid and liver functions; blood sugar level; lipid profile and folate and vitamin B12 levels were within the normal ranges. Infective screening for human immunodeficiency virus, hepatitis B, hepatitis C and syphilis yielded negative findings. There was no evidence of substance abuse as well. His paraneoplastic panel, autoimmune markers, cerebrospinal fluid and electroencephalogram findings were normal.

Brain CT showed no focal enhancing brain parenchymal lesion, but the ventricles and extra-axial subarachnoid spaces were prominent (left more than right), especially at the left frontal and temporal lobes. Brain magnetic resonance imaging (MRI) proceeded and showed cerebral atrophy with no thrombosis (**Figure 1**). Further positron emission tomography (PET) revealed fluorodeoxyglucose hypometabolism at the bifrontal cortex (left more than right) and left parietal and temporal cortices, which was suggestive of FTD.

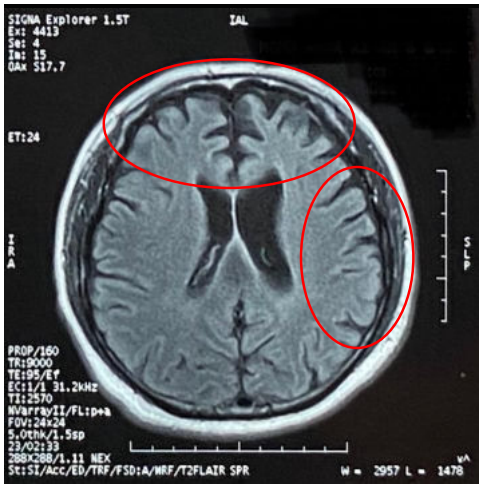


**Figure 1.** Brain magnetic resonance imaging showing cerebral atrophy over the bifrontal and left temporal lobes.

Owing to the presentation of behavioural changes (i.e. disinhibition, loss of empathy, apathy, hyperorality and complex compulsive behaviour), progressive aphasia and memory impairment based on MRI and PET findings, he was diagnosed with FTD.

Sertraline was changed to rivastigmine 6 mg OD and Epilim 200 mg BD. Despite treatment, his condition deteriorated over a few months, and eventually, he was unable to take care of himself and, at times, became incontinent. Previously a loving father, he no longer recognised his children. Within 12 months of the diagnosis, the patient was considered unfit to continue as a firefighter and was laid off with a pension. His working wife had to bear the responsibility as a parent to their children and his caretaker. Within 24 months of the onset of the first symptom, he required total nursing care. His case was referred to a social welfare department for financial and social support. As a caretaker, his wife received appropriate counselling from the attending consultant and was introduced to a carer support group.

## Discussion

FTD is the leading cause of dementia in adults younger than 65 years.[1] It usually begins insidiously from age of 45 to 65 years; nevertheless, in this case report, the patient exhibited dementia symptoms at the age of 37 years.

There are three subtypes of FTD based on

the main clinical presentation. About 50% of cases present with behavioural-variant FTD (bvFTD),[4] which is characterised by behavioural changes. Other cases present with deterioration in language ability – primary progressive aphasia – either as impaired speech production (progressive non-fluent aphasia) or impaired word comprehension and semantic memory (semantic dementia). Each subtype has distinct features, but as the disease progresses, the symptoms may overlap.[5] As in this case, patients mainly present with bvFTD.

Diagnosing bvFTD early in the disease course is challenging because the initial symptoms can mimic those of primary psychiatric disorders such as MDD. The similarities and differences in the clinical symptoms between bvFTD and MDD are shown in **Table 1**.

**Table 1.** Similarities and differences in the clinical symptoms between bvFTD and MDD.

| bvFTD | MDD |
|---|---|
| Apathy in bvFTD is often mistaken for anhedonia in MDD, as both present clinically as a lack of motivation and interest.<br>Impaired concentration<br>Low energy<br>Social withdrawal<br>Changes in appetite | |
| Behavioural symptoms (as listed in **Table 2**) | Sustained depressed mood<br>Feeling worthless<br>Suicidal thoughts |
| Persistent cognitive impairments with progressive executive dysfunction despite improvement in psychiatric symptoms | Following treatment, cognitive symptoms will improve concurrently with mood |

bvFTD, behavioural-variant frontotemporal dementia; MDD, major depressive disorder

Certain behaviours such as socially inappropriate behaviours and other behaviours depicted in **Table 2** are the salient features for distinguishing between bvFTD and MDD. Given the patient's poor insight into his behavioural changes, obtaining a history from his family or close friends was crucial for collecting collateral information to make an accurate diagnosis. In due course, neuropsychological testing and neuroimaging help in the diagnosis.

**Table 2.** Diagnostic criteria for bvFTD.[6]

| | |
|---|---|
| A. *Early behavioural disinhibition | One of these symptoms must be present:<br>i. Inappropriate social behaviour<br>ii. Loss of manners or social decorum<br>iii. Impulsivity |
| B. Early apathy or inertia | One of these symptoms must be present:<br>i. Apathy<br>ii. Inertia |
| C. Early loss of sympathy or empathy | One of these symptoms must be present:<br>i. Lack of response to other people's feelings and needs<br>ii. Reduced social interest, interrelatedness or personal warmth |
| D. Early perseverative, stereotyped or compulsive/ ritualistic behaviour | One of these symptoms must be present:<br>i. Simple repetitive movements<br>ii. Complex, compulsive or ritualistic behaviours, such as hoarding or repetitive storytelling<br>iii. Stereotypy of speech |
| E. Hyperorality and dietary changes | One of these symptoms must be present:<br>i. Altered food preferences<br>ii. Binge eating or increased consumption of alcohol or smoking of cigarettes<br>iii. Oral exploration or consumption of inedible objects |
| F. Neuropsychological profile: Executive/generation deficits with relative sparing of memory and visuospatial functions | One of these symptoms must be present:<br>i. Executive dysfunction<br>ii. Relative sparing of episodic memory<br>iii. Relative sparing of visuospatial skills |

Three of these behavioural/cognitive symptoms (A–F) must be present to fulfil the criteria for possible bvFTD.
*Refers to symptoms that present within the first 3 years
bvFTD, behavioural-variant frontotemporal dementia

It is critical to distinguish between FTD and primary psychiatric disorder since each has different prognoses, treatments and family counselling needs. A recent study showed that the median survival time for FTD from disease onset is 10.8 years.[5] As the disease progresses, patients will lose their dignity, self-esteem and sense of utility. Eventually, they will be forced into early retirement, which might impose financial strain on their family. Their children must deal with concerns of losing future relationships with the parent, and caregivers experience significant levels of burden and depression.[7] FTD also significantly impacts society, as most patients require institutionalisation around a year after being diagnosed.[8]

Although there is no cure for FTD at present, empowering the family with education and initiating early psychosocial intervention enable them to assist their loved one and relieve their uncertainty. Early recognition of FTD in primary care settings is essential in coordinating with an appropriate interdisciplinary team to manage the disease and in providing psychosocial support to caretakers by referring them to welfare institutions, carer support groups or counsellors. Palliative care is for a life-limiting condition, including dementia. It focuses on making a person's quality of life as good as possible and relieving distress. If it becomes available in Malaysia, integration with palliative care will be substantially helpful for treatment-refractory neurological disorders, such as the present case.[9]

## Conclusion

FTD is often misdiagnosed, as it may present with early symptoms similar to those of primary psychiatric disorders. A high index of suspicion, thorough psychosocial history-taking and family consultation for collecting more information are essential in the clinical assessment. The present case highlights the importance of continuity of care to re-examine depression symptoms that are not responding to treatment, thus avoiding unnecessary delay in diagnosing other neurological disorders.

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## Conflicts of interest

The authors declare no conflicts of interest related to this article.

## Author contribution

Fa'iza Abdullah: Writing, Editing, Literature review, Supervision
Anith Aqillah Abd Razak: Drafting, Writing, Literature review, Editing
Umi Kalsom Mohd Idris: Writing, Editing, Supervision

## Patient's consent for the use of images and content for publication

Informed consent was obtained from the patient's wife before preparing this case report.

**What is the implication to patients?**

- Increasing awareness among primary care practitioners regarding dementia at a young age is crucial.
- Obtaining a comprehensive history from patients or their family or colleagues is essential to avoid misdiagnosis of frontotemporal dementia.
- Addressing psychosocial issues helps to reduce caretakers' burden.

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