CASE REPORT

Exploring Mixed Variant Frontotemporal Dementia: A Comprehensive Clinical Analysis

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ABSTRACT

This case report elucidates the clinical presentation, diagnostic challenges, and management strategies in a patient diagnosed with the mixed variant of Frontotemporal Dementia (FTD). The patient exhibited a complex interplay of cognitive decline, behavioral changes, and psychotic features, posing diagnostic complexities. Comprehensive clinical, neuropsychological, and imaging assessments supported the diagnosis of FTD with late-onset psychosis. Pharmacological and non-pharmacological interventions were initiated to alleviate symptoms and enhance the patient's quality of life. Despite multifaceted interventions, the patient's condition demonstrated a progressive decline. This case highlights the diagnostic and therapeutic dilemmas encountered in managing the mixed variant of FTD and emphasizes the need for comprehensive evaluation and tailored interventions in similar clinical scenarios. The case highlights clear diagnostic differentiation between frontotemporal dementia and alcohol-induced cognitive impairment, supported by neuroimaging evidence of focal frontal and temporal atrophy. Further research is warranted to validate these findings and optimize management strategies for this challenging condition.
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INTRODUCTION

Frontotemporal Dementia (FTD) encompasses neurodegenerative disorders predominantly affecting the frontal and temporal lobes (1). Though less common than Alzheimer's disease, FTD accounts for a significant proportion of early-onset dementia cases. The mixed variant of FTD presents a unique challenge due to its combination of symptoms typically associated with different subtypes, such as behavioral variant FTD and primary progressive aphasia (2). Diagnosing mixed variant FTD presents significant challenges due to overlapping symptoms with other psychiatric or neurodegenerative conditions, particularly when psychotic features are involved. Differentiating FTD from alcohol-induced cognitive impairments adds complexity to diagnosis, as both conditions may present with disinhibition, memory loss, and behavioral changes. The mixed variant of FTD combines symptoms from both the behavioral variant (bvFTD) and primary progressive aphasia (PPA) (2), making clinical evaluation particularly difficult. This case outlines the presentation and management of a patient diagnosed with the mixed variant of FTD, characterized by a combination of cognitive decline, behavioral changes, and psychotic features specifically in young patients (3). Clinical assessments revealed significant impairment in cognitive and functional domains, supported by imaging findings indicative of FTD pathology. Management strategies incorporated pharmacotherapy and psychosocial interventions, yet the patient's condition exhibited a progressive decline. Existing literature indicates that early-onset FTD cases (diagnosed before the age of 65) are often misdiagnosed as psychiatric disorders, including late-onset psychosis or alcohol-related dementia. Early recognition of mixed FTD is critical as it allows for more precise treatment strategies and management, yet early diagnosis remains a significant challenge due to symptom overlap with conditions such as bipolar disorder or schizophrenia. Despite advancements in diagnostic techniques, the mixed variant remains understudied. This gap in knowledge underscores the importance of comprehensive case reports that elucidate rare presentations, which provide valuable insights for clinicians, researchers, and healthcare practitioners.

CASE REPORT

A 58-year-old male, formerly a foreman with a medical history including myocardial infarctions and deep vein thrombosis, presented with a one-year history of

progressive cognitive decline and behavioral changes. Over 18 months, he exhibited disinhibition, irritability, repetitive behaviors, and apathy, eventually developing delusions of persecution and difficulty recognizing familiar people. A five-year history of alcohol consumption added diagnostic complexity, as symptoms overlapped with alcohol-related cognitive decline. Mental Status Examination and neuropsychological assessments, including the Frontal Assessment Battery (FAB) and Temporal Assessment Battery, revealed significant impairments in conceptualization, inhibitory control, and both semantic and episodic memory.

Baseline investigations ruled out reversible causes. While a metallic implant precluded an MRI, a CT scan was utilized to identify structural changes (Figure 1). This focal atrophy, particularly in the left temporal lobe, favored a diagnosis of the semantic variant of Frontotemporal Dementia (FTD) with late-onset psychosis over the generalized atrophy typically seen in alcohol-induced impairment. Although an MRI was not performed for this specific patient, the clinical features aligned with characteristic patterns (Figure 2).

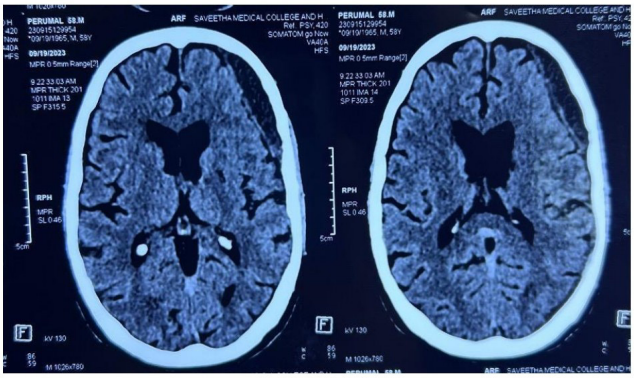

Figure 1: CT Brain suggests Frontotemporal Dementia, with evidence of significant atrophy in the frontal and temporal lobes and associated ventricular enlargement.

Pharmacological management included Haloperidol (1.5mg twice daily) and Quetiapine (25mg at bedtime) for psychotic symptoms, alongside Acamprosate to address alcohol cravings and Thiamine for nutritional support. Non-pharmacological interventions focused on caregiver psychoeducation and the Antecedent-Behavior-Consequence (ABC) Model to manage behavioral outbursts. Communication deficits were addressed through speech-language pathology using partner-supported techniques. Despite this multimodal approach, the patient's clinical trajectory showed progressive functional deterioration, necessitating a shift toward supportive care and the ongoing ethical prioritization of patient autonomy.

DISCUSSION

This case underscores the intricate nature of the mixed variant of frontotemporal dementia, accentuating the

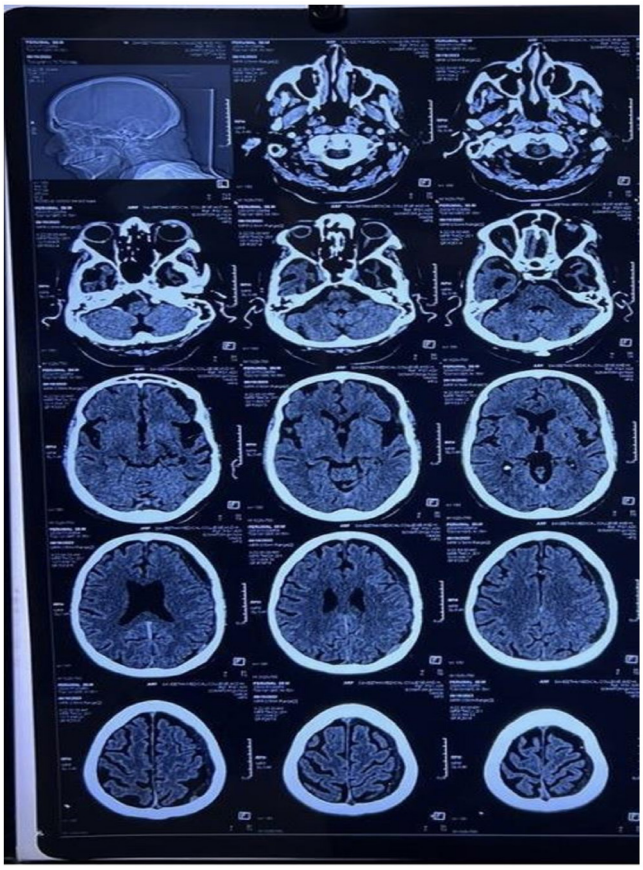

Figure 2: MRI brain suggests Frontotemporal Dementia (FTD), characterized by significant atrophy in the frontal and temporal lobes, with associated ventricular enlargement and widening of the Sylvian fissures.

diagnostic and therapeutic challenges encountered in clinical practice. Through a thorough examination of clinical, neuropsychological, and imaging data, this report contributes to the existing literature, enriching our comprehension of this rare presentation. The intersection of alcohol dependence and FTD further complicated the clinical presentation, as both conditions can independently contribute to cognitive decline and behavioral disinhibition. It serves as a valuable resource for clinicians, providing insights into the recognition and management of the mixed variant and highlighting the diverse manifestations within the frontotemporal dementia spectrum (4).

In distinguishing Frontotemporal Dementia (FTD) from alcohol-induced cognitive impairment, certain comparative clinical and neuroimaging patterns emerge as crucial diagnostic indicators. Alcohol-related cognitive impairment typically demonstrates diffuse cortical atrophy, particularly in the cerebellum and parietal regions, whereas FTD predominantly involves focal atrophy in the frontal and temporal lobes. In this case, imaging findings revealed marked left temporal and frontal atrophy with widening of the Sylvian fissures, a hallmark feature favoring FTD. Clinically, alcohol-induced cognitive deficits often show partial reversibility

with abstinence and thiamine supplementation, while the persistent and progressive deterioration observed in this patient, despite abstinence, further supports an FTD diagnosis. Additionally, the patient's distinct behavioral disinhibition, loss of empathy, and semantic language deficits are more aligned with the behavioral and semantic variants of FTD rather than alcohol-induced changes. This comparative analysis reinforces the diagnostic distinction between FTD and alcohol-related neurocognitive disorders and emphasizes the necessity for comprehensive neuropsychological and imaging assessments in overlapping presentations.

The single-case design limits the generalizability of findings, and the absence of biomarker data introduces potential bias. To mitigate this, a thorough review of relevant imaging, neuropsychological data, and differential diagnoses was performed, ensuring a robust diagnostic process. Future research involving larger sample sizes and biomarker analyses is essential to further validate these findings.

CONCLUSION

The mixed variant of frontotemporal dementia presents diagnostic and management challenges due to its heterogeneous nature. Comprehensive evaluation incorporating clinical, neuropsychological, and imaging assessments is essential for accurate diagnosis. Early intervention focusing on cognitive stimulation, behavioural management, and caregiver support is essential, though progression remains inevitable(5). Continued research is warranted to elucidate the underlying mechanisms and identify targeted interventions for this complex presentation.

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