Neuropsychiatry in Clinical Practice: The Challenge of Diagnosing Behavioral Variant Frontotemporal Dementia

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The behavioral variant of Frontotemporal dementia (bvFTD) is an insidious neurodegenerative disease associated with progressive degeneration of the frontal lobes, anterior temporal lobes, or both [1]. Alterations in social cognition represent the core symptoms of bvFTD resulting in emotional disengagement and socially inappropriate responses or activities [2,3]. As is apparent in revised consortium criteria, additional neuropsychiatric symptoms including apathy and stereotypical and impulsive behavior are prominent in the clinical presentation [4]. Consequently, both neurodegenerative diseases and primary psychiatric disorders are crucial in the challenging differential diagnosis.

The differentiation between bvFTD and Alzheimer’s disease (AD) has become easier by the use of biomarkers that are able to identify underlying AD pathology, such as the amyloid-β (Aβ) and tau [1,5]. However, to distinguish bvFTD from psychiatric disorders can still be difficult, particularly since biomarkers for bvFTD are less robust [6]. Previous studies indicated that as a result of symptomatic overlap between bvFTD and psychiatric disorders, bvFTD patients are clinically often mistaken for psychiatric patients and vice versa [7-10]. The current clinical criteria for bvFTD require that “if behavioral disturbance is better accounted for by a psychiatric diagnosis, a diagnosis of bvFTD has to be excluded” [4].

Despite clinical overlap, bvFTD patients do not often fulfill formal criteria for a psychiatric diagnosis, suggesting that it is valuable to apply formal criteria for psychiatric disorders [11]. Careful clinical phenotyping of overlapping symptoms can help to distinguish bvFTD from psychiatric disorders in clinical practice (Figure 1) [12,13].

Figure 1: Overlap and differentiation between bvFTD and psychiatric disorders in clinical practice.
According to current criteria, the diagnostic certainty of bvFTD increases when Frontotemporal abnormalities are found on neuroimaging. In a large cohort of patients with late-onset behavioral changes, MRI had a sensitivity of 70% and a specificity of 93% for a bvFTD diagnosis [4]. The additional [18F]FDG-PET, when the MRI was inconclusive, had a sensitivity of 90% at the cost of a lower specificity (68%) [16]. [18F]FDG-PET is mainly useful when Frontotemporal hypometabolism is absent to exclude bvFTD diagnosis. The interpretation of neuroimaging results should especially be taken with caution in cases with a psychiatric differential diagnosis where [18F]FDG-PET is the only abnormal finding on neuroimaging. In a large cohort of patients, MRI and [18F]FDG-PET can show a specificity of 90% at the cost of a lower sensitivity [16]. Genetic screening especially for C9orf72 repeat expansion is emphasized [19,20], particularly in cases with a remarkable (prolonged) disease course.

In clinical practice, bvFTD has a broad differential diagnosis including both neurodegenerative diseases and primary psychiatric disorders. The current criteria for bvFTD have clearly improved diagnostics but differentiating bvFTD from psychiatric disorders remains difficult. The challenge for the next decade is finding specific biomarkers for bvFTD on the one hand, and optimizing the neuropsychiatric diagnosis of bvFTD on the other hand. To this end, patient care for suspected bvFTD patients would be largely improved in a setting where neurologists and psychiatrists work hand in hand, ideally applying a consensus set of clinical rating scales next to their clinical expertise.

References


