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CASE REPORT

Logopenic Variant Primary Progressive Aphasia Presenting with Severe Depressive Syndrome and Suicidal Behaviour: A Case Report

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ABSTRACT

Background: Logopenic variant Primary Progressive Aphasia (lvPPA) is a language-predominant neurodegenerative syndrome characterized by progressive word retrieval deficits, impaired phonological processing, and hesitant speech. Unlike classical amnestic presentations of Alzheimer's disease, patients with LvPPA often present with language impairment as the earliest and most disabling symptom. Psychiatric manifestations may coexist and contribute to diagnostic complexity.

Case Presentation: A 62-year-old male presented with progressively worsening language impairment characterized predominantly by difficulty retrieving words and interrupted speech production. Family members reported that the patient frequently knew what he intended to communicate but was unable to retrieve appropriate words. Symptoms began approximately 18 months before evaluation and progressively worsened over time. Over the subsequent months, the patient developed depressive symptoms characterized by low mood, apathy, reduced motivation, feelings of worthlessness, and social withdrawal. Neuropsychiatric symptoms intensified and culminated in a suicidal attempt by ingestion of washing fluid approximately three months before presentation. Magnetic resonance imaging demonstrated minimal nonspecific white matter microangiopathic changes without major structural abnormalities or significant cortical atrophy. Florbetaben F18 amyloid PET imaging demonstrated diffuse cortical amyloid positivity involving frontal, temporal, parietal, and posterior cingulate–precuneus regions with minimal associated atrophy, supporting Alzheimer-spectrum pathology. The clinical profile and biomarker findings were considered most consistent with logopenic variant Primary Progressive Aphasia.

Conclusion: This case highlights the importance of recognizing atypical Alzheimer-spectrum presentations in individuals presenting with progressive language dysfunction and psychiatric symptoms.

Keywords: Logopenic variant Primary Progressive Aphasia; lvPPA; Alzheimer-spectrum disease; amyloid PET; aphasia; depression

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INTRODUCTION

Primary Progressive Aphasia (PPA) is a clinical syndrome characterized by gradual and progressive deterioration of language functioning caused by underlying neurodegenerative disease. Unlike typical dementias, in which episodic memory impairment often represents the earliest and most prominent feature, PPA initially presents with selective deterioration in language abilities while other cognitive domains remain relatively preserved during the early stages of illness. Patients may experience increasing difficulty with speaking, word retrieval, sentence formulation, comprehension, reading, or writing, depending on the specific language networks involved and the underlying neuropathology. [1]

Current diagnostic classifications recognize three principal variants of PPA: nonfluent/agrammatic variant, semantic variant, and logopenic variant. Each subtype demonstrates distinct clinical, neuroanatomical, and pathological characteristics. [2] Among these, logopenic variant Primary Progressive Aphasia (lvPPA) has emerged as a clinically important language-predominant neurodegenerative syndrome frequently associated with underlying Alzheimer-spectrum pathology. Unlike semantic and nonfluent variants, which are more commonly associated with frontotemporal lobar degeneration, lvPPA frequently demonstrates cortical amyloid deposition and atypical presentations of Alzheimer's disease. [2 3]

Clinically, lvPPA is characterized by progressive word-finding difficulty, pauses during spontaneous speech, impaired sentence repetition, and phonological retrieval deficits. Patients frequently report awareness of what they intend to communicate but experience difficulty retrieving the appropriate lexical content. Speech output often becomes hesitant and interrupted because of impaired word retrieval rather than motor speech dysfunction. In the early stages of illness, grammar, articulation, and single-word comprehension are relatively preserved, which may contribute to delayed recognition and diagnostic uncertainty. [2 4]

Neuroanatomically, lvPPA is commonly associated with dysfunction involving the left temporoparietal language network, particularly regions involved in phonological processing and verbal working memory. Structural neuroimaging findings in the early disease stages may remain subtle or nonspecific and can occasionally reveal minimal cortical atrophy despite clinically significant language dysfunction. Functional and molecular imaging modalities, particularly amyloid positron emission tomography (PET), have therefore become increasingly valuable in identifying underlying Alzheimer pathology and improving diagnostic accuracy in atypical neurodegenerative presentations. [3 5]

In addition to language impairment, patients with progressive aphasic syndromes may experience substantial psychiatric and behavioral manifestations, including depression, anxiety, apathy, emotional withdrawal, and suicidal ideation. Such symptoms may emerge particularly in individuals with preserved insight regarding progressive communication decline. Psychiatric manifestations can initially overshadow language dysfunction, resulting in delayed diagnosis or misattribution of symptoms to primary psychiatric illness. [6 7]

The present case describes a patient presenting with progressive language dysfunction characterized predominantly by word retrieval impairment accompanied by severe depressive symptomatology and suicidal behavior. Despite minimal structural abnormalities on MRI, Florbetaben F18 amyloid PET imaging demonstrated significant cortical amyloid positivity supporting an Alzheimer-spectrum neurodegenerative process consistent with logopenic variant Primary Progressive Aphasia. This case highlights the diagnostic challenges associated with atypical neurodegenerative presentations and emphasizes the importance of integrating clinical assessment with biomarker-supported neuroimaging findings.

CASE PRESENTATION

A 62-year-old male presented with a progressively evolving language disturbance characterized predominantly by impaired lexical retrieval and increasing difficulty in spontaneous verbal expression. According to collateral information obtained from family members, the patient consistently demonstrated awareness of intended communication but experienced significant

difficulty retrieving appropriate words during conversation. Initial symptoms reportedly emerged approximately 18 months before evaluation and followed a gradual but steadily progressive course. Over time, speech became increasingly hesitant and interrupted by frequent pauses, resulting in noticeable impairment in day-to-day communication.

During the subsequent course of illness, the patient developed prominent neuropsychiatric manifestations, including persistent low mood, apathy, reduced motivation, emotional withdrawal, and pervasive feelings of worthlessness. These symptoms progressively intensified and were associated with substantial psychosocial dysfunction. Approximately three months before presentation, the patient reportedly attempted suicide through ingestion of washing fluid, suggesting severe psychological distress in the context of preserved awareness of functional decline.

Neuroimaging evaluation was undertaken as part of the diagnostic workup. Magnetic resonance imaging (MRI) of the brain demonstrated minimal nonspecific white matter microangiopathic changes without significant cortical atrophy or focal structural abnormalities. Subsequent Florbetaben F18 amyloid positron emission tomography (PET) imaging demonstrated diffuse cortical loss of normal gray–white differentiation involving bilateral frontal, temporal, parietal, and posterior cingulate/precuneus regions, with overall findings interpreted as a positive amyloid scan indicative of frequent neuritic plaque deposition with minimal associated atrophy. The combination of progressive language-predominant symptomatology and biomarker-supported cortical amyloid pathology was considered highly suggestive of an Alzheimer-spectrum neurodegenerative process, with clinical findings most consistent with logopenic variant Primary Progressive Aphasia (lvPPA).

INVESTIGATIONS

Neuroimaging evaluation was performed as part of the diagnostic workup. Magnetic resonance imaging (MRI) of the brain demonstrated normal ventricular and cisternal anatomy appropriate for age, with no evidence of hydrocephalus, focal mass lesion, diffusion restriction, or hemosiderin deposition. Major vascular flow voids were preserved. Multiple scattered foci of FLAIR hyperintensities were identified within the white matter and interpreted as mild nonspecific microangiopathic changes. No significant cortical atrophy or focal structural abnormalities were identified on structural imaging.

Subsequent molecular neuroimaging with Florbetaben F18 amyloid positron emission tomography (PET) demonstrated diffuse loss of normal gray–white matter differentiation involving bilateral frontal, lateral temporal, and parietal cortices, as well as posterior cingulate and precuneus regions. The final radiological interpretation described the scan as positive for frequent amyloid neuritic plaques with minimal associated atrophy. The observed distribution pattern was considered highly suggestive of widespread cortical amyloid deposition and supportive of an underlying Alzheimer-spectrum neurodegenerative process. These findings provided important biomarker evidence in favor of logopenic variant Primary Progressive Aphasia (lvPPA), particularly in the context of relatively minimal structural abnormalities observed on MRI.

Additional laboratory investigations revealed thrombocytopenia, elevated creatine kinase levels, hyponatremia, and reduced iron saturation. These findings initially broadened the diagnostic considerations and prompted evaluation for alternative metabolic, systemic, and potentially autoimmune etiologies. However, the progressive language-predominant clinical course, combined with biomarker-supported cortical amyloid pathology, strongly favoured an underlying neurodegenerative etiology.

DISCUSSION

This case illustrates an atypical presentation of Alzheimer-spectrum disease in which language impairment preceded generalized cognitive decline. Progressive word-finding difficulty with preserved

awareness of intended communication represents a characteristic early feature of LVPPA.

A notable feature of this case was the discrepancy between relatively minimal MRI findings and markedly positive amyloid PET imaging. Structural imaging abnormalities may remain limited during early stages, whereas molecular imaging can provide greater diagnostic specificity.

Another clinically significant aspect was the presence of severe depressive symptoms and suicidal behaviour. Patients with insight-preserved neurodegenerative disorders may experience considerable emotional distress as they become aware of a progressive decline in communication abilities.

The coexistence of laboratory abnormalities further complicated clinical interpretation and initially broadened diagnostic considerations. Nevertheless, the longitudinal progression and biomarker profile strongly supported a neurodegenerative etiology.

CONCLUSION

Progressive word-finding difficulty in middle-aged and older adults warrants careful evaluation for atypical neurodegenerative syndromes, particularly when accompanied by preserved insight and evolving psychiatric symptoms.

This case demonstrates the diagnostic utility of Florbetaben F18 amyloid PET imaging in identifying Alzheimer-spectrum pathology in patients with minimal structural MRI abnormalities and language-predominant presentations.

Early recognition of LVPPA is clinically important not only for diagnostic clarification and prognostication, but also for management of associated psychiatric morbidity, including depression and suicide risk.

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The **Journal of Applied Therapeutics (JAT)** is an interdisciplinary, practice-oriented journal that focuses on translating scientific knowledge into meaningful, real-world healthcare applications. The inaugural issue reflects this philosophy through articles exploring the multidimensional impact of head injury and barriers to rehabilitation, an integrated neurobehavioral outpatient model aimed at improving treatment acceptance, the complexity of suicide and its legal, social, and clinical determinants, the importance of multidisciplinary pain management as a core component of modern medicine, and a case report highlighting atypical Alzheimer-spectrum disease presenting with language impairment, depression, and suicidal behaviour. Collectively, these articles emphasize that effective healthcare requires the integration of biological, psychological, social, ethical, and practical factors to address complex human problems in real-world settings.