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The Diagnostic Role of FDG-PET in Dementias Presenting with Language and Behavioral Symptoms

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ABSTRACT

Objective: This study aimed to assess the diagnostic contribution of Fluorodeoxyglucose Positron Emission Tomography (FDG-PET) in neurodegenerative dementias presenting with language-predominant symptoms, with or without accompanying behavioral changes, and to evaluate the correspondence between PET-derived metabolic patterns and cerebrospinal fluid (CSF) biomarkers.

Methods: Sixteen patients with language-predominant onset were categorized into two groups: Alzheimer's disease (AD) spectrum (n=8) and the frontotemporal lobar degeneration (FTLD) spectrum (n=8). All participants underwent clinical evaluation, neuropsychological testing, and FDG-PET imaging. Cerebrospinal fluid biomarkers [Amyloid Beta (Aβ42), total tau, and phosphorylated tau] were analyzed in eligible individuals (n=6). The two groups were compared across demographic, clinical, and neuroimaging parameters.

Results: FDG-PET revealed pronounced posterior temporoparietal hypometabolism in AD-spectrum patients, whereas FTLD-spectrum patients demonstrated frontal, insular, and anterior temporal metabolic involvement. Cerebrospinal fluid biomarker profiles consistently supported Alzheimer-type pathology among AD-spectrum cases. In the FTLD group, one patient exhibited borderline-low Aβ42; however, the corresponding FDG-PET pattern was characteristic of FTLD.

Conclusion: FDG-PET demonstrates a supportive role in differentiating AD from FTLD among patients presenting with language-predominant neurodegenerative syndromes. In scenarios where CSF biomarkers are unavailable or yield borderline values, FDG-PET-based metabolic signatures may offer additional guidance and can contribute to strengthening multimodal diagnostic frameworks in geropsychiatry settings.

Keywords: Alzheimer's Disease, biomarker, dementia, FDG-PET frontotemporal dementia, imaging, language impairment

INTRODUCTION

Dementias with predominant language involvement, particularly in older individuals, may exhibit substantial symptomatic overlap with psychiatric disorders such as depression, anxiety, psychosis, personality changes, and inappropriate behavioral disturbances (Aytulun and Aki 2022). Consequently, approximately 50% of patients initially receive a psychiatric diagnosis, and accurate diagnosis may be delayed by an average of 5–6 years (Ducharme et al. 2020, Vismara et al. 2020, Brodaty and Connors 2020, Flavell et al. 2025). Apathy, disinhibition, loss of empathy, compulsive behaviors, and personality changes are clinical features that can be observed in both dementia and psychiatric disorders (Bang et al. 2015, Vismara et al. 2020, Brodaty and Connors 2020, Flavell et al. 2025). Although language impairments (such as primary progressive aphasia) typically precede psychiatric symptoms, they may often be overlooked by clinicians and misinterpreted as psychiatric conditions (Turan et al. 2013, Vismara et al. 2020, Géraudie et al. 2021).

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Although neuroimaging and biomarkers contribute to diagnosis, their sensitivity and specificity remain limited in the early stages (Ducharme et al. 2020, Loi et al. 2025, Flavell et al. 2025). Particularly in genetic subtypes such as C9orf72 mutation carriers, psychiatric symptoms may precede overt dementia manifestations by several years (Ducharme et al. 2020, Vismara et al. 2020). In recent years, cerebrospinal fluid (CSF) biomarkers [Amyloid Beta 42 (Aβ42), total tau, and phosphorylated tau] have been accepted as the gold standard for the biological confirmation of Alzheimer's disease (AD) pathology. However, due to the invasive nature, limited availability across centers, and contraindications in certain patients, lumbar puncture cannot be routinely applied in all cases. In contrast, Fluorodeoxyglucose Positron Emission Tomography (FDG-PET) imaging provides significant differential diagnostic value between AD and Frontotemporal Lobar Degeneration (FTLD) by evaluating patterns of cortical hypometabolism. The lateral temporal–parietal and posterior cortical hypometabolism typical of AD can be clearly distinguished from the frontal-insular or anterior temporal patterns observed in FTLD (Foster et al. 2007, Minoshima et al. 2022, Ito et al. 2025).

In this study, CSF biomarkers and FDG-PET findings were jointly examined in 16 consecutive patients diagnosed with neurodegenerative dementia presenting with language-predominant onset. In cases without available CSF biomarkers, the diagnostic utility of FDG-PET in differentiating AD and FTLD spectra was specifically evaluated. Our aim was to contribute to the accurate biological classification of the commonly encountered clinical combination of "language impairment + behavioral change" in geropsychiatric practice and to analyze the concordance between FDG-PET and CSF findings within clinical decision-making processes.

METHODS

Participants

This study consists of a retrospective analysis of 16 consecutive patients who presented to the Hacettepe University, Faculty of Medicine Neurology outpatient clinic between 2021 and 2024 with predominant language impairment and were considered to have a neurodegenerative process. Patients were classified based on clinical evaluations obtained from medical records, neuropsychological test results (SMME, verbal/semantic fluency, Enhanced Cued Recall Test, Stroop test, Digit Span, Trail Making Test A and B, Clock Drawing Test, Boston Naming Test, and Gülhane Aphasia Test-II), behavioral findings, motor findings, CSF biomarkers, and FDG-PET imaging results (Güngen et al. 2002).

Clinical phenotypes were determined according to widely accepted diagnostic criteria in the literature. Cases with language-predominant onset were classified according to the Primary Progressive Aphasia (PPA) consensus criteria defined by Gorno-Tempini et al. (Gorno-Tempini et al. 2011). Patients fulfilling criteria for the logopenic variant PPA were evaluated within the AD spectrum, whereas those meeting criteria for semantic variant PPA or nonfluent/agrammatic variant PPA were classified within the FTLD spectrum. Final clinical classification was established through a multidisciplinary evaluation integrating neurological examination findings, neuropsychological profiles, structural neuroimaging findings, and—when available—CSF biomarker results. FDG-PET findings were not used as the primary determinant of diagnostic group classification; rather, they were employed as supportive data following clinical classification. Subsequently, the concordance of FDG-PET patterns with clinical phenotypes and CSF biomarkers was evaluated.

The study was conducted following approval from Hacettepe university health sciences ethics committee, and all participant data were processed in accordance with ethical standards.

Imaging

Patients were instructed to fast for 4–6 hours prior to imaging. When fasting blood glucose levels were below 160 mg/dL, 185 MBq of F-18 FDG was administered intravenously. Following injection, patients rested in a dim and quiet room for 60 minutes. FDG-PET imaging (GE Healthcare, Chicago, IL, USA) was performed 60–70 minutes after tracer administration. Image acquisition was conducted in a single-bed position focused on the cranial region, following a low-dose CT scan for attenuation correction. The acquired raw FDG-PET images were processed iteratively and visually assessed on an ADW 4.7 (GE Healthcare) workstation. Each patient's images were also evaluated using Cortex ID Suite 2.1 (GE Healthcare), comparing metabolic patterns with those of a normative database.

Cerebrospinal Fluid Analysis

Cerebrospinal fluid samples were obtained in the morning under sterile conditions, with patients in the lateral decubitus position, via standard lumbar puncture at the L3–L4 or L4–L5 interspace. To minimize blood contamination, the first 1–2 mL of CSF was discarded. Samples intended for analysis were collected exclusively in polypropylene tubes; glass or polystyrene materials were avoided due to the risk of amyloid beta adherence and falsely low measurements. Immediately after collection, samples were placed on dry ice and transported under strict cold-chain conditions to the Brain and Neurodegenerative Diseases Research Laboratory (BNHAL) at Istanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine.

Biomarker analyses at BNHAL were conducted according to standardized laboratory protocols. Cerebrospinal fluid levels of Amyloid Beta 42 (Aβ42), total tau, and phosphorylated tau (p-tau181) were measured using Single Molecule Array (Simoa) technology (Quanterix platform). Reports included laboratory-specific cutoff values validated through the Alzheimer's Association Global QC Program (cutoffs for AD pathology: Aβ42 <813 pg/mL; total tau >375 pg/mL; p-tau181 >52 pg/mL) and coefficients of variation (CV%). All biomarkers were confirmed to be consistent with international reference standards.

Statistical Analysis

All analyses were conducted at a descriptive level. Neuropsychological scores, rates of behavioral changes, and imaging patterns were summarized using percentage distributions and median (minimum-maximum) values. Due to the small sample size, no inferential statistical analyses were performed.

RESULTS

A total of sixteen patients diagnosed with neurodegenerative dementia with language-predominant onset were included in the study; eight were classified within the AD spectrum and eight within the FTLD spectrum. The demographic, clinical, and neuropsychological characteristics of the groups are presented in Table 1.

The rate of behavioral changes was 50% (4/8) in the AD-spectrum group and 65% (5/8, with one missing data point) in the FTLD-spectrum group. Motor findings were more prominent in the FTLD-spectrum group, with 50% of patients exhibiting motor involvement. In contrast, only one patient (12.5%) in the AD-spectrum group demonstrated motor findings.

The median mini-mental state examination (MMSE) score was 16 (range: 5–20) in the AD-spectrum group and 18 (range: 15–23) in the FTLD-spectrum group. Median verbal fluency scores were 3 (0–13) and 6 (0–11), while semantic fluency scores were 6 (0–13) and 10 (2–11), respectively. In both groups, some patients were unable to produce any responses in these tests.

Across the entire cohort, FDG-PET revealed hypometabolism most frequently in the temporal (84.2%), frontal (69.2%), and posterior cingulate/precuneus (46.2%) regions. Parietal hypometabolism was also observed in 46.2% of cases, while occipital, insular, and anterior temporal involvement were less frequent and observed at equal rates (15.4%) (Table 2).

Cerebrospinal fluid analysis was available in only six patients. Four patients with Aβ42 levels below the cutoff were considered consistent with AD biology. In the single FTLD case with borderline-low Aβ42, clinical classification was maintained as FTLD, as FDG-PET findings supported an FTLD pattern.

In the AD-spectrum group, all patients demonstrated parietotemporal, lateral temporal, or posterior parietal hypometabolism, consistent with the typical metabolic pattern of AD. In the FTLD-spectrum group, hypometabolism predominantly involved frontal, insular, anterior temporal, or frontoparietal regions, reflecting FTLD-specific patterns (Figure 1). Among patients with available CSF biomarkers, FDG-PET patterns were concordant with CSF findings in differentiating AD and FTLD (Figure 2).

Table 1. Demographic and clinical characteristics of the groups

	AD Spectrum	FTLD Spectrum
Number of patients	8	8
Age at onset, median (min–max)	60 (49–68)	64 (42–82)
Years of education, median (min–max)	5 (5–15)	8 (0–15)
Behavioral changes (%)	4 (50%)	5 (63%)
Any motor findings (%)	1 (12.5%)	4 (50%)
MMSE score, median (min–max)	16 (5–20)	18 (15–23)
Verbal fluency, median (min–max)	3 (0–13)	6 (0–11)
Semantic fluency, median (min–max)	6 (0–13)	10 (2–11)
Amyloid Beta 1–42 (pg/mL)	481.0 (256–745)	876.5 (790–963)
Phosphorylated tau (pg/mL)	35.6 (19–47)	27 (19–35)
Total tau (pg/mL)	327 (95–392)	133.5 (95–172)
Phosphorylated tau/Amyloid Beta (1–42)	0.064 (0.024–0.123)	0.030 (0.024–0.036)

MMSE: mini-mental state examination; AD: Alzheimer's disease; FTLD: frontotemporal lobar degeneration.

Table 2. Distribution of hypometabolism detected on FDG-PET

Region	AD Spectrum	FTLD Spectrum	Total (n)	Total (%)
Temporal lobe	7	4	11	84.6
Frontal lobe	6	3	9	69.2
Parietal lobe	4	2	6	46.2
Posterior cingulate / precuneus	4	2	6	46.2
Occipital lobe	2	0	2	15.4
Insula	0	2	2	15.4
Anterior temporal lobe	0	2	2	15.4

AD: Alzheimer's disease; FTLD: frontotemporal lobar degeneration.

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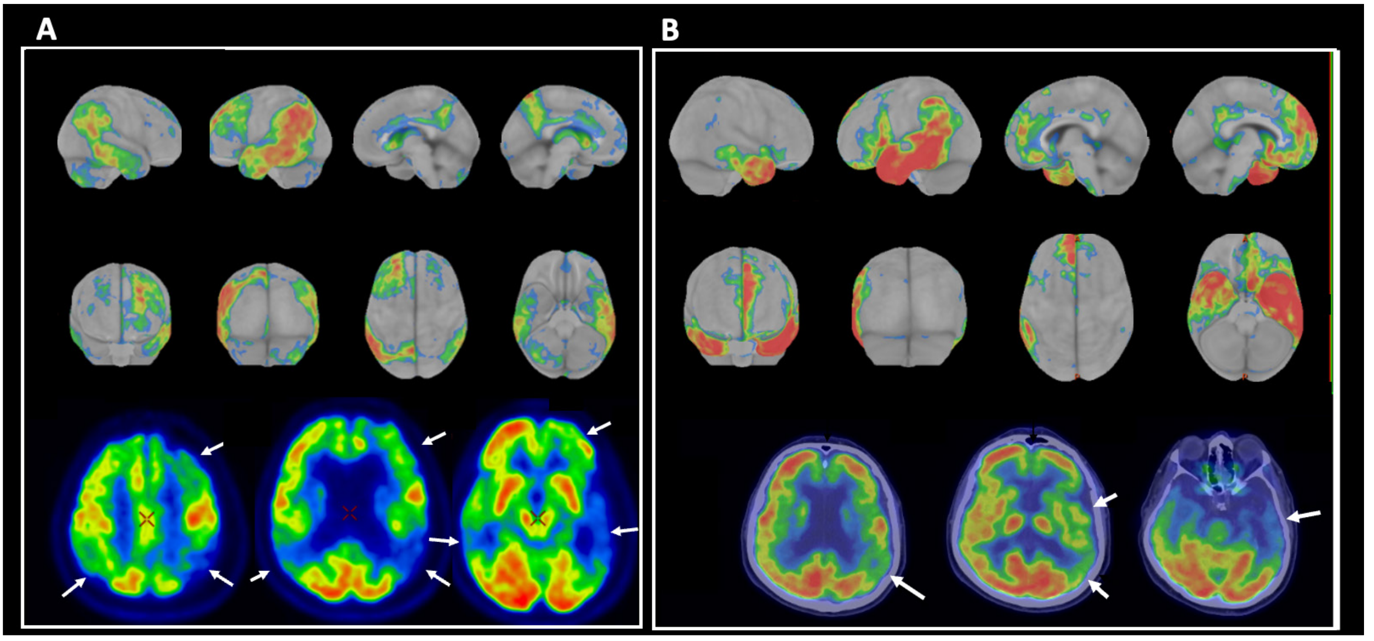

Figure 1. Metabolic difference maps relative to an age-matched normal population are shown in the upper two rows, while the bottom row presents FDG-PET images of the patients. (A: In a patient with language-predominant cognitive impairment clinically and neuropsychologically consistent with Alzheimer's disease, hypometabolism is observed in the bilateral parietotemporal and left frontal regions, more prominent on the left side. This metabolic distribution is consistent with the posterior cortical involvement pattern associated with Alzheimer's disease. Alzheimer pathology was further supported by CSF biomarkers in this patient. B: In a patient presenting with reduced speech fluency and agrammatic language production, consistent with the diagnostic criteria for nonfluent/agrammatic primary progressive aphasia, hypometabolism (arrows) is observed predominantly in the left temporal lobe, extending to the left temporoparietal and left frontal regions. Additionally, metabolic reduction is noted in the anterior right temporal lobe and in bilateral mesial frontal regions, more pronounced on the left. Cerebrospinal fluid biomarkers in this case did not meet the cutoff values for Alzheimer's pathology.)

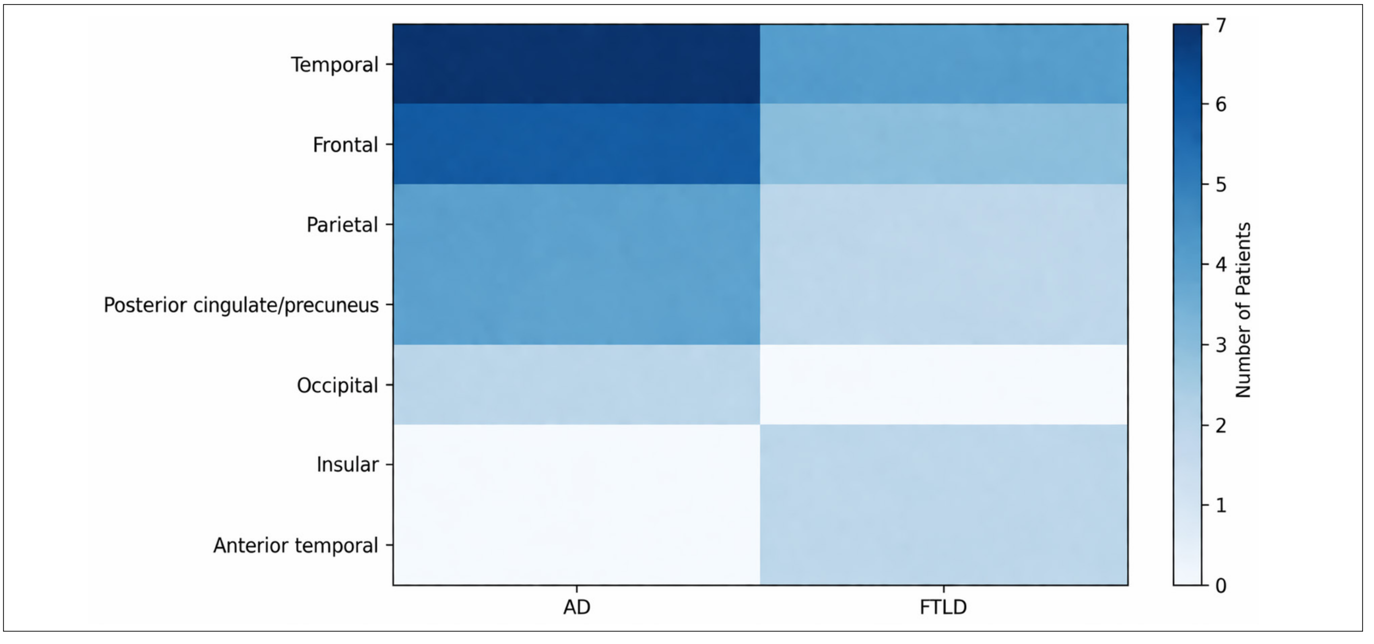

Figure 2. Comparison of FDG-PET hypometabolism patterns between AD-spectrum and FTLD-spectrum patients. The heatmap illustrates the distribution of hypometabolic regions across the two groups. Darker colors represent a higher number of affected patients. While AD-spectrum patients demonstrate prominent hypometabolism particularly in temporal, frontal, and posterior cingulate/precuneus regions, FTLD-spectrum patients exhibit a more limited and heterogeneous distribution of involvement.

DISCUSSION

This study presents a comprehensive analysis of the differentiation between AD-spectrum and FTLD-spectrum neurodegenerative dementias with language-predominant onset, integrating neuropsychological profiles, CSF biomarkers, and FDG-PET-based patterns of neural network involvement. The findings indicate that FDG-PET gains particular diagnostic relevance in cases where CSF biomarkers are limited or yield borderline results.

The neurobiological basis of language-onset neurodegenerative disorders is closely linked to the anatomical and functional characteristics of the affected neural networks. In AD-spectrum cases, early involvement is observed in the default mode network (DMN) and posterior temporoparietal networks, whereas in FTLD-spectrum cases, disruption typically begins within the salience network (SN), left inferior frontal gyrus, insula, anterior temporal pole, and dorsal frontoparietal executive networks (Ekinci et al. 2009, Hafkemeijer et al. 2016, Ranasinghe et al. 2016, Yıldırım and Soncu Büyükişcan 2019, Giorgio et al. 2023). The posterior cortical hypometabolism observed in the AD-spectrum group (posterior cingulate, precuneus, lateral temporal, and inferior parietal cortex) represents the metabolic signature of the DMN and constitutes a characteristic involvement pattern seen in both typical amnestic AD and logopenic variant PPA (Ossenkoppele et al. 2015, Mandelli et al. 2023). In the current cohort, FDG-PET findings in the AD spectrum showed high concordance with the logopenic variant PPA and Posterior Cortical Atrophy components described in the literature. In contrast, the FTLD-spectrum group demonstrated hypometabolism predominantly involving the anterior temporal, orbitofrontal, anterior insular, dorsolateral prefrontal, and dorsal anterior cingulate regions, consistent with predominant involvement of the salience and executive networks. This pattern directly corresponds to the metabolic signatures of semantic variant PPA (ventral anterior temporal network degeneration), nonfluent/agrammatic PPA (left inferior frontal-temporo-insular network involvement), and behavioral variant FTD (frontal-insular SN disruption) (Bejanin et al. 2020, Minoshima et al. 2022). This network-based differentiation provides meaningful support for clinical decision-making, particularly in FTLD cases lacking CSF biomarker evaluation (Minoshima et al. 2022).

The CSF profile of one patient within the FTLD spectrum was notable due to borderline low Aβ1-42 levels. In FTLD patients, CSF Aβ1-42 levels may vary widely and can fall below thresholds typically considered pathological for AD in some cases (Goossens et al. 2018). This reduction is often attributed to coexisting (comorbid) AD pathology or genetic factors such as APOE ε4 carrier status. However, reduced CSF Aβ1-42 levels may also be observed in FTLD patients in the absence of AD pathology (Goossens et al. 2018, Lleó et al. 2018). Therefore, reliance on CSF Aβ1-42 levels alone carries a risk of false-positive AD classification in FTLD patients. Combined biomarkers, such as the p-tau/Aβ1-42 ratio, are considered more reliable in reducing false positivity (Lleó et al. 2018, Mattsson-Carlgren et al. 2022). Furthermore, CSF biomarkers should not be interpreted in isolation but rather in conjunction with clinical and imaging findings (Leuzy et al. 2025).

FDG-PET is categorized within the "N" (neurodegeneration) component of the AT (N) biomarker framework and is capable of demonstrating characteristic hypometabolic patterns associated with AD (Perani et al. 2015, Ou et al. 2019, Chételat et al. 2020). When CSF biomarkers yield borderline results, the addition of FDG-PET may enhance diagnostic confidence and accuracy, thereby contributing to clinical decision-making (Perani et al. 2015, Massa et al. 2019, Chételat et al. 2020, Gjerum et al. 2021). Nevertheless, FDG-PET is not an absolute biomarker for defining AD biology. Although concordance between FDG-PET and CSF biomarkers exceeds 80%, while discordance may occur in approximately 20% of cases (Gjerum et al. 2021, Quispialaya et al. 2022). Clinical guidelines recommend the use of FDG-PET as a supportive tool when CSF biomarker values fall within borderline ranges for AD pathology (Chételat et al. 2020, Gjerum et al. 2021).

In conclusion, the diagnostic strength of FDG-PET arises from its ability to reflect disease-specific network dysfunction rather than merely anatomical involvement. In this study, posterior temporoparietal hypometabolism observed on FDG-PET in AD-spectrum patients was consistent with CSF biomarker profiles, confirming that FDG-PET–derived metabolic patterns align with biological diagnosis. This concordance suggests that, particularly in geropsychiatric practice, FDG-PET serves as a valuable tool in differential diagnosis in patients presenting with language impairment accompanied by behavioral symptoms, especially when CSF analysis is not feasible.

This study has several limitations. First, it is a single-center study with a relatively small sample size, which may limit the generalizability of the findings. Second, the retrospective design implies that clinical, neuropsychological, and imaging data were derived from medical records, and therefore some variables were not available for all patients. In particular, biomarker data are limited, as CSF analysis was performed only in a subset of patients. Additionally, FDG-PET findings were based on clinical reports and visual assessments rather than quantitative analyses, which may limit the detection of more subtle metabolic changes. The relatively small sample size also precluded detailed subgroup comparisons. Another limitation inherent to the retrospective nature of the study is the potential for FDG-PET findings to have indirectly influenced clinical classification in certain cases. Although clinical evaluation served as the primary basis for classification, FDG-PET findings may have contributed to diagnostic decision-making, particularly in cases with limited biomarker data. Therefore, the observed concordance between FDG-PET and clinical classification may partly reflect a degree of circularity (inclusion bias). Future studies incorporating larger cohorts, multicenter designs, and quantitative imaging analyses will be important to validate these findings.

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Ethics Committe Approval: The study protocol was approved by the Hacettepe University, Faculty of Health Sciences Ethics Committee on March 18, 2025, with decision number SBF 25/002.

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REFERENCES

Aytulun A and Aki ŞÖE (2022) Mild behavioral impairment: a new prodromal syndrome for dementia. Hafif davranışsal bozulma: demans öncülü yeni bir sendrom. *Turk Psikiyatri Derg* 33:280–9. https://doi.org/10.5080/u26980

Bang J, Spina S, Miller B et al. (2015) Frontotemporal dementia. *Lancet* 386, 1672–82. https://doi.org/10.1016/S0140-6736(15)00461-4

Bejanin A, Tammewar G, Marx G et al. (2020) Longitudinal structural and metabolic changes in frontotemporal dementia. *Neurology* 95:e140–54. https://doi.org/10.1212/WNL.0000000000009760

Brodaty H and Connors M (2020) Pseudodementia, pseudo-pseudodementia, and pseudodepression. *Alzheimers Dement (Amst)* 12:e12027. https://doi.org/10.1002/dad2.12027

Chételat G, Arbizu J, Barthel H et al. (2020) Amyloid-PET and 18F-FDG-PET in the diagnostic investigation of Alzheimer's disease and other dementias. *Lancet Neurol* 19:951–62. https://doi.org/10.1016/S1474-4422(20)30314-8

Ducharme S, Dols A, Laforce R et al. (2020) Recommendations to distinguish behavioural variant frontotemporal dementia from psychiatric disorders. *Brain* 143:1632–50. https://doi.org/10.1093/brain/awaa018

Ekinci G, Tuncer N, Midi I et al. (2009) P1-135: brain methobolic changes in mild cognitive impairment and early Alzheimer's disease patients. *Alzheimers Dement (Amst)* 5:P216 https://doi.org/10.1016/j.jalz.2009.04.140

Flavell J, Ahern E, Logan B et al. (2025) Factors associated with true-positive and false-positive diagnoses of behavioural variant frontotemporal dementia in 100 consecutive referrals from specialist physicians. *Eur J Neurol* 32:e70036. https://doi.org/10.1111/ene.70036

Foster N, Heidebrink J, Clark C et al. (2007) FDG-PET improves accuracy in distinguishing frontotemporal dementia and Alzheimer's disease. *Brain* 130:2616–35. https://doi.org/10.1093/brain/awm177

Géraudie A, Battista P, García A et al. (2021) Speech and language impairments in behavioral variant frontotemporal dementia: a systematic review. *Neurosci Biobehav Rev* 131:1076–95. https://doi.org/10.1016/j.neubiorev.2021.10.015

Giorgio J, Adams J, Maass A et al. (2023) Amyloid induced hyperexcitability in default mode network drives medial temporal hyperactivity and early tau accumulation. *Neuron* 112:676–86.e4. https://doi.org/10.1016/j.neuron.2023.11.014

Gjerum L, Andersen B, Bruun M et al. (2021) Comparison of the clinical impact of 2-[18F]FDG-PET and cerebrospinal fluid biomarkers in patients suspected of Alzheimer's disease. *PLoS One* 16:e0248413. https://doi.org/10.1371/journal.pone.0248413

Goossens J, Bjerke M, Van Mossevelde S et al. (2018) Diagnostic value of cerebrospinal fluid tau, neurofilament, and progranulin in definite frontotemporal lobar degeneration. *Alzheimers Res Ther* 10:31. https://doi.org/10.1186/s13195-018-0364-0

Gorno-Tempini ML, Hillis AE, Weintraub S et al. (2011) Classification of primary progressive aphasia and its variants. *Neurology* 76:1006–14. https://doi.org/10.1212/WNL.0b013e31821103e6

Güngen C, Ertan T, Eker E et al. (2002) Standardize Mini Mental Test'in türk toplumunda hafif demans tanisinda geçerlik ve güvenilirliği [Reliability and validity of the standardized Mini Mental State Examination in the diagnosis of mild dementia in Turkish population]. *Turk Psikiyatri Derg* 13:273–281.

Hafkemeijer A, Möller C, Dopper E et al. (2016) A longitudinal study on resting state functional connectivity in behavioral variant frontotemporal dementia and Alzheimer's disease. *J Alzheimers Dis* 55:521–37. https://doi.org/10.3233/JAD-150695

Ito K, Washimi Y, Kato T et al. (2025) 18F-FDG PET for the differential diagnosis of Alzheimer's disease and frontotemporal lobar degeneration: a multicenter prospective study in Japan. *J Alzheimers Dis* 106:293–303. https://doi.org/10.1177/13872877251338691

Leuzy A, Bollack A, Pellegrino D et al. (2025) Considerations in the clinical use of amyloid PET and CSF biomarkers for Alzheimer's disease. *Alzheimers Dement (Amst)* 21:e14528. https://doi.org/10.1002/alz.14528

Lleó A, Irwin D, Illán-Gala I et al. (2018) A 2-step cerebrospinal algorithm for the selection of frontotemporal lobar degeneration subtypes. *JAMA Neurol* 75:38–745. https://doi.org/10.1001/jamaneurol.2018.0118

Loi S, Eratne D, Santillo A et al. (2025) Biofluid biomarkers in distinguishing young-onset dementia from primary psychiatric disorders. *Curr Opin Psychiatry* 38:134–43. https://doi.org/10.1097/YCO.0000000000000979

Mandelli M, Lorca-Puls D, Lukic S et al. (2023) Network anatomy in logopenic variant of primary progressive aphasia. *Hum Brain Mapp* 44:4390–406. https://doi.org/10.1002/hbm.26388

Massa F, Farotti L, Eusebi P et al. (2019) Reciprocal incremental value of 18F-FDG-PET and cerebrospinal fluid biomarkers in mild cognitive impairment patients suspected for Alzheimer's disease and inconclusive first biomarker. *J Alzheimers Dis* 72:1193–207. https://doi.org/10.3233/JAD-190539

Mattsson-Carlgren N, Grinberg L, Boxer A et al. (2022) Cerebrospinal fluid biomarkers in autopsy-confirmed Alzheimer disease and frontotemporal lobar degeneration. *Neurology* 98:e1137–50. https://doi.org/10.1212/WNL.0000000000200040

Minoshima S, Cross D, Thientunyakit T et al. (2022) 18F-FDG PET imaging in neurodegenerative dementing disorders: insights into subtype classification, emerging disease categories, and mixed dementia with copathologies. *J Nucl Med* 63:2S–12S. https://doi.org/10.2967/jnumed.121.263194

Ossenkoppele R, Cohn-Sheehy B, La Joie R et al. (2015) Atrophy patterns in early clinical stages across distinct phenotypes of Alzheimer's disease. *Hum Brain Mapp* 36:4421–37. https://doi.org/10.1002/hbm.22927

Ou Y, Xu W, Li J et al. (2019) FDG-PET as an independent biomarker for Alzheimer's biological diagnosis: a longitudinal study. *Alzheimers Res Ther* 11:57. https://doi.org/10.1186/s13195-019-0512-1

Perani D, Cerami C, Caminiti S et al. (2015) Cross-validation of biomarkers for the early differential diagnosis and prognosis of dementia in a clinical setting. *Eur J Nucl Med Mol Imaging* 43:499–508. https://doi.org/10.1007/s00259-015-3170-y

Quispialaya K, Therriault J, Aliaga A et al. (2022) Discordance and concordance between cerebrospinal and [18F]FDG-PET biomarkers in assessing atypical and early-onset AD dementia cases. *Neurology* 99:e2428–36. https://doi.org/10.1212/WNL.0000000000201198

Ranasinghe K, Rankin K, Pressman P et al. (2016) Distinct subtypes of behavioral variant frontotemporal dementia based on patterns of network degeneration. *JAMA Neurol* 73:1078–88. https://doi.org/10.1001/jamaneurol.2016.2016

Turan Ç, Kesebir S, Meteris H et al. (2013) Afazi, prosopagnozi ve mani: semantik demans sağ temporal varyant tanılı bir olgu [Aphasia, prosopagnosia and mania: a case diagnosed with right temporal variant semantic dementia]. *Turk Psikiyatri Derg* 24:68–72.

Vismara M, Cirnigliaro G, Piccoli E et al. (2020) Crossing borders between frontotemporal dementia and psychiatric disorders: an updated overview. *J Alzheimers Dis* 75:661–73. https://doi.org/10.3233/JAD-191333

Yıldırım E and Soncu Büyükişcan E (2019) Default Mode Network Connectivity in Alzheimers Disease. Alzheimer Hastalığında Olağan Durum Ağı Bağlantısallığı. *Turk Psikiyatri Derg* 30:279–86. https://doi.org/10.5080/u23526