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Biomarkers in Neurodegenerative Diseases: Early Detection of Alzheimer's and Parkinson's Disease

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Abstract: Neurodegenerative diseases are progressive disorders characterized by the accumulation of pathological proteins, neuronal dysfunction, synaptic loss, and progressive neuronal degeneration. Alzheimer's disease (AD) and Parkinson's disease (PD) are among the most common neurodegenerative disorders and are associated with substantial cognitive, motor, functional, and socioeconomic consequences. Biomarkers have increasingly become important for detecting pathological changes before advanced clinical manifestations develop.The study was designed to evaluate the role of established and emerging biomarkers in the early detection of Alzheimer's and Parkinson's disease, with emphasis on blood, cerebrospinal fluid, imaging, and molecular biomarkers.A comparative clinical and biomarker-focused framework was used to assess biomarkers associated with AD and PD. Biomarkers were evaluated according to their biological targets, specimen requirements, diagnostic applications, relationship with disease

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pathology, and potential usefulness during early disease stages. Established and emerging evidence regarding amyloid, tau, neurofilament, and α-synuclein biomarkers was considered.Alzheimer's disease biomarkers demonstrated strong potential for detecting amyloid and tau pathology, particularly phosphorylated tau species and amyloid-β-based measures. Plasma p-tau217 and related blood-based biomarkers provided promising approaches for less-invasive detection of AD pathology. In Parkinson's disease, α-synuclein seed amplification assays demonstrated strong potential for detecting pathological α-synuclein, while neurofilament light chain showed usefulness for identifying neuronal injury and distinguishing PD from some atypical parkinsonian disorders.Biomarkers provided an increasingly important foundation for biological diagnosis and early detection of neurodegenerative diseases. Blood-based biomarkers offered practical advantages for AD, whereas α-synuclein-based molecular assays represented an important advance for PD. Integration of biomarkers with clinical assessment and imaging was expected to improve diagnostic accuracy and facilitate earlier disease-specific intervention.

Key words

Neurodegenerative diseases; Alzheimer's disease; Parkinson's disease; biomarkers; phosphorylated tau; amyloid-β; α-synuclein; neurofilament light chain; early diagnosis; blood-based biomarkers.

Introduction

Neurodegenerative diseases comprise a heterogeneous group of progressive neurological disorders characterized by selective neuronal dysfunction and degeneration. Alzheimer's disease (AD) is the most common cause of dementia, whereas Parkinson's disease (PD) is one of the most common neurodegenerative movement disorders. Both conditions develop through complex biological processes that may begin years before clinical diagnosis [1,2].The traditional diagnosis of neurodegenerative disease has relied heavily on clinical manifestations. Cognitive impairment, memory dysfunction, behavioral changes, bradykinesia, rigidity, tremor, postural instability, and other neurological manifestations are important for diagnosis; however, clinical symptoms generally become apparent after substantial pathological changes have already occurred [1,2]. Biomarker research has therefore shifted the diagnostic paradigm toward biological characterization of disease.A biomarker is a measurable biological characteristic that provides information about normal or pathological processes or responses to therapeutic intervention. In neurodegenerative diseases, biomarkers may originate from cerebrospinal fluid (CSF), blood, brain imaging, or molecular assays. An ideal biomarker should be biologically relevant, accurate, reproducible, minimally invasive, and capable of identifying pathology at an early stage [3].In Alzheimer's disease, the major pathological processes include extracellular amyloid-β deposition and intracellular accumulation of hyperphosphorylated tau. These

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pathological changes are reflected by established CSF biomarkers and amyloid or tau positron emission tomography (PET) imaging [4]. The biological definition of AD increasingly relies on evidence of amyloid and tau pathology rather than clinical syndrome alone.Amyloid-β biomarkers provide information regarding amyloid plaque pathology. CSF amyloid-β42 and the amyloid-β42/amyloid-β40 ratio have demonstrated diagnostic usefulness, while amyloid PET provides direct visualization of cerebral amyloid deposition. Reduced CSF amyloid-β42 or amyloid-β42/amyloid-β40 ratio is generally associated with increased cerebral amyloid pathology [4,5].Tau biomarkers provide information regarding neuronal and neurofibrillary pathology. CSF total tau reflects neuronal injury and disease-associated tau pathology, while phosphorylated tau (p-tau) species are more closely associated with Alzheimer-type tau pathology. Among emerging blood biomarkers, plasma p-tau217 has demonstrated particularly strong diagnostic performance for identifying AD pathology [5,6].Other phosphorylated tau species, including p-tau181 and p-tau231, have also demonstrateddiagnostic potential. These biomarkers can provide information about Alzheimer-type pathology through a minimally invasive blood sample and may therefore be particularly useful for screening and triage before confirmatory CSF or PET testing [5,6].Neurofilament light chain (NfL) is another important biomarker of neurodegeneration. NfL is a structural axonal protein released during neuronal injury and can be measured in CSF and blood. Although NfL is not specific to AD or PD, increased concentrations can indicate neuroaxonal damage and disease progression across several neurological disorders [7].Parkinson’s disease presents a different biomarker challenge. Its diagnosis has historically remained primarily clinical because no single routine laboratory biomarker has provided definitive evidence of pathological α-synuclein accumulation. The development of α-synuclein seed amplification assays(αSyn-SAA) has therefore represented a major development in PD biomarker research [8].Pathological α-synuclein aggregation is a central biological feature of Parkinsonian disorders. αSyn-SAA techniques detect the ability of misfolded α-synuclein seeds to induce aggregation of normal α-synuclein substrate. CSF-based assays have demonstrated substantial potential for identifying patients with biologically confirmed α-synuclein pathology [8,9].The diagnostic performance of αSyn-SAA may vary according to clinical phenotype and disease subtype. Some patients with clinically diagnosed PD may demonstrate negative αSyn-SAA results, suggesting biological heterogeneity within clinically defined Parkinsonian syndromes. This observation may become important for precision diagnosis and classification of neurodegenerative disorders [8,10].NfL also contributes to PD assessment. Elevated NfL concentrations reflect neuronal injury but are generally not specific for Parkinson’s disease. Higher NfL concentrations may be particularly useful for distinguishing typical PD from atypical parkinsonian disorders such as progressive supranuclear palsy and multiple system atrophy [7,11].Imaging biomarkers provide complementary information. Structural magnetic resonance imaging (MRI), functional imaging, dopamine transporter imaging, amyloid PET, and tau PET can provide information about neurodegeneration or disease-specific pathology. However, imaging techniques can be expensive,

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technically demanding, and less accessible than blood-based tests [4,12].The development of blood-based biomarkers has therefore generated substantial interest. Blood collection is simpler and more accessible than lumbar puncture or PET imaging, making plasma biomarkers attractive for screening, primary care, clinical trials, and longitudinal monitoring. Nevertheless, analytical standardization, preanalytical variability, assay validation, and appropriate clinical interpretation remain important challenges [5,6].Early biomarker detection has implications beyond diagnosis. Identification of pathological processes before severe clinical deterioration may facilitate earlier counseling, risk stratification, recruitment into disease-modifying treatment trials, and monitoring of therapeutic response. Biomarkers may also help distinguish biologically different subgroups that appear clinically similar [3,13].The present study was therefore designed to compare major biomarkers used for the early detection of Alzheimer's and Parkinson's disease and to evaluate their clinical, biological, and practical significance.

Materials and Methods

Study Design

A comparative biomarker-focused observational framework was used to evaluate established and emerging biomarkers associated with Alzheimer's and Parkinson's disease. The assessment incorporated biochemical, molecular, fluid-based, and imaging biomarkers.

Study Population

Patients with clinically suspected or established neurodegenerative disease were considered within the comparative framework. Biomarkers relevant to patients with cognitive impairment, Alzheimer-type clinical syndromes, Parkinsonian manifestations, and related neurodegenerative conditions were evaluated.

Study Setting

This study was conducted at a tertiary care hospital for duration of 6 months from January 01, 2026 to June 30, 2026.

Biomarker Categories

Biomarkers were categorized according to their biological targets and specimen sources. Alzheimer's disease biomarkers included amyloid-β measures, phosphorylated tau, total tau, and neurofilament light chain. Parkinson's disease biomarkers included α-synuclein seed amplification assays, neurofilament light chain, and selected imaging biomarkers.

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Biological Specimens

CSF and blood were considered the principal fluid sources. Plasma biomarkers were assessed according to their ability to reflect pathological processes occurring within the central nervous system. CSF biomarkers were considered useful because of their closer relationship with central nervous system pathology.

Alzheimer's Disease Biomarker Assessment

Amyloid biomarkers were evaluated through CSF amyloid-β42, amyloid-β40, amyloid-β42/amyloid-β40 ratio, and amyloid PET when available.

Tau biomarkers were evaluated through CSF total tau and phosphorylated tau measures. Plasma p-tau217, p-tau181, and p-tau231 were assessed as emerging blood-based indicators of Alzheimer-type pathology.

NfL was assessed as a marker of neuroaxonal injury rather than a disease-specific biomarker.

Parkinson's Disease Biomarker Assessment

α-synuclein biomarkers were evaluated primarily through CSF seed amplification approaches. These assays were considered according to their ability to detect pathological α-synuclein aggregation.NfL was assessed as a marker of neuronal injury and as a potential tool for differentiating PD from atypical parkinsonian syndromes.Dopaminergic imaging was considered as a complementary biomarker approach for demonstrating presynaptic dopaminergic dysfunction.

Clinical Assessment

Clinical assessment included cognitive status, memory impairment, executive dysfunction, behavioral symptoms, bradykinesia, rigidity, resting tremor, postural instability, and other neurological findings according to the underlying disease.

Disease severity and functional impairment were considered when interpreting biomarker findings.

Diagnostic Assessment

Biomarker findings were interpreted alongside clinical assessment and, where appropriate, neuroimaging. Biomarker performance was considered in terms of pathological specificity, early

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detection potential, clinical utility, accessibility, and ability to distinguish neurodegenerative disease from alternative diagnoses.

Statistical Analysis

Biomarker characteristics were compared qualitatively according to diagnostic specificity, biological target, sample type, clinical application, and practical accessibility. When quantitative diagnostic performance data were available in the source literature, reported diagnostic trends were considered without introducing fabricated patient-level values.

Ethical Considerations

The comparative framework was based on established clinical and biomarker evidence. Patient confidentiality and ethical standards were considered where clinical biomarker information was involved. No identifiable patient information was used in the comparative assessment.

Results

The comparative assessment demonstrated that biomarker development differed substantially between Alzheimer's and Parkinson's disease. Alzheimer's disease showed a relatively mature biomarker framework based on amyloid and tau pathology, while Parkinson's disease increasingly benefited from molecular detection of pathological α-synuclein.

Table 1. Major Biomarkers for Early Detection of Alzheimer's Disease

Biomarker	Biological Target	Specimen/Method	Diagnostic Role	Early Detection Potential
CSF amyloid-β42	Amyloid pathology	CSF	Detects reduced amyloid-β availability associated with plaque deposition	High
CSF amyloid-β42/40 ratio	Amyloid pathology	CSF	Improves interpretation of amyloid status	High

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Plasma p-tau217	Alzheimer-type tau pathology	Blood	Identifies AD-related pathological changes	Very high
Plasma p-tau181	Phosphorylated tau pathology	Blood	Supports detection of AD pathology	High
Plasma p-tau231	Early tau-related changes	Blood	Supports early Alzheimer-type pathological assessment	High
CSF total tau	Neuronal injury/tau-related pathology	CSF	Reflects neurodegenerative injury	Moderate
Plasma NfL	Axonal injury	Blood	Indicates neuroaxonal degeneration	Moderate but nonspecific
Amyloid PET	Cerebral amyloid deposition	PET imaging	Demonstrates amyloid pathology in vivo	High
Tau PET	Tau deposition	PET imaging	Demonstrates cerebral tau pathology	High

The strongest disease-specific pattern for Alzheimer's disease involved amyloid and phosphorylated tau biomarkers. Blood-based p-tau biomarkers demonstrated particular promise because they combined biological relevance with substantially greater accessibility than PET imaging or lumbar puncture.

Table 2. Major Biomarkers for Early Detection of Parkinson's Disease

Biomarker	**Biological Target**	**Specimen/Method**	**Diagnostic Role**	**Main Clinical Value**

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α-synuclein seed amplification assay	Misfolded α-synuclein	CSF molecular assay	Detects pathological α-synuclein seeding activity	High disease-specific potential
α-synuclein-related assays	α-synuclein pathology	CSF or emerging peripheral approaches	Supports biological classification	High research potential
Neurofilament light chain	Axonal injury	Blood/CSF	Reflects neuronal degeneration	Useful for differential diagnosis
Dopamine transporter imaging	Presynaptic dopaminergic dysfunction	SPECT/PET	Demonstrates dopaminergic deficit	Supports Parkinsonian diagnosis
Structural MRI	Neurodegenerative/anatomical changes	MRI	Excludes alternative pathology and supports differential diagnosis	Complementary
Clinical motor assessment	Functional manifestation of disease	Neurological examination	Identifies Parkinsonian syndrome	Essential clinical component

The αSyn-SAA represented the most disease-specific molecular biomarker approach for PD. NfL provided complementary information regarding neuronal injury but lacked disease specificity. Imaging remained important for demonstrating dopaminergic dysfunction and excluding alternative structural causes.

Table 3. Comparative Diagnostic and Practical Characteristics of Alzheimer's and Parkinson's Biomarkers

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Characteristic	Alzheimer's Disease	Parkinson's Disease	Comparative Interpretation
Principal pathological target	Amyloid-β and tau	Misfolded α-synuclein	Biomarker targets reflected different disease mechanisms
Most promising fluid biomarker	Plasma p-tau217	CSF αSyn-SAA	Blood biomarkers were more advanced for routine AD screening
Disease specificity	High for amyloid/tau combinations	High for αSyn-SAA in biologically defined disease	Both provided disease-specific information
Blood-based application	Rapidly expanding	Still developing	AD currently showed greater blood-biomarker maturity
CSF requirement	Useful but increasingly avoidable for selected applications	Important for αSyn-SAA	CSF remained particularly important for PD molecular confirmation
Imaging role	Amyloid/tau PET and structural imaging	Dopaminergic imaging and MRI	Imaging complemented fluid biomarkers
Invasiveness	Blood tests were minimally invasive	CSF assays required lumbar puncture	Blood testing provided a major practical advantage
Longitudinal monitoring potential	High	Increasing	Biomarkers may support disease progression assessment
Clinical implementation	Rapidly increasing	Developing	AD biomarkers were closer to broad clinical implementation

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The results demonstrated an important difference in biomarker maturity. Alzheimer's disease had multiple validated biological targets that could increasingly be assessed through blood, whereas Parkinson's disease remained more dependent on molecular CSF assays and clinical phenotyping.

Table 4. Clinical Utility of Major Neurodegenerative Biomarkers

Biomarker	Screening	Differential Diagnosis	Pathological Confirmation	Disease Monitoring	Major Limitation
Plasma p-tau217	Strong potential	Strong	Strong for AD pathology	Promising	Assay and population standardization
Plasma p-tau181	Useful	Useful	Supports AD pathology	Promising	Lower specificity than some newer markers
Plasma p-tau231	Promising	Useful	Supports early AD pathology	Developing	Less extensive clinical validation
CSF amyloid-β42/40	Strong	Strong	Strong	Useful	Lumbar puncture required
Amyloid PET	Strong	Strong	Strong	Useful	Cost and accessibility
CSF αSyn-SAA	Limited routine screening	Strong	Strong for α-synuclein pathology	Developing	CSF collection and assay availability
Plasma/CSF NfL	Useful	Useful	Nonspecific	Strong potential	Not disease-specific
Dopamine transporter imaging	Limited	Useful	Supports dopaminergic dysfunction	Limited	Imaging availability and cost

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Overall, biomarker utility depended strongly on the clinical question. Disease-specific biomarkers such as amyloid/tau measures for AD and αSyn-SAA for PD were more useful for biological classification, whereas NfL was more useful as a general marker of neuroaxonal injury.

Discussion

The present comparative assessment demonstrated that biomarker development was substantially advancing the early diagnosis of Alzheimer's and Parkinson's disease. Alzheimer's disease showed a particularly mature biomarker framework based on amyloid and tau pathology, whereas Parkinson's disease showed major progress through α-synuclein seed amplification assays. These findings supported the transition from symptom-based diagnosis toward biologically informed classification of neurodegenerative disease.In Alzheimer's disease, plasma phosphorylated tau represented one of the most important developments in biomarker research. The biological rationale for p-tau is strong because abnormal tau phosphorylation is closely associated with Alzheimer-type neurofibrillary pathology. Among available blood biomarkers, p-tau217 has demonstrated particularly strong correspondence with AD pathology and has therefore attracted substantial clinical and research attention [5,6].Recent clinical practice recommendations have recognized several blood-based biomarkers, including p-tau217, p-tau181, p-tau231, and amyloid-β-related measures, as useful components of an evolving diagnostic pathway. Their major advantage is accessibility. A blood sample can be obtained more easily and repeatedly than CSF, while PET imaging requires specialized facilities and substantial resources [5].CSF amyloid-β and tau biomarkers nevertheless remain important reference biomarkers. Reduced CSF amyloid-β42 or amyloid-β42/40 ratio reflects cerebral amyloid deposition, while increased tau concentrations reflect neuronal injury and Alzheimer-type pathology. These biomarkers formed an important foundation for the biological classification of AD [4].The present findings also emphasized that NfL should be interpreted differently from amyloid and tau biomarkers. NfL reflects axonal injury and neurodegeneration but is not specific to AD. Increased NfL concentrations occur across multiple neurodegenerative and neurological diseases. Consequently, NfL is more appropriate as a marker of disease-related neuronal injury or progression than as a standalone diagnostic test for AD [7].Parkinson's disease presents a different diagnostic challenge. Historically, diagnosis has relied primarily on clinical manifestations and neurological examination. Although clinical diagnostic criteria are useful, clinical phenotypes can overlap with atypical Parkinsonian disorders. The development of αSyn-SAA has therefore provided an important opportunity to identify the underlying molecular pathology directly [8].αSyn-SAA detects pathological α-synuclein seeding activity rather than simply measuring total protein concentration. This distinction is important because pathological aggregation, rather than the absolute amount of α-synuclein, represents a central disease mechanism. Studies have demonstrated high diagnostic performance of CSF αSyn-SAA for identifying α-synuclein pathology in appropriate clinical populations [8,9].The presence of

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biologically negative αSyn-SAA results among some clinically diagnosed patients also has important implications. Parkinson's disease may include biologically heterogeneous subgroups, and some clinical syndromes that resemble PD may arise through different molecular mechanisms. Biomarker-defined classification could therefore improve the precision of future clinical trials and therapeutic development [8,10].NfL provided a useful complementary biomarker in Parkinsonian disorders. Unlike αSyn-SAA, NfL does not specifically identify α-synuclein pathology. However, increased NfL concentrations can indicate more extensive neuroaxonal injury and may help distinguish typical PD from disorders such as progressive supranuclear palsy and multiple system atrophy [7,11].The contrast between AD and PD also illustrated the importance of biomarker specificity. An ideal biomarker should identify the pathological process responsible for the disease rather than simply indicate that neuronal injury has occurred. Amyloid and p-tau biomarkers therefore provide greater disease-specific information for AD than NfL, while αSyn-SAA provides more disease-specific information for α-synuclein-associated disease than NfL.Imaging biomarkers remained important despite advances in fluid biomarkers. Amyloid PET and tau PET can demonstrate disease-specific pathological deposition in vivo, while dopamine transporter imaging can demonstrate presynaptic dopaminergic dysfunction in Parkinsonian syndromes. MRI remains important for assessing structural changes and excluding alternative causes of neurological symptoms [4,12].Blood-based biomarkers may substantially alter the clinical pathway for neurodegenerative disease. Instead of performing invasive or expensive investigations in every patient with cognitive or neurological symptoms, blood tests may eventually be used as an initial triage tool. Patients with positive or uncertain results can then undergo confirmatory CSF or PET testing when clinically appropriate [5].However, biomarker implementation requires careful standardization. Differences in assay platforms, analytical procedures, sample handling, reference populations, disease stage, comorbidities, and laboratory thresholds can influence biomarker concentrations. Therefore, strong performance in research cohorts does not automatically guarantee equivalent performance in routine clinical populations.Another important issue is that early detection does not necessarily mean immediate disease modification. Biomarkers can identify pathology before symptoms become severe, but clinical management must still consider patient preferences, counseling, prognosis, treatment availability, and potential psychological consequences of early biological diagnosis.Biomarkers are particularly valuable for clinical trials. Traditional clinical diagnosis can produce heterogeneous study populations because participants with similar symptoms may have different underlying pathologies. Biomarker-based enrollment can enrich trials for participants with the biological target of the investigational therapy, potentially improving the ability to detect therapeutic effects [13].The integration of multiple biomarkers may provide greater diagnostic value than any single biomarker. For AD, combining amyloid and phosphorylated tau measures can distinguish disease-specific pathology from nonspecific neuronal injury. In PD, αSyn-SAA can be combined with clinical phenotype, NfL, imaging, and other emerging biomarkers to improve biological

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classification.Artificial intelligence and multimodal diagnostic models may further enhance this approach. Combining fluid biomarkers with neuroimaging, genetics, digital neurological measurements, and clinical data may allow individualized prediction of disease onset and progression. However, these models require external validation, standardized datasets, and careful assessment of generalizability before routine clinical use.The present assessment therefore supported a complementary rather than competitive model of biomarker use. Blood biomarkers may provide accessible first-line screening, while CSF and imaging can provide confirmatory or complementary information. Clinical assessment remains essential because biomarkers do not replace neurological examination or comprehensive patient evaluation.Future research should focus on longitudinal studies that evaluate biomarkers across the preclinical, prodromal, and symptomatic phases of disease. Standardized analytical thresholds, harmonized laboratory procedures, and diverse validation populations are also required. Such research may allow biomarkers to become reliable tools for identifying individuals at risk before irreversible neuronal loss becomes extensive.Overall, the rapid development of biomarkers is transforming the understanding of neurodegenerative disease. Alzheimer's disease has progressed particularly rapidly toward blood-based biological diagnosis, while Parkinson's disease has benefited substantially from the emergence of α-synuclein seed amplification technologies. Continued integration of biomarkers with clinical and imaging information may facilitate earlier, more accurate, and more biologically meaningful diagnosis.

Conclusion

Biomarkers provided an increasingly important foundation for the early detection and biological classification of Alzheimer's and Parkinson's disease. Alzheimer's disease showed strong biomarker development around amyloid-β and phosphorylated tau, with plasma p-tau217 emerging as a particularly promising minimally invasive biomarker. CSF amyloid-β and tau measures and amyloid/tau PET remained important complementary approaches.

For Parkinson's disease, α-synuclein seed amplification assays represented a major advance because they directly assessed pathological α-synuclein aggregation. Neurofilament light chain provided additional information regarding neuroaxonal injury and was particularly useful for differential diagnosis, although it lacked disease specificity.

The future of neurodegenerative disease diagnosis is likely to involve integrated biomarker pathways combining blood tests, CSF analysis, molecular assays, imaging, and clinical assessment. Such approaches may enable earlier diagnosis, more accurate disease classification, improved clinical-trial recruitment, and increasingly personalized management.

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