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Stability and Accuracy of a Diagnosis of Parkinson Disease Over 10 Years

Valtteri Räty,[1,2] Tomi Kuusimäki,[1,2] Joonas Majuri,[3] Tero Vahlberg,[4] Maria Gardberg,[5] Tommi Noponen,[6] Marko Seppänen,[7] Anna-Maija Tolppanen,[8] and Valtteri Kaasinen[1,2]

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Correspondence
Dr. Kaasinen
valtteri.kaasinen@tyks.fi

Abstract

Background and Objectives

Accurate diagnosis of Parkinson disease (PD) remains challenging, with variability and clinical uncertainty, especially in nonspecialized settings. Despite advancements in diagnostic criteria and biological markers, misdiagnosis continues to affect patient care and research. This study aimed to assess the long-term diagnostic stability of PD and evaluate the accuracy of initial diagnoses over time in a large, consecutive cohort diagnosed by neurologists, with or without movement disorder specialization.

Methods

We conducted a retrospective longitudinal analysis of patients diagnosed with PD between 2006 and 2020. Patient records were reviewed over a median follow-up period of 10 years, with more than half of the cohort tracked from motor symptom onset to death. Diagnostic evaluations included dopamine transporter (DAT) imaging and neuropathologic examinations for a subset of patients, based on clinical indications. Two movement disorder specialists cross-validated diagnoses through retrospective chart reviews.

Results

The cohort included 1,626 patients (mean age 69.0 years, 44.1% female). Of these, 10.6% (n = 172) had their diagnoses revised by treating neurologists, and 2.7% (n = 44) were revised based on chart reviews or neuropathologic findings. The median time to diagnosis revision was 22 months (interquartile range = 43). The most common revised diagnoses were vascular parkinsonism, progressive supranuclear palsy, and multiple system atrophy, with 4.7% (n = 77) classified as clinically undetermined parkinsonism. In a secondary analysis separating PD and dementia with Lewy bodies (DLB), the revision rate increased to 17.7%. DAT imaging had been performed on 588 patients and was more frequently used in revised cases. Postmortem neuropathologic examinations had been conducted in only 3% of deceased patients, with 64% confirming the initial PD diagnosis.

Discussion

This study demonstrates significant diagnostic instability in PD, with 13.3% of diagnoses revised, primarily within 2 years. When DLB is considered separately, the revision rate increases to 17.7%. Despite frequent DAT imaging and limited postmortem examinations, clinical uncertainty persists among practicing neurologists, contrasting with lower misdiagnosis rates in specialized centers. These findings highlight the need for systematic application of diagnostic criteria, regular reevaluation of diagnoses, more frequent autopsies, and the development of accessible diagnostic biomarkers.

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[1]Clinical Neurosciences, University of Turku, Finland; [2]Neurocenter, Turku University Hospital, Finland; [3]Department of Neurology, Helsinki University Hospital and University of Helsinki, Finland; [4]Department of Biostatistics, University of Turku and Turku University Hospital, Finland; [5]Tyks Laboratories, Pathology, Turku University Hospital and Institute of Biomedicine, University of Turku, Finland; [6]Department of Clinical Physiology, Nuclear Medicine, Turku PET Centre and Medical Physics, Turku University Hospital and Wellbeing Services County of Southwest Finland; [7]Department of Clinical Physiology, Nuclear Medicine, and Turku PET Centre, Turku University Hospital and Wellbeing Services County of Southwest Finland; and [8]School of Pharmacy, University of Eastern Finland, Kuopio.

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Glossary

CUPS = clinically undetermined parkinsonian syndrome; **DAT** = dopamine transporter; **DIP** = drug-induced parkinsonism; **DLB** = dementia with Lewy bodies; **EHR** = electronic health record; **ET** = essential tremor; **ICD-10** = International Classification of Diseases, 10th Revision; **IQR** = interquartile range; **MDS** = Movement Disorder Society; **MSA** = multiple system atrophy; **PD** = Parkinson disease; **PSP** = progressive supranuclear palsy; **SAA** = seed amplification assay; **VP** = vascular parkinsonism.

Introduction

Accurate diagnosis of Parkinson disease (PD) poses a significant challenge that affects both patient care and research outcomes. Previous meta-analyses of clinicopathologic studies have reported suboptimal diagnostic accuracy for PD, ranging from 73.8% to 79.6%, depending on the expertise of the diagnosing clinician.[1] Recent updates to clinical diagnostic criteria seem to have improved accuracy, and emerging biological definitions of the disease may prove valuable for the diagnosis and staging of PD in the long term.[2-4] However, even specialists frequently encounter difficulties in determining the precise etiology of parkinsonism.[5] Moreover, PD diagnosis codes in electronic health records (EHRs) often lack accuracy, and PD is frequently overdiagnosed, with diagnostic accuracy particularly compromised in early-stage patients, nursing home residents, and individuals evaluated by nonspecialists.[6-11]

Routine clinical practice rarely uses gold standard diagnostic procedures such as postmortem neuropathologic examination or evaluations by movement disorder specialists. This infrequent use results in a less clear understanding of diagnostic accuracy in clinical settings. Previous clinical follow-up cohort studies have reported a broad range of diagnostic error rates, from 5.0% to 30.4%, based on samples as large as 1,393 individuals.[12-18] The variability likely arises from differences in diagnostic methodologies, criteria, levels of expertise, and patient demographics.

To bridge this knowledge gap, our study aimed to systematically evaluate the diagnostic stability and accuracy of PD diagnoses in a large, unselected cohort of patients over a median follow-up of 10 years. By examining the complexities encountered in clinical settings, the study aimed to contribute to a deeper understanding of the factors affecting diagnostic precision and the variability that practitioners face when diagnosing PD.

Methods

The study cohort predominantly comprised outpatients diagnosed with PD across 4 medical institutions in southwestern Finland: Turku University Hospital and 3 regional hospitals. In Finland, specialized health care facilities manage both outpatient clinics and inpatient admissions, with the medical records from both settings being integrated into a single-registry system. Therefore, although the diagnoses were primarily made in outpatient settings, all patient data were retrieved from the hospital registry, which includes records from both outpatient and inpatient care. Patients were included if their EHRs contained at least 2 instances of the G20 code during the study period from January 1, 2006, to December 31, 2020. From October 2022 to April 2024, 2 movement disorder specialists conducted a thorough review of the EHRs. The initial evaluation was performed by V.K., who assessed all patient records. A second evaluation was performed by T.K. to validate the diagnostic classifications by reviewing a randomly selected subsample of 10% of the cases (n = 163). T.K. was blinded to V.K.'s results and used the same diagnostic criteria.

In both historical and contemporary Finnish care guidelines for PD, a comprehensive clinical evaluation by a neurologist is required for diagnosis, with specific diagnostic criteria recommended.[19] Historically, the UK Brain Bank Criteria[20] were used, whereas the Movement Disorder Society (MDS) Clinical Criteria[21] are currently endorsed. Furthermore, reimbursement for PD medications requires a clinically verified diagnosis made by a neurologist, accompanied by a written confirmation from the diagnosing neurologist. Consequently, all patients with PD included in this study were diagnosed by certified neurologists or were neurology residents supervised by a specialist in the field.

Auria Clinical Informatics, which manages the Turku University Hospital database, houses a comprehensive repository of clinical data.[22] This includes demographic information, clinical diagnoses, procedural details, inpatient and outpatient records, pathology reports, imaging results, treatments, medications, laboratory measurements, and clinical narratives. This database has been maintained in electronic format since January 1, 2004, and it consolidates information from the individual hospitals in the region. All clinical patients are automatically included in the registry if they have any contact with one of the participating hospitals, including a single visit or consultation. The data for this study were obtained exclusively from the Auria clinical data registry.

From 2006 to 2020, Finland did not have an officially structured subspecialty in movement disorders, a situation shared by many other countries.[23] Neurologists in Finland receive training in PD diagnostics and treatment as part of their general neurology education. Further expertise in

movement disorders is pursued on an individual basis, leading to variability in the depth of knowledge and experience among neurologists. As a result, the neurologists involved in diagnosing patients in this study comprised a diverse group with varying levels of specialization and interest in PD.

Evaluation of PD Diagnoses

Detailed documentation included variables such as age at initial PD diagnosis, sex, date of death (if applicable), date of first PD code appearance, date and results of brain dopamine transporter (DAT) imaging, diagnostic criteria used, final diagnosis date, and any changes in diagnosis made by clinicians or evaluators. Final diagnoses and their corresponding International Classification of Diseases, 10th Revision (ICD-10) codes were also recorded. For PD, the MDS clinical diagnostic criteria were applied, with modifications made as necessary for remote evaluations based on available chart information.[24] If a patient did not meet the PD diagnostic criteria but retained the PD diagnosis code by the end of the follow-up period, 2 evaluators reviewed the case and adjusted the diagnosis accordingly. Diagnostic evaluations adhered to specific criteria: multiple system atrophy (MSA) was classified based on core clinical features of autonomic dysfunction and poorly levodopa-responsive parkinsonism or cerebellar syndrome, supported by a striatal DAT defect.[25] Progressive supranuclear palsy (PSP) required documentation of vertical gaze palsy, postural instability, and levodopa-resistant akinesia, with additional support from midbrain atrophy or a striatal DAT defect if available.[26] Although the primary analysis was conducted with dementia not considered an exclusion criteria in accordance with the MDS-PD criteria,[21] diagnostic changes from PD to dementia with Lewy bodies (DLB) were also recorded. Classification of DLB used the 1-year rule, which stipulates that dementia occurs before or within 1 year after the onset of parkinsonism, particularly with fluctuating cognition and visual hallucinations.[27] Vascular parkinsonism (VP) was identified based on levodopa-resistant parkinsonism, especially in the lower body, combined with a history of strokes and cerebrovascular disease evident on brain CT or MRI.[28] Category of essential tremor (ET) was used in the presence of a long-standing, predominantly upper limb action tremor, with supporting features including a positive family history, normal DAT imaging, and/or a lack of levodopa response.[29] Drug-induced parkinsonism (DIP) was defined as tremor or parkinsonism associated with the initiation or dosage increase of a medication (e.g., neuroleptic), supported by normal or borderline DAT imaging. Smaller diagnostic categories were assessed individually using the available clinical and imaging information, guided by diagnostic guidelines and the evaluators' expertise. Finally, cases of parkinsonism that did not meet the MDS criteria for PD or fit other specific diagnoses were classified as clinically uncertain parkinsonian syndrome (CUPS). Patients were considered miscoded if there was no mention of PD in their EHRs and if there were no prescriptions of antiparkinsonian medications, despite having the PD diagnosis code (G20) in their records. These patients were subsequently excluded from the final analysis.

Dopamine Transporter Imaging

Brain DAT imaging results were collected retrospectively from existing patient records. DAT imaging had been conducted for clinical diagnostic purposes at the Turku University Hospital Department of Nuclear Medicine, following the routine clinical imaging protocol. The decision to perform DAT imaging was based on clinical judgment, particularly in cases with atypical symptoms or when diagnostic uncertainty arose, such as in suspected iatrogenic, vascular, or ET cases. DAT imaging was not routinely used for all PD diagnoses, but rather selectively to support clinical decision-making in complex cases. Imaging had been performed using one of 6 SPECT systems available at the facility. Patients had been administered 185 MBq of the radiopharmaceutical [^{123}I]FP-CIT intravenously, followed by a waiting period of approximately 3–4 hours before SPECT imaging. To minimize thyroid radiation exposure, patients had received a protective dose of either potassium perchlorate (250 or 300 mg) or potassium iodide tablets (130 mg) 30–60 minutes before radiopharmaceutical injection. The imaging acquisition had lasted 28–38 minutes, which was followed by reconstruction and both visual and quantitative analysis. SPECT images had been reconstructed using the Hermes Medical Solutions software (Stockholm, Sweden), using a 3-dimensional Ordered Subsets Expectation Maximization algorithm. The image acquisition and reconstruction followed the guidelines provided by the European Association of Nuclear Medicine.[30] The images had been further analyzed with BRASS analysis software (Hermes Medical Solutions, Stockholm, Sweden).

Specific binding ratios for DAT were calculated in 4 predefined subregions of the striatum: the left and right putamen and caudate nuclei, with the occipital cortex used as the reference region. Experienced nuclear medicine physicians had interpreted the imaging and quantitation results as part of the routine clinical diagnostic process. For this study, these results were retrospectively analyzed to assess the presence and severity of dopaminergic deficits, which were subsequently classified into 3 categories: normal, borderline, or abnormal.

Neuropathologic Examinations

Neuropathologic examination data were retrospectively collected from patient records. The examinations had been conducted at Turku University Hospital by 3 trained neuropathologists following standardized protocols. Briefly, autopsy brains had been fixed in 10% formalin for a minimum of 2 weeks, after which tissue samples had been collected from multiple brain regions, paraffin-embedded, sectioned, and stained with hematoxylin and eosin for histologic analysis. Immunohistochemistry had been performed on selected tissue sections, using routine stains for β-amyloid (RBT-AY, BioSB), tau (AT-8, Innogenetics), and alpha-synuclein (KM51, Novocastra Antibodies). Positive and negative controls were included on each slide to ensure accuracy.

Neurodegenerative disease diagnoses had been made according to contemporary neuropathologic criteria.[31-34] All cases were reviewed and confirmed by a single neuropathologist (M.G.).

Statistics

Statistical analysis was performed using IBM SPSS Statistics software (version 29.0; IBM, Armonk, NY). Descriptive statistics for demographic data are presented as means with SDs or medians with interquartile ranges (IQRs), depending on the data distribution. The χ^2 test was used to test the differences in categorical variables. Differences in continuous variables between binary groups were assessed using Mann-Whitney *U* tests. For comparisons among multiple diagnostic groups, parametric data were analyzed using general linear model 1-way analysis of variance, while nonparametric data were evaluated with Kruskal-Wallis tests. Post hoc Bonferroni corrections were applied to control for multiple comparisons. Correlations were assessed using Spearman rank correlation coefficients. Interrater agreement was quantified using Cohen κ statistics. Statistical significance was defined at a threshold of $p < 0.05$.

Standard Protocol Approvals, Registrations, and Patient Consents

This study was conducted in accordance with ethical standards for human experimentation and approved by the Turku University Hospital administration. Approval from an ethical standards committee was waived according to Finnish law because the research involved a retrospective analysis of existing patient medical records and did not require direct contact with patients. Consequently, informed consent from participants was not necessary. All patient data were handled in compliance with local regulations and institutional policies, ensuring confidentiality and anonymity.

Data Availability

Anonymized data not included in this article will be made available on reasonable request from qualified investigators. Interested parties may request access to the deidentified patient data, which includes clinical evaluations and diagnostic information relevant to the study. Requests should be directed to the corresponding author and will be considered based on the qualifications of the requesting investigator and the intended use of the data. Data will be shared for purposes of replicating procedures and results, in accordance with ethical guidelines and institutional policies.

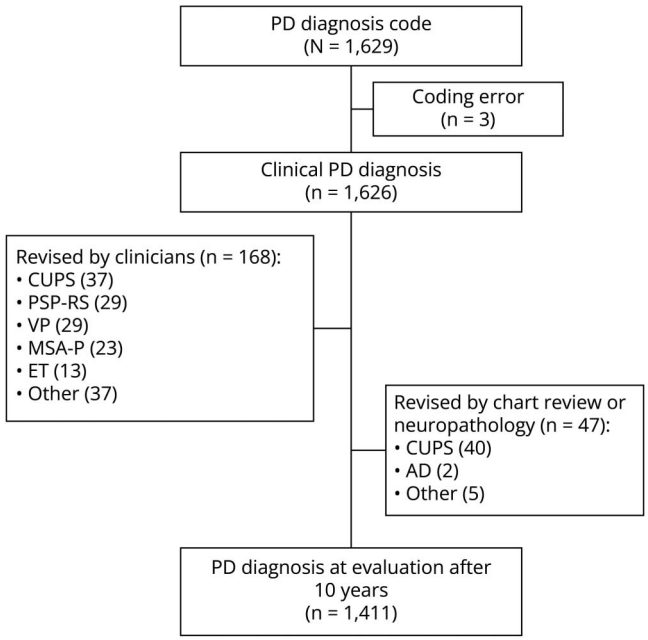
Results

Diagnostic Stability of PD

A total of 1,629 patients were initially diagnosed with PD using the ICD-10 code G20. After excluding 3 patients who were mistakenly coded with PD while receiving treatment for unrelated conditions, the final sample comprised 1,626 cases. Diagnostic stability was assessed through a retrospective chart review, with the mean time from the initial PD diagnosis to the evaluation of diagnostic stability being 10.2 years (SD = 6.1; median = 10.0 years, IQR = 9.0). Throughout the follow-up period, when changes from PD to DLB or monogenic PD were not considered as altered diagnoses (n = 1,411), 10.6% of patients (n = 172) had their PD diagnoses changed to non-PD diagnoses based on clinical evaluations. In addition, 2.7% of patients (n = 44) had their diagnoses revised following chart reviews or neuropathologic examinations. Thus, 13.3% of patients experienced diagnostic changes, including those ultimately classified as clinically undetermined, based on evaluations by treating neurologists, chart reviews, or neuropathologic examinations. Excluding patients with a final diagnosis of CUPS from the analysis reduced the revision rate to 9.8% (n = 138 of 1,411). A detailed flow chart of patient inclusion and exclusion criteria is shown in Figure 1, with final diagnoses outlined in Tables 1–4. When idiopathic PD, DLB, and monogenic PD were classified as separate diagnostic categories, the rate of diagnostic revisions increased to 17.7% (Table 2).

For diagnoses modified based on clinical evaluations, the median time interval between the initial PD diagnosis and the revised diagnosis was 22 months (IQR = 42.5). A Kruskal-Wallis test was performed to compare diagnostic delays in the 20 different diagnostic groups. The overall test yielded

Figure 1 Diagnostic Categorization Flowchart



The process of diagnostic categorization for patients included in the study outlining the sequence from initial PD diagnosis through subsequent evaluations. AD = Alzheimer disease; CUPS = clinically undetermined parkinsonian syndrome; ET = essential tremor; MSA-P = multiple system atrophy—parkinsonian subtype; PD = Parkinson disease; PSP-RS = progressive supranuclear palsy—Richardson syndrome; VP = vascular parkinsonism

Table 1 Summary of Final Diagnoses Based on Clinical Evaluations, Chart Reviews, and Neuropathologic Examinations

Diagnosis	n	Age at PD diagnosis, y, mean (SD)	Sex, % males	DAT imaging				Deceased at evaluation, %
				Performed, %, n	Abnormal, %, n	Borderline, %, n	Normal, %, n	
PD	1,338	68.5 (11.2)	55.5	34.1, 456[a]	99.3, 453	0.4, 2	0	48.4
CUPS	77	67.8 (10.4)	57.1	53.2, 41	24.4, 10	31.7, 13	43.9,18	49.4
DLB	69	74.3 (7.5)	59.4	29.0, 20	90.0, 18	5.0, 1	5.0, 1	75.4
VP	30	73.2 (9.3)	63.3	40.0, 12	16.7, 2	58.3, 7	25.0, 3	66.7
PSP-RS	29	71.8 (7.6)	65.5	41.4, 12	100, 12	0	0	89.7
MSA-P	24	65.2 (11.2)	45.8	54.2, 13	100, 13	0	0	75.0
ET	15	73.4 (8.4)	53.3	66.7, 10	0	40.0, 4	60.0, 6	26.7
DIP	12	73.5 (7.4)	41.7	66.7, 8	12.5, 1	12.5, 1	75.0, 6	58.3
CBS	6	71.1 (4.9)	50.0	83.3, 5	80.0, 4	20.0, 1	0	66.7
AD	4	78.3 (4.8)	75.0	0	0	0	0	100
Monogenic PD	4	37.8 (8.1)	25.0	100, 4	100, 4	0	0	25.0
NPH	4	77.4 (5.3)	50.0	25.0, 1	0	0	100, 1	100
Depression	3	69.3 (19.2)	33.3	33.3, 1	100, 1	0	0	66.7
PSP-CBS	2	73.0 (3.0)	100	0	0	0	0	50.0
FND	2	50.5 (3.0)	50.0	100, 2	0	0	100, 2	0
MND	2	60.8 (3.4)	100	50.0, 1	0	0	100, 1	50.0
Other[b]	5	65.7 (11.3)	100	60.0, 3	0	0	100, 3	60
Total	1,626	69.0 (11.1)	55.9	36.3, 590[a]	87.8, 518	4.9, 29	6.9, 41	51.2

Abbreviations: AD = Alzheimer disease; CBS = corticobasal syndrome; CUPS = clinically undetermined parkinsonian syndrome; DAT = dopamine transporter; DIP = drug-induced parkinsonism; DLB = dementia with Lewy bodies; ET = essential tremor; FND = functional neurologic disorder; MND = motor neuron disease; MSA-P = multiple system atrophy—parkinsonian subtype; NPH = normal pressure hydrocephalus; PD = Parkinson disease; PSP-CBS = progressive supranuclear palsy—corticobasal syndrome; PSP-RS = progressive supranuclear palsy—Richardson syndrome; VP = vascular parkinsonism.
[a] One PD patient underwent DAT imaging without an available result.
[b] Dystonia (n = 1), AD combined with DLB (n = 1), hepatic encephalopathy (n = 1), unspecified myalgia (n = 1), and unspecified tauopathy (n = 1).

a significant p value of 0.041, but post hoc pairwise comparisons with adjustment for multiple testing (using Bonferroni) revealed no statistically significant differences between any pairs of groups, with the lowest adjusted p value being 0.238. Patients with altered diagnoses were diagnosed with PD at a median age 2.3 years older than those with stable diagnoses ($p = 0.038$; Table 2 and Figure 2). There were no significant differences in sex distribution between the 2 groups.

DAT Imaging

DAT imaging was conducted for 588 patients, accounting for 36.2% of the total cohort. Among these, 87.9% (n = 517) of examinations were classified as abnormal, 4.9% (n = 29) as borderline, and 7.0% (n = 41) as normal, with 1 scan result missing. Patients with revised PD diagnoses underwent DAT imaging more frequently (50.7%, n = 109 of 215) compared with those with stable PD diagnoses (34.0%, n = 480 of 1,411) ($p < 0.001$). In addition, normal DAT imaging findings were observed more frequently in patients with revised diagnoses compared with those with stable PD diagnoses (36.7%, n = 40 of 109 vs 0.2%, n = 1 of 480; $p < 0.001$) (Table 2, Figure 2). DAT imaging was typically performed a median of 13 days before the PD diagnosis (IQR = 129 days). There were no significant differences in the timing of DAT imaging between patients with altered diagnoses and those with unchanged PD diagnoses (Table 2, Figure 2).

Among patients with revised diagnoses who underwent DAT imaging, the timing of imaging relative to the PD diagnosis did not correlate with the delay in diagnosis. However, for patients who underwent DAT imaging after their initial PD diagnosis, there was a significant positive correlation between the timing of the DAT imaging and the timing of the revised diagnosis ($r = 0.70$, $p < 0.001$, n = 50) (Figure 2), suggesting that diagnostic revisions were closely linked to the results of the DAT imaging. Detailed DAT imaging results according to diagnostic categories are presented in eTable 1.

Mortality and Neuropathology

During the follow-up period, 833 patients (51.2%) died. The distribution of mortality rates across different diagnostic

Table 2 Comparison of Clinical Characteristics Between Patients With Stable and Revised Diagnoses Based on Clinical Evaluations, Chart Reviews, and Neuropathologic Examinations

Diagnosis	Variable	Stable diagnosis	Revised diagnosis	*p* Value
PD including DLB and monogenic PD	n, % of all patients	1,411, 86.8	215, 13.2	—
	Age at PD diagnosis, y	70.0 (15.6)	72.3 (13.9)	0.038
	Sex, male/female, % males	785/626, 55.6	125/90, 58.1	0.49
	DAT imaging performed, yes/no, % performed	480/931, 34.0	109/106, 50.7	<0.001
	DAT imaging result, a/b/n/u, % abnormal[a]	475/3/1/1, 99.0	43/26/40/0, 39.4	<0.001
	Timing of DAT imaging relative to Lewy body disease diagnosis, d[b]	−14 (78)	16 (368)	0.065
PD excluding DLB and monogenic PD	n, % of all patients	1,338, 82.3	288, 17.7	—
	Age at PD diagnosis, y	69.5 (15.5)	73.2 (13.0)	<0.001
	Sex, male/female, % males	743/595, 55.5	167/121, 58.0	0.45
	DAT imaging performed, yes/no, % performed	456/882, 34.1	133/155, 46.2	<0.001
	DAT imaging result, a/b/n/u, % abnormal[a]	453/2/0/1, 99.3	65/27/41/0, 48.9	<0.001
	Timing of DAT imaging relative to PD diagnosis, d[b]	−14 (86)	−6 (329)	0.22

Abbreviations: DAT = dopamine transporter; DLB = dementia with Lewy bodies; IQR = interquartile range; PD = Parkinson disease.
Values are presented as median (IQR) or n. *p* Values are from Mann-Whitney *U* tests or χ^2 tests.
[a] Abnormal/borderline/normal/unknown.
[b] Negative values indicate that DAT imaging was conducted before diagnosis date.

categories is summarized in Table 1. Postmortem neuropathologic examinations were conducted for 25 patients (3.0% of deceased patients), as outlined in Table 5. Of these, 16 patients had their antemortem PD diagnoses confirmed through neuropathology. Six patients whose diagnoses had been revised to MSA or PSP before death were confirmed to have these conditions on postmortem examination. In addition, 3 patients (12% of neuropathologically examined patients) had their antemortem PD diagnoses revised to alternative conditions, including MSA, Alzheimer disease, or unspecified tauopathy, based on neuropathologic findings.

Diagnostic Cross-Validation

For interrater reliability in the multicategory classification of combined PD and DLB vs 6 other diagnostic categories, the Kappa statistic between the 2 designated movement disorder specialists was 0.90 (95% CI 0.81–1.0), indicating an excellent level of agreement. Cross-tabulations of the individual ratings are presented in eTable 2. The greatest disagreement was observed between PD and DLB cases, which may be attributed to their overlap within the same disease spectrum. When PD and DLB were classified as separate diagnostic categories, the Kappa statistic decreased to 0.68 (95% CI 0.55–0.82), indicating substantial agreement.

Discussion

This study reveals that approximately 13.3% of PD diagnoses made by neurologists are revised over a median follow-up period of 10 years. If DLB is considered a separate diagnostic category from PD, the revision rate increases to 17.7%. The majority of these diagnostic changes occur within the first 2 years after the initial diagnosis. Commonly revised diagnoses include CUPS, VP, MSA, and PSP. Notably, 10.6% of patients had their diagnoses modified directly by treating clinicians based on clinical evaluations.

The observed pattern of diagnostic changes aligns with findings from clinicopathologic studies, where patients misdiagnosed with PD often receive final diagnoses of MSA, PSP, DLB, VP, ET, or DIP.[1] However, the prominence of undetermined parkinsonism in this study highlights a significant clinical issue: many patients present with mixed phenotypes that defy clear-cut diagnostic categorization. Clinicians outside clinicopathologic specialized settings frequently encounter these challenges, which are exacerbated by the overlapping pathologic features of various neurodegenerative diseases.[35] Furthermore, even in specialist clinics, a proportion of parkinsonian patients remain uncategorized despite thorough evaluations, reflecting the substantial clinical uncertainty inherent in PD diagnostics,[5] particularly in cases of tremulous parkinsonism.[36] Although recent frameworks have introduced alpha-synuclein-based definitions of PD, which may enhance biomarker-based diagnostics in the future,[3,4] the diagnosis of PD and other parkinsonian syndromes remains primarily clinical in most regions worldwide. This approach relies on the evaluation of signs and symptoms that change over time. These temporal changes in phenotypes necessitate regular, critical reassessment of diagnoses and

Table 3 Diagnoses Revised Based on Clinical Evaluations

Altered diagnosis	n	Age at PD diagnosis, y, median (IQR)	Sex, % males	DAT imaging				Timing of DAT imaging relative to PD diagnosis, d, median (IQR)[a]	Delay from PD diagnosis to altered diagnosis, mo, median (IQR)
				Performed, % (n)	Abnormal, % (n)	Borderline, % (n)	Normal, % (n)		
CUPS	37	67.5 (14.3)	54.1	67.6 (25)	28.0 (7)	20.0 (5)	52.0 (13)	48 (864)	29 (63)
PSP-RS	29	72.8 (11.0)	65.5	41.4 (12)	100 (12)	0	0	−15 (411)	19 (31)
VP	29	74.9 (10.9)	65.5	41.4 (12)	6.9 (2)	58.3 (7)	25.0 (3)	4 (376)	16 (57)
MSA-P	23	67.1 (22.4)	47.8	56.5 (13)	100 (13)	0	0	−14 (69)	33 (29)
DLB	19	74.7 (9.9)	63.2	47.4 (9)	88.9 (8)	11.1 (1)	0	17 (323)	12 (45)
ET	13	70.8 (6.8)	53.8	69.2 (9)	0	33.3 (3)	66.6 (6)	168 (1,162)	12 (46)
DIP	12	74.5 (8.8)	41.7	66.7 (8)	12.5 (1)	12.5 (1)	75.0 (6)	48 (210)	9 (29)
CBS	6	73.1 (7.9)	50.0	83.3 (5)	80.0 (4)	20.0 (1)	0	−14 (179)	19 (22)
Monogenic PD	4	41.5 (13.0)	25.0	100 (4)	100 (4)	0	0	−46 (—)	32 (—)
NPH	4	78.7 (9.9)	50.0	25.0 (1)	0	0	100 (1)	97 (—)	3 (3)
Depression	3	76.5 (—)	33.3	33.3 (1)	100 (1)	0	0	−161 (—)	10 (—)
AD	2	77.9 (—)	100	0	0	0	0	—	17 (—)
Other[b]	10	59.8 (17.7)	90.0	60.0 (6)	0	0	100 (6)	366 (1,887)	15 (39)
Total	191	72.3 (14.5)	58.1	55.0 (105)	49.5 (52)	17.1 (18)	33.3 (35)	17 (368)	22 (43)

Abbreviations: AD = Alzheimer disease; CBS = corticobasal syndrome; CUPS = clinically undetermined parkinsonian syndrome; DAT = dopamine transporter; DIP = drug-induced parkinsonism; DLB = dementia with Lewy bodies; ET = essential tremor; IQR = interquartile range; MSA-P = multiple system atrophy—parkinsonian subtype; NPH = normal pressure hydrocephalus; PD = Parkinson disease; PSP-RS = progressive supranuclear palsy—Richardson syndrome; VP = vascular parkinsonism.
[a] Negative values indicate that DAT imaging was conducted before PD diagnosis date.
[b] PSP-CBS (n = 2), functional parkinsonism (n = 2), motor neuron disease (n = 2), dystonia (n = 1), AD combined with DLB (n = 1), hepatic encephalopathy (n = 1), and unspecified myalgia (n = 1).

effective communication with newly diagnosed patients regarding the instability associated with PD diagnoses.

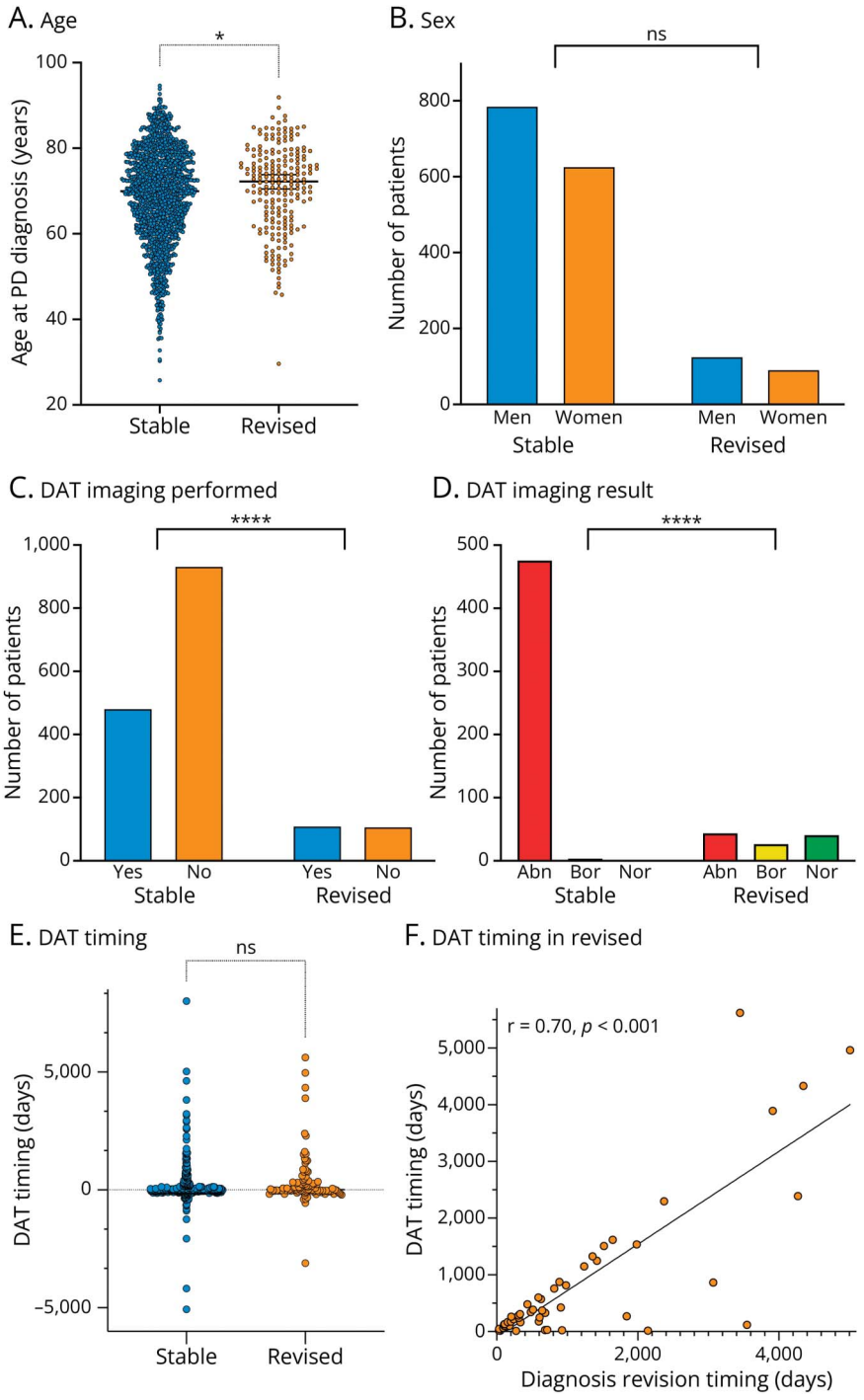
Another significant issue highlighted in this study is the overlap between PD and DLB, particularly in relation to the utility of the 1-year rule. In our secondary analysis, the application of this rule, which dictates the temporal sequence of cognitive and motor symptom onset, led to a notably higher identification of DLB cases by evaluators compared with the diagnoses made by clinicians. This discrepancy likely stems from the structured application of the 1-year rule. Although the rule is still endorsed as a pragmatic

Table 4 Diagnoses Modified Based on Chart Reviews

Altered diagnosis	n	Age at PD diagnosis, y, median (IQR)	Sex, % males	DAT imaging				Timing of DAT imaging relative to PD diagnosis, d, median (IQR)[a]
				Performed, %, n	Abnormal, %, n	Borderline, %, n	Normal, %, n	
DLB	50	78.0 (10.0)	58.0	22.0, 11	90.9, 10	0	9.1, 1	−54 (317)
CUPS	40	72.4 (12.8)	60.0	40.0, 16	18.8, 3	50.0, 8	31.2, 5	−22 (316)
ET	2	83.0 (—)	50.0	50.0, 1	0	100, 1	0	−28 (—)
AD	1	83.2 (—)	100	0	—	—	—	—
VP	1	84.3 (—)	0	0	—	—	—	—
Total	94	76.3 (10.8)	58.5	29.8, 28	46.4, 13	32.1, 9	21.4, 6	−28 (177)

Abbreviations: AD = Alzheimer disease; CUPS = clinically undetermined parkinsonian syndrome; DAT = dopamine transporter; DLB = dementia with Lewy bodies; ET = essential tremor; IQR = interquartile range; PD = Parkinson disease; VP = vascular parkinsonism.
[a] Negative values indicate that DAT imaging was conducted before PD diagnosis date.

Figure 2 Diagnostic Characteristics and DAT Imaging in Patients With Stable vs Altered Diagnoses



(A) Age distribution of patients with stable vs altered diagnoses. (B) Comparison of sex distribution between patients with stable and altered diagnoses. (C) Frequency of DAT imaging use in patients with stable vs altered diagnoses. (D) DAT imaging results by diagnostic category. (E) Timing of DAT imaging relative to the initial PD diagnosis across all patients. (F) Correlation between the timing of DAT imaging and the timing of revised diagnoses in patients who underwent DAT imaging after the initial PD diagnosis. DAT = dopamine transporter; PD = Parkinson disease.

tool for distinguishing between DLB and PD and it may serve as a potentially useful diagnostic threshold in clinical practice,[27] it can also be viewed as arbitrary. Although substantial group-level statistical differences have been documented between DLB and PD,[37] individual cases may present with few or no discernible differences, apart from potentially greater beta-amyloid burden in DLB or variations in the onset sequence of differential syndromes.[38] To address this ambiguity and the ongoing debate regarding the shared and distinct features of PD and DLB,[37] we conducted a subanalysis that separated the 2 diagnoses to evaluate syndrome-specific diagnostic accuracy.

Over the past 30 years, DAT imaging, with SPECT or PET, has been used as a method to enhance diagnostic accuracy for PD.[39,40] In our cohort, 36.2% of patients underwent DAT

Table 5 Diagnostic Outcomes Based on Neuropathologic Examinations

	Overall	PD confirmed postmortem	PD changed antemortem and changed diagnosis confirmed postmortem	PD changed postmortem
n	25	16	6	3
Age at PD diagnosis	66.1 (13.7)	67.5 (13.6)	57.9 (16.6)	71.0 (9.6)
Sex, % males	68.0	87.5	33.3	50.0
Neuropathologic diagnosis	—	PD	MSA (n = 4) PSP (n = 2)	MSA (n = 1) AD (n = 1) Tauopathy (n = 1)
DAT imaging performed, %	40.0	37.5	66.7	0
DAT imaging abnormal, %	100	100	100	na

Abbreviations: AD = Alzheimer disease; DAT = dopamine transporter; IQR = interquartile range; MSA = multiple system atrophy; PD = Parkinson disease; PSP = progressive supranuclear palsy.
Values are median (IQR), n or %.

imaging, indicating a high usage rate. The results indicate that patients with revised diagnoses underwent DAT imaging more frequently than those with stable PD diagnoses. This suggests 2 possibilities: first, DAT imaging may be prompted by atypical clinical features or progression in patients with altered diagnoses; second, DAT imaging may influence diagnostic decisions, reflecting its utility in differentiating parkinsonian syndromes, especially ET, VP, and DIP from PD. The correlation between the timing of DAT imaging and the date of diagnosis alteration supports the role of DAT imaging in influencing clinical decisions. Although DAT imaging can assist clinicians in making informed decisions and increase their confidence in revising diagnoses, its highly uneven global availability underscores the need for more accessible diagnostic methods.[41] In many cases of our study, clinical follow-up visits and high-dose levodopa tests provided sufficient information regarding the stability of PD diagnosis, often rendering additional DAT imaging unnecessary. DAT scans are not typically necessary for the standard clinical diagnosis of PD unless there are unusual clinical features that warrant further investigation. Ultimately, clinicians must weigh the risks and benefits to determine the best diagnostic method for each patient, recognizing that imaging, follow-up, and pharmacologic trials can be complementary rather than mutually exclusive. We also acknowledge the promising potential of biomarkers in aiding PD diagnosis, such as the alpha-synuclein seed amplification assay (SAA), although it remains in the developmental stage and has limitations. Although SAA may not ultimately be the ideal biomarker, we believe that a cost-effective, globally accessible diagnostic biomarker could improve diagnostic accuracy, especially in regions with limited access to neurologists and movement disorder specialists.

Postmortem confirmation of diagnoses was achieved in only 3% of deceased patients, reflecting a significant discrepancy between clinical practice and research settings. Low autopsy rates have also been documented in other clinical studies of parkinsonism, with a significant issue being the overall decline in autopsy rates across various cohorts.[12,14,42,43] Clinicopathologic studies, which often rely on brain bank materials, may be biased toward more severe or atypical cases due to low autopsy rates and selective patient sampling.[44,45] Routine clinical practice faces similar challenges because patients undergoing autopsy are often those with clinical uncertainty, potentially skewing diagnostic accuracy estimates.[12,46] To obtain a more representative understanding of diagnostic accuracy, large-scale, systematic, and consecutive neuropathologic examinations of community-dwelling patients with suspected PD are critically needed, complementing studies like those conducted in the Arizona Study of Aging and Neurodegenerative Disorders.[6,7,47]

Regarding diagnoses by movement disorder specialists, it is important to acknowledge that movement disorder neurology is not universally recognized as an official subspecialty of neurology, and the number of movement disorder specialists relative to the global PD population is low. In the United States, it has been estimated that 40% of patients with PD do not see a neurologist, and only 9% of patients visit a movement disorder neurologist during a calendar year.[48,49] In Europe and North Africa, subspecialty training for movement disorders exists only in a minority of countries.[23] Consequently, only a small percentage of PD diagnoses are conducted by specialists on a global scale, and the majority of patients with PD receive initial diagnoses from nonspecialists, either with or without adherence to diagnostic criteria, and without eventual neuropathologic confirmation. However, although the title of "movement disorder specialist" or a postgraduate qualification in movement disorders may suggest expertise, it does not automatically guarantee superior diagnostic accuracy compared with a competent general neurologist. Experienced clinicians who regularly see patients can be more accurate in diagnosing PD than specialists who may have less frequent clinical exposure and whose expertise may be more focused on research rather than routine clinical practice.

This study's cohort was predominantly composed of White individuals from Finland, which may limit the generalizability of our findings to more ethnically diverse populations. In addition, regional variations in diagnostic procedures and practices, while consistent with advancements in PD diagnostics, could have influenced the results. For patients still alive at the end of the follow-up period, diagnostic changes may have continued beyond the study's observation timeframe, which could influence the stability of the diagnoses observed. The study's focus on hospital-based patients excludes those from nursing homes or private practices, potentially impacting the representativeness of the sample. As a retrospective analysis, the study relied on patient records that could vary in completeness and accuracy, potentially influencing the robustness of the findings.

Strengths of the study include its large, unselected cohort of patients, which contrasts with the often selective recruitment seen in clinical trials or clinicopathologic studies. In addition, the study used a systematic approach to data collection, using consistent methodology and criteria throughout, which helps mitigate the subjective variability seen in studies with multiple evaluators.

The observed rate of diagnostic revisions (13.3%–17.7%, depending on the diagnostic categorization of PD and DLB) and the significant proportion of clinically indeterminate cases underscore the persistent challenges in achieving improved PD diagnostic accuracy. Notably, had the study been restricted to early-stage cases, the rate of diagnostic alterations would likely have been even higher. Therefore, despite recent advancements in clinical criteria and diagnostic technologies, variability in diagnoses continues to be a significant issue. This emphasizes the urgent need for ongoing refinement of diagnostic processes, enhanced clinical training for neurologists, more frequent use of postmortem diagnostic confirmation, and the development of widely accessible, cost-effective biomarkers.

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Author Contributions

V. Räty: drafting/revision of the manuscript for content, including medical writing for content; analysis or interpretation of data. T. Kuusimäki: drafting/revision of the manuscript for content, including medical writing for content; analysis or interpretation of data. J. Majuri: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. T. Vahlberg: analysis or interpretation of data. M. Gardberg: major role in the acquisition of data. T. Noponen: major role in the acquisition of data. M. Seppänen: major role in the acquisition of data. A.-M. Tolppanen: analysis or interpretation of data. V. Kaasinen: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data.

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