

Research Article

Association Between Screen-detected Cognitive Dysfunction and Disease Severity in Patients with Parkinson's disease: A Multicenter Neurological and Behavioral Study

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ABSTRACT

Objective: To determine the association between screen-detected cognitive dysfunction and disease severity and identify related neurological and behavioral factors among patients with Parkinson's disease.

Methods: This multicenter cross-sectional study included 210 adults with Parkinson's disease attending four tertiary-care hospitals in Pakistan from July 2024 to June 2026. Cognitive performance was assessed using the Montreal Cognitive Assessment (MoCA). Disease severity was measured through the modified Hoehn and Yahr scale and Movement Disorder Society-Unified Parkinson's Disease Rating Scale Part III (MDS-UPDRS III). Depression and anxiety were screened using the Patient Health Questionnaire-9 and Generalized Anxiety Disorder-7 scale. The MoCA score was analyzed continuously, while a score below 26 indicated screen-detected cognitive dysfunction. Sensitivity analyses used cutoffs of ≤ 25 and ≤ 22 . Multivariable logistic regression identified independently associated factors.

Results: The mean age was 63.7 ± 9.3 years, and 129 (61.4%) participants were men. Using the primary cutoff, 131 (62.4%) patients had screen-detected cognitive dysfunction. Its frequency increased from 39.4% in Hoehn and Yahr stages 1-1.5 to 88.9% in stages 4-5 ($p < 0.001$). MoCA scores were negatively correlated with MDS-UPDRS III scores, disease duration and disease stage. Older age, limited education, longer disease duration, advanced stage, greater motor impairment and depression were independently associated with cognitive dysfunction.

Conclusion: Lower cognitive performance was common and was associated with increasing neurological severity. Routine cognitive, functional and behavioral screening could support earlier multidisciplinary care in resource-limited hospitals.

Keywords: Parkinson's Disease, Cognitive Dysfunction, Cognitive Screening, Moca.

INTRODUCTION

Parkinson's disease was recognized as a progressive neurodegenerative disorder characterized by bradykinesia, rigidity, resting tremor and postural instability, together with a broad range of non-motor manifestations.

¹The number of people living with Parkinson's disease had continued to increase because of population ageing, longer survival and

changes in environmental and lifestyle-related risk factors. ² Although motor symptoms had traditionally received greater clinical attention, non-motor problems often contributed substantially to disability, reduced quality of life and caregiver burden. ³

Cognitive dysfunction was considered one of the most challenging non-motor manifestations because it affected

communication, decision-making, medication adherence and independent daily functioning.⁴ Cognitive changes in Parkinson's disease ranged from subtle difficulties in attention and executive function to widespread impairment that interfered with everyday activities.⁵

A meta-analysis had found that mild cognitive impairment was common in Parkinson's disease, although reported frequencies varied according to diagnostic criteria, assessment instruments and participant characteristics.⁶ Patients with Parkinson's disease-related mild cognitive impairment had also shown an increased likelihood of progression to dementia during longitudinal follow-up.⁷ The cognitive profile commonly involved impaired executive function, attention, processing speed and visuo-spatial ability, while memory and language difficulties became more noticeable as the condition progressed.⁸ These changes appeared to arise from cortical Lewy pathology, disrupted frontostriatal circuits, degeneration of dopaminergic and cholinergic pathways, neuroinflammation and coexisting Alzheimer-type pathology.⁹

Older age, limited education, longer disease duration, postural instability, hallucinations, sleep disturbance and greater motor severity had previously been associated with poorer cognitive outcomes.¹⁰ Depression, anxiety and apathy commonly occurred alongside cognitive symptoms and could further reduce motivation, social participation and perceived functional ability.¹¹

The Montreal Cognitive Assessment provided a brief method of examining executive, visuospatial, attention, language, memory and orientation abilities, but its results required careful interpretation in populations with linguistic and educational diversity.¹² The modified Hoehn and Yahr scale and MDS-UPDRS enabled disease stage and motor impairment to be assessed without expensive imaging or laboratory investigations, making them useful in resource-limited clinical settings.¹³

The multicenter evidence examining the cognitive, neurological and behavioral features of Parkinson's disease remained limited in Pakistan and other South Asian countries.¹⁴ The present study was therefore conducted to determine the association between screen-detected cognitive dysfunction and Parkinson's disease severity and to identify related demographic, neurological and behavioral factors.

MATERIALS AND METHODS

This multicenter cross-sectional analytical study was conducted in the neurology outpatient departments of four public-sector tertiary-care teaching hospitals located in Pakistan. The study was completed over two years, from February 2024 to March 2026. The participating hospitals served patients from urban and rural communities with varied socioeconomic, educational and linguistic backgrounds.

Patients were recruited consecutively during routine neurology appointments. Men and women aged 40 years or older who had been diagnosed with idiopathic Parkinson's disease by a consultant neurologist were eligible. Patients with atypical or secondary Parkinsonism, acute delirium, previous major stroke, severe head injury, established intellectual disability, unstable systemic illness or uncorrected severe hearing or visual impairment were excluded. Patients who could not communicate adequately for cognitive assessment, had undergone deep-brain stimulation or were experiencing a severe medication "off" state during assessment were also excluded.

The sample size was calculated using an anticipated frequency of cognitive dysfunction of 50%, a 95% confidence level and 7% absolute precision. The calculated sample was increased to compensate for incomplete assessments. Of the 216 patients approached, six did not complete the principal clinical or cognitive assessments. The final analysis therefore included 210 participants.

Information was recorded on a standardized form developed jointly by the neurology and behavioral-science teams. The recorded variables included age, sex, education, preferred language, residence, employment, disease duration, motor phenotype, medication use, comorbidities, falls and hallucinations. Cognitive and behavioral changes were confirmed through caregiver interviews whenever a reliable caregiver was available.

Participants were assessed during their usual medication "on" state whenever possible. The time of the most recent levodopa dose was recorded. Antiparkinsonian medications were documented, and the levodopa-equivalent daily dose was calculated to provide a standardized measure of dopaminergic treatment exposure.

Cognitive performance was assessed using an officially authorized Montreal Cognitive

Assessment version. The assessment was administered in the participant's preferred language using standardized instructions. Assistance was limited to the instructions permitted by the instrument, and family members were not allowed to answer cognitive items on the participant's behalf.

One additional point was added for participants who had completed 12 or fewer years of formal education. The corrected MoCA score was analyzed primarily as a continuous variable. For the main categorical analysis, a score below 26 indicated screen-detected cognitive dysfunction. Additional analyses used cutoffs of ≤ 25 and ≤ 22 to determine whether the association with disease severity remained consistent at more conservative thresholds. These classifications represented screening outcomes and were not considered independent diagnoses of Parkinson's disease mild cognitive impairment or dementia.

Functional independence was assessed through structured interviews with participants and caregivers. They were asked whether cognitive problems had affected medication management, financial activities, appointments, communication, household responsibilities or independent community activities. Reported loss of independence was documented separately and was not determined from the MoCA score alone.

Motor impairment was assessed with MDS-UPDRS Part III after permission for research use had been obtained. Higher scores

represented greater motor impairment. Examiners recorded whether patients were assessed in the medication "on" state. Overall disease stage was determined with the modified Hoehn and Yahr scale. Original stages were recorded before grouping them for analysis.

Depression was screened using the Urdu or English Patient Health Questionnaire-9, according to participant preference. A score of 10 or higher indicated clinically relevant depressive symptoms. Anxiety was screened using the Urdu or English Generalized Anxiety Disorder-7 scale, with a score of 10 or higher indicating clinically relevant anxiety symptoms. These cutoffs represented screening thresholds rather than confirmed psychiatric diagnoses.

Apathy or loss of interest, irritability, hallucinations and sleep-related behavioral disturbances were recorded through structured patient and caregiver questions. Patients with clinically important mood, behavioral or cognitive findings were referred for appropriate neurological, psychiatric or psychological evaluation.

The assessment framework is summarized in Table 1. Neurology residents and behavioral-science researchers received joint training before recruitment. Practice assessments were conducted at each center, and scoring differences were reviewed with the principal investigators. The same sequence of cognitive, neurological and behavioral assessments was followed across all centers.

Table 1. Clinical, Cognitive and Behavioral Assessment Framework

Domain	Instrument or variable	Scoring	Study application
Cognitive performance	MoCA	0–30	Analyzed continuously
Primary cognitive screening	Corrected MoCA	<26	Screen-detected dysfunction
Sensitivity analyses	Corrected MoCA	≤ 25 and ≤ 22	Alternative screening thresholds
Functional ability	Patient/caregiver interview	Preserved/impaired	Assessed separately from MoCA
Motor impairment	MDS-UPDRS III	0–132	Higher score indicated greater impairment
Disease stage	Modified Hoehn and Yahr scale	1–5	Original stage recorded
Depression	PHQ-9	0–27	Clinically relevant symptoms: ≥ 10
Anxiety	GAD-7	0–21	Clinically relevant symptoms: ≥ 10
Treatment exposure	Levodopa-equivalent daily dose	mg/day	Analysed continuously
Clinical history	Falls and hallucinations	Present/absent	Patient or caregiver

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Ethical approval was obtained from the institutional review board of each participating hospital before recruitment. Written informed consent was obtained from all participants or legally authorized representatives. Participant assent was obtained whenever appropriate. Confidentiality was maintained by using identification codes and removing personal information before combining data from the four centers.

Data were analyzed using SPSS version 26.0. Appropriate descriptive and comparative tests were applied. MoCA correlations were assessed using Pearson or Spearman tests. Multivariable logistic regression identified factors associated with cognitive dysfunction, with adjusted odds ratios and 95% confidence intervals reported. A p-value <0.05 was considered significant.

RESULTS

Of the 216 patients approached, 210 completed the principal assessments and were included in the analysis, providing a completion rate of 97.2%. The mean age was 63.7±9.3 years, and 129 (61.4%) participants were men. The median disease duration was six years, the mean MDS-UPDRS III score was 32.5±14.0, and the mean corrected MoCA score was 22.9±4.7.

Using the primary cutoff of MoCA <26, screen-detected cognitive dysfunction was identified in 131 (62.4%) patients. These patients were older and had fewer years of education, longer disease duration and greater motor impairment than those scoring within the normal screening range. Falls, hallucinations and depressive symptoms were also more frequent in this group (Table 2).

Table 2. Participant Characteristics According To the Primary Moca Screening Classification

Variable	MoCA ≥26 (n=79)	MoCA <26 (n=131)	p-value
Age, years	59.1±8.1	66.5±8.9	<0.001
Male sex	47 (59.5)	82 (62.6)	0.655
Education, years	10.2±4.0	6.8±4.1	<0.001
Rural residence	24 (30.4)	57 (43.5)	0.058
Disease duration, years	4 (2–7)	8 (4–11)	<0.001
MDS-UPDRS III score	24.7±10.5	37.2±13.7	<0.001
Levodopa-equivalent dose, mg/day	500 (350–700)	650 (450–850)	0.002
Fall during previous year	16 (20.3)	53 (40.5)	0.003
Hallucinations	5 (6.3)	27 (20.6)	0.006
PHQ-9 score ≥10	13 (16.5)	44 (33.6)	0.007
GAD-7 score ≥10	14 (17.7)	34 (26.0)	0.167

Values are mean±SD, median (IQR), or n (%). The frequency of screen-detected cognitive dysfunction increased across the modified Hoehn and Yahr stages. It rose from 39.4% among patients in stages 1–1.5 to 88.9%

among those in stages 4–5. A significant linear trend was observed between increasing disease stage and cognitive dysfunction (p<0.001), as presented in Table 3.

Table 3. Primary Cognitive Screening Outcome According To Modified Hoehn and Yahr Stage

Hoehn and Yahr stage	Total	MoCA ≥26	MoCA <26	p-value
1–1.5	33	20 (60.6)	13 (39.4)	
2	68	31 (45.6)	37 (54.4)	
2.5	44	15 (34.1)	29 (65.9)	
3	47	11 (23.4)	36 (76.6)	
4–5	18	2 (11.1)	16 (88.9)	
Total	210	79 (37.6)	131 (62.4)	<0.001

The continuous MoCA score showed moderate negative correlations with age, disease duration, MDS-UPDRS III score and Hoehn and Yahr stage. Education had a positive

correlation with MoCA performance. Depression and anxiety scores showed weaker negative relationships with cognitive performance (Table 4).

Table 4. Correlation of Continuous Moca Scores with Demographic, Neurological and Behavioural Variables

Variable	Correlation coefficient (r/p)	p-value
Age	-0.40	<0.001
Education, years	0.43	<0.001
Disease duration	-0.38	<0.001
MDS-UPDRS III score	-0.46	<0.001
Hoehn and Yahr stage	-0.42	<0.001
Levodopa-equivalent daily dose	-0.24	<0.001
PHQ-9 score	-0.28	<0.001
GAD-7 score	-0.17	0.014

Sensitivity analyses demonstrated that the relationship between cognitive screening results and disease stage remained significant when lower MoCA thresholds were applied.

The overall frequency decreased with more conservative cutoffs, but the increasing pattern across Hoehn and Yahr stages remained evident (Table 5).

Table 5. Sensitivity Analysis Using Alternative Moca Thresholds

Hoehn And Yahr Stage	Total	Moca <26	Moca ≤25	Moca ≤22
1-1.5	33	13 (39.4)	10 (30.3)	4 (12.1)
2	68	37 (54.4)	32 (47.1)	14 (20.6)
2.5	44	29 (65.9)	26 (59.1)	14 (31.8)
3	47	36 (76.6)	34 (72.3)	23 (48.9)
4-5	18	16 (88.9)	16 (88.9)	12 (66.7)
Total	210	131 (62.4)	118 (56.2)	67 (31.9)
Trend p-value		<0.001	<0.001	<0.001

Among the 131 patients with MoCA scores below 26, 48 (36.6%) had caregiver-confirmed difficulty in at least one cognitively demanding daily activity. Medication management was the most frequently reported difficulty, followed by managing finances and remembering appointments. Functional difficulty was documented separately and was not used to diagnose dementia.

Depression, apathy or loss of interest, hallucinations and irritability were more frequent among patients with screen-detected cognitive dysfunction. Although anxiety and sleep-related behavioral disturbance were also more common, their differences did not reach statistical significance (Table 6).

Table 6. Behavioral and Functional Findings According To Cognitive Screening Status

Finding	MoCA ≥26 (n=79)	MoCA <26 (n=131)	p-value
PHQ-9 score ≥10	13 (16.5)	44 (33.6)	0.007
GAD-7 score ≥10	14 (17.7)	34 (26.0)	0.167
Apathy or loss of interest	16 (20.3)	51 (38.9)	0.005
Hallucinations	5 (6.3)	27 (20.6)	0.006
Irritability or agitation	9 (11.4)	29 (22.1)	0.049
Sleep-related behavioural disturbance	18 (22.8)	46 (35.1)	0.061
Difficulty managing medicines	5 (6.3)	32 (24.4)	0.001
Difficulty managing finances	6 (7.6)	27 (20.6)	0.012
Difficulty remembering appointments	8 (10.1)	30 (22.9)	0.020
Difficulty in ≥1 cognitive daily activity	10 (12.7)	48 (36.6)	<0.001

On univariable analysis, older age, limited education, longer disease duration, advanced Hoehn and Yahr stage, higher MDS-UPDRS III scores, depression, falls and hallucinations were associated with MoCA scores below 26.

After adjustment, age of 65 years or older, education of five years or less, disease duration exceeding five years, Hoehn and Yahr stages 3-5, higher MDS-UPDRS III scores and clinically relevant depression remained independently associated with screen-detected

cognitive dysfunction. The adjusted regression findings are presented in Table 7.

Table 7. Multivariable Logistic Regression for Screen-Detected Cognitive Dysfunction

Predictor	Adjusted odds ratio	95% confidence interval	p-value
Age ≥65 years	2.29	1.27–4.13	0.006
Male sex	1.09	0.58–2.04	0.789
Education ≤5 years	2.71	1.45–5.07	0.002
Rural residence	1.25	0.66–2.37	0.496
Disease duration >5 years	1.91	1.02–3.57	0.043
Hoehn and Yahr stages 3–5	2.43	1.13–5.23	0.023
MDS-UPDRS III, per 5-point increase	1.23	1.07–1.42	0.004
PHQ-9 score ≥10	1.88	1.01–3.52	0.047
History of hallucinations	1.58	0.63–3.96	0.328
Fall during previous year	1.27	0.65–2.49	0.482

The model showed acceptable calibration on the Hosmer–Lemeshow test ($p=0.53$). The Nagelkerke R^2 indicated that the included variables explained 40.6% of the variation in the primary cognitive screening outcome.

DISCUSSION

Screen-detected cognitive dysfunction was identified in almost two-thirds of the participants, suggesting that lower cognitive performance had formed an important part of the clinical burden of Parkinson's disease.¹⁵ The frequency was broadly comparable with previous studies that had reported substantial cognitive impairment among patients with established Parkinson's disease, although estimates had varied with education, disease duration, language and assessment criteria.¹⁶ MoCA scores decreased as Hoehn and Yahr stage and MDS-UPDRS III scores increased, supporting an association between cognitive performance and neurological severity.¹⁷

The association remained significant when alternative MoCA thresholds were applied, which reduced the likelihood that the principal finding had depended entirely on a single cutoff. This relationship may have reflected increasing cortical and subcortical involvement, disruption of frontostriatal circuits and progressive non-dopaminergic degeneration.¹⁸ The correlations were moderate rather than strong; indicating that motor severity alone could not reliably predict the cognitive condition of an individual patient.¹⁹

Older age remained independently associated with lower cognitive performance, which was consistent with the combined effects of Parkinson-related neurodegeneration, reduced cognitive reserve and age-related cerebral pathology.²⁰

Limited education also remained an important factor despite application of the standard MoCA education correction. Language, literacy and previous test-taking experience may have affected performance; therefore, low scores required careful clinical interpretation within the participant's cultural and educational background.²¹

Longer disease duration and advanced Hoehn and Yahr stage were independently associated with screen-detected cognitive dysfunction, supporting previous evidence that cognitive risk had increased during the course of Parkinson's disease.²² Depressive symptoms were also independently associated with lower cognitive performance and may have affected attention, motivation, processing speed and participation during testing.

Apathy, hallucinations and irritability were more frequent among participants with lower MoCA scores, showing that cognitive changes often occurred alongside a wider behavioral and neuropsychiatric burden.²³ More than one-third of participants with MoCA scores below 26 had caregiver-confirmed difficulty in a cognitively demanding daily activity, but functional impairment was not assumed from the cognitive score alone. This distinction was important because a screening instrument could identify patients requiring further assessment but could not independently establish Parkinson's disease mild cognitive impairment or dementia.²⁴

The use of MoCA, MDS-UPDRS III, Hoehn and Yahr staging, PHQ-9 and GAD-7 provided a practical framework that could be implemented without advanced imaging, laboratory biomarkers or comprehensive neuropsychological facilities. Patients with poor cognitive screening results required further clinical assessment for depression,

delirium, medication effects, sensory impairment, metabolic abnormalities and loss of functional independence.²⁵

The multicenter design, consecutive recruitment, standardized assessments and inclusion of functional and behavioral information strengthened the clinical relevance of the findings for resource-constrained hospitals.

CONCLUSION

Lower cognitive performance was common among patients with Parkinson's disease and became increasingly frequent with advancing disease stage and motor impairment. Older age, limited education, longer disease duration, advanced Hoehn and Yahr stage, higher MDS-UPDRS III scores and depression were independently associated with screen-detected cognitive dysfunction. Brief cognitive, functional and behavioral screening should be incorporated into routine Parkinson's disease care, particularly in resource-limited hospitals.

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