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HEALTH AND REHABILITATION

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PREVALENCE AND CORRELATES OF AUTONOMIC DYSFUNCTION AMONG PATIENTS OF PARKINSONS DISEASE

Original Research

Shaher Bano[1]*, Husnain Nawaz Malik[2], Tayba Zain1, Asif Hashmat[3], Ayesha Zubair[1], Maham Syed[1]
[1]Trainee Neurology, Pakistan Emirates Military Hospital Rawalpindi, Pakistan.
[2]Pakistan Emirates Military Hospital Rawalpindi, Pakistan.
[3]Professor Neurology, Pakistan Emirates Military Hospital Rawalpindi, Pakistan.
Corresponding Author: Shaher Bano, Trainee Neurology, Pakistan Emirates Military Hospital Rawalpindi, Pakistan, shaherbanos95@gmail.com
Acknowledgement: The authors thank the neurology department staff for their valuable support throughout the study.

Conflict of Interest: None
Grant Support & Financial Support: None

ABSTRACT

Background: Autonomic dysfunction is a frequently overlooked non-motor manifestation of Parkinson's disease (PD), contributing significantly to morbidity and decline in quality of life. It encompasses a range of symptoms affecting various organ systems and is more likely to develop as the disease progresses. Despite its clinical relevance, autonomic dysfunction remains underdiagnosed, leading to challenges in patient care and functional independence.

Objective: This study aimed to determine the prevalence of autonomic dysfunction in PD patients and explore its association with patient demographics, disease severity, and pharmacological treatments.

Methods: A cross-sectional study was conducted over six months at a tertiary care neurology department, enrolling 130 PD patients diagnosed using the United Kingdom Parkinson's Disease Society Brain Bank Criteria. Autonomic function was evaluated using the Scales for Outcomes in Parkinson's Disease–Autonomic (SCOPA-AUT), covering cardiovascular, gastrointestinal, urogenital, sudomotor, and pupillomotor domains. Disease severity was assessed with the Hoehn & Yahr scale. Statistical analysis was performed using SPSS version 22. Descriptive statistics, chi-square tests, independent t-tests, and multivariate logistic regression were applied to examine the prevalence and predictors of autonomic dysfunction, considering age, disease duration, medication use, and clinical staging.

Results: Out of 130 participants, 63.1% exhibited autonomic dysfunction. Cardiovascular dysfunction was most prevalent (60.0%), followed by gastrointestinal (49.2%), urogenital (42.3%), sudomotor (36.2%), and pupillomotor (23.1%) symptoms. Patients with autonomic dysfunction were older (mean age 68.4 vs. 62.7 years; $p = 0.002$) and had longer disease duration (8.6 vs. 5.3 years; $p = 0.001$). Advanced Hoehn & Yahr stages (III–V) were significantly associated with autonomic symptoms (79.3% vs. 37.5%; $p < 0.001$). Levodopa dose and dopamine agonist use were also significant predictors ($p = 0.003$ and $p = 0.02$, respectively).

Conclusion: Autonomic dysfunction is highly prevalent in PD, particularly in older patients, those with prolonged disease duration, and those receiving higher doses of dopaminergic therapy. Early detection and personalized treatment strategies are essential for improving patient outcomes and quality of life.

Keywords: Autonomic Nervous System Diseases, Dopaminergic Agents, Levodopa, Parkinson Disease, Postural Hypotension, Quality of Life, SCOPA-AUT.

INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative disorder primarily characterized by motor dysfunction, including tremors, bradykinesia, rigidity, and postural instability (1,2). While motor impairments remain the hallmark of diagnosis, non-motor symptoms have gained increasing recognition for their significant contribution to disease burden, with autonomic dysfunction emerging as one of the most prevalent and disabling features (3). The autonomic nervous system (ANS), which regulates involuntary physiological processes, becomes impaired in a considerable proportion of PD patients, leading to a wide spectrum of complications that substantially deteriorate quality of life (4,5). Patients with PD commonly experience various manifestations of autonomic dysfunction, such as orthostatic hypotension, constipation, urinary difficulties, sexual dysfunction, and abnormal sweating (6,7). These symptoms often coexist and exacerbate the overall disease complexity, making clinical management more challenging and undermining patients' ability to maintain functional independence. Despite reports suggesting that 50–70% of individuals with PD develop some degree of autonomic impairment, the pathophysiological mechanisms behind this dysfunction remain inadequately understood. Evidence points toward the involvement of degenerative changes in both central and peripheral autonomic pathways, including the brainstem and sympathetic ganglia, as key contributors to this phenomenon (8). Several factors appear to influence the onset and severity of autonomic dysfunction in PD, including disease duration, the extent of motor symptomatology, and comorbid conditions such as diabetes mellitus, cardiovascular disease, and depression (9). Moreover, dopaminergic therapies—particularly levodopa, which remains the cornerstone of PD treatment—have been associated with autonomic side effects, though the underlying mechanisms have yet to be fully elucidated. Notably, early presentation of autonomic symptoms in PD has been linked to an increased risk of cognitive decline and the subsequent development of dementia (10), emphasizing the broader clinical implications of this non-motor domain.

Despite the well-documented prevalence of autonomic dysfunction in PD, significant gaps remain in the literature. Little is known about the predictive factors of autonomic involvement, its trajectory over the course of the disease, or its specific impact on quality of life and treatment outcomes. Additionally, the relationship between autonomic dysfunction and other disease-modifying elements, such as medication use, motor severity, and cognitive status, has not been comprehensively explored. These gaps hinder the development of targeted strategies aimed at improving patient outcomes. In light of these observations, the present study aims to investigate the prevalence of autonomic dysfunction among PD patients and to examine its associations with patient demographics, disease progression, and therapeutic interventions, with the broader objective of identifying predictive factors that may guide early diagnosis and personalized management approaches.

METHODS

This cross-sectional study aimed to investigate the prevalence and determinants of autonomic dysfunction among individuals with Parkinson's disease (PD). A total of 130 participants were recruited from the neurology outpatient department of Pakistan Emirates Military Hospital, Rawalpindi. All participants fulfilled the United Kingdom Parkinson's Disease Society Brain Bank Criteria for idiopathic PD. Patients with secondary parkinsonism or atypical parkinsonian syndromes were excluded. Additionally, individuals with comorbidities known to independently contribute to autonomic dysfunction—including diabetes mellitus, chronic kidney disease, and peripheral neuropathy—were excluded to reduce confounding influences (3,4). Following written informed consent, each participant underwent detailed clinical evaluation, including documentation of demographic variables (age and sex), disease-specific characteristics (duration of PD, Hoehn and Yahr staging), and current treatment regimens (levodopa dosage, dopamine agonist use, monotherapy versus combination therapy). Autonomic dysfunction was assessed using the Scales for Outcomes in Parkinson's Disease–Autonomic (SCOPA-AUT), a validated instrument that evaluates symptom severity across five autonomic domains: cardiovascular, gastrointestinal, urogenital, sudomotor, and pupillomotor functions. Cardiovascular autonomic dysfunction was further evaluated through standardized orthostatic blood pressure testing. Blood pressure measurements were obtained after participants rested supine for five minutes and then again three minutes after assuming a standing position, following recommended clinical protocols for assessing orthostatic hypotension (11).

Data analysis was conducted using SPSS version 22.0. Descriptive statistics, including frequencies and percentages, were used to present the distribution of autonomic symptoms across the sample. Chi-square tests and independent sample t-tests were utilized to examine

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associations between autonomic dysfunction and categorical or continuous demographic and clinical variables. Comparison of autonomic dysfunction between early-stage (Hoehn and Yahr stages I–II) and advanced-stage (stages III–V) PD patients was carried out using chi-square tests and the Mann–Whitney U test. For evaluating treatment-related effects, independent sample t-tests were employed for comparisons involving two groups (e.g., monotherapy vs. combination therapy), while ANOVA was used where more than two groups or dosage strata were considered. To identify independent predictors of autonomic dysfunction, multivariate logistic regression analysis was performed using age, disease duration, Hoehn and Yahr staging, and levodopa dosage as covariates. A p-value ≤ 0.05 was considered statistically significant across all analyses. The study protocol received ethical approval from the Institutional Review Board of Pakistan Emirates Military Hospital, Rawalpindi. All participants provided written informed consent prior to enrollment, and the study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

The study revealed that autonomic dysfunction was present in 63.1% of patients with Parkinson's disease. Among the affected individuals, cardiovascular symptoms were the most frequently reported (60.0%), followed by gastrointestinal (49.2%), urogenital (42.3%), sudomotor (36.2%), and pupillomotor dysfunctions (23.1%). This high prevalence reflects the broad systemic involvement of autonomic domains in PD, reinforcing the substantial impact of these non-motor symptoms on patient well-being. Age and disease duration showed significant associations with autonomic dysfunction. The mean age of affected individuals was 68.4 ± 8.1 years compared to 62.7 ± 7.3 years in those without autonomic symptoms ($p = 0.002$). Similarly, patients with autonomic dysfunction had a longer average disease duration of 8.6 ± 4.2 years versus 5.3 ± 3.7 years in unaffected individuals ($p = 0.001$). Autonomic symptoms were significantly more prevalent in patients at Hoehn and Yahr stages III–V (79.3%) compared to those at stages I–II (37.5%) ($p < 0.001$), indicating that disease severity is a strong determinant of autonomic involvement. No significant sex-based differences were observed ($p = 0.45$), suggesting that gender does not significantly influence autonomic symptom burden in this population. Regarding medication effects, patients experiencing autonomic dysfunction had a significantly higher daily levodopa dose (540 ± 110 mg) compared to those without symptoms (460 ± 95 mg) ($p = 0.003$). Dopamine agonist use was also more frequent in the symptomatic group (58.5%) than the asymptomatic group (39.6%) ($p = 0.02$). However, the comparison between monotherapy and polytherapy did not reveal a statistically significant difference in autonomic dysfunction prevalence ($p = 0.08$), suggesting that dosage rather than therapy type contributes more directly to symptom development.

Patients at advanced stages of PD (Hoehn and Yahr III–V) exhibited significantly higher autonomic symptom scores across all measured domains. Cardiovascular symptoms averaged 7.1 ± 3.4 compared to 4.2 ± 2.3 in early-stage patients ($p < 0.001$), while gastrointestinal (6.5 ± 2.7 vs. 3.8 ± 1.9, $p < 0.001$), urogenital (5.3 ± 2.4 vs. 2.6 ± 1.8, $p < 0.001$), and sudomotor scores (4.7 ± 2.1 vs. 2.1 ± 1.2, $p = 0.002$) were also significantly elevated in the advanced group. These findings affirm that autonomic dysfunction severity escalates in tandem with disease progression. Multivariate logistic regression identified several independent predictors of autonomic dysfunction. Each additional year of age was associated with an 8% increased risk (OR: 1.08, 95% CI: 1.03–1.14, $p = 0.004$), and each additional year of disease duration raised the odds by 21% (OR: 1.21, 95% CI: 1.11–1.31, $p < 0.001$). For every 100 mg increase in levodopa dose, the risk increased by 19% (OR: 1.19, 95% CI: 1.07–1.33, $p = 0.002$). Most notably, patients in Hoehn and Yahr stages III–V had more than a threefold increased risk of developing autonomic dysfunction (OR: 3.45, 95% CI: 1.89–6.31, $p < 0.001$). Domain-specific analysis revealed distinct patterns of autonomic dysfunction in relation to age, disease duration, and levodopa dosage. Cardiovascular dysfunction was associated with the highest mean age (69.2 years), the longest disease duration (9.1 years), and the highest levodopa dosage (560 mg/day), suggesting a strong linkage with disease advancement and cumulative medication exposure. Similarly, gastrointestinal dysfunction presented among individuals with a mean age of 68.1 years and a disease duration of 8.8 years, with levodopa use averaging 545 mg/day. Urogenital and sudomotor symptoms were also found predominantly in patients with extended disease courses and higher levodopa requirements, though to a slightly lesser extent. Pupillomotor dysfunction was least prevalent and occurred in relatively younger patients (mean age 65.9 years), with a shorter disease duration (7.6 years) and lower levodopa intake (500 mg/day). These findings underscore that age, chronicity of Parkinson's disease, and dopaminergic treatment intensity are not only associated with overall autonomic dysfunction but also show varying influence across different autonomic domains, with cardiovascular and gastrointestinal systems appearing to be the most sensitive to these risk factors.

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Table 1. Prevalence of Autonomic Dysfunction in Parkinson's Disease (PD)

Autonomic Dysfunction Domain	n (%) Affected (n = 130)
Cardiovascular	78 (60.0%)
Gastrointestinal	64 (49.2%)
Urogenital	55 (42.3%)
Sudomotor	47 (36.2%)
Pupillomotor	30 (23.1%)

Table 2. Demographic and Clinical Correlates of Autonomic Dysfunction

Variable	With Autonomic Dysfunction (n=82)	Without Autonomic Dysfunction (n=48)	p-value
Age (years) (Mean ± SD)	68.4 ± 8.1	62.7 ± 7.3	0.002
Sex (Male/Female)	55/27	30/18	0.45 (NS)
Disease Duration (years)	8.6 ± 4.2	5.3 ± 3.7	0.001
Hoehn & Yahr Stage III–V	65 (79.3%)	18 (37.5%)	<0.001

Table 3. Impact of Medication Use on Autonomic Dysfunction

Medication	With Autonomic Dysfunction (n=82)	Without Autonomic Dysfunction (n=48)	p-value
Levodopa (mg/day) (Mean ± SD)	540 ± 110	460 ± 95	0.003
Dopamine Agonist Use (%)	48 (58.5%)	19 (39.6%)	0.02
Mono vs. Polytherapy	56 (68.3%) / 26 (31.7%)	38 (79.2%) / 10 (20.8%)	0.08 (NS)

Table 4. Comparison Between Early & Advanced PD

Autonomic Dysfunction Domain	Early PD (n=42, Hoehn & Yahr I–II)	Advanced PD (n=88, Hoehn & Yahr III–V)	p-value
Cardiovascular Score (Mean ± SD)	4.2 ± 2.3	7.1 ± 3.4	<0.001
Gastrointestinal Score (Mean ± SD)	3.8 ± 1.9	6.5 ± 2.7	<0.001
Urogenital Score (Mean ± SD)	2.6 ± 1.8	5.3 ± 2.4	<0.001
Sudomotor Score (Mean ± SD)	2.1 ± 1.2	4.7 ± 2.1	0.002

Table 5. Multivariate Regression Analysis (Predictors of Autonomic Dysfunction)

Independent Variable	Odds Ratio (OR)	95% CI	p-value
Age (per 1-year increase)	1.08	(1.03 – 1.14)	0.004
Disease Duration (years)	1.21	(1.11 – 1.31)	<0.001
Levodopa Dose (per 100 mg)	1.19	(1.07 – 1.33)	0.002
Hoehn & Yahr Stage (III–V)	3.45	(1.89 – 6.31)	<0.001

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Table 6: Stratified Autonomic Domain Analysis

Domain	Mean Age (years)	Disease Duration (years)	Levodopa Dose (mg/day)
Cardiovascular	69.2	9.1	560
Gastrointestinal	68.1	8.8	545
Urogenital	67.8	8.4	535
Sudomotor	66.5	7.9	515
Pupillomotor	65.9	7.6	500

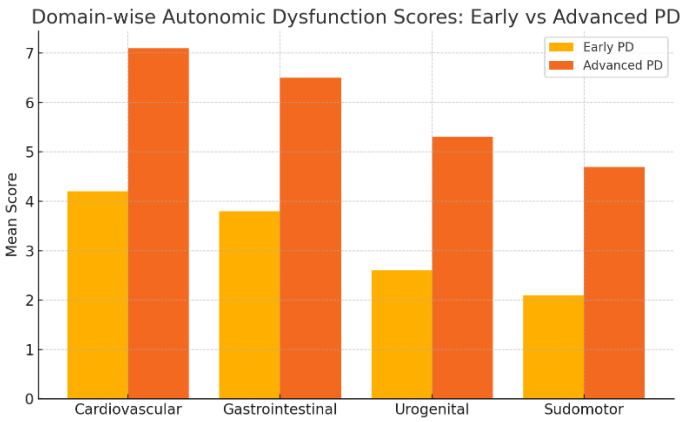

Figure 2 Domain-Wise Autonomic Dysfunction Score: Early vs Advances PD

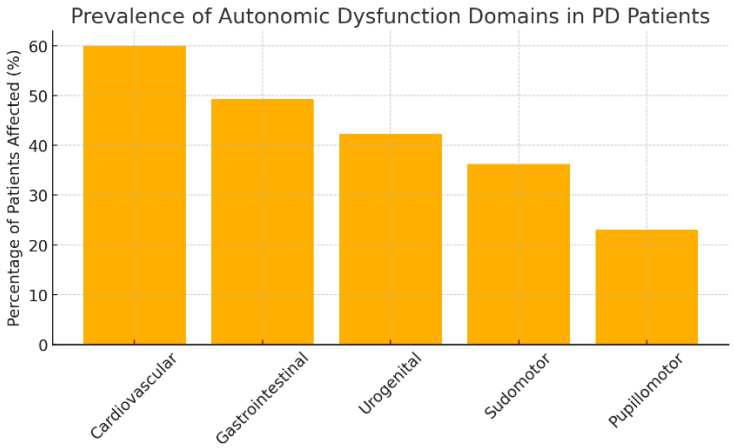

Figure 1 Prevalence of Autonomic Dysfunction Domains in PD Patients

DISCUSSION

Autonomic dysfunction emerged as a frequent yet often underrecognized non-motor manifestation in individuals with Parkinson's disease, with this study reporting a prevalence of 63.1%. Cardiovascular symptoms were the most predominant, followed by gastrointestinal, urogenital, sudomotor, and pupillomotor disturbances. These findings align with previous literature, which consistently identifies cardiovascular and gastrointestinal domains as early and prominent indicators of autonomic involvement in PD (11,12). The relatively lower prevalence of sudomotor and pupillomotor symptoms observed may be attributed to differences in population characteristics, symptom reporting patterns, or assessment methods, underscoring the need for more standardized and objective testing strategies across studies (13). A strong association was observed between increasing age, longer disease duration, and the presence of autonomic dysfunction. Older patients and those with disease duration exceeding 8.6 years demonstrated significantly higher symptom burden, highlighting the progressive nature of autonomic involvement in PD. These findings are supported by neuropathological evidence showing that chronic neurodegenerative changes, including α-synuclein deposition in autonomic centers and peripheral ganglia, contribute to functional deterioration over time (14). Patients in advanced Hoehn and Yahr stages (III–V) showed markedly higher rates of autonomic dysfunction compared to those in early stages, further validating the concept that autonomic symptoms intensify with disease progression. This reflects the broader neurodegenerative burden that accompanies motor decline in PD (15). The relationship between pharmacological treatment and autonomic dysfunction was also noteworthy. Patients with autonomic symptoms received higher daily doses of levodopa and were more frequently exposed to dopamine agonists. These findings are consistent with prior research suggesting that dopaminergic agents, particularly dopamine agonists such as pramipexole and ropinirole, can exacerbate orthostatic hypotension and related symptoms through peripheral vasodilation (16). Furthermore, long-term levodopa exposure has been implicated in the progressive disruption of central and peripheral autonomic pathways, supporting the observed dose-dependent effect (17). Interestingly, no significant difference in autonomic symptom prevalence was observed between those on monotherapy versus polytherapy, suggesting that the cumulative pharmacological load, rather than treatment regimen type, may be more influential in autonomic symptom development.

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Advanced-stage PD patients exhibited higher scores across all autonomic domains, particularly in cardiovascular and gastrointestinal categories. This domain-specific deterioration indicates a structured progression of autonomic dysfunction that parallels motor symptom severity. Such findings emphasize the need for routine screening of non-motor symptoms in clinical evaluations, especially in patients at later disease stages where these symptoms may contribute substantially to morbidity (18). The progressive trajectory of autonomic dysfunction mirrors that of motor impairment, reinforcing the importance of an integrated care approach that targets both symptom clusters in disease management (19). Multivariate regression analysis identified age, disease duration, levodopa dosage, and advanced Hoehn and Yahr staging as independent predictors of autonomic dysfunction. These results confirm that autonomic impairment arises from a complex interplay of aging, disease advancement, and pharmacological exposure. Such findings echo previous studies that have reported a similar constellation of risk factors contributing to the non-motor symptom burden in PD (20). While these factors provide a predictive framework, the variability in symptom presentation suggests that individual vulnerability and compensatory mechanisms may also play a role.

This study demonstrated several methodological strengths, including the use of the SCOPA-AUT, a validated and comprehensive tool for assessing autonomic symptoms across multiple domains. The relatively large sample size and clinical stratification enhanced the generalizability and internal validity of the results. However, the cross-sectional design limited causal interpretations, and the absence of a control group restricted direct comparisons with non-PD populations. The reliance on patient-reported symptoms introduces the potential for recall bias and underreporting, particularly for domains such as sudomotor and pupillomotor dysfunction that may be less readily perceived by patients. Additionally, the lack of objective autonomic function tests, such as tilt-table testing, heart rate variability analysis, or gastric emptying studies, constrains the diagnostic precision of the findings. Future research should prioritize longitudinal study designs to delineate the temporal dynamics of autonomic dysfunction progression and explore potential causal relationships. Incorporating objective autonomic assessments alongside subjective scales would provide a more robust understanding of the physiological basis and clinical impact of these symptoms. Investigations that examine therapeutic modulation of autonomic symptoms, including non-dopaminergic strategies or lifestyle interventions, would also contribute meaningfully to patient care. Furthermore, exploring biomarkers that predict autonomic involvement could facilitate earlier intervention and improve quality of life outcomes in Parkinson's disease.

CONCLUSION

This study concluded that autonomic dysfunction is a prevalent and clinically significant non-motor manifestation in Parkinson's disease, with cardiovascular and gastrointestinal domains most frequently affected. Its occurrence was more common among older patients, those with longer disease duration, and individuals receiving higher doses of dopaminergic therapy, particularly in advanced stages of the disease. These findings underscore the importance of early recognition and proactive management of autonomic symptoms, especially in patients with escalating treatment needs. Addressing this often-overlooked aspect of Parkinson's care through a combination of tailored pharmacological strategies and supportive non-pharmacological interventions may substantially improve patient quality of life and functional independence.

Author Contribution

Author	**Contribution**
Shaher Bano*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Husnain Nawaz Malik	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Tayba Zain	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Asif Hashmat	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Ayesha Zubair	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Maham Syed	Substantial Contribution to study design and Data Analysis

	Has given Final Approval of the version to be published

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