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Original Article

Gait Patterns and Balance Impairment in Parkinson's Disease With Correlation to Disease Severity

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ABSTRACT

Background and Purpose: Accurate and early diagnosis of Parkinson's disease (PD) remains challenging, as clinical assessments have notable limitations. Gait impairment, a hallmark motor symptom, appears in the early stages and is indicative of disease evolution. This study aimed to examine gait parameters and their associations with established clinical measures to assess their potential as diagnostic and monitoring biomarkers in a Korean PD cohort.
Methods: PD patients (n=18) and healthy controls (HCs, n=15) underwent gait assessments with MotionCore (JEIOS Inc.). Gait parameters were measured during both the 10-m walk (locomotion) and Timed Up and Go (TUG) tests, including stride length, cadence, single and double support time, toe-off time, and turning performance. Clinical assessments comprised the Hoehn & Yahr (H&Y) scale, Unified Parkinson's Disease Rating Scale Part III, Freezing of Gait Questionnaire, Korean version of the Mini-Mental State Examination, Geriatric Depression Scale, and Berg Balance Scale.
Results: Patients with PD demonstrated significantly shortened stride length and higher cadence. Reduced single support time, extended double support time, and delayed toe-off time were observed in the PD group. Integrating gait metrics from both the 10-m locomotion and TUG tests improved discrimination between PD patients and healthy controls, as well as among different H&Y stages. Turning-related measures were significantly higher in PD patients. Gait measures showed robust correlations with clinical indices, including motor severity, cognitive status, and balance scores.
Conclusions: Gait analysis identified prominent motor and balance deficits in PD patients that were strongly associated with clinical severity. Gait parameters hold considerable potential as objective digital biomarkers for the diagnosis and monitoring of PD progression.

Keywords: Parkinson Disease; Gait, Biomarker; Disease Progression

INTRODUCTION

Parkinson's disease (PD) is the most prevalent movement disorder, affecting over 1% of adults older than 60 years.[1] Furthermore, PD represents the second most frequent

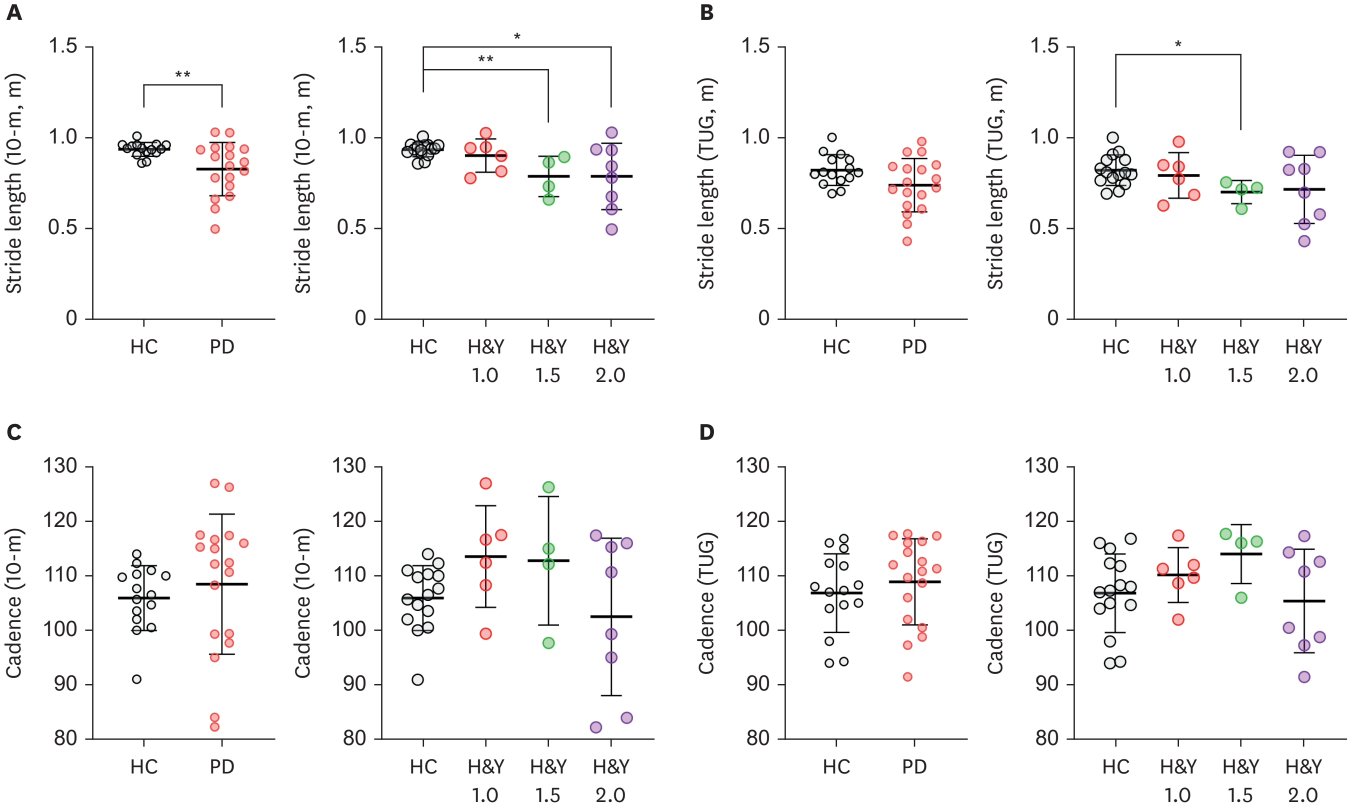

Fig. 1. Notable alterations in gait patterns among PD patients.
There was a significant reduction in stride length in both the 10-m walk (locomotion) and the TUG tests. (A) Stride length of the 10-m walk test were: HC: 0.935±0.0379, PD: 0.827±0.146; H&Y 1.0: 0.903±0.0914, H&Y 1.5: 0.789±0.110, H&Y 2.0: 0.789±0.182. (B) Stride length of the TUG test were: HC: 0.821±0.0843, PD: 0.738±0.146; H&Y 1.0: 0.792±0.126, H&Y 1.5: 0.700±0.0635, H&Y 2.0: 0.716±0.187. Cadence increased significantly in the 10-m walk and the TUG tests. (C) Evaluated cadence of the 10-m walk test were: HC: 106±5.95, PD: 108±12.9; H&Y 1.0: 114±9.33, H&Y 1.5: 113±11.8, H&Y 2.0: 103±14.4. (D) Evaluated cadence of the TUG test were: HC: 107±7.19, PD: 109±7.87; H&Y 1.0: 110±5.02, H&Y 1.5: 114±5.38, H&Y 2.0: 105±9.48.
Data are presented as mean ± standard deviation.
PD: Parkinson's disease, TUG: Timed Up and Go, HC: healthy control, H&Y: Hoehn and Yahr.
*$p<0.05$, **$p<0.01$.

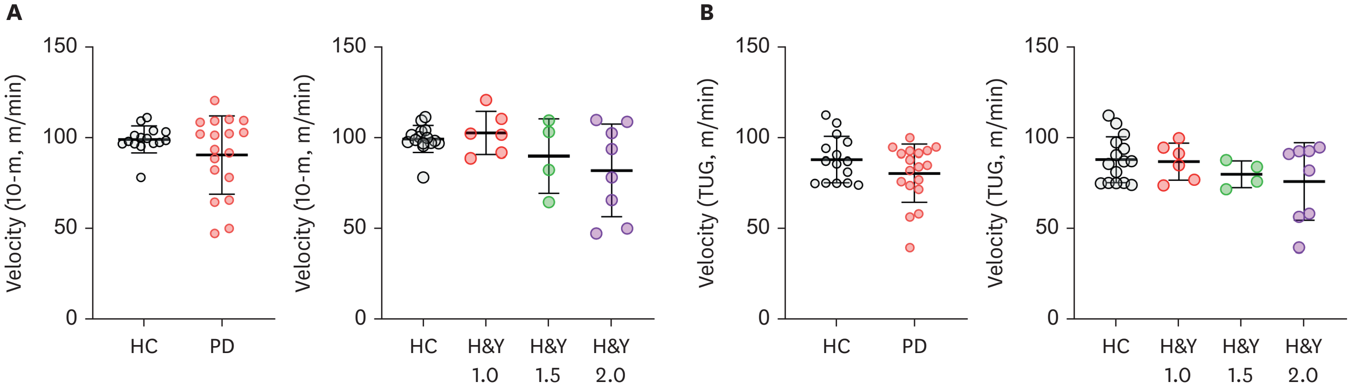

Fig. 2. Reduced velocity (walking speed) in PD patients.
Changes in velocity were observed in both the 10-m walk (locomotion) and the TUG tests. (A) Changes in velocity of the 10-m walk test were: HC: 99.1±7.43, PD: 90.5±21.6; H&Y 1.0: 102±11.8, H&Y 1.5: 89.8±20.4, H&Y 2.0: 81.9±25.4. (B) Changes in velocity of the TUG test were: HC: 87.9±12.7, PD: 80.4±15.9; H&Y 1.0: 86.8±10.3, H&Y 1.5: 79.8±7.37, H&Y 2.0: 75.8±21.4.
Data are presented as mean ± standard deviation.
PD: Parkinson's disease, TUG: Timed Up and Go, HC: healthy control, H&Y: Hoehn and Yahr.

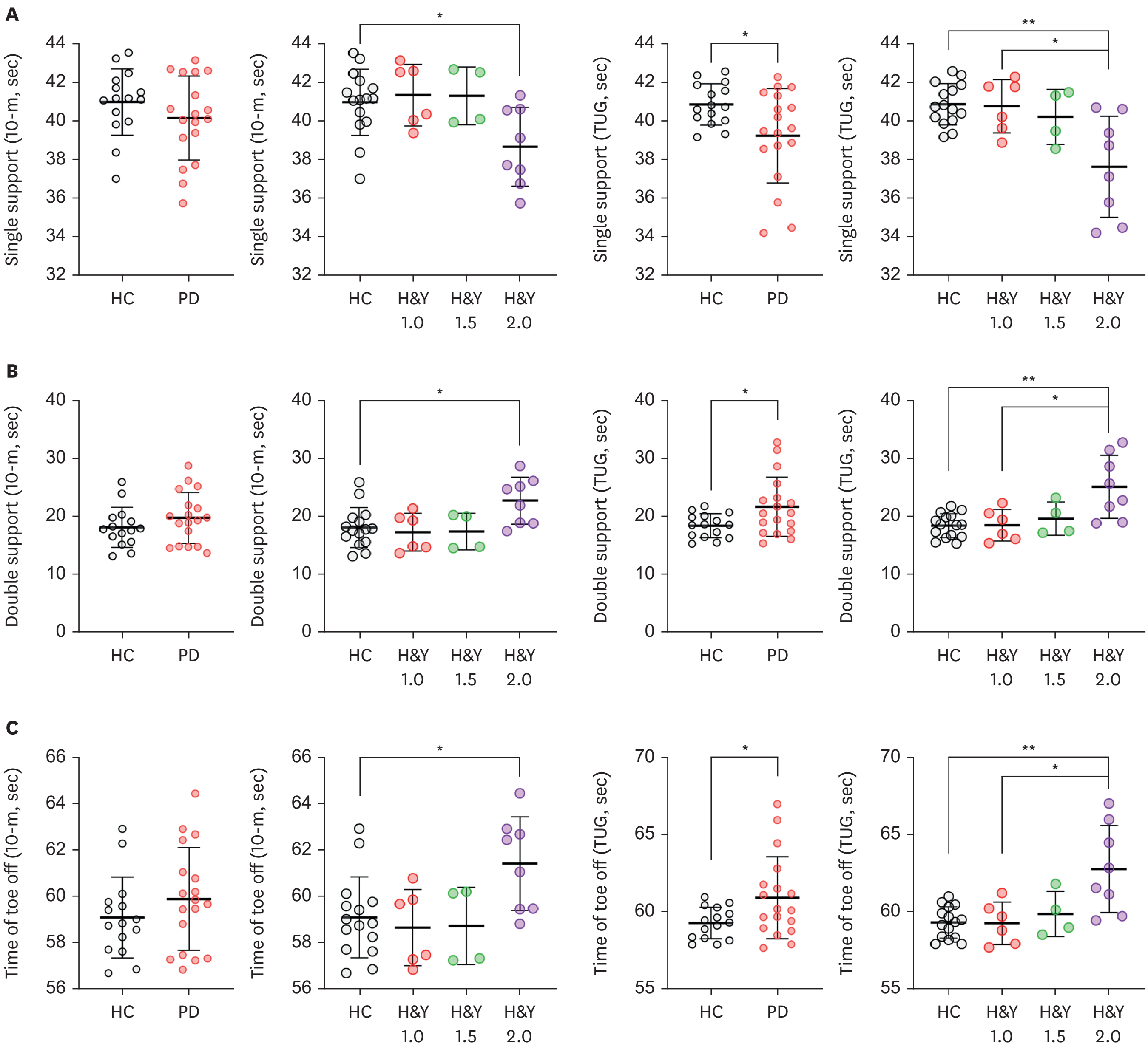

Fig. 3. Gait balance impairments in PD patients.

(A) Single support time showed a significant reduction. 10-m walk test: HC: 41.0±1.71, PD: 40.1±2.17; H&Y 1.0: 41.3±1.59, H&Y 1.5: 41.3±1.50, H&Y 2.0: 38.7±2.04. TUG test: HC: 40.8±1.07, PD 39.2±2.45; H&Y 1.0: 40.7±1.38, H&Y 1.5: 40.2±1.42, H&Y 2.0: 37.6±2.61. (B) Double support time was markedly increased. 10-m walk test: HC: 18.1±3.45, PD: 19.7±4.39; H&Y 1.0: 17.3±3.24, H&Y 1.5: 17.4±3.16, H&Y 2.0: 22.7±4.06. TUG test: HC: 18.5±2.07, PD: 21.7±5.10; H&Y 1.0: 18.5±2.74, H&Y 1.5: 19.7±2.86, H&Y 2.0: 25.1±5.43. (C) Toe off time was significantly delayed. 10-m walk test: HC: 59.1±1.75, PD: 59.9±2.22; H&Y 1.0: 58.6±1.65, H&Y 1.5: 58.7±1.66, H&Y 2.0: 61.4±2.02. TUG test: HC: 59.3±1.02, PD: 60.9±2.66; H&Y 1.0: 59.2±1.37, H&Y 1.5: 59.8±1.47, H&Y 2.0: 62.8±2.83.

Data are presented as mean ± standard deviation.

PD: Parkinson's disease, HC: healthy control, H&Y: Hoehn and Yahr, TUG: Timed Up and Go.

$^{*}p<0.05$, $^{**}p<0.01$.

Enhanced differentiation by integrating gait parameters across tests

To enhance the differentiation between HC and PD, gait parameters from both the 10-m walk and TUG tests were analyzed in combination. Stride length was markedly reduced in PD participants, with those at H&Y stage 1.5 exhibiting significant decreases compared to HC (**Fig. 4A**). Cadence was found to be elevated in PD, with statistically significant increases at H&Y stages 1.0 and 1.5, whereas no significant change was detected at stage 2.0 (**Fig. 4B**). Single support time demonstrated a decline in PD, and was significantly shorter in H&Y stage 2.0 when compared with both HC and H&Y stage 1.0 (**Fig. 4C**). Double support duration was

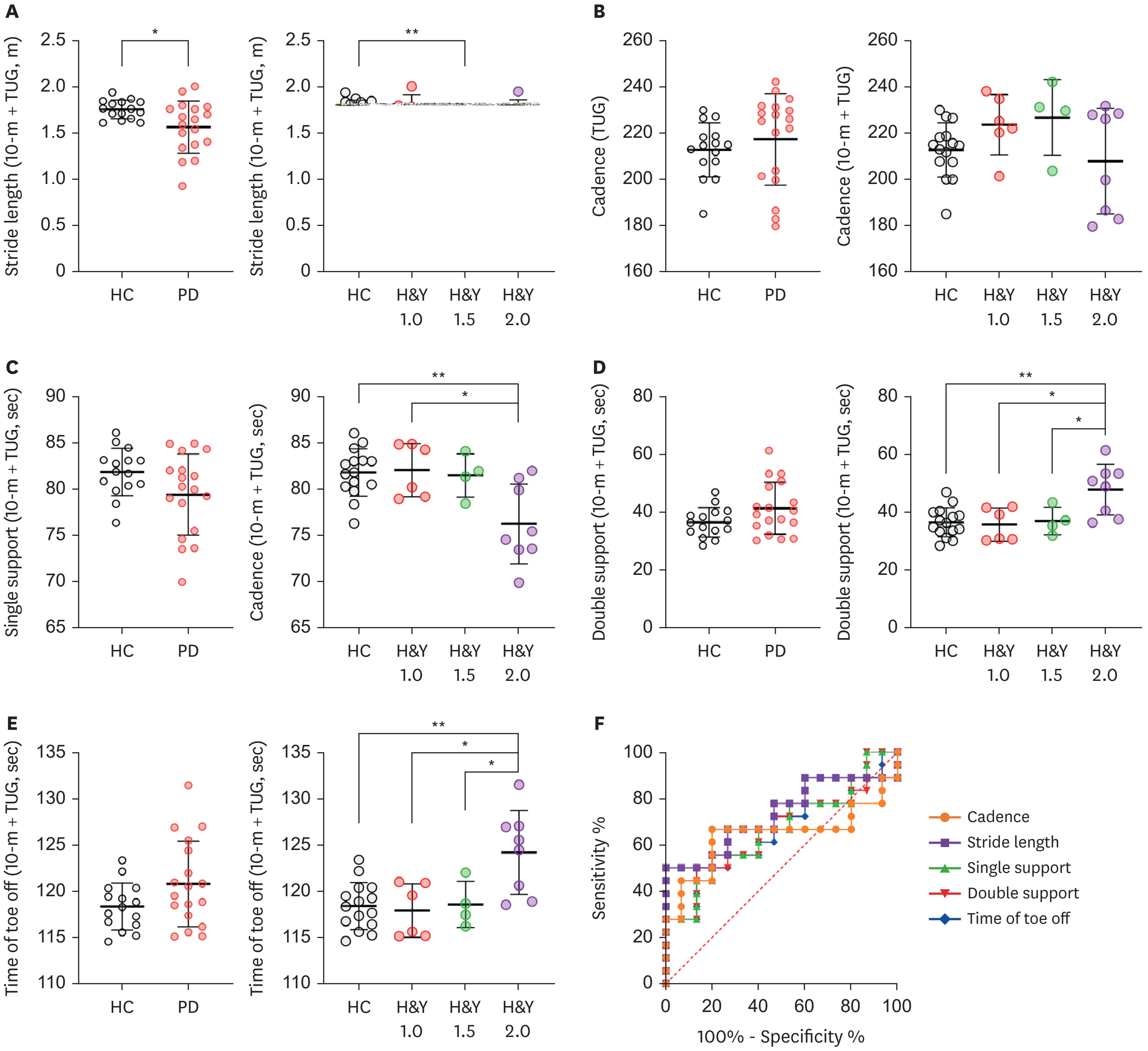

Fig. 4. Diagnostic significance of combining 10-m walk and TUG test according to H&Y stages.
(A) A significant reduction in stride length was observed. HC: 1.76±0.101, PD: 1.56±0.282; H&Y 1.0: 1.70±0.217, H&Y 1.5: 1.49±0.153, H&Y 2.0: 1.50±0.357. (B) Cadence was elevated in the PD and H&Y subgroups. HC: 213±11.8, PD: 217±19.8; H&Y 1.0: 224±13.1, H&Y 1.5: 227±16.4, H&Y 2.0: 208±23.0. (C) A marked decrease in single support time was detected. HC: 81.8±2.57, PD: 79.4±4.38; H&Y 1.0: 82.1±2.89, H&Y 1.5: 81.5±2.34, H&Y 2.0: 76.3±4.30. (D) Double support time was significantly increased. HC: 36.6±5.08, PD: 41.5±9.00; H&Y 1.0: 35.8±5.75, H&Y 1.5: 37.1±4.80, H&Y 2.0: 47.9±8.81. (E) A significant prolongation in the time of toe off was observed. HC: 118±2.53, PD: 121±4.63; H&Y 1.0: 118±2.87, H&Y 1.5: 119±2.49, H&Y 2.0: 124±4.52. (F) Receiver operating characteristic curve analyses assessed gait parameters as discriminative markers. Cadence: AUC=0.6407, specificity=80%, sensitivity=66.67%; Stride length: AUC=0.7296, specificity=100%, sensitivity=50%; Single support: AUC=0.6667, specificity=80%, sensitivity=55.56%; Double support: AUC=0.6667, specificity=86.67%, sensitivity=50%; Time of toe off: AUC=0.6556, specificity=86.67%, sensitivity=50%.
Data are presented as mean ± standard deviation.
TUG: Timed Up and Go, H&Y: Hoehn and Yahr, HC: healthy control, PD: Parkinson’s disease, AUC: area under the curve.
$^{*}p<0.05$, $^{**}p<0.01$.

stride length and cadence provide insight into PD motor manifestations, as reduced stride length often leads to compensatory increases in cadence to preserve walking speed despite shorter steps.[26] In the present study, we measured gait parameters in a Korean population and assessed their associations with clinical outcomes to validate their utility as digital biomarkers for PD. Gait analyses were conducted using both 10-m walk and TUG tests with MotionCore (JEIOS Inc.)

Stride length was markedly reduced in PD, whereas cadence increased as a compensatory response (**Fig. 1**). These patterns became increasingly evident with advancing H&Y stages, indicating that gait parameters reflect disease progression as documented in previous studies.[26] For the evaluation of gait balance, single support time, double support time, and toe off time were systematically examined. Among PD patients, there was a reduction in single support time accompanied by an increase in double support time, signifying a shift towards increased double support duration due to impaired balance (**Fig. 3**).[26] This compensatory adaptation was further evidenced by elevated cadence, as patients attempted to enhance stability by reducing the time spent on a single limb. These gait variables exhibited significant correlations with H&Y stage, with more pronounced associations evident in the TUG test. Toe off time was found to be delayed in PD patients and increased progressively with H&Y stage, representing challenges in initiating subsequent steps during ambulation. By combining assessments from the 10 m locomotion and TUG tests, the distinction between PD and HC, as well as differentiation across H&Y stages, was improved. Receiver operating characteristic analysis of gait metrics yielded moderate discriminative power, with AUC values between 0.64 and 0.73. Although specificity was relatively high (reaching 100% for stride length), sensitivity remained moderate (50%–66.67%) (**Fig. 4**). This result suggests enhanced ability to distinguish PD from HC, specifically by reducing false positive rates. Furthermore, assessment of turning parameters during the TUG test revealed significant increases in turning onset, effective turning radius, and effective movement area among PD patients (**Fig. 5**). Prior research established that anti-parkinsonian medications correlate with shorter turn durations and higher turning velocities in PD individuals.[27] Consistently, our results demonstrated relevant alterations in turning performance, supporting the utility of turning analysis as a valuable element in the comprehensive assessment of gait dysfunction in PD. Gait characteristics showed strong correlations with established clinical scales, including H&Y stage, UPDRS, and BBS, which are standard for evaluating motor dysfunction in PD (**Fig. 6**). In addition, gait parameters displayed inverse relationships with cognitive outcomes, underscoring the broader clinical relevance of gait analysis in PD.

However, this study has certain limitations. The sample size was limited, and there was an underrepresentation of female participants and controls compared to male participants. To mitigate this issue, future research should enroll larger and more balanced cohorts and utilize longitudinal designs to observe disease progression over time. Furthermore, cognitive impairment in PD involves multiple domains, and the MMSE applied in this study may lack sensitivity to detect executive dysfunction. The Montreal Cognitive Assessment may offer greater sensitivity for clinical relevance and should be considered in future investigations. Finally, although PD diagnosis in this cohort was supported by clinical ratings and 18F-FP-CIT PET, we did not perform a direct comparison between gait assessments and imaging outcomes. Incorporating neuroimaging findings could enhance understanding of the association between gait metrics and PD pathophysiology.

In summary, PD patients exhibited decreased stride length that was offset by an increase in cadence, along with impaired gait balance as evidenced by longer ground contact time and elevated double support time. Notably, gait instability resulted in longer turning time, as well as greater turning distance and area, features that robustly distinguished PD individuals from HC. Digital gait analysis offers objective, quantitative evaluation in clinical settings and may facilitate early detection of subtle motor deficits that appear prior to clear clinical or imaging evidence. Overall, these results reinforce the utility of gait characteristics as objective indicators for PD diagnosis and monitoring progression, and highlight their promise for development as digital biomarkers in clinical care.

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REFERENCES

1. Samii A, Nutt JG, Ransom BR. Parkinson's disease. Lancet 2004;363:1783-1793. PUBMED | CROSSREF
2. GBD 2016 Parkinson's Disease Collaborators. Global, regional, and national burden of Parkinson's disease, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Neurol 2018;17:939-953. PUBMED | CROSSREF
3. Su D, Cui Y, He C, Yin P, Bai R, Zhu J, et al. Projections for prevalence of Parkinson's disease and its driving factors in 195 countries and territories to 2050: modelling study of Global Burden of Disease Study 2021. BMJ 2025;388:e080952. PUBMED | CROSSREF
4. Chen PH, Wang RL, Liou DJ, Shaw JS. Gait disorders in Parkinson's disease: assessment and management. Int J Gerontol 2013;7:189-193. CROSSREF
5. Vingerhoets FJG, Snow BJ, Lee CS, Schulzer M, Mak E, Calne DB. Longitudinal fluorodopa positron emission tomographic studies of the evolution of idiopathic parkinsonism. Ann Neurol 1994;36:759-764. PUBMED | CROSSREF
6. Fearnley JM, Lees AJ. Ageing and Parkinson's disease: substantia nigra regional selectivity. Brain 1991;114:2283-2301. PUBMED | CROSSREF
7. Mancini M, Afshari M, Almeida Q, Amundsen-Huffmaster S, Balfany K, Camicioli R, et al. Digital gait biomarkers in Parkinson's disease: susceptibility/risk, progression, response to exercise, and prognosis. NPJ Parkinsons Dis 2025;11:51. PUBMED | CROSSREF
8. Mirelman A, Bonato P, Camicioli R, Ellis TD, Giladi N, Hamilton JL, et al. Gait impairments in Parkinson's disease. Lancet Neurol 2019;18:697-708. PUBMED | CROSSREF
9. Castilla-Cortázar I, Aguirre GA, Femat-Roldán G, Martín-Estal I, Espinosa L. Is insulin-like growth factor-1 involved in Parkinson's disease development? J Transl Med 2020;18:70. PUBMED | CROSSREF
10. Kalia LV, Lang AE. Parkinson's disease. Lancet 2015;386:896-912. PUBMED | CROSSREF
11. Horak FB, Mancini M. Objective biomarkers of balance and gait for Parkinson's disease using body-worn sensors. Mov Disord 2013;28:1544-1551. PUBMED | CROSSREF
12. Cai G, Shi W, Wang Y, Weng H, Chen L, Yu J, et al. Specific distribution of digital gait biomarkers in parkinson's disease using body-worn sensors and machine learning. J Gerontol A Biol Sci Med Sci 2023;78:1348-1354. PUBMED | CROSSREF
13. McNeely ME, Earhart GM. Medication and subthalamic nucleus deep brain stimulation similarly improve balance and complex gait in Parkinson disease. Parkinsonism Relat Disord 2013;19:86-91. PUBMED | CROSSREF
14. Vila MH, Pérez R, Mollinedo I, Cancela JM. Analysis of gait for disease stage in patients with Parkinson's disease. Int J Environ Res Public Health 2021;18:720. PUBMED | CROSSREF
15. Kim SM, Kim DH, Yang Y, Ha SW, Han JH. Gait patterns in Parkinson's disease with or without cognitive impairment. Dement Neurocogn Disord 2018;17:57-65. PUBMED | CROSSREF
16. Jeon H, Shin JH, Yu R, Kang MK, Lee S, Kim S, et al. Spatiotemporal gait parameters during turning and imbalance in Parkinson's disease: video-based analysis from a single camera. J Mov Disord 2025;18:87-92. PUBMED | CROSSREF

17. Luo Y, Qiao L, Li M, Wen X, Zhang W, Li X. Global, regional, national epidemiology and trends of Parkinson's disease from 1990 to 2021: findings from the Global Burden of Disease Study 2021. Front Aging Neurosci 2025;16:1498756. PUBMED | CROSSREF
18. Ramesh S, Arachchige ASPM. Depletion of dopamine in Parkinson's disease and relevant therapeutic options: a review of the literature. AIMS Neurosci 2023;10:200-231. PUBMED | CROSSREF
19. Postuma RB, Berg D, Stern M, Poewe W, Olanow CW, Oertel W, et al. MDS clinical diagnostic criteria for Parkinson's disease. Mov Disord 2015;30:1591-1601. PUBMED | CROSSREF
20. Burtscher J, Moraud EM, Malatesta D, Millet GP, Bally JF, Patoz A. Exercise and gait/movement analyses in treatment and diagnosis of Parkinson's disease. Ageing Res Rev 2024;93:102147. PUBMED | CROSSREF
21. Rizzo G, Copetti M, Arcuti S, Martino D, Fontana A, Logroscino G. Accuracy of clinical diagnosis of Parkinson disease: a systematic review and meta-analysis. Neurology 2016;86:566-576. PUBMED | CROSSREF
22. Moustafa AA, Chakravarthy S, Phillips JR, Gupta A, Keri S, Polner B, et al. Motor symptoms in Parkinson's disease: a unified framework. Neurosci Biobehav Rev 2016;68:727-740. PUBMED | CROSSREF
23. Hausdorff JM. Gait dynamics in Parkinson's disease: common and distinct behavior among stride length, gait variability, and fractal-like scaling. Chaos 2009;19:026113. PUBMED | CROSSREF
24. Koyama M, Yagishita A, Nakata Y, Hayashi M, Bandoh M, Mizutani T. Imaging of corticobasal degeneration syndrome. Neuroradiology 2007;49:905-912. PUBMED | CROSSREF
25. Sun YM, Wang ZY, Liang YY, Hao CW, Shi CH. Digital biomarkers for precision diagnosis and monitoring in Parkinson's disease. NPJ Digit Med 2024;7:218. PUBMED | CROSSREF
26. Zanardi APJ, da Silva ES, Costa RR, Passos-Monteiro E, Dos Santos IO, Kruel LFM, et al. Gait parameters of Parkinson's disease compared with healthy controls: a systematic review and meta-analysis. Sci Rep 2021;11:752. PUBMED | CROSSREF
27. Miller Koop M, Ozinga SJ, Rosenfeldt AB, Alberts JL. Quantifying turning behavior and gait in Parkinson's disease using mobile technology. IBRO Rep 2018;5:10-16. PUBMED | CROSSREF