



Association between cognitive decline and postural instability in patients with Parkinson's disease - A Cross Sectional correlation study

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Abstract

Background: Parkinson's disease is a progressive neurodegenerative disorder characterized by both motor and non-motor symptoms. Postural instability and cognitive decline are two significant manifestations that substantially impact quality of life and functional independence. This study aims to correlate the relationship between postural instability and cognitive decline that remains inadequately understood.

Methodology: The study was conducted at RVS College of Physiotherapy with ethical approval and informed consent. Twenty clinically diagnosed Parkinson's disease subjects of age group 50-80 years were selected in this study that satisfied the inclusion and exclusion criteria. Cognitive decline was measured by Addenbrooke's Cognitive Examination (ACE) scale focusing on attention, memory and visuospatial domains, and postural instability was measured by Mini-Balance Evaluation Systems Test (Mini-BESTest).

Results: There is a strong positive correlation between cognitive decline and postural instability ($r = 0.970$). This correlation was found to be statistically significant at the 0.001 level ($p < 0.001$). This indicates that as cognitive decline increases, postural instability also increases significantly.

Conclusion: The strong positive correlation demonstrates that cognitive impairment is closely related with balance deficits in patients with Parkinson's disease.

Keywords: Parkinson's disease, Cognitive decline, Postural instability, Addenbrooke's Cognitive Examination, Mini-BESTest.

Introduction

Parkinson's disease is a progressive disorder of the central nervous system with both motor and non-motor symptoms. Motor symptoms includes the cardinal features of bradykinesia, tremor, rigidity and postural instability. Non-motor symptoms may precede the onset of motor symptoms by 10 years. The non-motor symptoms include excessive daytime sleepiness, fatigue, pain, altered bladder function, difficulty speaking and swallowing, apathy and cognitive problems (reduced concentration and attention, slowed thinking, confusion, and in some cases dementia). It was initially described by James Parkinson in 1817 as "shaking palsy." The disease was later termed "maladie de Parkinson" by Charcot ¹.

The origins of Parkinson's disease are intricate and not completely understood. It is a disorder that causes degeneration of the nervous system. There is a decrease in neurons (nerve cells) located in specific parts of the brain, encompassing a zone referred to as the substantia nigra. Latin for "black substance." The neurons located in this region produces a neurotransmitter (a chemical messenger that enables communication between neurons) called dopamine. Dopamine assists in regulating movement. As the number of cells in the substantia nigra decreases, there is less dopamine available in the brain. This loss of dopamine is the reason for disruption of normal movement pattern. Degeneration of neurons in different areas of the brain accounts for some of the non-motor symptoms of the disease ².

Among the wide spectrum of motor and non-motor manifestations in Parkinson's disease, two features assume particular clinical and functional significance: Postural instability and cognitive decline. These complications not only contribute substantially to disability and mark advanced disease progression but also provides essential data on fall frequency, enabling targeted prevention measures. Difficulty maintaining stable upright posture represents one of the defining characteristics of Parkinson's disease, typically becoming evident as the condition advances and contributing significantly to falls, physical injuries and diminished life satisfaction ³.

The brain regions and pathways involved in postural control extend well beyond the dopamine-producing cells that primarily degenerate in Parkinson's disease, which explains why balance problems may respond incompletely to dopamine replacement medications ⁴.

The pattern of cognitive difficulties typically observed in the condition includes problems with mental flexibility and planning, maintaining focused attention, understanding spatial relationship, and retrieving information from memory, reflecting dysfunction primarily in brain circuits connecting frontal and deeper brain structures as well as posterior brain regions ⁵.

Recent scientific work has revealed the intricate connections between postural control and cognitive function in Parkinson's disease, challenging earlier assumptions that these represented completely separate symptom categories ⁶.

Multiple studies have demonstrated a significant correlation between these two domains. Patients with greater postural instability exhibit more pronounced cognitive deficits, particularly in executive function and attention, which are crucial for maintaining balance during complex motor tasks. This relationship becomes increasingly apparent during dual task paradigms, where simultaneous cognitive and motor demands reveal the brain's limited capacity to allocate attentional resources effectively in Parkinson's disease ⁷.

As cognitive decline progresses, fewer resources remain available for postural maintenance, creating a vicious cycle wherein cognitive impairment exacerbates postural instability and vice versa ⁸.

The correlation between postural instability and cognitive decline stems from overlapping neural networks affected by Parkinson's disease pathology. The basal ganglia, particularly the substantia nigra pars compacta, undergo dopaminergic degeneration that disrupts both motor control circuits and cognitive processing loops ⁹.

The dorsolateral prefrontal cortex, which governs executive functions, receives substantial dopaminergic input and plays a critical role in postural adjustments and gait planning. Degeneration of this pathway impairs both cognitive flexibility and adaptive postural responses ¹⁰.

Methodology

Materials and Methods:

This cross-sectional correlation study was conducted in the Physiotherapy Outpatient Department of R.V.S. College of Physiotherapy, Sulur, Coimbatore, over a period of three months. A total of 20 participants with clinically diagnosed Idiopathic Parkinson's disease were randomly selected based on the predefined inclusion and exclusion criteria. The study included male and female individuals aged 80–40 years presenting with mild to moderate Parkinson's disease, who are able to walk independently or with minimal assistance. Participants with severe dementia, severe orthopaedic or cardiovascular conditions

limiting mobility, Other neurological conditions (Stroke, Multiple sclerosis, cerebellar disorders) and who have psychiatric illness affecting cognition or compliance were excluded from the study.

After obtaining written informed consent, demographic details were recorded, and all participants underwent assessment of the cognitive decline which was measured by the Addenbrooke's Cognitive Examination scale and postural instability measured by the Mini-BESTest.

Measurement procedure

Addenbrooke's Cognitive Examination:

Cognitive function was assessed using the Addenbrooke's Cognitive Examination scale, with evaluation restricted to the attention, memory and visuospatial domains. Prior to administration, the procedure was explained to the subject, and verbal consent was obtained. The attention and orientation, visuospatial domain and memory assessed the subject's ability to maintain attention, orientation to time and place, and perform basic mental tasks such as counting and recall. The visuospatial and executive functional domain evaluated the subject's visuospatial processing and constructional abilities through tasks such as figure copying and spatial judgement. The memory domain was evaluated the subject's recognition and recall. Scores obtained from the selected domains were recorded according to the standardized ACE scoring criteria. Higher scores indicated better cognitive performance, while the lower scores suggested greater cognitive decline.



Figure 1: Addenbrooke's Cognitive Examination.

Mini-BESTest:

Patient position: The subject was asked to stand barefoot or with regular footwear on a firm, level surface in a comfortable posture.

Therapist position: The examiner stood close to the subjects to provide guarding for safety during balance tasks without providing physical assistance unless required.

Procedure: The test consists of task assessing dynamic balance across four components: anticipatory postural adjustments, reactive postural control, sensory orientation, and dynamic gait. Before test administration, the procedure was explained to the subject, and a demonstration was provided when necessary. Each task was performed according to standardized Mini-BESTest guidelines. The subject was encouraged to perform each task to the best of their ability, and rest periods were provided as required to avoid fatigue. Each item was scored on a three-point ordinal scale (0-2), where the higher score indicated better balance performance and lower score indicated postural instability. The total Mini-BESTest score was calculated by summing the scores of individual items.



Figure 1: Mini-BESTest.

Data analysis and Results:

The study was aimed to investigate the association between cognitive decline and postural instability in patients with Parkinson's disease. Statistical analysis revealed there is a very strong positive correlation between cognitive decline and postural instability in patients with Parkinson's disease (Pearson $r = -0.970$). The relationship is highly significant ($p < 0.001$), meaning it is extremely likely to be due to chance.

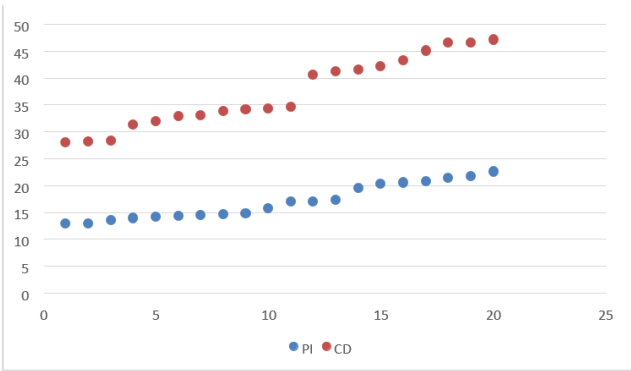


Figure 3: Correlation between cognitive decline and postural instability.

Table 1: Correlation values of cognitive decline and postural instability

		CD	PI
CD	'Pearson Correlation	1	0.970
	Significance (p)		<0.001
	N	20	20
PI	Pearson Correlation	0.970	1
	Significance (p)	<0.001	
	N	20	20

*0.001 Level of significance

Results

Twenty clinically diagnosed patients with Parkinson's disease participated in the study. Cognitive decline was measured using the

Addenbrooke's cognitive examination and postural instability was measured using the MINI-BESTest. Karl Pearson's correlation analysis revealed a very strong positive correlation between CD and PI ($r = -0.970$, $p < 0.001$). The 95% confidence interval ranged from 0.924 to 0.988, suggesting a consistent and reliable relationship. This indicates that as the cognitive impairment increases, the postural instability also increases significantly in patients with Parkinson's disease

Discussion

The present study demonstrated a significant strong relationship between cognitive decline and postural instability in patients with Parkinson's disease. These findings support previous research showing that patients with greater postural instability exhibit more pronounced cognitive deficits, particularly in executive function and attention, which are crucial for maintaining balance during complex motor tasks. As cognitive decline progresses, fewer resources remain available for postural maintenance, creating a vicious cycle wherein cognitive impairment exacerbates postural instability and vice versa. Therefore, cognitive and postural assessment should be considered as important components in the evaluation and rehabilitation of patients with Parkinson's disease.

Conclusion

The study concludes that there is a very strong, statistically significant positive correlation between cognitive decline and postural instability in patients with Parkinson's disease. As the cognitive decline increases, postural instability also increases creating a vicious cycle, suggesting that cognitive decline influences postural instability. These results underline the importance of incorporating cognitive assessment and cognitive-motor training strategies into balance rehabilitation programs to reduce fall risk and improve functional outcomes.

References

1. Sullivan, S. B. (2024). Physical Rehabilitation (8th Indian Ed.). Jaypee Brothers Medical Publishers.

2. American Parkinson Disease Association. (2017). Parkinson's Disease: Causes, Symptoms and Treatment.

3. Grimbergen, Y. A., Munneke, M., & Bloem, B. R. (2009). Falls in Parkinson's disease. *Current Opinion in Neurology*, 22(4), 309-315.

4. Takakusaki, K., Tomita, N., & Yano, M. (2016). Substrates for normal gait and pathophysiology of gait disturbances with respect to the basal ganglia dysfunction. *Journal of Neurology*, 255(Suppl 4), 19-29.

5. Kehagia, A. A., Barker, R. A., & Robbins, T. W. (2010). Neuropsychological and clinical heterogeneity of cognitive impairment and dementia in patients with Parkinson's disease. *The Lancet Neurology*, 9(12): 1200-1213.

6. Mak, M. K., Wong-Yu, I. S., Shen, X., & Chung, C. L. (2017). Long-term effects of exercise and physical therapy in people with Parkinson disease. *Nature Reviews Neurology*, 13(11), 689-703.

7. Yogev-Seligmann, G., Hausdorff, J. M., & Giladi, N. (2008). The role of executive function and attention in gait. *Movement Disorders*, 23(3), 329-342.

8. Brauer, S. G., Woollacott, M. H., & Shumway-Cook, A. (2011). The influence of a concurrent cognitive task on the compensatory stepping response to a perturbation in balance-impaired and healthy elders. *Gait & Posture*, 15(1), 83-3.
9. Peterson, D. S., & Horak, F. B. (2016). Neural control of walking in people with parkinsonism. *Physiology*, 31(2), 95-107.
10. Takakusaki, K. (2017). Functional neuroanatomy for posture and gait control. *Journal of Movement Disorders*, 10(1), 1-17.