

A Hospital Survey Associated with Parkinson's Disease

Amruta Baban Bhagat, Ms. Priya S. Shinde, Trupti M. Waydande

YSPM's Yashoda Technical Campus, Satara, Maharashtra, India

Abstract

Parkinson's disease (PD) is a long-term, progressively worsening neurological condition that mainly affects movement. It develops due to the gradual loss of dopamine-producing neurons in the substantia nigra region of the brain. PD is recognised as the second most common neurodegenerative disorder after Alzheimer's disease and is seen more frequently in older adults. The condition is classically associated with motor symptoms such as resting tremor, slowness of movement (bradykinesia), muscle stiffness, and difficulties with balance and posture. In addition to these motor features, individuals with Parkinson's disease often experience a variety of non-motor symptoms, including cognitive decline, emotional disturbances, sleep-related problems, and autonomic system dysfunction.

This review article presents an in-depth discussion of Parkinson's disease, focusing on its epidemiology, underlying disease mechanisms, risk factors, clinical presentation, diagnostic guidelines, and available treatment options. Current therapeutic strategies are outlined, with particular attention to pharmacological management such as levodopa, dopamine agonists, monoamine oxidase-B inhibitors, and supportive treatments aimed at managing non-motor symptoms. The article also incorporates findings from hospital-based survey observations that reflect patient experiences, commonly prescribed therapies, and observed clinical outcomes. Overall, this review seeks to improve awareness and understanding of Parkinson's disease, while highlighting the critical role of early detection, coordinated multidisciplinary care, and continued research in improving the quality of life and outcomes for affected individuals.

Index Terms

Bradykinesia, Dopaminergic neurons, Neurodegenerative disease, Parkinson's disease, Review article

I. INTRODUCTION

Parkinson's disease (PD) is a slowly progressive neurodegenerative condition that was first identified by James Parkinson in 1817. The disorder primarily affects movement as a result of the gradual loss of dopamine-producing neurons in the substantia nigra pars compacta of the midbrain. Parkinson's disease has a profound effect on patients' quality of life and represents an increasing challenge for healthcare systems across the globe. Beyond the well-recognized motor symptoms, PD is also characterized by a wide range of non-motor features, many of which may appear early in the disease course and contribute to the complexity of diagnosis and long-term management

II. EPIDEMIOLOGY

Parkinson's disease is recognized as the second most prevalent neurodegenerative condition worldwide.

Its occurrence increases significantly with age, with nearly 1% of the population aged 65 years and older affected by the disease. Epidemiological data also show a higher prevalence among males than females, with an estimated male-to-female ratio of about 1.5:1. Recent studies highlight a steady and substantial increase in the global burden of Parkinson's disease, largely driven by aging populations. This growing trend underscores the urgent need for improved preventive measures, early detection, and more effective treatment approaches.

III. PATHOPHYSIOLOGY

Parkinson's disease is characterized by distinct pathological changes, most notably the progressive loss of dopamine-producing neurons and the formation of Lewy bodies within the brain. These Lewy bodies are abnormal intracellular aggregates

primarily composed of alpha-synuclein and ubiquitin. The degeneration of dopaminergic neurons disrupts dopamine signaling in the basal ganglia, a brain region essential for the regulation of movement, ultimately leading to the motor symptoms seen in Parkinson's disease. In addition to dopamine deficiency, several other mechanisms contribute to neuronal damage, including mitochondrial dysfunction, increased oxidative stress, chronic neuroinflammation, and defects in protein clearance systems. Importantly, these pathological processes are believed to start many years before the onset of noticeable clinical symptoms.

IV. RISK FACTORS AND ETIOLOGY

The development of Parkinson's disease is influenced by a combination of genetic susceptibility and environmental exposures. Several gene mutations, including LRRK2, PARK7, PINK1, PRKN, and SNCA, have been linked to inherited forms of the disease. In addition to genetic factors, environmental elements such as exposure to pesticides, heavy metals, and a history of head injury have been identified as important contributors to disease risk. Advancing age is the strongest and most consistent risk factor for Parkinson's disease. Moreover, underlying biological mechanisms such as oxidative stress and mitochondrial dysfunction are believed to play a key role in neuronal damage and the progression of the disorder.

V. SIGNS AND SYMPTOMS

A. Motor Symptoms

- Resting tremor
- Bradykinesia
- Muscle rigidity
- Postural instability
- Gait disturbances

B. Non-Motor Symptoms

- Cognitive impairment
- Depression and anxiety
- Sleep disturbances
- Autonomic dysfunction
- Sensory abnormalities

VI. DIAGNOSIS

The diagnosis of Parkinson's disease is largely based on clinical assessment, including a detailed medical history and thorough neurological examination. A key requirement for diagnosis is the presence of bradykinesia along with either resting tremor or muscle rigidity. Additional features that support the diagnosis include an unequal involvement of symptoms on the two sides of the body and noticeable improvement following levodopa treatment. Imaging studies and laboratory tests are not routinely used to confirm Parkinson's disease but are helpful in ruling out alternative neurological disorders.

VII. TREATMENT STRATEGIES

A. Pharmacological Treatment

Levodopa continues to be the most effective therapy for controlling motor symptoms in Parkinson's disease and is widely regarded as the cornerstone of treatment. It is typically prescribed in combination with a peripheral decarboxylase inhibitor, such as carbidopa, to enhance its effectiveness and reduce peripheral side effects. Other pharmacological options, including dopamine agonists, monoamine oxidase-B (MAO-B) inhibitors, catechol-O-methyltransferase (COMT) inhibitors, and amantadine, may be used either alone or in combination with levodopa. The choice of therapy is guided by the stage of the disease, symptom severity, and individual patient needs.

B. Management of Non-Motor Symptoms

Management of non-motor symptoms in Parkinson's disease is tailored to each patient and focuses on improving overall quality of life. Pharmacological interventions may include antidepressants, anxiolytics, sleep-promoting agents, and medications aimed at managing autonomic disturbances. In addition to drug therapy, a multidisciplinary care approach is essential. Support from physiotherapy, occupational therapy, speech therapy, and nutritional counseling plays a crucial role in addressing functional limitations and enhancing long-term patient outcomes.

VIII. HOSPITAL-BASED SURVEY OBSERVATIONS

Findings from hospital-based surveys involving patients with Parkinson's disease indicated a slow and progressive onset of symptoms, with motor impairments being the most prominent clinical

features. These symptoms were found to have a considerable effect on patients' ability to perform daily activities. The majority of patients were treated with levodopa-centered therapeutic regimens, often supplemented with medications such as trihexyphenidyl and amantadine to improve symptom control. Interactions with patients highlighted the critical role of timely diagnosis, early initiation of treatment, and the need for regular follow-up to achieve better disease management and clinical outcomes.

IX. CHALLENGES AND FUTURE PERSPECTIVES

The management of Parkinson's disease continues to face several important challenges, including the absence of dependable biomarkers for early detection, the limited availability of therapies that can slow or halt disease progression, and significant differences in how patients respond to treatment. Current research efforts are increasingly directed toward the development of neuroprotective approaches, advances in regenerative medicine, and the adoption of personalized treatment strategies. These evolving areas of research hold promise for improving long-term clinical outcomes and enhancing the quality of life for individuals living with Parkinson's disease.

X. CONCLUSION

Parkinson's disease is a multifaceted neurodegenerative condition that presents with a combination of motor and non-motor symptoms, significantly affecting patients' daily lives. Timely diagnosis, well-planned pharmacological treatment, and a multidisciplinary approach to care are crucial in managing the disease and enhancing quality of life. Ongoing research aimed at understanding the underlying disease processes and developing innovative therapeutic options offers hope for improved disease management and the possibility of more effective treatments in the future.

References

11. Lim, S.Y. and Lang, A.E. (2010) 'The nonmotor symptoms of Parkinson's disease-An overview', *Movement Disorders*, 25(SUPPL. 1). <https://doi.org/10.1002/mds.22786>.

12. Picillo, M. et al. (2016) 'Gender and non-motor fluctuations in Parkinson's disease: A prospective

1. Armstrong, M.J. and Okun, M.S. Diagnosis and Treatment of Parkinson Disease: A Review. *JAMA*, 2020.

2. Bloem, B.R., Okun, M.S. and Klein, C. Parkinson's disease. *The Lancet*, 2021.

3. Beitz, J.M. Parkinson's disease: A review. *Frontiers in Bioscience*, 2014.

4. Dar, P.A. et al. Catalepsy: A scientific model for Parkinsonism. 2012.

5. Dorsey, E.R. et al. The emerging evidence of the Parkinson pandemic. *Journal of Parkinson's Disease*, 2018.

6. Fullard, M.E. et al. (2017) 'Utilization of rehabilitation therapy services in Parkinson disease in the States', *Neurology*, <https://doi.org/10.1212/WNL.0000000000004355>. 89(11), pp. 1162–1169.

7. Gazewood, J.D., Richards, D.R. and Clebak, K. (2013) 'Parkinson disease: An update', *American Family Physician*, 87(4), pp. 267–273.

8. Greenamyre, J.T. et al. (2010) 'Lessons from the rotenone model of Parkinson's disease', *Trends in Pharmacological Sciences*, 31(4), pp. 141–142. <https://doi.org/10.1016/j.tips.2009.12.006>.

9. Gubellini, P. et al. (2006) 'Chronic high-frequency stimulation of the subthalamic nucleus and L-DOPA treatment in experimental parkinsonism: Effects on motor behaviour and striatal glutamate transmission', *European Journal of Neuroscience*, 24(6), pp. 1802–1814. <https://doi.org/10.1111/j.1460-9568.2006.05047>.

10. Jain, S. (2011) 'Multi-organ autonomic dysfunction in Parkinson disease', *Parkinsonism and Related Disorders*, 17(2), pp. 77–83. <https://doi.org/10.1016/j.parkreldis.2010.08.022>

Parkinsonism and Related Disorders, 27, pp. 89–92. <https://doi.org/10.1016/j.parkreldis.2016.04.001>.

13. Politis, M. et al. (2010) 'Parkinson's disease symptoms: The patient's perspective', *Movement Disorders*, 25(11), pp. 1646–1651. <https://doi.org/10.1002/mds.23135>.

14. Postuma, R.B., Gagnon, J.F. and Montplaisir, J. (2010) 'Clinical prediction of Parkinson's disease: Planning for the age of neuroprotection', *Journal of Neurology, Neurosurgery and Psychiatry*, 81(9), pp. 1008–1013.
<https://doi.org/10.1136/jnnp.2009.174748>

