Epidemiology of Parkinson Disease

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KEYWORDS

• Parkinson disease • Epidemiology • Neuroepidemiology
• Neurodegenerative disorder

KEY POINTS

- Parkinson disease (PD) is the second most common neurodegenerative disorder, presenting with bradykinesia, rigidity, tremor, and postural instability.
- Incidence rates of PD are 8 to 18 per 100,000 person-years based on prospective population-based studies with either record-based or in-person case finding.
- Nonmotor symptoms include autonomic dysfunction, sleep disorders, mood disorders, cognitive abnormalities, and pain and sensory disorders. Hallucinations and dementia predict later nursing home placement.
- There are 18 PD-related gene loci that have been identified to date, with at least 7 disease-causing genes.
- PD is a very complicated condition for physicians to optimally manage, with specific rehabilitation needs and complicated psychosocial dynamics, all that evolve with time.

INTRODUCTION

Epidemiology

Parkinson disease (PD) is the second most common neurodegenerative disorder after Alzheimer disease and refers to the clinical presentation of bradykinesia, rigidity, tremor, and postural instability. The main known risk factor is increased age. The estimated prevalence of PD in industrialized countries is 0.3% in the general population, 1.0% in people older than 60 years, and 3.0% in those aged 80 years and older, with incidence rates of PD of 8 to 18 per 100,000 person-years based on prospective population-based studies with either record-based or in-person case finding.[1–3] The median age of onset is 60 years; the mean duration of the disease from diagnosis

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to death is 15 years, although patients can live for decades with PD.[4,5] Sex differences exist in PD.[6] In addition to older age, male sex is recognized as a prominent risk factor in developing PD. Both incidence and prevalence of PD are 1.5 to 2.0 times higher in men than in women.[7] Age at onset is 2.1 years later in women (53.4 years) than in men (51.3 years). Women present with a milder PD phenotype, as evidenced by higher presentation of tremor (67%) than men (48%) and a slower rate of motor impairment. Animal studies suggest that estrogen may play a neuroprotective role against cell death of striatal dopaminergic neurons. Nonmotor symptoms that are more prevalent in women are anxiety, depression, and constipation, whereas men suffered more from daytime sleepiness, drooling, and sexual symptoms.[8]

Natural History

The natural history of disability and progression of motor symptoms in PD has been well studied since the seminal article by Hoehn and Yahr in 1967.[9] The Hoehn and Yahr (H&Y) scale is the most commonly used system for describing the progression of PD. The transition from H&Y scale stage II to stage III is considered a pivotal milestone in PD when gait and balance impairment result in disability in many gait-dependent activities, such as walking, dressing, bathing, and housework. In one longitudinal study, the time to reach H&Y scale III was 7.73 years.[10] Male sex, gait disorder, lack of tremor, and lack of asymmetry as presenting clinical features are associated with poorer long-term survival.[11]

Clinical Subtypes

Subtypes of PD have emerged, with patients classified according to distinct clinical features, such as motor phenotype, or age of onset.[12] Patients presenting with tremor at onset have a slower progression of disease than those with a postural-instability-gait difficulty (PIGD) phenotype.[13] The PIGD form of PD is associated with a faster rate of cognitive decline and a higher incidence of dementia, whereas those with tremor-predominant PD start to show signs of dementia only after PIGD symptoms develop.[14] Patients with late-onset PD (aged >60 years) are often characterized by the PIGD subtype,[15] whereas young-onset PD (aged 20–40 years) presents more often with tremor, rigidity, dystonia and a higher rate of levodopa-related motor complications, such as dyskinesias.[12]

Nonmotor Symptoms

In addition to motor dysfunction, nonmotor symptoms (NMSs) of PD are recognized to play an extremely important role in adversely affecting the quality of life of patients with PD and may precede the formal diagnosis by decades.[17] The Braak hypothesis[16] suggests that Lewy bodies, the cellular abnormalities seen in neurons of Parkinson diseased brains, are found in multiple brain stem nuclei, causing non motor symptoms prior to their appearance in the areas of the brain that cause motor symptoms. In recent studies, at least one NMS was reported by almost 100% of patients with PD.[18–20] Some suggest that the NMSs of dementia and hallucinations are the strongest predictors of nursing home placement for patients with PD.[18] NMSs are broadly classified as autonomic dysfunction, sleep disorders, mood disorders, cognitive abnormalities, and pain and sensory disorders. Of these, dysautonomia, rapid eye movement (REM) sleep behavior disorder (RBD), depression, and olfactory disturbance have been shown to often predate the onset of motor symptoms of PD.

Dysautonomia

Dysautonomia associated with PD mainly consists of gastrointestinal dysfunction, genitourinary abnormalities, and cardiovascular dysfunction with orthostatic