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Parkinson's Disease: Assessment, Diagnosis, and Management

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ABSTRACT

Parkinson's disease (PD) is a progressive, chronic, neurodegenerative condition. The estimated prevalence of PD in the United States is 0.3%. Prevalence is estimated to be as high as 5% in people 85 years and older. Etiologic factors include genetics and environmental conditions. Pathophysiology consists of a loss of dopamine-producing neurons and a reduction in dopamine. Clinical presentation includes primary motor, secondary motor, and nonmotor symptoms. Diagnosis is primarily a clinical diagnosis. Clinical management consists of early and late medical management and quality of life interventions. Surgical intervention consists of deep brain stimulation.

Keywords: motor complications, movement disorder, neurodegenerative disorder, Parkinson's disease, parkinsonism

Parkinson's disease (PD) is a progressive, chronic, incurable, neurodegenerative condition. PD is the 2nd most collective neurodegenerative disease after Alzheimer disease. In the United States, PD prevalence is estimated at 0.3% of the population. The prevalence of PD increases as age increases, with an estimated prevalence of 5% in people 85 years and older.[1,2] As our US population increases in age, the likelihood of nurse practitioners (NPs) encountering PD patients in various clinical practice sites increases. Therefore, this article provides NPs with an overview of PD assessment, diagnosis, and management practices to facilitate the clinical management of this patient population.

ETIOLOGY

The etiology of PD remains unknown in most patients. PD is considered a nonhereditary disorder with etiologic associations to environmental factors.[3] The associated environmental risk factors include exposure to well water, pesticides, herbicides, industrial chemicals, wood pulp mills, farming, and rural residence. The association between PD etiology and environmental factors is weak but indicates that they are an important contributing agent that needs to be considered in etiologic PD risk assessment.

PD is not considered a hereditary condition; however, genetic risk factors should be considered as an etiology, along with environmental factors.[4] Several genes have been linked to PD, a condition in which there is an interaction of environmental factors and genetic factors, which may promote etiologic development.

The first gene linked to PD was alpha-synuclein in the 1990s. Alpha-synuclein gene mutation may be an etiologic factor in PD. Genetic mutations are known to cause disease or support the development of a disease's clinical manifestation.[5] About 15% of PD patients have a positive family history for PD, known as familial cases. Familial PD cases may be caused by mutations in the *LRRK2*, *PARK2*, *PARK7*, *PINK1*, or *SNCA* gene or by alterations in other genes that have not yet been identified. In addition, alterations in certain genes, including *GBA* and *UCHL1*, do not cause PD but appear to modify the risk of developing PD in some families. NPs should consider both the environmental and genetic etiologic risk factors in PD patients' history and clinical assessment.

PATHOPHYSIOLOGY

The main brain structures affected by PD are the substantia nigra pars compacta and the basal ganglia. The basal ganglia controls fine motor movements, and is composed of numerous subcortical nuclei, including the striatum, amygdaloid body, and claustrum. PD symptoms are a result of a loss of dopamine-producing neurons from the substantia nigra pars compacta. The loss of these neurons results in a deficiency of dopamine, a neurotransmitter in the striatum. Dopamine is vital to movement because it promotes the transmission of messages that initiate and control movement and balance. At the time of clinical presentation of PD when motor and balance symptoms occur, up to 70%-80% of the dopamine neurotransmitters may already be lost. Casey[6] proposed that the pathology underlying PD may not be restricted to the substantia nigra or dopamine loss. Gazewood et al[7] suggested that pathologic changes may be detected up to 20 years before the onset of motor symptomatology.

The aging process may be an accentuating or contributing pathophysiologic factor in PD clinical presentation. Dopamine levels decrease as a person ages; therefore, the reduction in dopamine from the aging process and pathophysiologic changes associated with PD may facilitate symptom advancement and support the development of new PD symptoms.

Clinical Presentation

The clinical presentation of PD consists of primary and secondary motor symptoms. In addition, PD clinical presentation may include nonmotor symptoms.

Primary Motor Symptoms

PD is challenging to diagnose in the early stages because the first signs and symptoms are often subtle and vague. There are 4 cardinal symptoms of PD known through the acronym TRAP: Tremor at rest, Rigidity, Akinesia (or bradykinesia), and Postural instability.[8] PD patients often present with diverse lifestyles and profiles; therefore, motor and nonmotor signs and symptoms should be evaluated in the context of each patient's needs and goals.[9]

Resting tremor occurs in the early stages of the disease and is the most common and easily recognized PD symptom. Approximately 70% of PD patients experience a unilateral tremor in the hand or foot.[2] The tremor is described by the patient as "shakiness" or "nervousness" and may have an intermittent presentation. Classically, the tremor is a resting uncontrollable tremor and disappears with the use of the affected limb.[8]

Rigidity is characterized by increased resistance and often described as stiffness in the limbs.[10] Rigidity may be associated with pain. The pain often originates in the shoulder and is commonly misdiagnosed as arthritis, bursitis, or rotator cuff injury.[8]

Bradykinesia, the most characteristic symptom of PD, presents as slowness of movement.[2] Bradykinesia encompasses difficulties with planning, initiating, and executing movement and with performing sequential and simultaneous tasks. Performing repetitive movements, such as finger tapping, and activities of daily living, such as buttoning a shirt, cutting food, or brushing teeth, become difficult.[10]

Postural instability, the imbalance and loss of righting reflexes, is the most common cause of falls contributing to the risk of hip fractures in the PD population. This coordination and balance symptom is generally a manifestation of late-stage PD, which occurs after the onset of other primary motor symptoms.[10]

Secondary Motor Symptoms

In addition to the primary motor or cardinal symptoms, PD patients may exhibit a number of secondary motor symptoms known to impact activities of daily living and quality of life.[10] Dysarthria (motor speech disorder), hypophonia (soft speech), dysphagia (difficulty swallowing), and sialorrhea (drooling or excessive salivation) are frequently observed in PD patients and may be more disabling than the cardinal features. Orofacial-laryngeal bradykinesia and rigidity are thought to cause these symptoms.[8]

Freezing of gait is a secondary motor symptom not explained by bradykinesia or rigidity. With gait freezing, the PD patient will hesitate before stepping forward or experience the inability to continue movements when already in motion. The gait freezing increases the client's fall risk potential.[10]

Micrographia is the shrinkage of handwriting and progresses with increased amounts of writing. Micrographia occurs as a result of bradykinesia.[10]