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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]

Mask-like expression, a secondary motor symptom, results from a combination of rigidity and bradykinesia. The PD patient's face will appear less expressive than usual.[10]

Several patients with PD may experience unwanted movement accelerations that also pose a fall risk potential. These are body movements that occur rapidly in an uncoordinated manner. These quick movements are especially troublesome in speech and mobility. Accelerated movement presentation may also cause tachyphemia, also known as cluttering (rapid rate speech that is not understandable).[10]

Nonmotor Symptoms

Nonmotor symptoms are symptoms that do not involve movement, coordination, physical tasks, or mobility. Nonmotor symptoms are an underappreciated aspect of PD clinical presentation. Nonmotor symptoms may precede motor symptoms and a PD diagnosis by several years. Nonmotor symptoms include autonomic dysfunction, cognitive/neurobehavioral disorders, and sensory and sleep abnormalities.[8]

Autonomic dysfunction is common in the PD patient. Features include orthostatic hypotension, sweating dysfunction, sphincter dysfunction, erectile dysfunction (ED), urinary bladder retention, urinary tract infections, and constipation.[10]

Cognitive/neurobehavioral disorders may be as disabling as motor symptoms. Depression, anxiety, apathy, and hallucinations frequently occur in PD patients. PD patients may also present with dementia. As PD progresses, the brain experiences microscopic deposits of alpha-synuclein (Lewy bodies), resulting in memory changes, decreased attention ability, difficulty planning, and problems with decision making. In addition to cognitive and affective disorders, obsessive-compulsive and impulsive behaviors, such as craving, binge eating, pathologic gambling, and compulsive shopping, tend to occur in PD patients. There is also a fascination with repetitive handling, examining, sorting, and arranging of objects.[8] These symptoms are referred to as *hedonistic homeostatic dysregulation*, which is a neuropsychological behavior disorder associated with the use of dopaminergic drugs, especially a dopamine agonist. Therefore, NPs should monitor the presentation of these nonmotor symptoms when managing the PD patient with a dopamine agonist.

Sleep disturbances, specifically rapid eye movement (REM) behavior disorder, is a substantial risk factor for the development of PD. REM behavior disorder is a preparkinsonian state that occurs in approximately one third of PD patients and is characterized by violent dreams accompanied by hitting or kicking motions, yelling, screaming, and other potentially dangerous motor activity during REM sleep.[10-12]

Fatigue is a common nonspecific complaint. Fatigue can have both mental and physical causes. Muscle stiffness, depression, slow movement, insomnia, and PD medications can also cause fatigue symptoms.[10] PD patients can experience a loss of smell (anosmia). The loss of smell may occur before the onset of other PD signs and symptoms and is a strong predictor of beginning long-term cognitive problems. Table 1 summarizes primary, secondary, and nonmotor PD symptoms.

Diagnosis

Differential diagnoses to consider in patients presenting with PD clinical symptoms are essential tremors, vascular parkinsonism, drug-induced parkinsonism, dementia, progressive supranuclear palsy, and multisystem atrorphy.[7] A differential diagnosis of PD is primarily based on a clinical diagnosis. A clinical diagnosis of PD requires the presence of the cardinal signs of distal resting tremor of 3-6 Hz, rigidity, bradykinesia, and asymmetrical onset. In addition, patients diagnosed with PD must respond to an adequate therapeutic levodopa or a dopamine agonist challenge. The clinical diagnosis of PD is further supported by progressive functional and motor impairment. Both computed tomographic (CT) scanning and magnetic resonance imaging (MRI) provide a limited role in the differential diagnosis of PD because they do not show a specific PD pathologic finding. CT or MRI scans are used to facilitate the differential diagnosis of PD and rule out brain tumor or cerebral vascular accident (stroke). The US Food and Drug Administration (FDA) has approved the use of ioflupane iodine-123 injection or DaTscan (GE Healthcare B.V., Eindhoven, The Netherlands) to be used to detect dopamine transporters in suspected PD cases.

Table 1. Primary, Secondary, and Nonmotor Signs and Symptoms of Parkinson's Disease

Primary motor signs and symptoms
- Tremor at rest
- Rigidity
- Bradykinesia
- Postural instability

Secondary motor signs and symptoms
- Dysarthria
- Hypophonia
- Dysphagia
- Sialorrhea
- Dystonia
- Freezing
- Micrographia
- Mask-like expression
- Unwanted accelerations

Nonmotor signs and symptoms
- Depression
- Anxiety
- Apathy
- Hallucinations
- Obsessive-compulsive behaviors
- Impulsive behaviors

The DaTscan uses radioactive ioflupane iodine-123 to determine the amount of dopamine available in a person's brain. DaTscan is not used to diagnose PD but rather to assist with confirming a PD diagnosis. Clinical providers who do not diagnose PD frequently are encouraged to refer suspected cases to clinicians who are experienced and frequently diagnose PD as a means of reducing clinical diagnostic errors.[2]

CLINICAL MANAGEMENT

Clinical management involves motor symptom treatment, which does improve functional ability. Clinical symptomatic management may impact PD prognosis. Clinical management consists of early and late medical management. PD patients should be followed up at least every 6-12 months; however, follow-up care depends on clinical symptom progression and response to pharmacologic management.

Early Medical Management

Once patients with PD develop functional disability, the American Academy of Neurology recommends initiating early medical management. General guidelines for treatment regimens exist; however, each client must have an individual evaluation to determine the most effective initial pharmacologic agent or combination of agents that is most likely to impact the loss of dopamine in the brain and the initial presenting clinical symptoms. Levodopa, nonergot dopamine agonists, and monoamine oxidase B (MAO-B) inhibitors can be used for initial therapy.[7] Table 2 summarizes the FDA—approved medications for PD.

Levodopa, administered with carbidopa, remains the primary treatment for symptomatic patients. Carbidopa inhibits peripheral metabolism of levodopa, thereby increasing levels of cerebral levodopa to a therapeutic concentration.[7] The combination of levodopa/carbidopa (Sinemet; Bristol-Myers Squibb Pharma Company, New York, NY) is the most effective agent available to treat PD motor symptoms. The early use of levodopa/carbidopa may result in dyskinesias.[7]

Nonergot dopamine agonists pramipexole (Mirapex; Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT) and ropinirole (Requip; GlaxoSmithKline, Middlesex, UK) directly stimulate dopamine receptors in the brain and are used alone or in combination with levodopa/carbidopa to treat PD motor symptoms. These 2 pharmacologic agents are not as effective as levodopa in treating motor symptoms; however, the nonergot dopamine agonists do have a lower incidence of dyskinesias.[7]

Ergot-derived dopamine agonists cabergoline bromocriptine (Parlodel; Validus Pharmaceuticals LLC, Parsippany, NJ) and pergolide (Permax; Eli Lilly and Company, Boston, MA) should not be used as a first-line treatment because of the risk of serosal fibrosis and cardiac valvulopathies.[7] If used, baseline and annual transthoracic echocardiograms, chest x-ray, and testing of erythrocyte sedimentation rate and renal function are required.[7]

MAO-B inhibitors block the action of the MAO enzyme that breaks down dopamine and may be used in early medical management. Fewer incidences of dyskinesia and adverse effects occur with MAO-B inhibitors compared with levodopa/carbidopa. MAO-B inhibitors are less effective in treating motor symptoms than dopamine agonist.[7]

Table 2. Food and Drug Administration–Approved Medications for Parkinson's Disease

Drug Class/Drug[a]	Indication	Adverse Effects
Carbidopa/Levodopa	Primary treatment for symptomatic PD; improves ability to complete activities of daily living	Dyskonesias, somnolence, confusion, dystonia
Dopamine agonists		
Nonergot: Pramipexole (Mirapex); Ropinirole (Requip)	Useful for early disease, can be used as monotherapy	Nausea, vomiting, daytime sleepiness, hallucinations, orthostatic hypotension
Ergot: Bromocriptine (Parlodel); Pergolide (Permax)	Useful for early disease, less risk of developing motor complications	Pulmonary fibrosis, cardiac valve fibrosis, erythromelalgia
MAO-B inhibitors		
Selegiline (Eldepryl); Rasagaline (Azilect)	Useful to treat motor complications in late disease, may be used as monotherapy early in disease	Risk of serotonin syndrome
COMT inhibitors		
Entacapone (Comtan); Tolcapone (Tasmar)	Useful for treating motor complications in patients taking Levodopa, decreases "off time," mild improvement in activities of daily living	Diarrhea, exacerbates Levodopa adverse effects, Tolcapone is associated with fatal liver toxicity, urine discoloration: red, orange, or brown
Injectable dopamine agonist		
Apomorphine (Apokyn)	Reduces "off time" in late disease	Tachycardia, chest pain, dizziness, sleepiness, regular subcutaneous injections
NMDA receptor inhibitor		
Amantadine (Symmetrel)	Useful for treating dyskinesias in late disease	Hallucinations, edema, hypotension
Anticholinergics		
Benztropine (Cogentin); Trihexyphenidyl (Artane)	Useful for treatment of tremors in cognitively functioning patients < 60 years old	Dry mouth, dry eyes, constipation, hypotension, cognitive impairment, urinary retention

NMDA = N-methyl-D-asparate; PD = Parkinson's disease.

[a] Trade names: Apokyn (Mylan Lab, Canonsburg, PA); Artrane (TAJ Pharmaceuticals Ltd., Mumbai, India); Azilect (Teva Pharmaceutical Industries Ltd., North Wales, PA); Cogentin (Qualtest Pharmaceuticals, Huntsville, AL); Comtan (Novartis Pharmaceuticals Corporation, East Hanover, NJ); Eldepryl (Apotex Corp., Weston, FL); Mirapex (Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT); Parlodel (Validus Pharmaceuticals LLC, Parsippany, NJ); Permax (Eli Lilly and Company, Boston, MA); Requip (GlaxoSmithKline, Middlesex, UK); Symmetrel (Lannett Company, Inc., Philadelphia, PA); Tasmar (Valeant Pharmaceuticals International, Inc., Steinbach, MB, Canada).

Late Medical Management

As PD progresses, additional motor complications, such as dyskinesias and motor fluctuations, develop, and initial medical management becomes no longer as effective. "On time" refers to the time when medication is effectively controlling the disease's symptoms, and "off time" occurs when disease symptoms recur gradually or abruptly. As PD progresses, on time becomes shorter, causing complications that can impair quality of life.[7]

Dopamine agonists, nonergot dopamine agonists, MAO-B inhibitors, and catechol O-methyltransferase (COMT) inhibitors may be used as adjunctive therapy to decrease off time. COMT inhibitors tolocapone (Tasmar; Valeant Pharmaceuticals International, Inc., Steinbach, MB, Canada) and entacapone (Comtan; Novartis Pharmaceuticals Corporation, East Hanover, NJ) are administered with levodopa and prolong symptom relief. The COMT inhibitor tolcapone (Tasmar) is rarely prescribed because it is associated with fatal hepatotoxicity.[7] A Cochrane review concluded that dopamine agonists were most effective with reducing off time.[13]

Surgery

Despite optimal medical management, most PD patients will develop disabling symptoms that may make them a candidate for deep brain stimulation (DBS). Candidacy for DBS is a careful and considerate process. The PD patient must be screened for depression and suicidal ideation before DBS therapy is considered an option. DBS assists with dyskinesia and gait freezing.

DBS targets either the subthalamic nucleus or the globus pallidus interna to effectively improve motor function and reduces motor fluctuations and dyskinesias. DBS may or may not decrease antiparkinson medication use (may or may not increase on time). DBS relief of motor symptoms may improve quality of life. DBS does not slow pathologic progression but impacts clinical symptom presentation.[7]

Alternative Therapy Management

Alternative therapies may provide symptomatic relief to the PD patient. Acupuncture is widely used and may have symptomatic benefits for motor and nonmotor symptoms.[14] Manual therapy and exercise interventions (aerobic exercise), including chiropractic manipulation, osteopathic manipulation, and Trager therapy, may benefit patients with PD; however, no research findings refute or confirm this position.[14] Trager therapy is a type of massage that involves rhythmic touch and movement exercises. The goal of Trager therapy is to generate positive feelings, connect mind and body, promote relaxation, improve mobility, and clear the mind. Trager therapy is used in PD patients to reduce muscle rigidity.

Quality of Life and Caregiver Issues

Nonmotor symptom fatigue may occur early during PD presentation. As PD progresses, nonmotor symptoms significantly decrease quality of life. Early recognition and treatment of these symptoms will improve quality of life for both patients with PD and their caregivers. Table 3 presents a list of resources to assist both the PD patient and primary caregiver.

Orthostatic hypotension, ED, urinary incontinence, and constipation may appear late in PD progression. These symptoms negatively impact the PD patient's quality of life. The PD patient must exercise caution to reduce the incidence of falls that further reduce their ability to remain physically active and socially engaged. This fall risk increases the need for the primary caregiver to remain aware of the environmental conditions that may increase the PD patient's fall potential. Interventions to impact the fall risk potential include activities such as using ambulation aids like laser canes or walkers, eliminating moveable rugs from the client's environment, reducing glare and clutter in the home, using nonskid surfaces, placing handrails along stairs/steps and in the bathroom, raising the toilet seat level, and lowering the bed level.

Freezing during movement is associated with increasing the PD client's fall risk. Interventions to impact fall risk associated with freezing includes weight shifting from 1 leg to the next while in motion, listening to music and stepping with the rhythm, focusing on a floor target to step on, and imagining a line on the floor to step on while walking.

ED also impacts both PD patients' and their significant others' sexual health. Clients experiencing ED can be treated with different modalities, such as cognitive/behavioral and pharmacologic interventions. ED management consists of sexual counseling, vacuum pump devices, penile implantation, and pharmacologic management, such as sildenafil and apomorphine.

Depression is another symptom that compounds the impact of PD symptoms on the patient's quality of life. The Beck Depression Inventory may be used as a screening tool for patients in whom depression is suspected. The common treatments for depression,

Table 3. Parkinson's Disease Resources

- The Michael J. Fox Foundation for Parkinson's Research https://www.michaeljfox.org.
- Parkinson's Disease Foundation http://www.pdf.org.
- National Parkinson Foundation http://www.parkinson.org.
- The Parkinson Alliance http://www.parkinsonalliance.net.
- American Parkinson Disease Association http://www.apdaparkinson.org.
- Davis Phinney Foundation http://www.davisphinneyfoundation.org.

cognitive behavioral therapy and pharmacologic therapy, should also be implemented in the PD patient. Pharmacologic therapies include the selective serotonin reuptake inhibitors citalopram (Celexa; Forest Pharmaceuticals, Inc., St. Louis, MO), sertraline (Zoloft; Pfizer, New York, NY), and fluoxetine (Prozac; Eli Lilly and Company) and the serotonin and norepinephrine reuptake inhibitors venlafaxine (Effexor; Pfizer) and duloxetine (Cymbalta; Eli Lilly and Company). The tricyclic antidepressants should be used with caution in treating depression in PD patients. Tricyclic antidepressants can cause anticholinergic adverse effects and exacerbate orthostatic hypotension.[15]

The ability to communicate may negatively impact quality of life. Speech therapy is frequently used to treat the development of dysarthria. Individual speech therapy aimed at phonatory efforts may improve speech volume and improve the client's ability to communicate.

The Lee Silverman Voice Treatment (LSVT) is to increase vocal intensity in PD patients. LSVT focuses on a set of voice exercises that are practiced intensively, 4 sessions per week during a 4-week period. The goal of LSVT is to improve vocal performance by maximizing vocal effort ("think loud, think shout") and sensory perception of vocal effort. LSVT has resulted in normal speech and voice quality in some patients.

CONCLUSIONS

PD is a progressive, chronic, incurable, neurodegenerative disorder that can be clinically diagnosed and managed symptomatically. Clinical management focuses on decreasing loss of dopamine in the brain, preserving functional state, and managing associated symptoms, such as ED and cognitive deficits. In addition, clinicians must focus on the risk reduction strategies for PD, such as fall prevention. Clinicians are capable of positively impacting the clinical management of PD symptoms and improving quality of life. JNP

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