

Parkinson Disease: An Overview for the Management Techniques Via Physical Therapy

Abdulaziz Ali Hasan Alameer¹, Muhammed Ali Mofareh Alhuraysi², Mohammad Ismail Alhazmi³, Wael Abdulghani Mohammed Al-Ghamdi⁴, Saeed Abdullah Aziz Alshehri⁵, Sultan Mohammed Maashi⁶, Khalid Hassan Ali Al-Ghashmari⁷

Abstract

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by motor symptoms such as bradykinesia, resting tremor, and rigidity, as well as non-motor symptoms including sleep disturbances, mood disorders, and autonomic dysfunction. It affects approximately 1% of individuals aged 60 and older, with both genetic and environmental factors contributing to its etiology. The pathophysiology involves the degeneration of dopaminergic neurons in the substantia nigra and the accumulation of alpha-synuclein in Lewy bodies. This article provides an overview of PD, focusing on its clinical presentation, pathophysiology, and management strategies, with an emphasis on the role of physical therapy in improving functional outcomes and quality of life. The review synthesizes current literature on PD, including its epidemiology, etiology, histopathology, and clinical features. It also examines evidence-based management techniques, particularly the integration of physical therapy into multidisciplinary care. PD is diagnosed clinically, with physical therapy playing a critical role in addressing motor and non-motor symptoms. Key interventions include gait and balance training, strength and flexibility exercises, and task-specific activities. Physical therapy has been shown to improve mobility, reduce fall risk, and enhance overall well-being, though long-term adherence remains a challenge. Non-motor symptoms, such as depression and autonomic dysfunction, require a holistic approach involving pharmacological and non-pharmacological strategies. PD is a multifaceted disorder requiring comprehensive management. Physical therapy is a vital component of care, offering significant benefits in improving motor function and quality of life. However, sustained outcomes depend on ongoing support and adherence to exercise programs. A multidisciplinary approach, including physical therapists, physicians, and other healthcare professionals, is essential for optimizing patient outcomes.

Keywords: Parkinson's Disease, Physical Therapy, Bradykinesia, Alpha-Synuclein, Gait Disturbances, Multidisciplinary Care.

Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder primarily characterized by motor symptoms such as bradykinesia (generalized slowing of movements), often accompanied by resting tremor or rigidity [1][2][3]. In addition to these core features, non-motor symptoms are also prevalent, including olfactory dysfunction, sleep disturbances, mood disorders, excessive salivation, constipation, and rapid eye movement (REM) sleep behavior disorder, which involves abnormal limb movements during sleep [1][2][3]. These non-motor manifestations can significantly impact patients' quality of life and may even precede the onset of motor symptoms. Epidemiologically, PD affects approximately 1% of individuals aged 60 and above, making it one of the most common neurodegenerative disorders in the elderly population [1]. The pathophysiology of PD involves the degeneration of dopaminergic neurons in the substantia nigra, a region of the brain critical for motor control, along with the presence of Lewy bodies, which are abnormal protein aggregates [1]. While the majority of cases are idiopathic, approximately 10% of patients have a genetic predisposition, with these cases typically manifesting at a younger age [1]. Clinically, PD has an insidious onset and progresses gradually. Tremor is frequently the initial symptom, later accompanied by bradykinesia and rigidity [1]. Postural instability, a hallmark of advanced disease, often emerges in later stages and can severely impair mobility and increase the risk of falls [1]. Autonomic dysfunction, including symptoms such

¹ Ksa, Ministry of Health, Jazan Health Cluster

² Ksa, Ministry of Health, Jazan Health Cluster.

³ Ksa, Ministry of Health, Rehabilitation Center in King Fahad Hospital, Jazan.

⁴ Ksa, Ministry of Health, Aseer Central Hospital.

⁵ Ksa, Ministry of Health, Khamis Mushayt General Hospital

⁶ Ksa, Ministry of Health, Aseer Central Hospital

⁷ Ksa, Ministry of Health, King Abdul Aziz Specialist Hospital - Taif

as orthostatic hypotension and gastrointestinal issues, may also occur and can sometimes precede motor symptoms, complicating early diagnosis [1]. The diagnosis of PD is primarily clinical, relying on patient history and physical examination. In cases of diagnostic uncertainty, single-photon emission computed tomography (SPECT) scans may be utilized to differentiate PD from other neurological conditions [1]. Overall, the multifaceted nature of PD necessitates a comprehensive approach to diagnosis and management.

Etiology

The etiology of Parkinson's disease (PD) has undergone significant advancements in understanding over the past century. In 1919, post-mortem examinations revealed that the loss of pigmentation in the substantia nigra of the midbrain was a hallmark feature of PD [4][5]. By the 1950s, it was further discovered that the pigmented neurons lost in this region were dopaminergic, and the depletion of dopamine within subcortical motor pathways was identified as a critical mechanism underlying the movement disorders characteristic of PD [4][5]. This understanding highlighted the central role of the basal ganglia, a group of nuclei involved in motor control, in the pathophysiology of PD. The striatum, a key component of the basal ganglia, receives both excitatory and inhibitory inputs from various cortical regions, and the loss of dopaminergic neurons disrupts this circuitry, leading to the motor symptoms observed in PD [4][5]. Environmental factors have also been implicated in the etiology of PD. Exposure to pesticides, herbicides, and proximity to industrial plants have been linked to an increased risk of developing the disease [4][5]. Additionally, the injection of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), a chemical that accumulates in mitochondria and induces Parkinsonian-like symptoms, has provided further insights into the role of environmental toxins in PD pathogenesis [4][5]. Oxidative stress and the generation of free radicals have also been proposed as mechanisms contributing to neuronal damage, particularly in thalamic nuclei [4][5]. Genetic factors play a significant role, as the risk of PD is higher among siblings of affected individuals, with these cases often presenting at a younger age [4][5]. Pathologically, abnormal aggregation of alpha-synuclein is a major component of Lewy bodies, which are found in the brains of PD patients at autopsy. Dysfunctional alpha-synuclein is believed to contribute to the disease's etiology, and current research is focused on understanding and preventing its aggregation and propagation [4][5]. These findings underscore the multifactorial nature of PD, involving both genetic and environmental influences, and highlight the importance of ongoing research into its underlying mechanisms.

Epidemiology

Parkinson's disease (PD) has a global prevalence of 1 to 2 cases per 1,000 individuals at any given time, with the frequency rising significantly with age. Specifically, it affects approximately 1% of the population aged 60 years and older, making it one of the most common neurodegenerative disorders in the elderly [1][2]. Among patients with PD, 5% to 10% exhibit a genetic predisposition, often presenting with earlier-onset symptoms compared to idiopathic cases [1][2]. Both the incidence and prevalence of PD increase progressively with advancing age, reflecting its strong association with aging. Additionally, epidemiological studies have consistently shown that PD is more prevalent in men than in women, suggesting potential gender-related differences in susceptibility or underlying biological mechanisms [1][2]. These findings highlight the demographic and genetic factors that contribute to the distribution and burden of PD worldwide.

Pathophysiology

The pathophysiology of Parkinson's disease (PD) has been significantly advanced through genetic and molecular research. In 1999, the identification of several members of an Italian family in New Jersey with PD spurred investigations into the genetic basis of the disease [6]. Studies on families with rare autosomal dominant forms of PD revealed that the accumulation of alpha-synuclein, particularly in the substantia nigra, plays a central role in the disease's pathogenesis. This accumulation leads to the degeneration of dopaminergic neurons and a subsequent reduction in dopamine levels within the basal ganglia, which are critical for regulating muscle tone and movement [6]. The aggregation of alpha-synuclein may result from genetic mutations, such as the PARK-1 mutation identified in the Contursi kindred or may be triggered by

environmental factors that remain poorly understood [6]. Recent research has also explored the possibility of an infectious etiology contributing to PD. Evidence suggests that the earliest pathological changes in PD may originate in the myenteric plexus of the gastrointestinal tract (GIT), from where the pathology ascends to involve the dorsal motor nucleus of the vagus nerve, sleep centers in the pons, and eventually the midbrain [6]. This "gut-to-brain" hypothesis provides a plausible explanation for the early non-motor symptoms of PD, such as gastrointestinal motility issues and REM sleep behavior disorder, which often preceded motor symptoms by several years [6]. While the traditional view of PD centers on dopamine depletion in the substantia nigra, emerging evidence suggests a more complex and widespread pathophysiology. The deposition of alpha-synuclein in various brain regions, potentially influenced by both genetic and environmental factors, underscores the multifaceted nature of the disease [6]. Ongoing research continues to investigate the mechanisms underlying alpha-synuclein aggregation and its role in PD, highlighting the need for a broader understanding of the disorder beyond dopaminergic dysfunction.

Histopathology

The histopathology of Parkinson's disease (PD) is marked by distinct macroscopic and microscopic features. Macroscopically, the loss of dopamine pigment in the substantia nigra is often visible to the naked eye in post-mortem examinations of PD cases [6]. Microscopically, PD is characterized by the presence of Lewy bodies and Lewy neurites, which are neuronal inclusions composed primarily of aggregated alpha-synuclein. Lewy bodies are found within neuronal cell bodies, while Lewy neurites are observed in neuronal processes. Together, these inclusions are referred to as Lewy-related pathology and are a hallmark of PD [6]. A defining feature of neurodegenerative diseases, including PD, is selective neuronal loss, which is most pronounced in the substantia nigra pars compacta. However, Lewy pathology in PD is not confined to this region; it extends to other areas of the brain and beyond [6]. Braak and colleagues proposed a staging system for PD based on the distribution of alpha-synuclein pathology. According to this model, the earliest pathological changes occur in the dorsal motor nucleus of the vagus nerve in the medulla and the anterior olfactory nucleus in the olfactory bulb [6]. As the disease progresses, pathology spreads to the locus ceruleus in the pons and then to dopaminergic neurons in the substantia nigra. In advanced stages, the basal forebrain, amygdala, and medial temporal lobe structures become affected, with cortical areas involved in the final stages [6]. This progression suggests a predictable pattern of neurodegeneration that begins in lower brain regions and ascends to higher cortical areas. Recent findings have expanded the understanding of PD as a multisystem disorder rather than a purely central nervous system (CNS) disease. Evidence indicates that autonomic neurons in the peripheral nervous system may also be affected, supporting the notion that PD involves multiple organ systems [6]. This has fueled hypotheses about the potential role of cell-to-cell transmission of pathological alpha-synuclein or other transmissible agents. One prominent theory suggests that the disease process may begin in the gut, with pathological agents traveling retrogradely through the vagus nerve to the brain, offering a possible explanation for the early gastrointestinal symptoms often observed in PD patients [6]. These insights highlight the complexity of PD pathology and underscore the need for further research into its mechanisms and systemic effects.

History and Physical Examination

The history and physical examination of Parkinson's disease (PD) reveal a constellation of motor and non-motor symptoms that evolve over time. One of the earliest and most recognizable features of PD is a resting tremor, which is typically unilateral and often prompts patients to seek medical attention. This tremor, described as a "reemerging tremor," may disappear temporarily during voluntary movements, such as picking up an object, only to reappear when the patient is at rest or distracted [6]. While tremor is a common early symptom, it is not universally present and is not required for a PD diagnosis. Bradykinesia, or slowness of movement, is a core diagnostic feature of PD. Patients often report difficulty performing routine tasks, such as buttoning clothes or walking, and may exhibit delayed responses to external stimuli. During clinical examination, bradykinesia can be assessed by asking patients to perform rapid, repetitive movements, such as tapping their finger and thumb together or tapping their foot on the floor. These tasks are typically performed slowly and with reduced amplitude in PD patients [6]. Rigidity, the third hallmark of PD, is characterized by increased muscle tone, which can be observed as "lead pipe" or "cogwheel" rigidity during physical examination. Patients often appear stiff and may struggle to rise from a chair without

assistance. Reduced arm swing while walking, particularly on one side, is another common manifestation, reflecting the asymmetric nature of PD at onset [6]. The combination of bradykinesia and rigidity can lead to additional features, such as micrographia (small, cramped handwriting), which has been retrospectively observed in historical figures like Adolf Hitler, suggesting he may have suffered from PD [6].

Gait disturbances, though typically a late-stage feature, are highly characteristic of advanced PD. Patients may exhibit a flexed posture, reduced arm swing, and festination (short, shuffling steps). Other gait abnormalities include camptocormia (severe forward bending of the trunk), retropulsion (tendency to fall backward), and turning "en bloc" (taking multiple small steps to turn). Freezing of gait, particularly when encountering obstacles like doorways, is also common. While these gait abnormalities are not early features, they are often diagnostic in later stages, though they can overlap with other conditions, such as normal pressure hydrocephalus [6]. Non-motor symptoms are integral to PD and often precede motor manifestations. Patients are frequently questioned about constipation, drooling, mood disorders (particularly depression), REM sleep behavior disorder, and anosmia (loss of smell). These symptoms reflect the widespread neurodegeneration mediated by alpha-synuclein deposition beyond the substantia nigra. Autonomic dysfunction, including orthostatic hypotension, urinary retention, difficulty swallowing, and erectile dysfunction, is also common and often resistant to treatment. Additionally, depression is prevalent in PD, and as the disease progresses, dementia and significant cognitive decline become increasingly common [6]. These features underscore the multifaceted nature of PD and the importance of a comprehensive clinical evaluation.

Evaluation

The evaluation of Parkinson's disease (PD) primarily relies on a thorough history and physical examination, focusing on identifying the characteristic motor and non-motor symptoms described earlier. Movement disorder clinics often utilize standardized tools such as the Unified Parkinson's Disease Rating Scale (UPDRS) to systematically assess and quantify various aspects of the disease, including mentation, behavior, mood, activities of daily living, tremors, motor function, and complications related to treatment [7][8][9]. However, there are no specific laboratory tests or imaging studies that definitively diagnose PD, making clinical evaluation the cornerstone of diagnosis. A critical step in the evaluation process is ruling out medication-induced parkinsonism, which can mimic idiopathic PD. Certain medications, particularly traditional antipsychotics, can cause extrapyramidal side effects that are clinically indistinguishable from PD. However, these drug-induced cases typically lack the non-motor features of PD, such as anosmia or REM sleep behavior disorder, which can help differentiate them from true PD [7][8]. Additionally, it is essential to exclude other neurodegenerative disorders that may present with similar symptoms. Alpha-synucleinopathies, such as Lewy body dementia (LBD) and multiple system atrophy (MSA), as well as tauopathies like progressive supranuclear palsy (PSP), can overlap with PD in their motor manifestations. However, each condition has distinguishing features: PSP is characterized by vertical gaze paralysis, LBD presents with early dementia and hallucinations, and MSA is marked by early autonomic dysfunction and pyramidal or cerebellar signs [7][8][9].

While clinical examination is often sufficient for diagnosing PD, imaging studies such as MRI can help exclude other conditions that may present similarly, including normal pressure hydrocephalus, subcortical stroke, or mass lesions. In select cases, a dopamine transporter (DAT) scan may be used to assess dopaminergic loss in the basal ganglia, though its interpretation can be challenging, and its routine use is not recommended [8][10][11]. A practical and widely accepted method to confirm PD is observing a clear and sustained response to levodopa treatment, which is typically not seen in other parkinsonian syndromes [8][10][11]. It is also important to differentiate PD from essential tremor, a common source of diagnostic confusion in clinical practice. Imaging studies, including MRI, can help exclude structural causes such as stroke, hydrocephalus, or Wilson disease, while lumbar puncture may be performed to rule out normal pressure hydrocephalus [7][8]. Overall, a comprehensive and systematic approach to evaluation is essential for accurate diagnosis and management of PD.

Treatment and Management

The treatment and management of Parkinson's disease (PD) involve a combination of pharmacological, non-pharmacological, and surgical interventions tailored to the patient's symptoms and disease stage. Levodopa, typically combined with Carbidopa to enhance central nervous system bioavailability and reduce side effects, remains the cornerstone of pharmacological treatment for PD [12][13][14]. Dopamine agonists such as Pramipexole and Ropinirole are often used in younger patients due to their fewer side effects, though they are less effective than levodopa. Anticholinergics or Amantadine may be prescribed if tremor is the predominant symptom, while Selegiline is used in early disease stages to provide mild symptomatic relief [12][13][14]. Most antiparkinsonian medications offer effective symptom control for 3 to 6 years, after which disease progression often leads to reduced responsiveness. Younger patients generally require more aggressive treatment compared to older individuals. A multidisciplinary approach is essential for optimal PD management. Structured physical therapy programs can improve balance, gait, and stability, enabling patients to maintain an active lifestyle. Unique interventions such as music therapy, cycling, and boxing have shown promise in addressing movement-related challenges. For instance, patients who struggle with walking may find dancing easier. Non-motor symptoms, including depression, caregiver fatigue, constipation, REM sleep behavior disorder, paranoia, and psychosis, must also be addressed, as they can arise from either the disease itself or its treatment [12][13][14].

As PD progresses, patients may develop motor complications such as "on/off" fluctuations or dyskinesias. Adjusting the pharmacokinetics of levodopa, such as using delayed-release formulations or continuous gastrointestinal infusion, can help manage these issues. Additionally, balancing medication timing and combining therapies can significantly improve symptom control [12][13][14]. For patients unresponsive to pharmacological measures, deep brain stimulation (DBS) has emerged as a promising surgical option. DBS modulates the excitatory and inhibitory signals in the subthalamic nucleus or globus pallidus, offering hope for advanced PD patients, though its long-term benefits and high costs remain areas of debate [12][13][14]. Non-motor symptoms, such as psychiatric, autonomic, and sensory issues, pose significant management challenges. Treatment strategies include Sildenafil for erectile dysfunction, Modafinil for daytime somnolence, polyethylene glycol for constipation, and levodopa for periodic limb movements during sleep. Dementia is managed with cholinesterase inhibitors, while depression and anxiety are treated with SSRIs. Psychotic symptoms may require antipsychotics or Pimavanserin, and impulse behaviors are addressed through cognitive behavioral therapy [12][13][14]. Exercise has been shown to improve gait, balance, and flexibility, though its benefits are not sustained, and the risk of falls remains high. Speech therapy may also benefit some patients. Overall, a comprehensive, individualized approach is crucial for managing the multifaceted challenges of PD.

Physical Therapy

Physical therapy (PT) plays a critical role in the comprehensive management of Parkinson's disease (PD), addressing motor and non-motor symptoms to improve functional independence, mobility, and quality of life. As PD progresses, patients experience a range of motor impairments, including bradykinesia, rigidity, postural instability, and gait disturbances, which significantly impact daily activities. Physical therapy interventions are tailored to these specific challenges, aiming to enhance movement efficiency, reduce fall risk, and maintain overall physical function. One of the primary goals of physical therapy in PD is to improve gait and balance. Gait disturbances, such as shuffling steps, festination, and freezing of gait, are common in later stages of the disease and contribute to a high risk of falls. PT programs often incorporate exercises that focus on stride length, step height, and rhythmic movement patterns. Techniques such as auditory cueing (e.g., using a metronome or music) and visual cues (e.g., stepping over lines or laser-guided pathways) have been shown to improve gait parameters and reduce freezing episodes. Balance training, including weight-shifting exercises and activities that challenge postural stability, helps patients maintain equilibrium and reduces the likelihood of falls [15][16].

Strength and flexibility training are also integral components of PT for PD. Muscle rigidity and bradykinesia often lead to decreased range of motion and muscle weakness, further limiting mobility. Stretching exercises target tight muscles, particularly in the trunk and limbs, while resistance training helps maintain muscle

strength and endurance. These interventions not only improve physical function but also counteract the secondary effects of immobility, such as joint stiffness and contractures [15][16]. Task-specific training is another key element of PT for PD. This approach involves practicing functional activities, such as rising from a chair, turning, or reaching for objects, to improve motor control and coordination. By repeatedly performing these tasks, patients can relearn movement strategies and enhance their ability to perform daily activities independently. Additionally, aerobic exercise, such as cycling, walking, or dancing, has been shown to improve cardiovascular fitness, motor function, and overall well-being in PD patients. Dance therapy, in particular, combines physical activity with cognitive engagement, offering both motor and psychosocial benefits [15][16].

Physical therapy also addresses non-motor symptoms, such as fatigue and depression, which are common in PD. Regular physical activity has been shown to reduce fatigue and improve mood, contributing to a better quality of life. Furthermore, PT programs often include education on energy conservation techniques and strategies to manage fatigue during daily activities [15][16]. Despite the benefits, the effects of physical therapy in PD are not always sustained, and ongoing exercise is essential to maintain improvements. Home exercise programs, guided by physical therapists, encourage patients to remain active and continue practicing the skills learned during therapy sessions. However, adherence to these programs can be challenging, particularly as the disease progresses and motivation declines [15][16]. In conclusion, physical therapy is a vital component of PD management, addressing both motor and non-motor symptoms to enhance functional independence and quality of life. Through targeted interventions such as gait and balance training, strength and flexibility exercises, task-specific practice, and aerobic conditioning, physical therapists help patients navigate the physical challenges of PD. While the benefits of PT are well-documented, maintaining long-term adherence to exercise programs remains a challenge, underscoring the need for ongoing support and motivation for PD patients.

Differential Diagnosis

Parkinson's disease (PD) must be differentiated from other conditions that present with similar motor and non-motor symptoms. Essential tremor is a common mimic, characterized by action tremor rather than the resting tremor seen in PD. Huntington's chorea, a genetic disorder, presents with choreiform movements and cognitive decline, distinguishing it from PD. Dementia with Lewy bodies (DLB) shares pathological features with PD, such as Lewy body formation, but is distinguished by the early onset of dementia and hallucinations. Progressive supranuclear palsy (PSP) presents with vertical gaze palsy, postural instability, and early falls, which are less common in early PD. Neuroacanthocytosis, a rare disorder, is associated with acanthocytes on blood smear and orofacial dyskinesias. Normal pressure hydrocephalus (NPH) presents with the triad of gait disturbance, urinary incontinence, and cognitive decline, often distinguishable from PD by imaging findings [17][18].

Prognosis

The progression of PD varies widely among individuals and can be influenced by several factors. Males with postural instability or gait difficulties at diagnosis tend to experience faster disease progression. Older age at onset, the presence of dementia, and poor response to dopaminergic medications are associated with earlier nursing home admission and reduced survival. Conversely, patients presenting primarily with tremor often have a more benign and protracted disease course. Those diagnosed at an older age with hypokinesia or rigidity typically experience rapid progression. Within ten years of diagnosis, most patients develop significant disability. The mortality rate in PD is three times higher than that of the general population, and while treatment can alleviate symptoms, the overall quality of life remains poor for many patients [17][18].

Complications

PD is associated with numerous complications that significantly impact patients' lives. Depression is common, affecting up to 50% of patients, and can exacerbate motor and cognitive symptoms. Dementia develops in approximately 80% of patients over the course of the disease, leading to profound cognitive decline and functional impairment. Laryngeal dysfunction, including dysphonia and dysphagia, increases

the risk of aspiration pneumonia, a leading cause of mortality in PD. Autonomic dysfunction manifests as orthostatic hypotension, constipation, urinary dysfunction, and sexual dysfunction, further reducing quality of life. Kyphosis, a postural deformity, can lead to cardiopulmonary impairment due to restricted lung capacity and increased strain on the cardiovascular system. These complications highlight the multifaceted nature of PD and the need for comprehensive, multidisciplinary care [17][18]. In summary, PD requires careful differentiation from other neurodegenerative and movement disorders. Prognosis varies based on clinical features, with older age, dementia, and gait instability predicting a more rapid decline. Complications such as depression, dementia, autonomic dysfunction, and postural deformities contribute to the high morbidity and mortality associated with the disease, underscoring the importance of early intervention and holistic management.

Postoperative and Rehabilitation Care

Patients diagnosed with Parkinson's disease (PD) require long-term follow-up due to the progressive nature of the disease and the emergence of motor and non-motor complications. Medication adjustments are often necessary to manage evolving symptoms, such as bradykinesia, rigidity, and tremor. Additionally, many patients develop neuropsychiatric complications, including impulsivity, psychosis, paranoia, and excessive daytime somnolence, which require careful monitoring and intervention. Rehabilitation programs, including physical therapy, occupational therapy, and speech therapy, are essential to maintain functional independence and address gait disturbances, postural instability, and communication difficulties. A multidisciplinary approach ensures comprehensive care tailored to the patient's changing needs [15][16].

Consultations

Effective management of PD involves consultations with a diverse interprofessional team, including:

- Psychiatrist: To address mood disorders, psychosis, and impulse control disorders.
- Neurosurgeon: For evaluation of deep brain stimulation (DBS) or other surgical interventions.
- Speech therapist: To manage dysphonia, dysphagia, and communication challenges.
- Urologist: For autonomic dysfunction-related urinary issues.
- Dietitian: To address nutritional needs, weight loss, and constipation.
- Gastroenterologist: For gastrointestinal motility disorders.
- Otolaryngologist: To evaluate and manage laryngeal dysfunction.
- Physical therapist: To improve mobility, balance, and gait [15][16].

Other Issues

Guidelines for managing non-motor symptoms in PD include:

- Sildenafil for erectile dysfunction.
- Polyethylene glycol for constipation.
- Modafinil for excessive daytime somnolence.
- Methylphenidate for fatigue. These interventions aim to improve quality of life and address the diverse manifestations of PD [15][16].

Enhancing Healthcare Team Outcomes

PD is the most common motor disorder in the U.S., and its progressive nature necessitates an interprofessional team approach to optimize patient outcomes. Physicians, nurses, pharmacists, social workers, and physical therapists each play a critical role in managing the multifaceted challenges of PD. Nurses are particularly vital in educating patients and families, monitoring daily living difficulties, assessing fall risks, and providing psychosocial support. They also facilitate referrals to appropriate specialists and therapists. Social workers address psychosocial issues, including personality changes, depression, and cognitive decline, while also assisting with end-of-life planning, financial concerns, and disability applications [15][16]. Pharmacists are essential in monitoring medication regimens to ensure safety and efficacy, particularly given the polypharmacy common in PD patients. They also alert clinicians to potential adverse drug reactions or interactions. This collaborative approach ensures comprehensive care, addressing both motor and non-motor symptoms while improving the overall quality of life for patients [15][16].

Outcomes

PD is a progressive, incurable disorder associated with reduced lifespan compared to the general population. Disease progression varies, but advanced stages are often marked by falls, gait difficulties, and dementia, leading to significant disability. The quality of life for most patients is poor, underscoring the importance of early intervention and holistic management. While pharmacological and surgical treatments can alleviate symptoms, the chronic and degenerative nature of PD necessitates ongoing support from an interprofessional team to address the physical, emotional, and social challenges faced by patients and their caregivers [17][18][15]. In conclusion, the management of PD requires a coordinated, patient-centered approach involving multiple healthcare professionals. By addressing the diverse manifestations of the disease and providing tailored interventions, the interprofessional team can enhance patient outcomes and improve quality of life despite the progressive nature of PD [19].

Conclusion

Parkinson's disease (PD) is a complex neurodegenerative disorder characterized by both motor and non-motor symptoms that significantly impact patients' quality of life. The progressive nature of the disease, marked by the degeneration of dopaminergic neurons and the accumulation of alpha-synuclein, necessitates a comprehensive and multidisciplinary approach to management. Physical therapy (PT) has emerged as a cornerstone of PD care, offering targeted interventions to address motor impairments such as bradykinesia, rigidity, and postural instability. Through gait and balance training, strength and flexibility exercises, and task-specific activities, PT helps patients maintain functional independence and reduce the risk of falls. Techniques such as auditory and visual cueing have proven particularly effective in improving gait parameters and reducing freezing episodes, which are common in advanced stages of the disease. In addition to motor symptoms, PT also addresses non-motor manifestations, including fatigue and depression, through regular physical activity and energy conservation strategies. However, the benefits of PT are not always sustained, highlighting the need for ongoing exercise and adherence to home-based programs. This underscores the importance of patient education and motivation, as well as the role of caregivers in supporting long-term engagement with therapeutic activities. The management of PD extends beyond physical therapy, requiring a holistic approach that includes pharmacological treatments, such as levodopa and dopamine agonists, and surgical options like deep brain stimulation for advanced cases. Non-motor symptoms, including autonomic dysfunction, sleep disturbances, and cognitive decline, further complicate management and necessitate tailored interventions. A multidisciplinary team, comprising neurologists, physical therapists, psychiatrists, and other specialists, is essential for addressing the diverse challenges posed by PD. In conclusion, while PD remains an incurable and progressive disorder, physical therapy plays a pivotal role in enhancing motor function, improving quality of life, and mitigating the impact of non-motor symptoms. The integration of PT into a comprehensive care plan, supported by a collaborative healthcare team, offers the best opportunity for optimizing patient outcomes. Future research should focus on innovative PT techniques and strategies to improve long-term adherence, ensuring that patients with PD can maintain their independence and well-being for as long as possible.

References

- Mirpour S, Turkbey EB, Marashdeh W, El Khouli R, Subramaniam RM. Impact of DAT-SPECT on Management of Patients Suspected of Parkinsonism. *Clin Nucl Med*. 2018 Oct;43(10):710-714.
- Alexoudi A, Alexoudi I, Gatzonis S. Parkinson's disease pathogenesis, evolution and alternative pathways: A review. *Rev Neurol (Paris)*. 2018 Dec;174(10):699-704.
- Kabra A, Sharma R, Kabra R, Baghel US. Emerging and Alternative Therapies For Parkinson Disease: An Updated Review. *Curr Pharm Des*. 2018;24(22):2573-2582.
- Ehgoetz Martens KA, Shine JM, Walton CC, Georgiades MJ, Gilat M, Hall JM, Muller AJ, Szeto JYY, Lewis SJG. Evidence for subtypes of freezing of gait in Parkinson's disease. *Mov Disord*. 2018 Jul;33(7):1174-1178.
- Chung SJ, Yoo HS, Lee HS, Oh JS, Kim JS, Sohn YH, Lee PH. The Pattern of Striatal Dopamine Depletion as a Prognostic Marker in De Novo Parkinson Disease. *Clin Nucl Med*. 2018 Nov;43(11):787-792.
- Gratton C, Koller JM, Shannon W, Greene DJ, Maiti B, Snyder AZ, Petersen SE, Perlmutter JS, Campbell MC. Emergent Functional Network Effects in Parkinson Disease. *Cereb Cortex*. 2019 Jun 01;29(6):2509-2523.
- Berg D, Adler CH, Bloem BR, Chan P, Gasser T, Goetz CG, Halliday G, Lang AE, Lewis S, Li Y, Liepelt-Scarfone I, Litvan I, Marek K, Maetzler C, Mi T, Obeso J, Oertel W, Olanow CW, Poewe W, Rios-Romenets S, Schäffer E, Seppi K, Heim B, Slow E, Stern M, Bledsoe IO, Deuschl G, Postuma RB. Movement disorder society criteria for clinically established early Parkinson's disease. *Mov Disord*. 2018 Oct;33(10):1643-1646.
- Creaby MW, Cole MH. Gait characteristics and falls in Parkinson's disease: A systematic review and meta-analysis. *Parkinsonism Relat Disord*. 2018 Dec;57:1-8.
- Odin P, Chaudhuri KR, Volkmann J, Antonini A, Storch A, Dietrichs E, Pirtošek Z, Henriksen T, Horne M, Devos D, Bergquist F. Viewpoint and practical recommendations from a movement disorder specialist panel on objective measurement in the clinical management of Parkinson's disease. *NPJ Parkinsons Dis*. 2018;4:14.
- Shen Y, Huang JY, Li J, Liu CF. Excessive Daytime Sleepiness in Parkinson's Disease: Clinical Implications and Management. *Chin Med J (Engl)*. 2018 Apr 20;131(8):974-981.
- Goodarzi Z, Hanson HM, Jette N, Patten S, Pringsheim T, Holroyd-Leduc J. Barriers and Facilitators for Guidelines with Depression and Anxiety in Parkinson's Disease or Dementia. *Can J Aging*. 2018 Jun;37(2):185-199.
- Rogers G, Davies D, Pink J, Cooper P. Parkinson's disease: summary of updated NICE guidance. *BMJ*. 2017 Jul 27;358:j1951.
- Biundo R, Weis L, Fiorenzato E, Antonini A. Cognitive Rehabilitation in Parkinson's Disease: Is it Feasible? *Arch Clin Neuropsychol*. 2017 Nov 01;32(7):840-860.
- Rosenquist PB, Youssef NA, Surya S, McCall WV. When All Else Fails: The Use of Electroconvulsive Therapy for Conditions Other than Major Depressive Episode. *Psychiatr Clin North Am*. 2018 Sep;41(3):355-371.
- Eggers C, Dano R, Schill J, Fink GR, Timmermann L, Voltz R, Golla H, Lorenzl S. Access to End-of-Life Parkinson's Disease Patients Through Patient-Centered Integrated Healthcare. *Front Neurol*. 2018;9:627.
- Espay AJ, Aybek S, Carson A, Edwards MJ, Goldstein LH, Hallett M, LaFaver K, LaFrance WC, Lang AE, Nicholson T, Nielsen G, Reuber M, Voon V, Stone J, Morgante F. Current Concepts in Diagnosis and Treatment of Functional Neurological Disorders. *JAMA Neurol*. 2018 Sep 01;75(9):1132-1141.
- Wanneveich M, Moisan F, Jacqmin-Gadda H, Elbaz A, Joly P. Projections of prevalence, lifetime risk, and life expectancy of Parkinson's disease (2010-2030) in France. *Mov Disord*. 2018 Sep;33(9):1449-1455.
- Ophey A, Eggers C, Dano R, Timmermann L, Kalbe E. Health-Related Quality of Life Subdomains in Patients with Parkinson's Disease: The Role of Gender. *Parkinsons Dis*. 2018;2018:6532320.
- Zafar, S., & Yaddanapudi, S. S. (2023). Parkinson disease. In *StatPearls* [Internet]. StatPearls Publishing.

مرض باركنسون: نظرة عامة على تقنيات العلاج الطبيعي

الملخص:

الخلفية: مرض باركنسون (PD) هو اضطراب تنكسي عصبي تدريجي يتميز بأعراض حركية مثل بطء الحركة، الرعاش أثناء الراحة، والتصلب، بالإضافة إلى أعراض غير حركية تشمل اضطرابات النوم، اضطرابات المزاج، والخلل الوظيفي الذاتي. يؤثر المرض على حوالي 1% من الأشخاص الذين تبلغ أعمارهم 60 عامًا أو أكثر، مع مساهمة العوامل الوراثية والبيئية في أسبابه. تتضمن الفيزيولوجيا المرضية تدهور الخلايا العصبية الدوبامينية في المادة السوداء وتراكم بروتين ألفا سينوكلين في أجسام ليوي.

الهدف: تقديم نظرة عامة على مرض باركنسون مع التركيز على الأعراض السريرية، والفيزيولوجيا المرضية، واستراتيجيات الإدارة، مع تسليط الضوء على دور العلاج الطبيعي في تحسين النتائج الوظيفية وجودة الحياة.

الطرق: تستعرض هذه المراجعة الأدبيات الحالية حول مرض باركنسون، بما في ذلك علم الأوبئة، والأسباب، والتغيرات المرضية، والخصائص السريرية. كما تناقش تقنيات الإدارة المبنية على الأدلة، وخاصة دمج العلاج الطبيعي ضمن رعاية متعددة التخصصات.

النتائج: يُشخص مرض باركنسون سريريًا، ويلعب العلاج الطبيعي دورًا حيويًا في معالجة الأعراض الحركية وغير الحركية. تشمل التدخلات الرئيسية تدريب المشي والتوازن، وتمارين القوة والمرونة، والأنشطة الموجهة للمهام. أثبت العلاج الطبيعي فعاليته في تحسين الحركة، وتقليل خطر السقوط، وتعزيز الرفاهية العامة، على الرغم من أن الالتزام طويل الأمد لا يزال يمثل تحديًا. تتطلب الأعراض غير الحركية، مثل الاكتئاب والخلل الذاتي، نهجًا شاملاً يتضمن استراتيجيات دوائية وغير دوائية.

الختامة: مرض باركنسون اضطراب متعدد الجوانب يتطلب إدارة شاملة. يُعد العلاج الطبيعي عنصرًا أساسيًا في الرعاية، حيث يوفر فوائد كبيرة لتحسين الوظيفة الحركية وجودة الحياة. ومع ذلك، تعتمد النتائج المستدامة على الدعم المستمر والالتزام ببرامج التمارين. يعد النهج المتعدد التخصصات، الذي يشمل أخصائي العلاج الطبيعي والأطباء وغيرهم من المهنيين الصحيين، ضروريًا لتحسين نتائج المرضى.

الكلمات المفتاحية: مرض باركنسون، العلاج الطبيعي، بطء الحركة، ألفا سينوكلين، اضطرابات المشي، الرعاية متعددة التخصصات.