A Comprehensive Review of Parkinson's Disease Management

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ABSTRACT

Parkinson's disease (PD) is a neurodegenerative disorder caused by the loss of dopamine-producing neurons in the brain, leading to movement issues like tremors and stiffness. The exact cause is unknown, but genetics and environmental factors are thought to be involved. While there is no cure, current treatments focus on managing symptoms through medications or surgery. This review aims to explore new ways to treat PD by looking at synthetic and natural drugs that could slow or stop disease progression. The review discusses the factors contributing to PD, such as genetics, environmental factors, mitochondrial dysfunction, and oxidative stress. It also reviews current treatment options, like levodopa and dopamine agonists, highlighting their limitations. Novel therapeutic approaches are examined, including natural compounds like mucuna pruriens and bacopa monnieri, which show promise in studies for improving dopamine production and neuroprotection. Furthermore, the study explores cutting-edge treatments like stem cell therapy, gene therapy, and optogenetics as potential future options for PD. It stresses the importance of early diagnosis and biomarker development for personalized treatment. By considering both traditional and new approaches, this review aims to advance the treatment options available for Parkinson's disease and improve outcomes for those affected by the condition.

Keywords: *Parkinson's disease (PD), dopamine, biomarker, MAO-B inhibitors, COMT inhibitor*

INTRODUCTION

Idiopathic Parkinsonism, also known as paralysis agitans or "shaking palsy," is the most prevalent type of Parkinsonism. It was initially identified by James Parkinson in 1817. The loss of pigmented, dopaminergic neurons in the substantia nigra pars compacta, along with the development of intracellular inclusions called Lewy bodies, is the pathological characteristic of Parkinson's disease.[1] Symptomatic PD is accompanied by a loss of 70–80% of these dopamine-containing neurons. In the absence of treatment, Parkinson's disease (PD) worsens over five to ten years, leading to a rigid, akinetic state when individuals are unable to take care of themselves. Complications of immobility, such as pulmonary embolism or aspiration pneumonia, commonly lead to death. The prognosis of Parkinson's disease has drastically changed due to the advent of efficient pharmaceutical treatment; in the majority of patients, good functional mobility can be maintained for many years.

Although patients who receive proper treatment have a significantly longer life expectancy, total mortality is still greater than in the general population. Numerous other brain regions, such as the cerebral cortex, hippocampus, and brainstem, are impacted by Parkinson's disease. Parkinson's disease (PD) is a degenerative disorder of the central nervous system that belongs to a group of conditions called movement disorders.[2] It is progressive, which means that its symptoms worsen with time, and chronic, which means that it lasts for a long time. People may start to have issues with movement, tremor, stiffness in the limbs or trunk of the body, or poor balance as nerve cells (neurons) in certain areas of the brain deteriorate or die. People may find it difficult to walk, talk, or perform other basic duties when these symptoms worsen.

Although certain PD cases are inherited and linked to particular genetic abnormalities, the exact origin of PD remains unknown. The majority of instances are sporadic, meaning that the illness usually does not run in families.[3] The most prevalent form of Parkinsonism, in which disorders of other causes produce features and symptoms that closely resemble Parkinson's disease, is believed to be caused by a combination of genetic susceptibility and exposure to one or more unknown environmental factors that trigger the disease.[4] There are instances when the cause of parkinsonism is known or suspected, or where the symptoms are caused by another disorder, even though the majority of cases have no known origin. Although there is currently no cure for Parkinson's disease (PD), research is being conducted, and drugs or surgery can frequently significantly reduce motor symptoms.[5]

HISTORICAL PERSPECTIVE

• In 1817, James Parkinson provided the first systematic documentation of Parkinson's disease (PD). Descriptive diagnoses have gradually given way to mechanical discoveries in the decades that have followed.

• Therapeutic Milestones: Levodopa's accidental discovery in the 1960s transformed the treatment of Parkinson's disease and laid the groundwork for dopaminergic treatments. Its shortcomings in treating the course of the disease and motor consequences, however, highlighted the necessity for creative solutions.[4,5]

• Lessons from the Past: Current treatment paradigms have been improved by failed attempts at neuroprotection and disease modification, which emphasize early intervention and foster accuracy in finding legitimate pharmacological targets.

AETIOLOGY

Parkinson's disease results in a reduction in dopamine production because of the death

or damage of neurons in the brain, especially in the substantia nigra. Movement is impacted by aberrant neuron firing patterns brought on by dopamine loss. Non-motor symptoms including weariness and blood pressure dysregulation can arise from the loss of nerve endings that produce the neurotransmitter norepinephrine in people with Parkinson's disease. It is currently unknown how exactly Lewy bodies, which are protein deposits in injured brain cells, contribute to Parkinson's disease, even though they are also observed in PD patients. Parkinson's disease has also been connected to mitochondrial dysfunction, environmental variables such exposure to chemicals, and genetic abnormalities. According to research, spontaneous cases of PD may also have changes in the genes that cause inherited disorders. The energy-producing parts of the cell, the mitochondria, contribute to Parkinson's disease (PD) by generating oxidative stress, which harms other parts of the cell. The precise cause of cell loss and death in Parkinson's disease is still unclear, despite evidence connecting a number of factors to the condition.[5,6]

PATHOGENESIS

The balance of dopamine (DA), a neurotransmitter essential to the proper operation of the extrapyramidal motor system (which regulates posture, support, and voluntary motion), is upset when dopamine-producing neurons in the substantia nigra of the midbrain degenerate. Baseline ganglia acetylcholine (Ach) It is not until 80% of the neurons in the substantia nigra are gone that symptoms appear.[2,5,6] The pathogenesis mechanism depicted in Figure 1.

CLASSIFICATION OF DRUGS USED IN PARKINSON'S DISEASE MANAGEMENT

It is of two types: -

- Synthetic drugs
- Natural drugs

Synthetic Drugs: -

Any medication used to treat Parkinson disease symptoms or other parkinsonian disorders is an anti-Parkinson medicine. Levodopa, amantadine, dopamine-receptor agonists, and the so-called COMT (catechol-O-methyltransferase) inhibitors, MAO-B (monoamine oxidase B) inhibitors, and muscarinic receptor antagonists are the main anti-Parkinson medications.[7,8,9]

Natural Drugs: -

A shortage of dopamine in the brain causes Parkinson's disease, a degenerative disorder that impairs motor skills. Although there isn't a cure, drugs and surgery can help control the symptoms. Parkinson's disease may be treated with natural therapies such as fish oil, cocoa flavanols, bacopa

monnieri, mucuna pruriens, and creatine monohydrate.[10] (Table 1)

DRUG THERAPY

Levodopa: Because dopamine is unable to get through the brain barrier, levodopa, a precursor to dopamine, is administered as a medication to treat Parkinson's disease. Levodopa undergoes decarboxylation to produce the active form, dopamine. In the gut, it is rapidly decarboxylated.[10,11] Levodopa is absorbed well by the small bowel but mostly decarboxylated in the periphery. It improves bradykinesia, rigidity, and other motor issues in Parkinson's. Psychological well-being also improves.[12,13] Effect tolerance develops over time. Anorexia, nausea, cardiovascular problems, mental disorders, hyperkinesia, and on-off phenomena are among the side effects. MAO inhibitors must be stopped prior to usage, vitamin B6 lessens their effects, and some medications can reverse the effects of levodopa.[2,9,12]

Carbidopa: Dopa decarboxylase is inhibited by carbidopa. It works to lessen the peripheral conversion of levodopa to dopamine because it cannot cross the blood-brain barrier. Therefore, when levodopa and carbidopa are administered together:

a. It can decrease the dosage of levodopa

b. It can reduce toxic side effects of levodopa

Levodopa + Carbidopa: Levodopa, sometimes referred to as L-DOPA, is the primary treatment for Parkinson's disease (PD). People with Parkinson's disease have lower levels of dopamine, which levodopa helps the brain's nerve cells manufacture. Carbidopa is frequently used in conjunction with levodopa to facilitate its delivery to the brain.[14] This combination helps reduce tremors and other motor symptoms in the early stages of PD, allowing individuals to lead active lives for longer periods. While Levodopa helps with symptoms like bradykinesia and rigidity, it may not address balance issues effectively. Some side effects of Levodopa/carbidopa include nausea, low blood pressure, restlessness, and drowsiness. Long-term use may lead to dyskinesias, which are involuntary movements.[15] To manage these side effects, dosage adjustments or additional medications like amantadine may be necessary.[16] Additionally, some people may suffer abrupt "off periods" or wearing-off effects. Due to the possibility of severe adverse effects, it is imperative that Levodopa not be stopped suddenly without medical care. In general, levodopa aids in symptom management but neither cures PD nor reverses its course.[17] The action of L+C drug are illustrated in Figure 2.

- Dopaminergic agonists

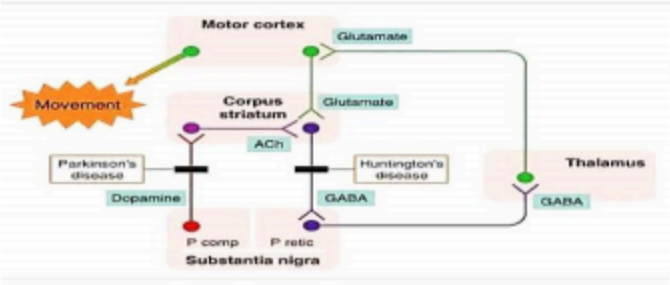

Figure 1: Pathogenesis of PD

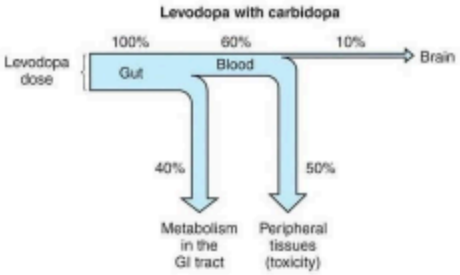

Figure 2: Action of L+C Drug

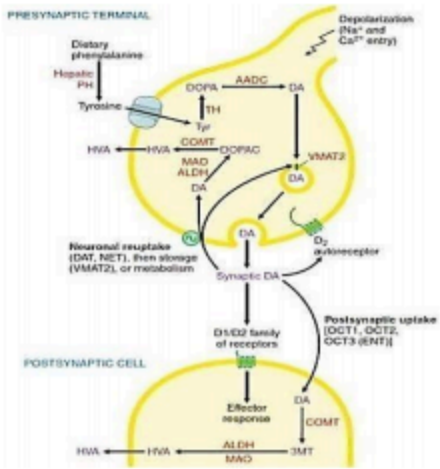

Figure 3: Drug Therapy for PD

When used alone or in combination with levodopa, dopamine agonist medications such as apomorphine, pramipexole, ropinirole, and rotigotine mimic dopamine in the brain to treat the symptoms of Parkinson's disease. They persist longer than levodopa but are marginally less effective.[16] Hallucinations, confusion, tiredness, and abrupt sleepiness are just a few of the possible side effects. When taken with levodopa, the ergot derivative bromocriptine can make you feel queasy and lightheaded. Ergot derivatives such as pergolide and cabergoline, which are used to treat parkinsonism, can also damage the heart valves. In mild and severe cases, pramipexole—which is not an ergot derivative—works well and lessens the requirement for levodopa.[17,18] While rotigotine, which is administered as a skin patch, is licensed for early Parkinson's disease but its efficacy in later stages is unknown, ropinirole, a pure D receptor agonist, is useful in moderate instances and helps with levodopa fluctuations. Mental problems, low blood pressure, nausea, and vomiting are possible side effects.[19]

- Mao-B Inhibitors

Parkinson's disease (PD) symptoms can be lessened by taking medications like selegiline and rasagiline, which block the monoamine oxidase B (MAO-B) enzyme that breaks down dopamine in the brain.

Table 1: Synthetic drugs commonly used in the treatment of PD

Classification	Generic Name	Brand Name	Uses
Dopamine Agonists	Pramipexole	Mirapex	Used to treat Parkinson's disease by stimulating dopamine receptors.
Dopamine Agonists	Ropinirole	Requip	Helps improve movement control in Parkinson's patients.
Levodopa and Carbidopa	Levodopa + Carbidopa	Sinemet	Used to increase dopamine levels in the brain, easing motor symptoms.
COMT Inhibitors	Entacapone	Comtan	Enhances the effects of levodopa therapy by preventing its breakdown.
MAO-B Inhibitors	Selegiline	Eldepryl	Increases dopamine levels in the brain by inhibiting the enzyme MAO-B.
MAO-B Inhibitors	Rasagiline	Azilect	Helps reduce the breakdown of dopamine, improving motor function.
Anticholinergics	Benztropine	Cogentin	Used to reduce tremors and rigidity associated with Parkinson's disease.
NMDA Receptor Antagonists	Amantadine	Symmetrel	Used to treat symptoms of Parkinson's, particularly dyskinesia.
Dopamine Precursor	Levodopa	Sinemet	Primary treatment for Parkinson's to replenish dopamine.
Dopamine Agonists	Apomorphine	Apokyn	Rapid relief from "off" episodes in advanced Parkinson's disease.
Adenosine A2A Antagonist	Istradefylline	Nourianz	Helps reduce Parkinson's symptoms by regulating dopamine levels.

Selegiline has been demonstrated to improve levodopa response and postpone the need for levodopa medication, which may lessen wearing-off. Although it is usually well taken, adverse symptoms like nausea, orthostatic hypotension, or sleeplessness are possible.[20] Certain drugs, such as meperidine or fluoxetine, should not be taken with selegiline. Although its ability to decrease the progression of the illness is still up for debate, rasagline, another MAO-B inhibitor, is used to treat PD symptoms with or without levodopa. Usually used in 5 mg doses with meals, selegiline is a selective irreversible inhibitor of MAO-B that can intensify and prolong the antiparkinsonian action of levodopa.[5,9]

COMT (Catechol-O-Methyltransferase) Inhibitors: The enzyme known as catechol-O-methyltransferase, or COMT, degrades dopamine. By preventing dopamine breakdown, medications such as entacapone and tolcapone prolong the

Table 2: A Comprehensive Overview of Novel Strategies for Anti-Parkinsonism Drug Delivery

Drug Dosage Form	Strategy	Working Mechanism	Examples/ Drugs	Key Benefits
Controlled-Release Tablets	Sustained drug release to maintain stable plasma concentrations.	Matrix or reservoir systems ensure gradual drug release over time, reducing fluctuation.	Sinemet CR (Carbidopa /Levodopa)	Reduces "on-off" phenomena and improves patient adherence.
Transdermal Patches	Non-invasive delivery through skin, ensuring continuous drug administration.	Drugs permeate the skin layers and enter systemic circulation via diffusion.	Rotigotine (Neupro patch)	Avoids first-pass metabolism, provides steady drug levels, and improves compliance.
Nanoparticle Formulations	Targeted delivery using nanoparticles to improve drug bioavailability and brain penetration.	Nanocarriers like liposomes or polymeric nanoparticles cross the blood-brain barrier (BBB).	Levodopa-loaded nanoparticles	Enhances BBB penetration, reduces required dosage, and minimizes systemic side effects.
Inhalable Formulations	Rapid relief of symptoms through pulmonary delivery.	Direct delivery to the bloodstream via alveoli ensures faster onset of action.	Inbrija (Levodopa inhalation powder)	Provides immediate relief from "off" episodes.
Oral Disintegrating Tablets (ODTs)	Rapid dissolution in the oral cavity for patients with swallowing difficulties.	Dissolves quickly in saliva and is absorbed through the oral mucosa or GI tract.	Parcopa (Carbidopa/Levodopa ODT)	Convenient for patients with dysphagia, ensuring better compliance.
Implantable Devices	Long-term and localized drug delivery through implants.	Releases drugs in a controlled manner directly into targeted areas of the body.	Levodopa-carbidopa intestinal gel (Duodopa)	Reduces dosing frequency, provides steady-state plasma concentrations, and improves symptom control.
Microneedle Patches	Minimally invasive, ensuring painless delivery through microneedles.	Drug-loaded microneedles penetrate the skin and release drugs into the dermal layer for systemic absorption.	Experimental Rotigotine microneedle patch	Enhanced patient comfort, no professional assistance needed, and steady drug delivery.
Intranasal Delivery	Direct drug delivery to the brain via the nasal cavity.	Bypasses the BBB through olfactory and trigeminal nerve pathways.	Apomorphine intranasal spray	Rapid onset of action for managing acute motor symptoms.
Hydrogel Systems	Injectable systems forming localized depots for sustained drug release.	Drugs are entrapped in hydrogel matrices, releasing gradually as the gel degrades.	Experimental Levodopa hydrogel	Localized and sustained release minimizes systemic side effects and dosing frequency.
Liposome-Based Delivery	Encapsulation of drugs in lipid vesicles to enhance brain targeting.	Liposomes shield drugs from enzymatic degradation and facilitate transport across the BBB.	Liposome-encapsulated dopamine derivatives	Prolonged drug half-life and improved BBB penetration.
Gene Therapy Vectors	Delivery of therapeutic genes to restore dopamine production or prevent neurodegeneration.	Uses viral vectors (e.g., AAVs) to introduce genes encoding enzymes for dopamine synthesis in targeted neurons.	AAV2-GAD (Glutamic acid decarboxylase vector)	Potential to modify disease progression and restore dopamine function.

of levodopa by reducing "off periods." Diarrhea, nausea, insomnia, light-headedness, urine discolouration, stomach pain, low blood pressure, or hallucinations are some of the adverse effects that these medications may induce. Tolcapone needs to be closely watched because it can occasionally cause serious liver problems. Levodopa's effect is boosted by lowering COMT, which lowers peripheral metabolism and produces a steadier response over time. The antiviral medication amantadine can help reduce the symptoms of Parkinson's disease and levodopa-induced dyskinesia, but it can also have negative side effects such edema, hallucinations, and insomnia.[20]

Anticholinergic: These medications, which reduce the activity of the neurotransmitter acetylcholine and can be especially useful for tremor, include trihexyphenidyl, benztropine, and ethopropazine. Dry mouth, constipation, urine retention, hallucinations, memory loss, impaired vision, and disorientation are possible adverse effects.[5]

Central antimuscarinic drugs: Synthetic substances called antimuscarinic medications improve tremor, rigidity, and other symptoms of Parkinson's disease by inhibiting ACh receptors in the central nervous system. Constipation, memory problems, dry mouth, and blurred vision are some of the side effects. Each patient needs a customized course of treatment because drug reactions differ and total symptom alleviation might not be achievable.[19] The effects of drug therapy are illustrated in Figure 3.

INNOVATIVE TECHNOLOGIES IN DRUG DELIVERY

Systems Based on Nanotechnology

• Precision at the Nanoscale: Lipid nanoparticles and other nano-formulations enable effective medication administration across the blood-brain barrier (BBB), improving targeted specificity and bioavailability.

• Polymeric Nanocarriers: Designed to limit the frequency of doses and lessen systemic side effects, these technologies offer regulated, prolonged drug release.

Infusion Systems and Implantable Devices

• Continuous Drug Delivery: Levodopa-carbidopa gel infused intra jejunally maintains steady plasma concentrations, reducing motor swings.

• Wearable Technology: As an alternative to surgical procedures, subcutaneous apomorphine pumps have become a minimally invasive option for severe Parkinson's disease care.

Intranasal and Transdermal Delivery Methods

• Non-Invasive Modalities: By avoiding gastrointestinal metabolism, transdermal

patches, such rotigotine, provide for consistent medication delivery.

• Intranasal Pathways: By using the olfactory pathway, intranasal formulations provide quick symptom alleviation by delivering medications straight to the central nervous system.

Intelligent Drug Distribution Methods

• Systems that respond: Closed-loop systems with biosensors represent a paradigm leap toward personalized therapy by monitoring biomarkers in real time and adjusting drug release accordingly.

• Micro-Robotic Carriers: To precisely deposit drugs in intricate brain microenvironments, micro-scale robotic systems are being developed.[7,9,12,14]

NON-PHARMACOLOGICAL MANAGEMENT

Surgical Interventions

Improvements in Deep Brain Stimulation (DBS): Adaptive DBS systems limit adverse effects while increasing therapeutic efficacy by modifying stimulation parameters in real-time depending on neural feedback.

With millimeter-level accuracy, focused ultrasound is a non-invasive method that reduces the need for invasive surgery by targeting ablation of brain areas that cause tremors.[8]

Rehabilitation and Lifestyle Changes

Exercise for Neuroprotection: It has been demonstrated that physical therapies like dance therapy, tai chi, and resistance training improve neuroplasticity and motor function.

Cognitive and Occupational Therapies: Personalized programs that target fine motor skills and executive dysfunction enable patients to perform activities of daily living and foster autonomy.

Dietary Treatments

Metabolic Modulation: By promoting mitochondrial biogenesis, the ketogenic diet, which places a strong emphasis on fatty acid metabolism, has shown promise in reducing neurodegeneration.

Gut-Brain Axis: Dysbiosis may worsen the pathophysiology of Parkinson's disease, according to new research. In an effort to restore the balance of the gut microbiota, probiotics and prebiotics are being researched as supplemental treatments.[10]

Biomarker Development and Early Diagnosis

• Imaging Innovations: Dopaminergic neuronal loss and α-synuclein deposition can be seen thanks to developments in PET and SPECT imaging, which helps in early diagnosis.

• Peripheral Biomarkers: As less intrusive diagnostic methods, blood-based tests for inflammatory cytokines and α-synuclein

oligomers are becoming more and more popular.

• Genetic Screening: As mutations like LRRK2 and PARK7 have been found, genetic screening is becoming a vital tool for identifying those who are at risk and customizing preventive measures.

Multidisciplinary and patient-centred methods

• Holistic Care Models: A thorough approach to managing Parkinson's disease is ensured by the collaboration of neurologists, physical therapists, dietitians, and mental health specialists.

• Telemedicine and Digital Health: As wearable technology and smartphone apps proliferate, remote monitoring becomes easier, allowing for real-time symptom tracking and customized therapy modifications.

• Empowering Patients and Caregivers: Support groups and educational programs address the psychosocial aspects of Parkinson's disease and promote psychological resilience and informed decision-making.[18,19]

CHALLENGES IN PARKINSON'S DISEASE MANAGEMENT

• *Symptom Management vs. Disease Modification:* While current treatments focus on treating motor and non-motor symptoms, they are unable to change the underlying neurodegenerative process. Effective disease-modifying treatments are still elusive despite a great deal of research.

• *Treatment-Related Complications:* Levodopa, the mainstay of PD treatment, is linked to motor fluctuations, dyskinesias, and a gradual decline in effectiveness with prolonged use. Likewise, impulse control issues brought on by dopamine agonists might make adherence and quality of life more difficult.

• *Complexity of Non-Motor Symptoms:* Autonomic dysfunction, depression, and cognitive impairment frequently do not respond to conventional treatments, indicating gaps in comprehensive care.

• *Heterogeneity of Disease Progression:* The necessity for individualized approaches is highlighted by the fact that variations in disease presentation and progression make it more difficult to produce one-size-fits-all treatments.[3,5,6]

FUTURE DIRECTIONS

Innovative research, collaboration, and patient-centred treatment are essential to the improvement of PD management.

Cutting-Edge Subjects of Study

Nanotechnology offers novel medication delivery methods that ensure accurate and sustained therapeutic release. Precision medicine is being made possible by the integration of artificial intelligence (AI) and

machine learning into diagnostic procedures and therapeutic approaches.

Global Projects

Through comprehensive data sharing, international collaborations such as the Parkinson's Progression Markers Initiative (PPMI) are improving our understanding of Parkinson's disease. Accelerated research and improved access to healthcare depend on increased funding and public awareness campaigns.

Strategies Focused on the Patient

Multidisciplinary teams in community-based care models offer patients all-encompassing support. Self-management programs encourage resilience and independence by empowering people to actively participate in their treatment.[1,19]

NOVEL WORK ON ANTI-PARKINSONISM DRUGS

The creation of novel anti-Parkinsonian medications and the procedures involved in their development are essential to enhancing the management of Parkinson's disease (PD) in light of the shortcomings of existing medications. New medications frequently aim to delay or stop the disease's course, reduce both motor and non-motor symptoms, and target the pathophysiology of the condition.[20,21] This article describes the new anti-Parkinsonian medications and how they are made.[4]

Below is a tabulated summary of novel drug dosage form strategies for anti-Parkinsonism drugs, detailing their mechanisms, examples, and key benefits. (Table 2)

NEW DEVELOPMENTS IN DRUG RESEARCH FOR PARKINSON'S DISEASE

UB-312 Immunotherapy: Vaxxinity created UB-312, an immunotherapy that targets alpha-synuclein, a protein that builds up in Parkinson's patients' brains and forms harmful clumps. By encouraging the production of antibodies by the immune system against these aberrant proteins, the treatment may lessen their accumulation and delay the course of the illness. According to encouraging results from early-stage clinical trials, it could revolutionize disease-modifying treatment.[22,23]

BIIB122 (LRRK2 Inhibitor: The LRRK2 gene is implicated in some genetic variants of Parkinson's disease, and Biogen's BIIB122 targets mutations in this gene. Degeneration results from aberrant neural activity linked to LRRK2 mutations. BIIB122 seeks to reduce the progression of the disease by blocking the mutated protein. Its safety and efficacy for patients with genetic alterations are being investigated in ongoing trials, providing targeted therapy for a subgroup of Parkinson's patients.[24,25]

Nilotinib: Nilotinib was first used to treat cancer, but it has also showed promise in treating Parkinson's disease, especially in patients with dementia. The accumulation of harmful proteins like alpha-synuclein is caused by the Abl kinase enzyme, which is inhibited by the medication. Early research has shown that nilotinib improves both cognitive and motor function, giving hope to people who have both motor symptoms and cognitive decline.[26,27]

Exenatide: This diabetes medication works by imitating the effects of the hormone GLP-1, which aids in blood sugar regulation. According to research, it possesses neuroprotective properties that help Parkinson's sufferers with their motor symptoms and increase dopamine production. Exenatide is a promising treatment option that may improve motor function and preserve brain cells, according to early-phase clinical trials.[28,29]

Gene Therapy (AAV2): The goal of gene therapy using adeno-associated virus (AAV2) vectors is to transfer genes that cause the brain to produce dopamine again. This strategy may be able to reverse motor symptoms and delay the progression of the disease. With promising results from early studies, more research is being done to ensure long-term efficacy and improve gene delivery techniques.[30]

CONCLUSION

The literature review on Parkinsonism and antiparkinsonian drugs mentioned above will help people understand the condition better and, in turn, help eradicate it. Effective care of Parkinson's disease necessitates a multidisciplinary approach due to its complex nature. Emerging therapies seek to address the underlying illness and enhance quality of life, whereas current medicines concentrate on controlling symptoms. There is optimism for a time when Parkinson's disease (PD) may be better controlled or maybe treated thanks to developments in tailored medication, neuroprotective techniques, and technology integration. To overcome current obstacles and guarantee fair access to care, cooperation between researchers, physicians, and legislators will be essential.

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