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PHARMACOLOGICAL MANAGEMENT OF DEPRESSION AND HALLUCINATIONS IN PARKINSON'S DISEASE: A COMPREHENSIVE REVIEW

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ABSTRACT

Purpose: The aim of this study is to present available treatment options for depression and hallucinations in Parkinson's disease (PD). The review discusses the application of serotonin-norepinephrine reuptake inhibitors (SNRIs, e.g., duloxetine), selective serotonin reuptake inhibitors (SSRIs, e.g., paroxetine, escitalopram), pimavanserin, pramipexole, and repetitive transcranial magnetic stimulation (rTMS) in the treatment of depressive symptoms in PD. Moreover, the therapeutic use of atypical antipsychotics such as clozapine, quetiapine, and pimavanserin in managing hallucinations in PD is addressed.
Materials and Methods: This review is based on literature identified via Google Scholar.
Results and Conclusions: SSRIs are among the most commonly prescribed agents in the treatment of depression associated with Parkinson's disease, although robust evidence supporting their efficacy in large cohorts remains limited. Other therapeutic options include SNRIs and tricyclic antidepressants. In cases of Parkinson's disease-related hallucinations, the initial approach involves reducing dopaminergic medication doses to rule out drug-induced psychosis. If symptoms persist, second-generation atypical antipsychotics may be introduced. Clozapine and quetiapine are preferred over agents such as olanzapine and risperidone due to a more favorable motor side effect profile. Pimavanserin stands out as a promising agent due to its minimal adverse effects and high safety profile in this patient population.

KEYWORDS

Parkinson's Disease, Depression, Hallucinations, Atypical Antipsychotics, Serotonin-Norepinephrine Reuptake Inhibitors (SNRI), Selective Serotonin Reuptake Inhibitors (SSRI)

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Introduction

Parkinson's disease (PD) affects a large population of patients, as it is the second most common neurodegenerative disorder worldwide, following Alzheimer's disease. It is a progressive condition, and its prevalence increases with age [1,2]. The disease affects approximately 1% of individuals over 60 years old, with around one million people affected globally [3]. Individuals under the age of 40 may also develop the disease, in which case it is often genetically determined [4]. The motor symptoms of Parkinson's disease include bradykinesia, muscular rigidity, resting tremor, and postural instability. These symptoms result from the loss of dopaminergic neurons in the substantia nigra [1]. A clinical diagnosis typically requires the presence of at least two of the following symptoms: bradykinesia, rigidity, or resting tremor [4]. Confirmation of the diagnosis is achieved through symptom resolution following the initiation of dopaminergic therapy [5].

Non-motor symptoms such as anxiety, hallucinations, sleep disturbances, cognitive dysfunction, and depression are less widely recognized. Depression affects about 50% of PD patients and is considered the most common non-motor symptom [1]. Impulse control disorders, frequently induced by antiparkinsonian medications, are also observed. These are more prevalent in men, in patients with early-onset PD, and in younger individuals [6].

Symptoms and Pathogenesis of Depression in Parkinson's Disease

Symptoms of depression include mood lowering, cognitive decline, and psychomotor disturbances. Some patients may also experience sleep disorders and appetite changes [7]. Depression in PD exhibits similar features. Authors note that geriatric patients particularly present with low mood, somatization, increased pain sensitivity, anxiety, pessimism, and irritability [8]. Both neuroanatomical and neurochemical changes contribute to the development of depression in PD [3]. Although the exact causes remain unclear, multiple factors are implicated [8].

One key psychological theory posits that depression in PD arises due to emotional responses to progressive disability [9]. Another, biological theory attributes depression to pathological changes in central neurotransmission affecting serotonergic, dopaminergic, and noradrenergic systems. Neuroimaging studies of depressed PD patients have demonstrated reduced blood flow in the cingulate gyrus and frontal cortex [8].

Clinicians often focus primarily on motor symptoms, neglecting non-motor symptoms like depression, which leads to underdiagnosis and undertreatment, despite their significant impact on quality of life [10]. Studies show that depressed PD patients respond less effectively to dopaminergic therapy, experience more rapid disability progression, and have higher mortality rates. Depression symptoms may precede or follow the onset of motor symptoms and may be triggered by diagnosis-related stress [11,12].

Therapeutic Management of Depression in Parkinson's Disease

It is essential to identify and eliminate external stressors exacerbating depressive symptoms where possible. Psychosocial interventions such as group therapy, psychoeducation, and cognitive-behavioral techniques have shown efficacy in PD-related depression. Endocrine disorders, such as untreated hypothyroidism, should be ruled out, as they may reduce treatment efficacy. Optimizing motor symptom control is also critical to preserve physical function and mitigate depression linked to disability [8].

Pharmacotherapy

The impact of SNRI and SSRI antidepressants on depression, apathy, and freezing of gait (FOG) in PD was evaluated in a multicenter, randomized, prospective trial conducted at five hospitals in Osaka, Japan, between January 2014 and June 2016. Eligible participants were over 40 years old with a PD diagnosis and a QIDS-J score of ≥6 (indicative of depression). The SNRI group included 14 men and 13 women; the SSRI group included 13 men and 12 women. Patients were randomly assigned to receive duloxetine (SNRI) or paroxetine/escitalopram (SSRI). Common side effects in the SSRI group included nausea (5 patients), vomiting (2), and somnolence (2). In the SNRI group, tremor was the most frequent side effect (2 patients) [2].

Both drug classes effectively reduced the severity and frequency of FOG. Although no significant differences in efficacy were observed between the groups, the small sample size limited definitive conclusions. Only duloxetine was used in the SNRI group, and paroxetine or escitalopram in the SSRI group, making inter-drug comparison difficult. Both SSRIs and SNRIs may help alleviate both motor and non-motor symptoms of PD, potentially improving overall disease severity. However, the lack of a placebo control limits the reliability of the findings.

Pimavanserin in PD-Related Depression

Pimavanserin is a selective serotonin 5-HT2A receptor antagonist/inverse agonist, with limited affinity for 5-HT2C receptors. A U.S. clinical study evaluated pimavanserin in monotherapy (21 patients) and as adjunct therapy with antidepressants such as bupropion, duloxetine, escitalopram, fluoxetine, paroxetine, sertraline, and vortioxetine (26 patients). The trial included 47 PD patients over 50 years old with depression. After eight weeks, 60% experienced a ≥50% reduction in HAMD-17 scores, and 44.4% achieved remission within six weeks [13].

Despite the promising results, the study had limitations: small sample size, short duration (eight weeks), and lack of a placebo group. However, the low incidence of adverse effects (21 reported, one serious) supports further research into pimavanserin as a potentially safe treatment for depression in PD. Compared to other medications, it has a low incidence of motor-related side effects and a favorable safety profile.

Clinical Study in China

A Chinese clinical study evaluated 131 of 328 hospitalized PD patients (based on HAMD-17 and Hoehn & Yahr scores). Patients were divided into four treatment groups: (1) conventional PD therapy only; (2) conventional therapy + escitalopram 10 mg; (3) conventional therapy + pramipexole (0.375 mg to 1.5 mg/day over three weeks); (4) conventional therapy + repetitive transcranial magnetic stimulation (rTMS, 5 Hz for 20 minutes, 5 times per week). Depression outcomes were assessed after four weeks [14].

Results suggested that escitalopram, pramipexole, and rTMS all reduced HAMD-17 scores more than conventional therapy alone. Escitalopram and pramipexole appeared more effective than rTMS, though neither was clearly superior. Limitations included short duration, no long-term follow-up, and no true control group beyond standard treatment.

Physical Activity

Physical activity significantly benefits PD patients by improving physical function, reducing mortality, enhancing mood, quality of life, and cognitive function. Combining pharmacotherapy with rehabilitation yields better outcomes. Kinesiotherapy should be tailored to disease stage and introduced at diagnosis, continuing throughout life. Optimal results occur during levodopa's peak effect. Techniques include the Bobath concept, PNF method, Peto method, biofeedback, group games with music therapy, sensory stimulation, and dance. Physical activity is cost-effective and low-risk, but underutilized—only 50% of PD patients exercise regularly, and just 25% receive exercise instruction [15].

Psychosis in Parkinson's Disease

Psychosis is another common non-motor symptom, affecting 26%–82.7% of PD patients. Visual hallucinations occur in 22%–38%, auditory hallucinations in up to 22% [16]. Contributing factors include dopaminergic medications and neurodegenerative changes [11]. Hallucinations arise from disruptions in neural networks, especially those involved in attention and default-mode activity. Thalamic dysfunction, particularly reduced white matter connectivity in the posterior thalamus, has been observed in PD psychosis.

Hallucinations increase the risk of nursing home placement and mortality. Visual hallucinations—often involving people or animals—are most common. Patients may also experience object misidentification, passage hallucinations, or peripheral hallucinations, which typically intensify over time and often occur in the evening or at home [17].

Hallucinations

The prevalence of psychotic disorders in the general population is estimated at 4%–7%. Hallucinations may occur persistently as a symptom of an underlying disease. They are also observed in acquired deafness (auditory hallucinations), in ocular diseases, and in disorders of the afferent visual pathways (visual hallucinations). Hallucinations have also been described in patients with hormonal imbalances in endocrine diseases (e.g., Hashimoto's disease). Psychotic symptoms may be triggered by deficiencies in vitamin B12 or vitamin D, chromosomal disorders (e.g., Prader-Willi syndrome), and autoimmune diseases. The literature also cites sleep disorders (e.g., narcolepsy), numerous neurological diseases and injuries, and cardiovascular disorders as causes of hallucinations. Hallucinations may also appear intermittently in response to external stimuli causing intense emotional stress. Substances such as alcohol, alcohol withdrawal, toxins, or certain medications (e.g., antimalarial drugs) may also induce hallucinations [18].

Hallucinations in Parkinson's Disease and Therapeutic Approaches

Treatment of the underlying symptoms in Parkinson's disease (PD) may induce hallucinations [18]. A major challenge is distinguishing de novo PD psychosis from drug-induced psychosis caused by chronic dopaminergic therapy (affecting approximately 30% of patients). Therefore, initial management of hallucinations should involve reducing the dose of dopaminergic medications. If hallucinations persist, second-generation atypical antipsychotics should be considered [16]. In advanced stages, patients may become unaware of their hallucinations, which may be accompanied by delusions of guilt, religious or persecutory ideation, or theft-related delusions. Effective treatment of hallucinations is difficult due to the potential exacerbation of motor symptoms of PD [17]. The worsening of motor symptoms by certain antipsychotics is linked to postsynaptic D2 receptor blockade in patients who already have reduced presynaptic dopamine levels. The first atypical antipsychotic shown to effectively treat hallucinations without worsening motor symptoms was clozapine [19]. Clozapine and quetiapine do not exacerbate motor symptoms, in contrast to olanzapine and risperidone. Clozapine requires hematological monitoring due to the risk of leukopenia and agranulocytosis [16]. Pimavanserin demonstrates a favorable safety profile and lacks motor side effects [16]. However, antipsychotics overall may negatively affect mortality rates, exacerbate hallucinations, and increase the risk of stroke [17].

A multicenter, randomized, double-blind, placebo-controlled trial was conducted in the Netherlands to evaluate whether treatment of mild visual hallucinations in PD with rivastigmine (a carbamate-class dual acetylcholinesterase and butyrylcholinesterase inhibitor) delays the progression to psychosis. The study enrolled 91 individuals aged 40 and older with PD and visual hallucinations (rated 1 or 2 points on the Movement Disorder Society–Unified Parkinson's Disease Rating Scale, MDS-UPDRS) occurring within four weeks before study onset. Patients were randomized to receive rivastigmine (3–6 mg twice daily; n = 46) or placebo (n = 45) for 24 months. Patients were withdrawn upon developing psychosis requiring antipsychotic

treatment. Psychosis developed in 4 out of 46 patients in the rivastigmine group and 5 out of 45 in the placebo group [20]. Outcomes for patients rated 1 point on MDS-UPDRS were similar to those rated 2. In the author's opinion, the study's credibility is limited by its small sample size and the inability to objectively assess the presence and nature of hallucinations, which may go unreported if patients believe them to be real or if they are too subtle to recognize.

Pimavanserin, a selective 5-HT2A inverse agonist, was approved by the FDA in 2016 for the treatment of hallucinations in PD. An open-label extension (OLE) trial evaluated the durability of response to pimavanserin (34 mg/day) over an additional four weeks of treatment (total 10 weeks). The study included 459 participants from 14 countries, with 424 evaluated at week 4. Patients were enrolled after completing one of three prior double-blind, placebo-controlled core studies. Assessments included the Parkinson's Disease–adapted Scale for the Assessment of Positive Symptoms (SAPS-PD), Clinical Global Impression scales for improvement and severity, and caregiver burden scales. Participants previously on placebo demonstrated improvement after four weeks of pimavanserin treatment. Those who had already received pimavanserin maintained efficacy in the OLE phase. The study was funded by ACADIA Pharmaceuticals and published in 2021—five years after the drug's FDA approval, and was not intended for regulatory submission [21].

This large-scale clinical trial demonstrated pimavanserin's efficacy in treating PD-related hallucinations. In the OLE phase, patients who switched from placebo to pimavanserin showed outcomes comparable to those receiving pimavanserin from the start. However, the study lacked a control group.

Conclusions and Summary

For patients with both motor and non-motor symptoms, selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) can be effectively used, reducing symptoms of depression and gait instability to a similar extent. These agents also demonstrate comparable safety profiles. Due to their broader mechanism of action, SNRIs and tricyclic antidepressants (TCAs) may be slightly more effective than SSRIs. If side effects from SSRIs occur—especially nausea, which is less common with SNRIs—a switch to an SNRI may be considered.

A clinical trial showed that escitalopram was more effective than pramipexole after four weeks in reducing depressive symptoms, without worsening motor function and with improved quality of life. Both escitalopram and pramipexole showed stronger antidepressant effects than high-frequency transcranial magnetic stimulation (TMS), which may be considered as an adjunct therapy in PD with depression.

Pimavanserin is superior to other agents due to its lack of motor side effects, making it a valuable option in PD patients with depression or hallucinations.

The trial with rivastigmine did not demonstrate efficacy in slowing psychosis progression in PD, and further properly designed studies are warranted. Physical activity tailored to the patient's current condition should be incorporated into PD management. Rehabilitation promotes functional improvement, delays disability, positively influences the patient's psychological state, and enhances quality of life [15]. Effective treatment of depression, hallucinations, and psychotic symptoms reduces the risk of malnutrition and significantly decreases mortality in these patients [22].

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