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Psychiatric Disorders and Parkinson's Disease: Between Symptomatic Overlap and Therapeutic Challenges – A Case Report

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Abstract | **Case Report**

Parkinson's disease (PD) is a neurodegenerative disorder primarily affecting the dopaminergic system, characterized by motor symptoms (tremor, rigidity, akinesia) as well as numerous psychiatric manifestations (depression, anxiety, psychosis, apathy) that significantly impair quality of life. The reported case involves a 74-year-old female patient with a 40-year history of bipolar disorder and several years of PD. The recent course was marked by a major depressive episode with psychotic symptoms. The introduction of an antipsychotic (aripiprazole) worsened the motor symptoms, while increasing L-Dopa doses intensified psychosis. A therapeutic compromise was achieved with quetiapine, which was better tolerated in terms of motor function, though less effective than clozapine. The discussion highlights the high prevalence of psychiatric disorders in PD, their impact on disease progression, and the challenge of balancing motor treatment with psychiatric symptom control. Literature supports the superior efficacy of clozapine for Parkinson's psychosis, but also recognizes the pragmatic value of quetiapine in certain clinical contexts. ***Conclusion:*** The management of PD requires a personalized, multidisciplinary approach to reconcile the treatment of both motor and psychiatric symptoms while minimizing side effects.

Keywords: Parkinson's disease, Bipolar disorder, Quetiapine, Clozapine, Aripiprazole, L-Dopa.

INTRODUCTION

Parkinson's disease (PD) is a chronic neurodegenerative disorder affecting the dopaminergic system, primarily in the substantia nigra. It is classically associated with motor symptoms such as bradykinesia, rigidity, and resting tremors. However, this motor symptomatology alone does not capture the complexity of the disease. Non-motor symptoms, especially psychiatric ones—depression, anxiety, hallucinations, behavioral disturbances—are frequently observed and significantly contribute to the deterioration of quality of life. These psychiatric manifestations are not mere comorbidities; they intertwine in a vicious cycle, potentially accelerating disease progression or complicating treatment. This bidirectional link between neurological and psychiatric domains poses major therapeutic challenges. We illustrate this interaction through a clinical case.

CLINICAL CASE

Mrs. M.A.I., 74 years old, married, and mother of two children, has been followed for over 40 years for type II bipolar disorder with a predominance of depressive episodes. Her psychiatric history includes several hospitalizations, with recurrent use of mood stabilizers and antidepressants.

She has also been diagnosed with Parkinson's disease for several years and is treated with Madopar® (levodopa + benserazide). Her medical history includes significant comorbidities: valvular heart disease requiring anticoagulant therapy with Sintron®, and controlled hypertension.

She was admitted to our department for worsening mood symptoms: depressed mood, psychomotor slowing, anhedonia, social withdrawal, along with the emergence of persecutory delusions and auditory hallucinations. A diagnosis of major depressive episode with psychotic features was made. Treatment was initiated with fluoxetine (20 mg/day), lamotrigine (200 mg/day), and aripiprazole (10 mg/day). After one week, the patient developed marked parkinsonian syndrome: worsening tremors, hypokinesia, and gait difficulties.

A neurological consultation concluded that this was a decompensation of PD probably induced by aripiprazole. The L-Dopa dosage was increased, but this modification worsened the psychotic symptoms. Faced with this therapeutic dilemma, the aripiprazole dose was increased to 15 mg/day, but the tremors became disabling. A second neurological opinion recommended replacing aripiprazole with a motor-friendly antipsychotic. Clozapine was excluded due to her cardiovascular history. Quetiapine was gradually introduced, allowing a better balance between controlling psychiatric symptoms and preserving motor function.

DISCUSSION

Psychiatric disorders (depression, anxiety, apathy, psychosis) in PD are extremely frequent, affecting up to 60% of patients in the advanced stages. In a multicenter study of late-stage PD patients, 92% had at least one persistent neuropsychiatric symptom, the most common being apathy (38.9%), depression (34.5%), and anxiety (23.8%). Recent reviews confirm these findings, reporting depression in 20–40% of patients, apathy in ≈40%, and psychotic symptoms in 15–30%.

Delusions and hallucinations, as seen in our patient, are often induced or exacerbated by dopaminergic treatments—particularly agonists—and by neurodegenerative progression. The prevalence of psychosis is correlated with disease duration, advanced stage, and the presence of dementia.

A bidirectional relationship exists between psychiatric manifestations and PD progression: depression often precedes the motor phase, while psychosis complicates management by causing medication non-adherence, which in turn worsens motor symptoms. This interaction increases functional disability and negatively impacts quality of life.

Regarding quetiapine as a treatment for Parkinson's psychosis, several open-label studies report psychotic improvement in about 80% of patients without significantly worsening mobility. However, randomized controlled trials (RCTs) have not confirmed these results, showing quetiapine to be ineffective compared to placebo, or comparable to clozapine without clear clinical superiority. A recent meta-analysis confirms that clozapine remains the most effective treatment, while quetiapine, although better tolerated, offers limited benefits.

In our case, quetiapine was chosen pragmatically due to clinical and hematological constraints (valvular disease, anticoagulation) making clozapine unsuitable. Its favorable motor profile, despite potentially lower efficacy against delusional psychosis, was an acceptable compromise.

Apathy, frequently underdiagnosed, affects up to 40% of PD patients. It severely impacts quality of life—sometimes more than motor symptoms—and is often correlated with cognitive decline and daytime sleepiness. In our patient, signs of withdrawal and disengagement were present from the depressive phase, confirming the coexistence of apathy and a depressive episode.

CONCLUSION

Managing Parkinson's disease requires a delicate balance between motor and psychiatric treatments. Therapeutic adjustments can exacerbate both psychiatric and motor symptoms, making management challenging. A multidisciplinary approach and personalized care are essential to improve quality of life and optimize treatment while minimizing side effects.

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