

## Case Report

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# Parkinson's Disease and Late Onset Psychosis – a case report and clinical dilemma therein

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### ABSTRACT

Psychosis is a common disorder comorbid with Parkinson's disease (PD) and is seen often in the later stages of the illness. Symptom severity due to psychosis varies and ranges from frank delusions and hallucinations to just minor dreams, paranoia and illusions. The pathogenesis of psychosis in PD is not fully understood from a neurobiological perspective. The management of psychosis in PD needs a careful approach considering the opposing groups of drugs i.e. antipsychotics that block dopamine and anti-parkinsonian drugs that are pro-dopamine in nature. We report herewith a case of psychosis in PD and the clinical dilemmas that arose due to the same.

**Key words:** psychosis, parkinsonism, Parkinson's disease, hallucinations, antipsychotics

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### INTRODUCTION

Psychotic symptoms like hallucinations and delusions occur in 4-25% of patients with Parkinson's disease (PD) and it is assumed that the main precipitant of psychotic phenomena is dopamine excess secondary to treatment with Levo-dopa [1]. It is important to note that psychosis in PD has been reported even in the absence of pro-dopamine drugs [2]. It is a distressing disorder as the psychopathology caused by psychosis further disrupts the movement disorder component. Even treatment is tough as most antipsychotics have drug induced parkinsonism as a side effect [3]. Psychosis in PD is known to worsen the disease course and cause greater distress to patients and relatives [4]. We report herewith a case of gradual progressive parkinsonism that developed concurrent psychotic features. Treating the psychosis and calibrating dopamine levels in various circuits was the main therapeutic dilemma ahead. Neuroimaging findings of the patient further pointed towards a probable organic etiology of psychotic features leading to further vexing of the problem.

### CASE REPORT

A 70 year old, right handed, 4<sup>th</sup> standard educated man who retired from chemical industry was brought by his son to psychiatry out patient department with a chief complaint of suspiciousness towards neighbors since the past one year sleep disturbances of recent onset (past month). He had an altercation with one of his neighbors over a parking issue in the society where he lived and since then started developing

persecutory delusions against his neighbor. He began to feel that the neighbor was plotting against him and would do something to harm him. Gradually, his persecutions became systematized and even though the neighbor had shifted to a new house and new people began to live in place, the patient claimed that the new people were part of the conspiracy and were brought in purposely by the previous neighbor. The patient developed olfactory hallucinations in form of smelling chemicals which he claimed to be dispersed into his house by the neighbors to harm and suffocate him. The patient used to shut all doors and windows to prevent the so called chemical gas from entering the house. This led to social dysfunction and severe distress in the patient and his family as multiple complaints were lodged by patient in police against the new neighbors and frequent altercations would go on with neighbors due to behavioral changes in patient. There was no history of symptoms similar to this prior to a year when the symptoms had started.

On examination, the patient was detected to have resting pill rolling type of tremor and his son gave a history of general slowing of movements since past 5 years which the family considered to be age related and did not seek treatment for. His blood pressure, blood sugar levels, serum lipid profile, Thyroid function & serum Vitamin B12 levels was found to be within normal limits. No focal neurological signs were detected. MMSE score was 25/30 at first visit. MRI Brain findings were quite alarming and gave a complex picture of chronic lacunar infarct in left thalamus, left corona radiata and left hemi-pons along with ischemic changes in bilateral fronto-parietal subcortical and deep white matter, periventricular region and bilateral putamina.

Hence a neurology reference was sought (to rule out an organic etiology) and the patient was started on Levodopa-Carbidopa combination (100mg+10mg) for his Parkinson's symptoms. Simultaneously to control his psychotic features, we started him on Quetiapine (25mg at night) and Clozapine (25mg at night) which helped him in sleep as well. The dosage were gradually optimized up to Quetiapine 50 mg (sustained release formulation). Improvement in cognitive and motor symptoms was noted and delusions and hallucinations were completely brought under control within a period of 3 months of treatment. A final diagnosis of parkinson's disease (probably vascular in origin) with psychosis and multi-infarct dementia was made. The patient was also started on Donepezil 10mg at night for his cognitive symptoms. No further deterioration in MMSE scores were noted in future visits.

## DISCUSSION

The primary symptoms of Parkinson's disease usually result from greatly reduced activity of dopamine secreting cells caused by cell death in the pars compacta region of the substantia nigra [5]. In our case, adjusting the perfect equilibrium of dopamine in mesolimbic connections and nigro-striatal pathways was the difficult task ahead. It has been reported that Quetiapine, Clozapine and Aripiprazole are the safest among the atypical antipsychotics and the monotherapy with antipsychotics in cases of psychosis and PD often fails due to poor efficacy or worsening of Parkinson's symptoms [6]. Hence, we opted for the regimen of a combination of Quetiapine and Clozapine together and we optimized dosages after evaluating for both psychotic and parkinson's symptoms on follow up. These drugs were also chosen as the patient had complained of poor sleep and sleep problems in PD are known to further precipitate psychotic features [7]. One more interesting finding in the case was multiple infarcts and ischemic changes over the broad circuit involving thalamus, basal ganglia & fronto-parietal regions of brain which gave the complex picture of psychosis and cognitive, behavioral dysfunction with a probable vascular origin to the parkinsonism and a multi-infarct dementia like clinical picture. These findings also highlight that parkinsonism and psychosis when seen in old age may have multiple etiologies and may be a product of neurotransmitter abnormalities and cerebrovascular compromise [8]. It is also vital that clinicians be aware of the same as the drug treatment in these cases may be three pronged i.e. treatment of PD, treatment of psychosis and management of an underlying cerebrovascular pathology. There is also a need for vigilant consultation-liaison between psychiatrists and neurologists when handling such cases where the picture and symptomatology may be of a neuropsychiatric nature.

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