Journal of Young Pharmacists, 2024; 16(1):137-138.
https://www.jyoungpharm.org

Case Report

Chronic Neuroleptic Therapy and Progressive Parkinsonism: A Case Report

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ABSTRACT

This case report presents the challenging clinical scenario of a middle-aged male patient with a decade-long history of psychiatric illness on chronic neuroleptic therapy. The patient's symptoms initially manifested as tremors a year ago, subsequently progressing to resting tremors, head titubation, and impaired mobility. Typically, Drug-Induced Parkinsonism (DIP) occurs within three months of initiating neuroleptic treatment, this case presents a unique and prolonged timeline, raising questions about the underlying aetiology complexed with patients' imaging studies revealing age related atrophy. The bilateral and symmetrical motor signs observed align with DIP characteristics, although studies report asymmetrical signs, introducing diagnostic complexities. This case emphasizes the need for further research on understanding the effect of chronic neuroleptic use, age-related structural alterations, and the potential unmasking of underlying Parkinsonism for improving diagnostic accuracy and tailoring effective management strategies for patients with similar challenging presentations.

Keywords: Prolonged neuroleptic exposure, Parkinsonian symptoms, Drug-Induced Parkinsonism, Anti-psychotic therapy, Extrapyramidal symptoms, Age-related cerebral atrophy.

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Received: 17-10-2023;
Revised: 04-12-2023;
Accepted: 10-01-2024.

INTRODUCTION

Drug-Induced Parkinsonism (DIP) is an extrapyramidal adverse effect resulting from the use of various drugs, with anti-psychotic medications being the primary and most common contributors.[1] Diagnosing Drug-Induced Parkinsonism (DIP) distinctively from Idiopathic Parkinson's Disease (IPD) poses challenges, hindering precise prevalence data collection. Despite this difficulty, DIP accounts for a prevalence ranging from 0.09% to 2.7%, positioning it as the second most prevalent cause of Parkinsonism.[2] Extrapyramidal Symptoms (EPS) primarily stem from the blockade of D2 receptors in the mesolimbic and mesocortical pathways within the basal ganglia.[2] Notably, these symptoms can endure in approximately 25% of patients even after discontinuing the causative drugs. Risk factors for Drug-Induced Parkinsonism (DIP) include advanced age, female gender, organic brain damage, atrophy, and dementia, highlighting the importance of recognizing these factors in clinical assessments.[3] Here we present a case of a 56-year-old patient presenting Parkinson-like symptoms while on antipsychotic and antiepileptic medications, highlighting the challenges in the diagnosis and treatment of drug-induced Parkinsonism.

CASE PRESENTATION

A 56-year-old male patient, previously diagnosed with psychiatric illness for the past decade, was being managed with medications including Tab. Risperidone 2 mg, Tab. Chlorpromazine 100 mg, Tab. Trihexyphenidyl 2 mg, and Sodium Valproate 500 mg BD. He had a history of a Generalized Tonic-Clonic Seizure (GTCS) a year ago, on physical examination, the patient had mild tremors which was managed with sodium valproate and trihexyphenidyl, and was advised to continue the antipsychotics prescribed previously. Presently the patient came with complaints of abnormal movements of both upper limb and lower limb since one month, generalized weakness since 15 days, and unable to walk since fifteen days. On examination, he exhibited resting tremors, head titubation, and cogwheel rigidity bilaterally. CT and MRI showed age-related cerebral atrophy, chronic bilateral corona radiata infarcts, and hypertensive leukoencephalopathy. He was also displaying behavioural issues such as irritability, verbal and physical aggression, and suspiciousness towards his wife. There was no history of substance abuse, psychiatry illness in family, no withdrawal symptoms, and at initial presentation the patient denied any illness.

DOI: 10.5530/jyp.2024.16.19

Upon referral to the psychiatry department, it was determined that the patient was suffering from drug-induced Parkinsonism consequently; Tab. Chlorpromazine and Tab Sodium Valproate were discontinued. He was prescribed Tab. Risperidone 2 mg (0-0-1), Tab. Trihexyphenidyl 2 mg (1-1-0), Tab. Levodopa 110 mg (1/2-1/2-1/2), and Tab. Clonazepam 0.25 mg (0-0-1). Despite some improvement in symptoms, extrapyramidal symptoms persisted.

Upon discharge on the 11th day, the patient was prescribed Tab. Levodopa 110 mg (1/2-1/2-1/2) for 15 days, Tab. Clonazepam 0.25 mg (0-0-1), Tab. Aspirin 75 mg (0-1-0), Tab. Atorvastatin 20 mg (0-0-1), Tab. Risperidone 2 mg (0-0-1), and Tab. Trihexyphenidyl 2 mg (1-1-0). He was advised to follow up in the outpatient department for further evaluation and management.

DISCUSSION

This case presents a unique challenge in understanding the etiology of drug-induced Parkinsonism (DIP) in a patient with a decade-long history of neuroleptic use. Typically, DIP manifests within three months of drug initiation;[4] however, this patient's symptoms, which started as tremors a year ago, progressed significantly, leading to resting tremors, head titubation, and impaired mobility. The progressive nature of the symptoms after a long duration of neuroleptic therapy raises questions about the underlying mechanisms.

Several studies propose that antipsychotic medications might unmask preclinical Parkinson's Disease (PD).[3] However, MRI findings in our case, indicating age-related atrophy and infarcts, suggest that structural changes in the brain, such as dopaminergic neuronal loss and alterations in white matter microstructure, especially in elderly patients, could predispose them to parkinsonism.[4-6] These findings align with studies indicating that these factors might contribute to the progressive nature of symptoms seen in our patient.

Symmetry and bilaterality in motor signs, as observed in this case, are common features of DIP, although some studies report asymmetrical signs, possibly indicative of emerging PD.[2] The lack of clarity in distinguishing these signs further complicates the diagnosis. Notably, the patient's favorable response to levodopa treatment, though inconsistent in cases of DIP,[7,8] raises questions about the underlying pathology. However, careful consideration is warranted, especially given the patient's history of mania, as responses to levodopa in such cases can be unpredictable.

Further insights into the complex interplay between chronic neuroleptic use, age-related brain changes, and the potential unmasking of underlying PD are essential. Longitudinal studies examining patients with prolonged neuroleptic exposure and detailed neuroimaging assessments could shed light on the mechanisms leading to progressive Parkinsonism. Additionally, exploring the variations in treatment responses, especially concerning levodopa, in patients with different psychiatric profiles could provide valuable insights for tailored therapeutic interventions.

CONCLUSION

In conclusion, this case emphasizes the intricate nature of DIP in the context of chronic neuroleptic therapy and age-related brain changes. Understanding the underlying mechanisms and clarifying the diagnostic criteria for distinguishing DIP from emerging PD are crucial. Further research addressing these complexities will enhance our diagnostic accuracy and pave the way for more effective management strategies tailored to individual patient profiles.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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Cite this article: Rao A, Reddy S, Nyamagoud SB, Swamy AHV. Chronic Neuroleptic Therapy and Progressive Parkinsonism: A Case Report. J Young Pharm. 2024;16(1):137-8.