

Advances in Clinical and Medical Research

Genesis-ACMR-5(2)-76
Volume 5 | Issue 2
Open Access
ISSN: 2583-2778

Drug-Induced Parkinsonism as it Compares with Parkinson Disease

Daniel Benharroch*

Department of Pathology, Soroka University Medical Centre, and Faculty of Health Sciences, Ben Gurion University of the Negev, Beer Sheva, Israel

***Corresponding author:** Daniel Benharroch, Department of Pathology, Soroka University Medical Centre, and Faculty of Health Sciences, Ben Gurion University of the Negev, Beer Sheva, Israel

Citation: Benharroch D. (2024) Drug-Induced Parkinsonism as it Compares with Parkinson Disease. *Adv Clin Med Res.* 5(2):1-4.

Received: December 15, 2023 | **Published:** February 18, 2024

Copyright © 2024 genesis pub by Benharroch D. CC BY-NC-ND 4.0 DEED. This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives 4.0 International License., This allows others distribute, remix, tweak, and build upon the work, even commercially, as long as they credit the authors for the original creation.

Abstract

Drug-induced Parkinsonism is associated primarily with several psychiatric medicines, notably, anti-psychotic medications, lithium and more. The reversibility of the condition depends on the patient's capacity to give up the drug consumption, without major withdrawal effects. Drug-related Parkinsonism shares several features with Parkinson disease. These belong to one of two categories of signs and symptoms: they may have motor or non-motors characteristics. Both categories may present similar symptoms and show different traits, but the clinical features differ. Using imaging and a brain scan, diagnosis of Parkinson disease is achievable by [¹⁸F]F-DOPA/PET-CT that is positive by contrast with that of normal individuals. No such contrasted figure has been displayed regarding drug-induced parkinsonism. It is assumed that the course of Parkinson disease should prevail over the drug-associated type. A case is hereby reported, that displays the fortuitous occurrence of a drug-induced parkinsonism which may be more consistent with Parkinson disease.

Case-Report | Benharroch D, et al. *Adv Clin Med Res* 2024, 5(2)-76.

DOI: [https://doi.org/10.52793/ACMR.2024.5\(2\)-76](https://doi.org/10.52793/ACMR.2024.5(2)-76)

Introduction

Parkinsonism has a wide disparity. It is a strongly progressive condition. Two of the variants have drawn our attention: the first was qualified by its strong association with psychotropic drugs. In theory, the medicine interruption should resolve the neurological condition. But is it conclusive? [1].

Drug-induced Parkinsonism exhibits some of the symptoms better known from PD. Among the motor symptoms, brady kinesia, with its slower movements, is due to an abnormal basal ganglia function; tremor; walking disturbances; drag-one's-feet; micrography; hypophonia; dysphagia of liquids; hypomimia; frozen facies; loss of stable posture [3].

Among the non-motor symptoms, one notes mild cognitive impairment (MCI); dementia; constipation; nocturnal hypersalivation; anosmia; urogenital impairment; Alzheimer disease [4, 5]. Anti-PD therapy – levodopa and apomorphine have persisted over the years. The urogenital dysfunction may be among the earliest symptoms of PD [6-12]. The treatment of drug-induced parkinsonism (DIP) is based essentially on the restriction of the psychotropic medication of concern. This depends on the capacity of the patient to do well in the absence of the given therapeutic agent. Therefore, the course of DIP may see more advantageous, as a progressive deterioration does not occur. However, the capacity to sustain an arrest of the antipsychotic drug is not homogenous. A rare incidence of PD, secondary to protracted parkinsonism, may be found especially in DIP.

Drug-associated Parkinsonism Versus PD

It is generally accepted that a symptom that characterizes any blend of PD, may also determine the features of DIP. The disease severity varies in both sorts. However, a general impression grants a more severe condition in PD. An attempt at an elucidation concerns PD being as a rule, a progressive neurodegenerative condition leading the elderly patient in an obligatory down hill course.

It is not unusual for the neurologist to declare that his patient suffers 'only' from parkinsonism, implying that his disease is milder than the PD considered at first. Furthermore, the patient is discharged with little or no instructions. "He will find a way to learn how to organize his life!". The issue is compounded by unusual cases. Here, the diagnosis might exhibit a more significant disparity between an apparently classical PD and a suggested Parkinsonian case.

A priori, the neurologist should find his way by using special imaging. The technique, called [18F] F-DOPA/PET-CT has been found to be highly sensitive in PD patients when compared with normal individuals. But we have no way to sort out the technique when comparing PD with Parkinsonism or more specifically, with drug-induced Parkinsonism [13,14].

The Report of a Case

This 58-year-old man exhibited urogenital dysfunction, constipation, right hand tremor, nocturnal hypersalivation and long-standing anosmia (unrelated with COVID-19). He was found dragging his legs and complaining of dysphagia to liquids. He was later shown to have MCI [15-17]. The overall picture was

consistent with PD, but it could be consistent as well with Parkinsonism. However, a thorough exploration of the patient's medical history, further revealed that at the age of 29, he had been diagnosed with bipolar affective disorder, including a psychotic (manic) episode, treated with antipsychotic agents, with minimal doses, to the present day.

The last evolution suggests drug-induced parkinsonism. Impressed by the patient's long list of PD-like symptoms, the neurologist required a DOPA/PET-CT. The imaging resolution excluded the involvement of the basal ganglia by PD. But the technique is based on the comparison of the imaging of PD with normal individuals, but not of PD with patients with parkinsonism [18]. It seems that the control (comparison) is not optimal in this case!

Conclusions

The patient reported hereby is suspected of suffering from Parkinsonism, more probably of drug-induced Parkinsonism. The DOPA/PET-CT examination was negative. Based on this patient's history, a query is raised concerning the comparison between PD and drug-induced Parkinsonism, on which the literature has remained silent. It seems that we don't know how to handle a [18F] F-DOPA/PET-CT examination that evaluates a PD diagnosis against a drug-induced Parkinsonism singling out.

Acknowledgements

Many thanks to Kibbutz Sde Boker.

The authors declare 'No conflicts of interest exist'.

No funding was considered necessary.

References

1. Galoppin M, Berroir P, Soucy JP, Suzuki Y, Lavigne GJ, et al. (2020) Chronic neuroleptic-induced Parkinsonism examined with Positron Emission Tomography. *Mov Disord.* 35(7):1189-98.
2. Chen M, Mor DE. (2023) Gut-to-brain alpha-Synuclein transmission in Parkinson's disease: evidence for Prion-like mechanism. *Int J Mol Sci.* 24(8):7205.
3. Haehner A, Hummel T, Reichmann H. (2014) Mental dysfunction in Parkinson's disease. *J of Parkinson's Dis.* 4:189-195.
4. Haddad R, Denys P, Arlandis S, Giannantoni A, Popolo GD, et al. (2020) Nocturia and nocturnal polyuria in neurological patients: from epidemiology to treatment. *Eur Urol Focus.* 6(5):922-34.
5. Batla A, Tayim N, Pakzad M, Panicker JN. (2016) Treatment options for urogenital dysfunction in Parkinson disease. *Curr Treat Options Neurol.* 18(10):45.
6. Graham JN Jr, Desroches BR, Weiss JP. (2014) Nocturia causes vary with each decade. *Curr Opin Urol.* 24(4):358-62.
7. Koza Z, Ayajuddin M, Das A, Chaurasia R, Phom L, et al. (2023) Sexual dysfunction precedes motor defects, dopaminergic neuronal degeneration, and impaired dopamine metabolism: insight from Drosophila model of Parkinson disease. *Front Neurosci.* 17:1143793.
8. Piccardo A, Cappoccio R, Bottoni G, Cecchin D, Mazzella, et al. (2021) The role of the deep convolutional neural network as an aid to interpreting brain[(18)F]DOPA PET/CT in the diagnosis of Parkinson's disease. *Eur Radiol.* 31(9):7003-11.

9. Darcourt J, Schiazza A, Sapin N, Dufour M, Ouvrier MJ, et al. (2014) 18F-FDOPA PET for the diagnosis of Parkinsonian syndromes. *Q J NuclMed Mol Imaging*. 58(4):355-65.
10. Crockett RA, Wilkins KB, Aditham S, Bronte-Stewart HM. (2023) No laughing white matter: cortical cholinergic pathway and cognitive decline in Parkinson's disease. medRxiv. 05 01 23289348.
11. Shi Z, Zhang J, Zhao P, Li X, Liu S, et al. (2023) Characteristics of mild cognitive impairment and associated factors in MSA patients. *Brain Sci*. 13(4):582.
12. Andrade EIN, Manxhari C, Smith KM. (2023) Pausing before verb production is associated with mild cognitive impairment in Parkinson disease. *Front Hum Neurosci*. 17:1102024.
13. Shin HW, Hong SW, Youn YC. (2022) Clinical aspects of the differential diagnosis of Parkinson's Disease and Parkinsonism. *J Clin Neurol*. 18(3):259-70.