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Late-onset Psychosis Leads to the Diagnosis of Parkinson's Disease: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. Author ACT wrote the case report, performed the literature research, made the submissionand all the final corrections of the manuscript. Authors ACT, AK, GNP, OS, PK, ES, GD and ID were all responsible for the patient's treatment during her hospitalization in the Neurology and the Psychiatry departments and reviewed the final version of the manuscript. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Psychotic symptoms are common in Parkinson's disease (PD) and are associated with poorer quality of life, significant patient morbidity, early mortality, and caregiver burden. We present a case of a 49-year old female patient with late-onset psychosis, which led to the diagnosis of PD. This clinical case combines all the rarely presented symptoms related to PD psychosis, and the diagnosis was formed after a thorough clinical, imaging, and neuropsychological examination.

Keywords: Parkinson's disease; psychosis; neuroleptics.

1. INTRODUCTION

Parkinson's disease (PD) psychosis refers to a spectrum of illusions, hallucinations, and delusions that occur throughout the disease course. Evolving literature highlights the importance of recognizing and treating PD psychosis and understanding its role as a clinical biomarker of disease stage, distribution, and future progression [1]. According to the National Institute of Neurological Disorders and Stroke (NINDS) and National Institute of Mental Health (NIMH) workgroup, the psychiatric symptomatology forms a continuum progressing over the course of PD [2].

Large patient series suggest that up to 50% of PD cases will present hallucinations during the disease course [1,3] and the Parkinson's Progression Marker's Initiative (PPMI) reported that the prevalence of psychosis at the time of PD diagnosis reaches the 3% [4]. Moreover, PD patients who reported hallucinations at the time of PD diagnosis have an increased risk of Dementia in the next few years [1].

We present a case of a middle-aged woman whose psychotic symptoms led to the diagnosis of a neurodegenerative disease, the PD.

2. CASE REPORT

A 49-year old female patient presented with auditory hallucinations of threatening voices, persecutory and somatic ("there are snakes in my head") delusions, in the emergency department in August 2018. At that time, she had no movement disorder complaints or other known comorbidities. She was hospitalized and treated as late-onset psychosis. In the beginning, she was treated with IV olanzapine with inadequate response, while IV haloperidol was used subsequently, combined with biperiden, and lorazepam. She was discharged with haloperidol per os, as no signs of a motor/extrapyramidal dysfunction were apparent. Her treatment was reversed to haloperidol decanoate due to her lousy compliance with the per os treatment.

She had a daughter with an unknown psychiatric history and a son diagnosed with Schizophrenia. According to the family, two years earlier, the patient presented depressive symptoms which gradually deteriorated.

She had no-compliance with the treatment, and a second hospitalization followed in January 2019. She received the previous therapy with haloperidol decanoate and biperiden. In March 2019, she presented with axial impairment, high rigidity, bradykinesia, unable to stand and walk, behavioral, and sleep disorder. At first, her clinical condition was treated as drug-induced parkinsonism. Despite the stop of haloperidol and the high doses of biperiden, her extrapyramidal signs were irreversible. Clozapine at a low dose was initiated, and the positive psychotic symptoms remitted. Neurologists consulted the addition of clonazepam and levodopa with gradual but poor outcomes. She removed to Neurology department for further investigation. Her neurological examination revealed a well-oriented patient with great nuchal rigidity, muscular strength 5/5 in upper limbs, 4/5 in proximal lower limbs but 2/5 distally (drop foot bilaterally), deep tendon reflexes symmetrical in upper and lower limbs, plantar reflexes were normal, extrapyramidal rigidity was present in upper and lower limbs as well, nasal-ocular reflex inexhaustible and sucking reflex present bilaterally. A brain MRI was performed with no specific pathological findings other than brain atrophy and few chronic vascular lesions in corona radiata (see Fig. 1). She was further investigated with a neurophysiological study, which revealed impairment of α -somatic motor neurons in two levels (cervical and lumbar-sacral spinal cord). Therefore, she was genetically tested with C9ORF72 for possible FTD/ALS, which was negative. She was investigated for Wilson's disease and other heavy metals, with normal findings. She investigated further with (DAT-scan), 123I-loflupane which pathological, and the subsequent 123-I-IBZM confirmed the diagnosis of idiopathic Parkinson's Disease (see Fig. 2).

Her cognitive status was impaired as assessed by the neuropsychological battery of Addenbrooke (ACE-R: 52/100), MMSE: 25/30 with multiple cognitive deficits (memory, attention, visual-spatial, learning ability) and that was in favor of the presence of a neurocognitive disorder as well.

The patient left the hospital in improved clinical condition, able to stand and walk few steps with bilateral support, with significant muscular atrophy due to the long period of bed rest, treated with a low dose of clozapine and high doses of levodopa.

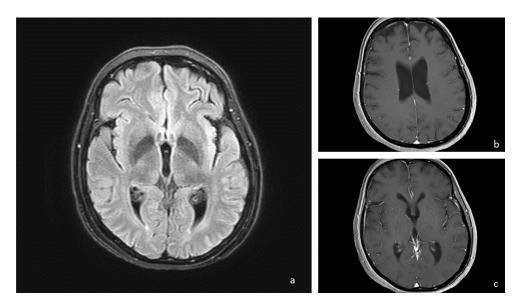


Fig. 1. Brain MRI. a. Axial Tw2 FLAIR at Basal Ganglia; b,c. Axial Tw1 with contrast showing mild frontal lobe atrophy regarding patient's age

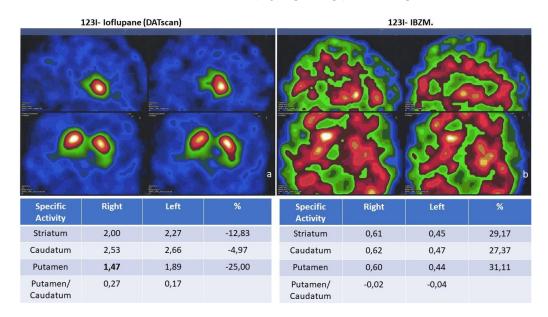


Fig. 2. a. basal ganglia SPECT with 123I - Ioflupane (DATscan); b. basal ganglia SPECT with 123I- IBZM

3. DISCUSSION

Our patient presented mood disorder, followed by psychotic episode with flattened affect, auditory hallucinations, and somatic delusions, without fluctuation of her cognitive status and any prominent movement disorder at first. The prominent extrapyramidal signs were triggered by the initiation of antipsychotic treatment in an underlying neurodegenerative disease.

According to the current diagnostic criteria, psychosis in PD should be presented after the diagnosis of PD [2]. However, studies are suggesting "pre-motor" symptoms in drug naïve, de novo PD patients or early in the disease process, but these cases are much less common [5]. The patient's auditory hallucinations and delusions are also rare symptoms in PD related psychosis [6]. She also did not have any fluctuation in her cognitive function. However, the

motor phenotype of PD of our patient with no tremor and more axial impairment, higher rigidity. and bradykinesia is related to more likely to develop psychosis [7]. Because of the lack of an initial neurological examination at the beginning of the psychotic symptoms, we cannot be certain about the timeline of the diagnosis of psychosis in Parkinson's disease. However, the motor phenotype, the pre-existing mood and sleep disorder, and the response to the low-dose clozapine strengthen our notion that our patient might had prodromal symptoms that were worsened by neuroleptic drugs making the diagnosis of early-onset psychosis in Parkinson's Disease likely [8]. The family history of her schizophrenic son may be related to a genetic risk factor, as well. The neurocognitive disorder that was diagnosed is also strongly associated with the early presentation of psychotic symptoms in PD, while the second consists of an independent risk factor for dementia.

4. CONCLUSION

The prominent extrapyramidal symptoms after initiation of antipsychotic treatment may be the clue of the diagnosis of an underlying neurodegenerative disease, and further investigation should be considered. There is a need for close monitoring and early intervention if the clinical features suggest a new onset of a movement disorder.

CONSENT

As per international standard informed and written patient consent has been collected and preserved by the authors.

ETHICAL APPROVAL

As per international standard written ethical permission has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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