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Association between Antidiabtic Medications and Worsening of Parkinson's Symptoms: A Case Report

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Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript

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Case Study

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ABSTRACT

Introduction: Parkinson's disease (PD) is one of the most prevalent neurologic disorders, leading to progressive disability; it is characterized by tremors, slow movements, stiffness in arms and legs, and balance impairment; PD symptoms can be slowed but not stopped by treatment such as a combination of Carbidopa/ Levodopa. Although it's widely used for PD, it risks dyskinesia, orthostatic hypotension, and dizziness. The prevalence of PD in Saudi Arabia has been estimated to be 27 per 100,000 populations, and the occurrence of PD in the U.S. is approximately 20 cases per 100,000 people per year.

Case presentation: A 61 years old male presented with worsening PD symptoms, especially dysarthria symptom; he had a history of diabetes with A1C of 8.5%, on metformin, insulin glargine, liraglutide, and linagliptin, with good adherence, and he had a history of Parkinson on levodopa/carbidopa. Even there are no known drug-drug interactions between antidiabetic medication and levodopa/ carbidopa, he reported that coadministration of antidiabetic medications with levodopa/carbidopa cause PD symptoms worsening, especially dysarthria worsening. This drug-drug interaction was noticed when the patient tried to stop all of his antidiabetic medication except insulin mixtard, when he noticed dysarthria symptoms improved. He is currently on insulin mixtard for diabetes with an A1C of 6.7%.

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Conclusion: Unintentional drug-drug interaction poses a challenge to the healthcare provider, so this report highlights the importance of adverse drug-drug interaction of antidiabetic with levodopa/carbidopa, its presentation, and management.

Keywords: Parkinson's disease; Diabetes mellitus; levodopa/carbidopa; antidiabetic medication.

1. INTRODUCTION

Parkinson's Disease (PD) is a chronic, progressive neurodegenerative disease characterized by motor and nonmotor features. The disease clinically influences patients, families, and caregivers through its dynamic degenerative ramifications for adaptability and muscle control. The motor appearances of PD are credited to the insufficiency of striatal dopaminergic neurons. The term parkinsonism is an appearance perplexing used to portray the motor components of PD, which join tremer, bradykinesia.

individuals may have PD in their 30s and 40s, sex contrasts in the recurrence of PD are reflected in a 3:2 male to females, with conceded starting in females attributed to the neuroprotective effects of estrogen on the nigrostriatal dopaminergic system [1].

Wearing-off is a complication that can occur after a few years of using levodopa to treat Parkinson's. During wearing-off [WO] symptoms of Parkinson's start to return or deteriorate before the next dose of levodopa is expected [2].

Motor fluctuations ordinarily happen when levodopa is wearing off, however they can occur at different occasions as well. This is called 'end of the dose wearing off' or just 'wearing off.' Sometimes the impacts of wearing off happen rapidly. These are called "switching off" or "on/out [2].

The study found young age of Parkinson's onset, longer duration of disease, longer time on levodopa treatment, a higher dose of levodopa, and dopamine agonist were associated with WO phenomena. WO is considered a levodopa-related complication that usually occurs in the late stage of PD. WO was seldom screened in patients treated without levodopa [3].

There is a developing proof recommending that patients with Type 2 diabetes have an expanded danger of fostering Parkinson's disease and offer comparative dysregulated pathways common underlying pathological mechanisms. Although the details of the pathogenesis of PD remain to be further defined, a growing body of evidence links insulin resistance to PD, and while the underlying mechanisms remain unclear, there is accumulating evidence suggesting that alpha-synuclein can interfere with normal insulin signaling via its action on inflammation and the AKT pathway [4].

To highlighted the importance of unintentional drug-drug interaction, which may pose a challenge to a healthcare provider, and the importance of adverse drug-drug interaction of antidiabetic with levodopa/carbidopa, its presentation, and management.

2. CLINICAL PRESENTATION

A 61 years old male with a known case of Parkinson's disease presented to his scheduled appointment in a diabetic clinic complained of dysarthria worsening. He had a history of Parkinson's disease since five years ago on (carbidopa25 mg/levodopa 25 mg), diabetes since ten years ago with A1C of 8.5%, was on insulin glargine 10 u daily, and metformin 750 mg 2 tabs once daily, linagliptin 5 mg daily, and liraglutide 1.6 mg. He mentioned that anti-diabetic medication affects levodopa/ carbidopa and causes PD symptoms to worsen.

His past medical history was notable for hypertension was on lisinopril 2.5 mg currently stopped due to hypotension, dyslipidemia with LDL of 3.97 mmol/L, HDL 1.10 mmol/L, and cholesterol 5.96 mmol/L on atorvastatin 20 mg daily, PBH on solifenacin 5mg daily, IHD s/p CABG on clopidogrel 75 mg daily, prostate cancer s/p prostatectomy, colon resection, and GERD on pantoprazole 40 mg.

When his diabetes was controlled with A1c of 6% and fasting blood glucose of 100 mg/dL, he switched to metformin only with a dose of 750 mg daily, then he experienced it's effect on (carbidopa 25 mg/levodopa 25 mg) response when he decided to stop taking it. His glucose worsened again with an A1C of 8.5%, so he started on liraglutide 0.6 mg with metformin 850 mg after that. His A1C improved to 6.7%, but he

can't tolerate dysarthria worsening, so he switched to insulin mixtard 20 u morning and 10 u pm only for his diabetes with no PD symptoms worsening.

3. DISCUSSION

Parkinson's disease (P.D.) is viewed as quite possibly the most widely recognized neurodegenerative disease [5], and Diabetes mellitus (D.M.) is the most common chronic metabolic disease [6].

There are numerous likenesses among D.M and P.D. Clinical highlights of the two illnesses result from the obliteration of specific cells, in particular pigmented dopamine cells in P.D. and pancreatic beta cells in D.M. The deficiency of these cells brings about diminished insulin in D.M.and dopamine in P.D [7-8]. Both disorders are ongoing sicknesses. The two illnesses result from a decline in a particular substance: dopamine in P.D. and insulin in D.M. In addition, the two disorders emerge because of the annihilation of specific cells, dopaminergic cells in P.D, and pancreatic beta-cell in D.M. As of late, numerous epidemiological and trial studies showed an association among D.M and P.D There are normal basic systems in the pathophysiology of the two sicknesses. These hidden systems incorporate mitochondrial brokenness, oxidative pressure, hyperglycemia, and irritation. Insulin resistance is the hallmark of D.M, primarily type 2 diabetes mellitus (T2DM) [8]. The association between T2DM and P.D. has been previously reported since patients with T2DM appear to have an increased risk of developing P.D. In a large cohort of 8 million people, Pablo Fernandez et al. showed a higher rate of post-PD after T2DM. However, some studies have shown the opposite or the absence of a relationship between these diseases [9].

Parkinson's disease (P.D.) is one of the most well-known neurodegenerative disease and a main source of death and inability. P.D. is perhaps the most widely recognized neurodegenerative disorder. The Parkinson's Disease Foundation reports that around 1 million Americans at present have the disorder. The rate of P.D. in the U.S. is around 20 cases for every 100,000 individuals each year (60,000 every year), with the mean period of beginning near 60 years [1]. P.D. affects ∼2% of the population over 65 years of age, and its prevalence increases as the population ages. The etiology of Parkinson's relies upon a blend of genetic factors (10% of cases) and conceivably natural variables. Besides, most instances of Parkinson's are idiopathic, and the specific etiology stays unclear [10-11].

Motor features in P.D. patients include tremer, inflexibility, and bradykinesia. The motor features of P.D. might correspond with the patient's age at beginning explicitly; tremert toward the start is twice in patients more than 64 years contrasted with those more youthful than 45 years old. What's more, difficulties identified with the term of treatment, for instance, the relationship of dystonias and dyskinesias with the length of levodopa treatment, are more in patients analyzed at more youthful ages (45 to 55 years old) [1].

Epidemiological investigations have reported that T2DM expands the danger of Parkinson's disease. What's more, clinical investigations portrayed that side effects of Parkinson's disease were essentially more awful after the beginning of T2DM [9].

We reported a case of unintentional drug-drug interaction between antidiabetic medication and levodopa/carbidopa, leading to P.D. symptoms worsening, especially dysarthria declining in 61 years old males with known cases of diabetes since ten years ago and Parkinson's disease since five years ago. He can't tolerate the worsening of PD symptoms with antidiabetic medication except for insulin mixtard.

4. CONCLUSION

This case highlighted the importance of unintentional drug-drug interaction, which may pose a challenge to a healthcare provider, and the importance of adverse drug-drug interaction of antidiabetic with levodopa/carbidopa, its presentation, and management .

A few epidemiological and exploratory investigations have uncovered the relationship between diabetes (DM) and Parkinson's disease (PD).

More investigations could prompt the disclosure of a single medication that can be utilized in the treatment of both DM and PD.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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