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Drug Induced Dyskinesia- Cinitapride In Grey Zone: A Case Report

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Abstract:
Tardive dyskinesia (TD) is a movement disorder that causes involuntary, repetitive body movements and is commonly seen in patients who are on long-term treatment with antipsychotic medications. However, several other classes of medications with different mechanisms are also associated with TD [1]
Case: *We present a case report of 59 year old male who presented to us with involuntary movements of bilateral upper limb and mouth from the last 1 year. History revealed that the patient was on tablet cinitapride from the last 2 years. Examination revealed involuntary oro-lingual movements and upper limb movements (right more than left). To the best of our knowlwdge, this is one rare case scenario of drug induced dyskinesia been ever reported for the drug cinitapride.*
Keywords: *Drug-induced, tardive dyskinesias, cinitapride, movement disorder*

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I. Introduction:

The tardive dyskinesia (TD) form of dyskinesia gets its name from the slow—or tardive—onset of involuntary movements of the face, lips, tongue, trunk, and extremities.[2] The pathophysiology of TD lacks a universally accepted theory and mechanism. Several hypotheses have been proposed that include prolonged blockade of postsynaptic dopamine receptors leading to dopamine receptor supersensitivity, gamma-aminobutyric acid (GABA) depletion, cholinergic deficiency, oxidative stress, altered synaptic plasticity, neurotoxicity, and defective neuroadaptive signaling.[3] The most commonly implicated drugs include antipsychotics, anti-emetics (metoclopramide and prochlorperazine) and some calcium channel antagonists with dopamine receptor blocking properties (cinnarizine and flunarizine). The minimum duration of exposure to the drug is three months, or one month in adults aged over 60 years.[4] Cinitapride is a novel gastrointestinal (GI) prokinetic agent used in many countries as a treatment for functional dyspepsia, gastroesophageal reflux, hiatus hernia, constipation, and other functional GI disorders. Its substituted benzamide structure confers agonist actions at 5-HT4 and 5-HT1 serotonergic receptors and antagonist actions at D2 dopaminergic and presynaptic 5-HT2 serotonergic receptors in the myenteric plexus, promoting acetylcholine release.[5,6]

II. Case:

A 59 year old male known case of DM and HTN since 22 years, hypothyroidism since 10 years and CKD-ESRD on MHD since 3.5 yrs, presented with complaint of involuntary movements bilateral upper limbs and mouth since 1 year. These movements were gradual in onset, irregular, random with amplitude of movements more in Right UL. They persisted during sleep and were aggravated during stress. He was on T.Trihexphenidyl 1mg BD and T. Primidone 25mg BD since 1 year from elsewhere but showed no improvement. Patient also complained of occasional falling of objects from hands. Apart from Inj Insulin, T. Cilinidipine, T. Thyrox & treatment of CKD, Patient was on T. Cinitapride since 2 years. Examination revealed involuntary lip smacking movements , jaw and upper limb movements (Right>Left). Power and tone were normal. There was no rigidity and bradykinesia, Finger nose test and diadokokinesis was also normal. Laboratory findings including CBC, LFT, TFT and electrolytes were normal, MRI Brain showed no structural lesions. Patient was advised to stop T. Cinitapride and advised not to take any other prokinetics. T. Primidone 25mg BD and T. Trihexphenydyl 1mg BD were continued. Patient showed decrease in amplitude of involuntary movements 6 weeks after cessation of Cinitapride.

III. Discussion:

We searched google scholar, Pubmed indices but till date tardive dyskinesia has not much been reported as yet by cintapiride which we observed in this case.

Earlier, as reported by ***Aaron de Souza et al*** in 2017 which had shown a patient with a presumptive diagnosis of drug-induced Parkinsonism and tremor improving on stoppage of cinitapride after 6 months. This improvement on cessation of drug as seen in our case also shows a possible role of cinitapride in movement disorders.[7]

Sang-Wook Hong et al in 2022 showed 3 patients developing parkinsonism and tardive dyskinesias with Mosapride even though it didn't have direct action on dopamine receptors like cinitapride, which later improved on drug stoppage. Hence compelling us to explore more regarding this unexplored area.[8]

IV. Conclusion:

This case has made us more intuitive to take much more closer observation in history taking as well as the examination with its close interactive associations with various other drug intakes, which sometimes the patient also seems to declare less to his/her physician, making it a bigger mask of actual prevalence of drug induced movement disorders among the patients. Till date not many cases have been reported with cinitapride, hence compelling us to be cautious in prescribing it for long term usage as prevention is better than cure.

Declaration of patient consent: The patient has given consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity but anonymity cannot be guaranteed.

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