ISSN: 0975-766X
CODEN: IJPTFI
*Available Online through* *Research Article*
www.ijptonline.com

# A CASE REPORT ON ARIPIPRAZOLE INDUCED PARKINSONISM AND ITS MANAGEMENT IN PATIENT OF MYOCLONIC TO GENERALIZED TONIC CLONIC SEIZURE WITH PSYCHOSIS NOS

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*Received on: 20-11-2019* *Accepted on: 29-12-2019*

**Abstract**

Parkinsonism is neurological syndrome characterized by six cardinal features tremor, rigidity, bradykinesia, flexed posture, loss of postural reflexes, and the freezing phenomena. Drug induced parkinsonism is most common adverse effect induced by drugs including antipsychotics, anticonvulsants, calcium channel blockers and lithium; which may arises as a result of dopamine receptor D2 blockage in striatum via extrapyramidal pathway and hence causing reduced stimulation of cortex. A case of 19 years male was bought to hospital with chief complains of flash of light in front of eyes, rolling of tongue, whole body rigidity, frothing from mouth, slurred speech, hallucination followed by episode of seizure for 2-3 minutes, sometimes preceded by jerking of neck on one side followed by loss of consciousness and unable to remember the episode after gaining consciousness. All characteristics were continuously repetitive from past 3 years, occurring more than once a week.

Patient also complaining unprovoked anger outbreak and muttering to self in the last 5 months with tremor, rigidity and reduced reflex symptoms. Patient was taking regular medication, sodium valproate for control of seizures and aripiprazole for aggressive behavior. Based on chief complaints, medical history and basic clinical features and laboratory data examination, patient was diagnosed with myoclonic to generalized tonic-clonic seizure with drug induced parkinsonism, Hyperammonemia and vitamin deficiency. In order to control the symptoms of drug induced parkinsonism, aripiprazole was stopped immediately and for

controlling seizures, sodium valproate, lamotrigine, levetiracetam, clonazepam and lorazepam were started while ropinirole for parkinsonism as well as trihexyphenidyl and quetiapine for aggressive behavior was included in therapy. At the time of discharge, patient was found to be well managed with parkinsonism and follow up was recommended for continued therapy for seizure. Such cases of antipsychotic drug therapy, therapeutic drug monitoring should be performed in continuous manner and the offending drug should be stopped immediately with suitable alternative drug.

Health care professionals need to be made aware of these adverse effects associated with anti-psychotic therapy via conduction of quality-based seminars, conferences, published medical literature and learning programs.

**Keywords:** Aripiprazole, Parkinsonism, Sodium valproate, Ropinirole, Trihexyphenidyl, Quetiapine.

## Introduction

Drug Induced Parkinsonism (DIP) is the most common movement disorder induced by drugs that affect dopamine receptors[1-2]. Since the clinical manifestations of DIP are very similar to those of Parkinson's disease (PD), patients with DIP are frequently misdiagnosed as having PD [1-3].

Typical antipsychotics are the most common causes of DIP. However, atypical antipsychotics, which were thought to be free from Extrapyramidal symptoms (EPS), can also induce parkinsonism. Although motor symptoms are initial indicators of Parkinson's disease (PD), the associated neuropsychiatric symptoms cause the greatest disability in advanced stages of the disease. Psychosis in PD patients is characterized mainly by the presence of visual hallucinations. This may be accompanied by illusions and paranoid delusions, the latter being the most serious symptoms, leading to hospitalization and even suicide attempts. Managing psychosis is one of the most difficult challenges in the care of PD, and thus more treatments are required. Several antipsychotic agents have been proposed, including quetiapine, risperidone, olanzapine and clozapine. Aripiprazole belongs to atypical antipsychotic drug which has been associated with fewer EPS that are thought to arise from a greater than 80% D2 receptor occupancy rate in the striatal area of the basal ganglia[4-10].

## Case Report

A case of 19 years male was bought to hospital with chief complains of flash of light in front of eyes, rolling of tongue, whole body rigidity, frothing from mouth slurred speech, hallucination followed by episode of

seizure for 2-3 minutes, sometimes preceded by jerking of neck on one side followed by loss of consciousness and unable to remember the episode after gaining consciousness. All characteristics were continuously repetitive from past 3 years, occurring more than once a week. Patient also complaining unprovoked anger outbreak and muttering to self in the last 5 months with tremor, rigidity and reduced reflex symptoms. Patient was on Sodium valproate 500mg BD (anti-epileptic) and Aripiprazole 7.5mg HS (antipsychotic), Propanolol 20mg OD to maintain BP and Lorazepam 1mg SOS for seizures. Laboratory tests like CBC were performed with high value of RDW (14.30%) indicative of vitamin deficiency and higher value of ammonia (198.49) indicative of hyperammonemia. Based on chief complaints, medical history and basic clinical features and laboratory data examination, patient was diagnosed with myoclonic to generalized tonic-clonic seizure with psychosis NOS and drug induced parkinsonism vitamin B12 deficiency and hyperammonemia.

In order to control the symptoms of drug induced parkinsonism, Aripiprazole was stopped immediately and for controlling seizures, Sodium valproate 500mg BD, Lamotrigine 12.5mg HS, Levetiracetam 5ml BD, Clonazepam 5mg HS and Lorazepam 1mg HS were started while Ropinirole 0.25mg TDS for parkinsonism as well as Trihexyphenidyl 2mg and Quetiapine 25mg HS for aggressive behavior 10 DNS with Injection Optineuron TDS for Vitamin B12 deficiency was included in therapy. At the time of discharge, patient was found to be well managed with parkinsonism and Anticonvulsants were recommended for continued therapy for seizure.

**Discussion**

The blockage of $D_2$ receptors by antipsychotic drugs in the striatum leads to stimulation of GABA- and encephalin-containing striatal neurons at the origin of the indirect pathway without alteration of the direct pathway, followed by stimulation of the subthalamic nucleus. This leads to increased GABAergic inhibition of the thalamocortical projection by facilitation of the inhibitory projection from the globus pallidus/substantia nigra pars reticulate. Chronic $D_2$ receptor blockade also induces changes in the direct pathways of the basal ganglia-motor loop to cause orolingal dyskinesia[11]. In this particular case, patient developed Aripiprazole induced parkinsonism via nigrostriatal pathway which was not tolerated by patient. So, Aripiprazole was stopped immediately and alternate antipsychotic drug was prescribed by physician

along with antiparkinsonism drugs and anticonvulsants for epilepsy. At the time of discharge, patient was well treated for drug induced parkinsonism and follow up was done for seizures management.

**Conclusion**

Study concluded that any patient undergoing treatment of psychosis should be regularly monitored and therapeutic drug monitoring is essential to avoid parkinsonism as a side effect in them. ADR monitoring should be done and any case of adverse reactions should be assessed and managed accordingly. In case of polypharmacy, all the medications taken by patient should be kept under scrutiny to avoid any type of ADRs or drug interactions.

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