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Original Research Paper

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CASE REPORT OF SEIZURE PROBABLLY DUE TO OLANZAPINE

Dr. Pooja Dixit	Resident Doctor , Dept.of Psychiatry , [Ravindranathtagore medical college udaipur] Rajasthan
Dr. Anilkumar khatik*	Resident Doctor ,Dept.of Psychiatry,[Ravindranathtagore medical college udaipur] Rajasthan *Corresponding Author
Dr. Sumit Mittal	Resident Doctor ,Dept.of Psychiatry[Ravindranathtagore medical college udaipur] Rajasthan

ABSTRACT Second generation antipsychotics may be associated with electroencephalogram abnormalities. Occurance of seizure with Olanzapine is very rare. Some time olanzapine can cause lower seizure threshold. We report the case of a 61 year old female with delusional disorder who developed new-onset generalized tonic-clonic seizure probably due to Olanzapine. Electroencephalogram showed epileptiformdischarges.The seizure risk associated with Olanzapine was reviewed.

KEYWORDS : Olanzapine, seizure, side effects,drug-induced seizure.

INTRODUCTION:

Olanzapine is an atypical antipsychotic primarily used to treat schizophrenia and bipolar disorder. Common side effects include weight gain, movement disorders, dizziness, feeling tired, constipation, and dry mouth. Other side effects include low blood pressure with standing, allergic reactions, neuroleptic malignant syndrome, high blood sugar, seizures, gynecomastia, erectile dysfunction, and tardive dyskinesia.[1] Olanzapine can lower seizure threshold and induce epileptiform discharges.(2)

Olanzapine is one of the most commonly used antipsychotic agents, and chemically simillar to clozapine. The dose-related enticement of seizure by Clozapine though is well known, and that of Olanzapine rare cause seizure. Among the trials conducted for FDA approval of drugs, the incidence of seizure was significantly higher in the Clozapine and Olanzapine groups.[3]The premarketing trials have found incidence of seizures at 0.88%, which is comparable to other conventional antipsychotics.[3] Despite its proconvulsant liability, the literature reporting seizures are sparse. Few case reports of fatal status epilepticus and myoclonic status have attributed Olanzapine as the causative agent.[4,5] Hereby we are reporting a case of seizure in a patient taking Olanzapine .

CASE:

A 61 year old female brought by her son to psychiatric emergency with complaint of frothing from mouth, tonic-clonicmovement,uprolling of eyes and loss of conciousness 3hourback. Two year back she was diagnosed as delusional disorder(documented).and she was on Tab olanapine10mg.and was maintained on this dose. But because of her husband died 1 month back she discontinue the medication. after that she gradually started irritability, suspiciousness for that 2days back she again started tab olanzapine 20mg of the counter. She has no history of previous sezuire attack, no history of any substance abuse. No any medical history. On eamination she was concious, partly oriented. No any other neurological finding present. computed tomographic scan and metabolic investigations were normal.EEG showed generalized epileptiform discharge. Olanapine was stopped and started sodim valproate for sezuire. After 3days she was discharge on tab sodium valproate 500mg bd and tab haloperidole 5mg bd.nextfollowup we stopped the anticonvulsant.with no recurrence of seizures and did not require anticonvulsant therapy.

DISCUSSION:

In this case study patient was develop seizures because she suddenly stared olanzapine over the counter in higher dose than previous dose. Seizure occur ed only when patient on olanzapine.and did not have seizure in the brief follow-up period of stopping it. No other alternative explanation could be found for sudden appearance of seizures in our case. The adverse event got repeated in our case but the trial for attribution by stopping and restarting the medicines could not be done owing to high fatal risk of the seizure. The objective evidence in the means of abnormalepileptiform discharge on EEG was noted. Thus concluding according to Naranjo algorithm with a score of 5, the seizure occurring in our case was probably due to Olanzapine.[6] As none of these mono-neurotransmitters can explain the differential seizurogenic potential of psychotropic drugs, dual neurotransmitter/receptor imbalances have been hypothesized. Drugs with higher dopamine:acetylcholine imbalance, serotonin:acetylchloine imbalance, D1:D2 antagonism, alpha 1:alpha 2 antagonism, and alpha 1:D2 receptor affinities are observed with higher rate of convulsions. Drugs with more affinity on dopamine receptors in cortical compared to subcortical (hippocampal/nigrostriatal) areas have also been noted to increase seizure liability.[7] Olanzapine is known to cause highest EEG changes, in 35-45% of cases, [8,9,10,11]among the non-Clozapine newer antipsychotics. [8,9,10,11] Atypicals have high propensity to cause EEG changes compared to typical antipsychotics.[12] Generalized/focal symmetrical theta and delta waves are more commonly found abnormal activities followed by asymmetrical slow waves, sharp waves with phase reversals, and spike-and-slow wave patterns. The later severe epileptic changes were noted in up to 11-15% of cases on Olanzapine.[6,10,11] EEG changes were noted at around 4-7 months of starting Olanzapine in most of the literature.[3,4,10] As abnormal epileptiform discharge on EEG could be seen in most of the reported cases including our case, so regular EEG monitoring should be done in high risk patient.

CONCLUSION:

Electroencephalogram showed epileptiform discharges; results of computed tomographic scan and metabolic investigations were normal. His antipsychotic was changed to haloperidol, and the patient showed a significant improvement in psychotic symptoms with no recurrence of seizures and did not require anticonvulsant therapy. Olanzapine has a profile similar to that of clozapine and shares its seizure-inducing potential. Typical antipsychotics such as haloperidol might be a safer option for such patients. it would be beneficial to monitor EEG as a seizure preventive strategy in high risk patients on olanzapine like Old age,

organicity, epilepsy, hypertension, bipolar disorders, comorbid OCD (Behere, 2009), after the cost effectiveness being evaluated.

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