CASE REPORT

Olanzapine-induced Convulsive Status Epilepticus: A case report from Eastern Nepal

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Abstract

The premarketing surveillance showed that the incidence of olanzapine-induced seizure was 0.88%. However, the data of post-marketing surveillance of olanzapine-induced seizure was limited. The causal effect of olanzapine-induced seizure was not established yet. We report a case of a 48-year-old female who had olanzapine-induced convulsive status epilepticus. Olanzapine was used for the treatment of post-capsular ischemic stroke hemichorea. Olanzapine can induce generalized tonic-clonic, focal and myoclonic seizure. An olanzapine-induced seizure is often self-limited with drug withdrawal. However, few patients with olanzapine-induced therapy need anticonvulsant therapy. Olanzapine can induce the seizure especially in patients with multiple medical co-morbidities.

Keywords: Olanzapine, Seizure, Status Epilepticus

INTRODUCTION

Olanzapine is an atypical antipsychotic that has affinity for dopamine, serotonin, histamine, adrenergic and muscarinic receptors. 1 The atypical antipsychotics have a higher risk for electroencephalographic changes than typical antipsychotics. 2 Among the atypical antipsychotics, not only clozapine but also olanzapine and quetiapine carries a higher risk for seizure than expected. This literature was confounded by the presence of medical co-morbidities, preexisting seizure disorder, and drug withdrawal. 3 The premarketing surveillance showed the incidence of olanzapine-induced seizure was 0.88%. 4 However, the data regarding the post-marketing surveillance of olanzapine-induced seizure is limited. The causal effect of olanzapine-induced seizure was not established yet. We report a case of a 48-year-old female who had olanzapine-induced convulsive status epilepticus.

CASE-REPORT

A 48-year-old female presented to the medical outpatient department of Internal Medicine of B.P. Koirala Institute of Health Sciences with a history of sudden onset of abnormal irregular jerky movement of the left hand for 12 hours. It was aggravated by lifting the hand and relieved by resting state and sleep. It was restricted to the left hand. She also had a complaint of a decrease in left grip strength. There was no history of a headache, fever, trauma and illicit drug intake. Past history- She was a case of type 2 diabetes mellitus and was taking Metformin 2gm and Glimepiride 4 mg per day. There was no history of any psychiatric co-morbidities and seizure disorder. She was a non-smoker and non-alcoholic. On examination finding Glasgow coma scale score - 15/15, BP 100/60 mmHg, Pulse rate- 72 beat /minute regular rhythm, Respiratory rate- 16 cycle /minute, temperature-98°F. On Central nervous system examination revealed left wrist flexion power- 4/5 and left wrist extension power 4/5. She had semi-purposive, non-repetitive, non-rhythmic and irregular movement in left hand. Other central examination, respiratory system, cardiovascular and gastrointestinal system revealed no abnormality. The provisional diagnosis of hemichorea was made.
INVESTIGATIONS
The baseline investigation findings of the patient are illustrated in Table 1. As shown in figure 1, the Computed tomography of the patient showed the lacunar infarct of the right internal capsule. The diagnosis of Post-internal capsule stroke chorea was made. Electroencephalogram was done on 2nd of seizure activity which showed the generalized epileptiform discharges is shown in Figure 2.

Table 1: Baseline investigation reports of the Patient

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Value</th>
<th>Normal value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin</td>
<td>13 g/dl</td>
<td>11-16 g/dl</td>
</tr>
<tr>
<td>Total leucocyte</td>
<td>10500 cell/mm cu</td>
<td>40000-11000 cell/mm cu</td>
</tr>
<tr>
<td>Platelet</td>
<td>400000 cell/mm cu</td>
<td>150000-400000/mm cu</td>
</tr>
<tr>
<td>Random blood sugar</td>
<td>350 mg/dl</td>
<td>&lt;140 mg/dl</td>
</tr>
<tr>
<td>Urea</td>
<td>32 mg/dl</td>
<td>10-50 mg/dl</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.7 mg/dl</td>
<td>0.5-1.5 mg/dl</td>
</tr>
<tr>
<td>Sodium</td>
<td>141 mmol/L</td>
<td>136-145 mmol/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.8 mmol/L</td>
<td>3.5-5 mmol/L</td>
</tr>
<tr>
<td>Calcium total</td>
<td>9.5 mg/dl</td>
<td>8.4-10.2 mg/dl</td>
</tr>
<tr>
<td>Ionized calcium</td>
<td>0.94 mg/dl</td>
<td>1.05-1.27 mg/dl</td>
</tr>
<tr>
<td>Urine ketones</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>HBAC</td>
<td>5.2%</td>
<td>5.5-6.4%</td>
</tr>
<tr>
<td>Serum bicarbonate</td>
<td>22 mmol/L</td>
<td>20-24 mmol/L</td>
</tr>
</tbody>
</table>

Abbreviation: HBAC - Glycated haemoglobin, g/dl-gram/deciliter, mg/dl-milligram/deciliter

Figure 2: Electroencephalography of a patient showing generalized epileptiform discharges

TREATMENT:
We prescribed aspirin 300mg followed by 75 mg once daily, atorvastatin 20 mg once daily, Injection Glargine 14 IU Subcutaneous once daily. One second day of admission Olanzapine 2.5 mg tablet was prescribed once daily for hemichorea. After four hours of intake of Olanzapine, she had Generalised tonic-clonic seizure repeatedly for one hour without regaining consciousness. The seizure was treated initially with and lorazepam 5 mg three doses but was not controlled. Injection Levetiracetam 2000 mg in 100 ml normal saline was given over one hour. After initiating levetiracetam, the seizure was controlled and she regained consciousness after six hours. Repeated Computed Tomography of the head showed no new changes. She was discharged on Tablet levetiracetam 500 mg twice a day, injection glaring 14 IU once a day, injective glulisine 10 IU SC thrice a day, Tablet aspirin 75 mg once daily, and tablet atorvastatin 20 mg once daily.

FOLLOW UP
She followed our outpatient department after two weeks. There was no new seizure activity. Tablet levetiracetam was tapered to 250 mg twice daily.

DISCUSSION:
We reported a case of Olanzapine-induced convulsive status epilepticus in a 48-year-old patient with Post-capsular stroke hemichorea. She recovered from status epilepticus with the
stoppage of olanzapine and use of Levetiracetam. Chorea is an unusual complication and observed in less than 1% of cases of acute stroke. Post-stroke chorea is treated with the typical or atypical antipsychotic agents. Our patient had post-stroke chorea due to the involvement of right internal capsule ischemic infarct. She was treated with Olanzapine 5 mg as the advantage of using atypical antipsychotics in the management of post-stroke chorea are a lower rate of parkinsonism, delayed dyskinesia and recurrent stroke. 5-7 Olanzapine was used to treat the chorea as reported by Dipple HC. 8 Olanzapine intake induced the convulsive status epilepticus in a 41-year-old patient taking olanzapine for bipolar disorder. Our patient recovered from the convulsive status epilepticus after withholding the olanzapine especially in the individual who has multiple co-morbidities. Psychotic disorder and seizure disorder can occur concurrently, Physicians must be cautious regarding the use of olanzapine for management of psychotic symptoms in a seizure disorder.

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CONFLICT OF INTEREST: None

REFERENCES: