CASE REPORT

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OLANZAPINE INDUCED SEIZURES: A CASE REPORT

OLANZAPİN İLE İNDÜKLENMİŞ NÖBETLER

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Abstract

Galactorrhea is defined as non-puerperal lactation and frequently occurs as an adverse drug reaction due to typical antipsychotics. Furthermore antidepressants, especially SSRIs, cause galactorrhea since the introduction of imipramine to psychiatry practice. Although galactorrhea ususally accompany increased prolactin levels, in some cases prolactin levels could be in normal range. To date there are two case reports of normoprolactinemic galactorrhea due to sertraline and here we report a patient who developed normoprolactinemic galactorrhea 1 month after initiating sertraline 50 mg/day.

Keywords: Sertraline, Galactorrhea, Normoprolactinemic

Özet

Galaktore doğuma bağlı olmaksızın süt salgılanması olarak tanımlanır ve tipik antipsikotiklere bağlı advers ilaç reaksiyonu olarak sıklıkla ortaya çıkar. Ayrıca, imipraminin psikiyatri pratiğine girişiyle birlikte antidepresanlar, özellikle SSRI'lar, da galaktoreye sebep olmaktadır. Genellikle galaktore prolaktin seviyesindeki artışa eşlik ederken, bazı olgularda prolaktin seviyesi normal aralıkta olabilir. Bugüne kadar sertraline bağlı iki normoprolaktinemik galaktore olgu raporu vardır ve bu çalışmada günde 50mg sertralin başlandıktan bir ay sonra normoprolaktik galaktore geliştirmiş bir hasta rapor edilmiştir.

Anahtar Kelimeler: *Sertralin, Galaktore, Normoprolaktinemik*

1. Introduction

Olanzapine is one of the most commonly used atypical antipsychotic agents. Though closely related to Clozapine structurally, the seizurogenic potential of olanzapine is limited. The premarketing trials have found incidence of seizures at 0.88% which is comparable to other conventional antipsychotics (Alper, Schwartz, Kolts, and Khan, 2007). But, few case reports of fatal status epilepticus (Wyderski, Starrett, Abou-Saif, 1999) and myoclonic status (Camacho, García-Navarro, Martínez, Villarejo, and Pomares, 2005) have attributed olanzapine as the causative agent. Hereby we are reporting a case of seizure in a patient receiving Olanzapine.

2. Case

A 58-year-old female was brought to the Emergency Department (ED) with "alteration of mental state." She reportedly experienced a single generalized tonic-clonic seizure 2 hrs back. She had recurrence of generalized tonic clonic seizure within half an hour of reaching Emergency Department. Her psychiatric history revealed that she was diagnosed as paranoid schizophrenia 2.5 years back and was on T. Olanzapine 7.5 mg/day. Informant reported that she missed medicine for previous three days and restarted the same day. Within one hour of taking medicine she had this first seizure attack. This was the first seizure in her life time. There was no history of alcohol and other substance use or any significant medical illness.

On examination in the ED, she was confused. The neurological examination was otherwise, unremarkable. Her vital signs were stable. Complete blood count, electrolytes, and liver function tests were in the normal range. MRI brain showed no abnormalities. EEG showed generalized epileptiform discharge.

Olanzapine was discontinued and was treated with anti epileptics Phenytoin and Lacosamide and. T Haloperidol 5 mg was started. Since she tolerated the drug and showed no further seizure, she was discharged on the same dose after 4 days.

3. Discussion

The present case suggests precipitation of seizures by Olanzapine which was restarted rapidly at relatively higher previously prescribed dose. The occurrence of seizure when patient was on Olanzapine and no recurrence during the brief follow up period of stopping

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it rules out other alternative explanations. 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 abnormal EEG was noted. According to Naranjo Algorithm (Naranjo et al., 1981) with a score of 6, the seizure occurring in our case was probably due to olanzapine.

Among second generation antipsychotics no drug is out of risk in inducing seizure. A possible mechanism postulated includes Dopamine D2 receptor antagonism, Histaminergic H1 antagonism, Alfa 1 antagonism, Chronic Alfa 2 receptor and sigma 1 receptor changes, and reduction of GABA neurotransmission as a common final pathway (Torta, and Monaco, 2002).

Olanzapine is known to cause highest EEG changes, in 35-45% of cases (Centorrino et al., 2002), among the nonclozapine newer antipsychotics. An abnormal EEG could be seen in most of the reported cases of seizure including our case. No prospective data regarding mean duration of appearance of changes have been done. But dose and duration both were not found to be correlating with the EEG changes. Abrupt changes in doses are noted to increase the risk (Lee, Crismon, and Dorson, 1999). As seen in our case, patient had long term Olanzapine use and had discontinued and restarted which led to seizures.

No consensus exists regarding ideal way of managing these seizures. Stopping Olanzapine with monitoring for the possible cholinergic rebound effects and starting a typical antipsychotic like Haloperidol (Behere, Anjith, Rao, Venkatasubramanian, and Gangadhar, 2009) could be recommended. Considering persistence of abnormal EEG and rare risk of fatal status epilepticus, cover of anticonvulsants at least until the normalization of EEG could also be recommended. Moreover, 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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