

A Case of Delusion During Epilepsy Treatment Following Lamotrigine Add-On

Epilepsi Tedavisi Sırasında Lamotrijin Ekleme ile Ortaya Çıkan Sanrılar



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Summary

Epilepsy may appear in comorbidity with other neurological disorders. Nevertheless, epileptic seizures and antiepileptic drugs may be responsible for the development of cognitive dysfunctions and mental disorders. Some epileptic seizures may develop with mood disorders, including depressive symptoms. Described in the present report is the case of a 23-year-old male patient with an 8-year history of partial epilepsy and a familial history of bipolarity. He had been treated for partial-onset seizures with carbamazepine and oxcarbazepine, and lamotrigine (LTG) was gradually added to treat depressive symptoms, reaching a twice-daily dosage of 100 mg. The patient developed paranoid delusions involving his neighbors, delusions which subsided after dosage was decreased to 150 mg/day. Seizure control was good, and psychiatric symptoms resolved when the dosage was decreased. Psychotic episodes resulting from rapid increase in LTG serum level has been documented.

Keywords: Acute psychosis; bipolar disorder; epilepsy; delusion; lamotrigine.

Özet

Epilepsi ruhsal bozukluklarla birlikte görülebilir, bununla beraber epileptik ataklar ve antiepileptik ilaçlar epilepsisi olan hastalarda bilişsel ve ruhsal bozuklukların gelişiminde sorumlu tutulabilmektedir. Bazı epileptik ataklar, depresif semptomlar dahil duygudurum bozukluklarına eşlik edebilir. Yirmi üç yaşında, sekiz yıllık pariyel başlangıçlı epilepsi öyküsü olup karbamazepin ve okskarbazepin alırken kontrolden çıkan ve kardeşinde bipolar bozukluk olan 23 yaşındaki erkek hastaya depresif semptomlarının da olması nedeniyle lamotrijin (LTG) eklenmiş ve kademeli olarak günde 2x100 mg dozuna çıkartılmış, 200 mg/g dozunda, hastada komşuları tarafından takip edildiği ve dinlendiği yönünde paranoid sanrılar çıkmış, LTG'nin 150 mg/g dozuna düşürüldüğünde psikiyatrik bulgu ve belirtiler kaybolmuştur. Lamotrijinin azaltılmasından sonraki bir hafta içinde hezeyanların hafiflemesi ve giderek kaybolması nedeniyle psikiyatrik bir tedavi planlanmadı. Literatürde bildirilen benzer olgular gibi olan bu olguda da genellikle LTG dozunun hızlı artırılması sonucunda psikotik ataklara ve psikiyatrik sunumların başlamasına neden olabileceğini ve ayrıca birlikte kullanılan tedavilerin LTG serum seviyelerinde artışa neden olabileceğini düşündürmektedir.

Anahtar sözcükler: Akut psikoz; bipolar bozukluk; hezeyan; epilepsi; lamotrijin.

Intruduction

Epilepsy may appear in comorbidity with other neurological disorders. Due to interaction with antiepileptic treatment, patients with seizures may develop cognitive dysfunction

and mood disorders that include depressive symptoms.

^[1] Lamotrigine (LTG) has been approved to treat a variety of seizure types in adults and children, including partial-onset seizures.^[2] The main psychiatric guidelines highlight



the considerable usefulness of the drug as mood stabilizer for the treatment of patients with bipolar disorder.^[3] However, psychiatric problems have been reported in epileptic patients, or those with mental disorders, primarily bipolar disorder.^[4]

Case Report

The case of a 23-year-old male patient with an 8-year history of partial-onset seizures and a sibling with bipolar disorder is described in the present report. While he experienced depressive episodes, the patient had no history of behavioral problems, psychotic features, or mental retardation. Seizures had been uncontrolled for 3 months, despite treatment of carbamazepine 2x400 mg and oxcarbazepine 2x300 mg initiated 7 years prior. LTG was added to treat accompanying depressive symptoms. Following administration of LTG 25 mg per week, dosage was increased to 100 mg twice daily, and seizures ceased. The patient's mother grew anxious about her son's fears that the neighbors were following him. Mental assessment revealed that the patient also believed the neighbors were listening to him through the walls of his home. He was diagnosed with paranoid delusions caused by LTG treatment. Delusions gradually subsided within 1 week of dosage being decreased to 150 mg per day, and no additional psychiatric treatment was needed. Informed consent was taken from the patient.

Discussion

Psychiatric problems have been reported in epileptic patients or patients with mental disorders, primarily bipolar disorder, treated with LTG. Affective switches of bipolar patients following LTG add-on has been reported, as has the resolution of symptoms following cessation of LTG without the need for additional psychiatric treatment.^[5-8]

Margolese et al.^[8] reported hypomanic symptoms in a patient with epilepsy who was partially responsive to bupropion after LTG add-on. It was concluded that LTG has potentiating antidepressant properties, likely through its ability to decrease glutamate release, and that it is an effective adjunctive treatment in partially responsive unipolar depression.

Matsuo et al.^[9] described an epileptic patient who experienced psychotic episodes after 300 mg LTG was added to treatment and 3 others who experienced delusions after

addition of 500 mg LTG. In each case, symptoms resolved after LTG treatment was ceased, without additional medical psychiatric intervention. Brandt et al.^[10] reported 6 similar cases of psychotic episodes.

Martin et al.^[11] also reported psychotic symptoms following addition of LTG to treatment of epilepsy, and Polselli et al.^[12] reported epileptic attack with loss of consciousness, delusional thinking, auditory hallucinations, and agitation. It was suggested that by reducing glutamate release, LTG triggered acute psychosis. This risk may be higher in patients prone to psychiatric disorders, particularly those with a history of mood disorder such as bipolarity.

It has been reported that LTG may cause affective switches and acute psychotic episodes, including delusions and/or hallucinations. Some affective episodes or symptoms of depression may cause or trigger epileptic seizures, as in the present case. Psychiatric problems may occur, particularly in patients genetically vulnerable to mood disorders, due to rapid dose increases, or with changes to medication that increases LTG serum levels. These may be resolved by reduction or cessation of LTG treatment. In the present case, psychotic features occurred with rapid onset and resolved with decrease in LTG dose, in accordance with reported findings.

It is recommended that LTG be administered as slowly as possible, particularly in patients with family history of psychotic disorders, particularly bipolar disorder, and in cases of polypharmacy.

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