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Lamotrigine induced Blepharospasm in a bipolar depression type-ii: A case report

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Abstract
The association of extrapyramidal side effects (EPS) with the use of conventional antipsychotics is well established, however, EPS can occur during treatment with anticonvulsant medications as well. There are several case reports of the development of involuntary movement disorders including chorea and tic disorder after lamotrigine (LTG) treatment. However, LTG-induced involuntary eyelid movement disorder is extremely rare. Most of this side effect was reported in patients with partial and generalized seizures, and only very few cases were in nonepileptic patients. In contrast, we present the case of 42 years old bipolar patient who developed blepharospasm during LTG therapy and again during re-challenge with the same agent.
The focus of discussion will be on common side effects of this medication and previous reports of lamotrigine associated blepharospasm. We will also discuss a possible underlying mechanism.

Keywords: Lamotrigine, blepharospasm, anticonvulsants, extrapyramidal side effects, bipolar

Introduction
Lamotrigine (LTG) is a broad-spectrum antiepileptic drug used to treat both seizures and bipolar disorders. LTG is thought to exert its mood stabilizing effects through preferential inhibition of voltage sensitive sodium channels, thereby stabilizing neuronal membranes and consequently modulating the presynaptic release of excitatory amino acid transmitters (e.g., glutamate and aspartate) [1]. Common side effects of LTG areskin rash, dizziness, headache, ataxia, nausea, vomiting, and diplopia. The association of movement disorders with the use of anticonvulsant medications is well established, however, veryfew number of lamotrigine induced Blepharospasm have been reported in the literature [2]. Hence, we present a case having no pre-existing movement disorder that developed blepharospasm with lamotrigine.

Case Report
A 42 year old married female patient presented to OPD with chief complaint of difficulty keeping her eyes open, excessive blinking and irritation for the last 7 days. Her score on Jankovic Rating Scale [3] (JRS)was 4 (severe). The patient was a diagnosed case of bipolar depression type–II(BPD II); currently taking lithium, olanzapine, fluoxetine, lamotrigine and benzodiazepine for the last 2 months. On follow up assessment MADRS score showed 34 indicating moderate level of depression. Therefore, the dose of lamotrigine was planned to be gradually increased to 200 mg. But after increase of lamotrigine from 50 mg to 75 mg patient exhibited excessive eye blinking to the verge of closure, rest of the other ocular examination was within normal limits. She could not voluntarily control her eye blinking; and the frequency of eye blinking did not change during conversation. Cranial nerve function and other neurologic examinations were also within normal limits. Her routine blood investigations, EEG and MRI did not reveal any abnormality. No other abnormal move mentse. g, tremors, tics, myoclonic jerks etc. were noted.
Since, blepharospasm appeared to be temporally related to lamotrigine and Naranjo adverse reaction probability scale [4] (Naranjo Scale) showed score of 7. The drug was stopped, her symptoms abated within one week and JRS score was of 1 (minimal).To confirm the causality, rechallenge test was given with 25 mg of lamotrigine and within 2 days patient reported excessive involuntary eye blinking. Naranjo scale score of 9, thus, establishes the definite diagnosed case of lamotrigine induced blepharospasm.

Discussion

Blepharospasm is a form of focal dystonia characterized by motor symptoms (difficulty keeping the eyes open and excessive blinking) and sensory symptoms (irritation of eyes and photophobia). It is classified into three groups, ie, essential, secondary and drug induced [5]. Some psychotropic medications are consistently associated with the development of movement disorders, while lamotrigine is not often included in that group. On rare occassions, it has been associated with the development of EPS. Verma *et al*. reported a patient who developed blepharospasm 4 months after administration of LTG, which disappeared within 1 month after the cessation of LTG [6].

The mechanism for Blepharospasm could be due to altered direct dopaminergic tone or indirect glutamate pathways of either AMPA or Kainatebut the exact cause is unknown. The possible explanation could be that LTG inhibits presynaptic excitatory Glu neurotransmitters, that decreases striatal dopaminergic system [7]. (Figure 1) Dopamine depletion increases the nigral inhibition of superior colliculus which reduces nucleus raphe mediated inhibition of trigeminal reflex [8]. Further neurochemical studies are required to delineate LTG induced pathophysiology of blepharospasm.

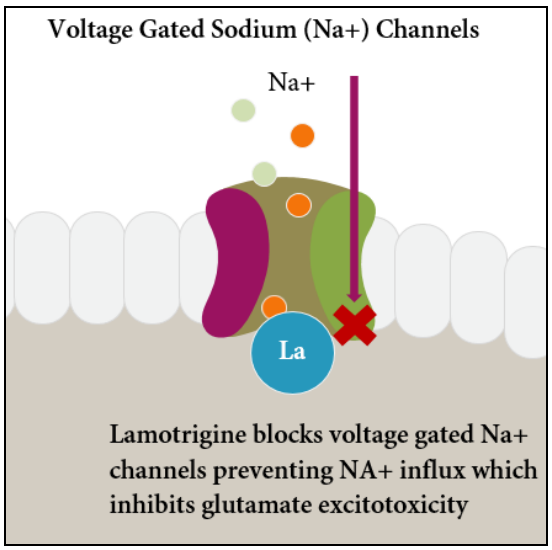

Fig 1: Mechanism of Action-Lamotrigine

Conclusion

It is, therefore, recommended that physicians should be aware of lamotrigineinduced blepharospasm in their clinical practice.

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