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A Case Report on Topiramate Induced Myopia

MOHAMMAD A AKBARIAN and MASSOUD MEHRPOUR

Department of Neurology, Iran University of Medical Sciences, Tehran, Iran.

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ABSTRACT

This is a case report of acute myopia following topiramate administration. There are several reports of this side effect recently and fortunately all of them were reversible. In our case we tried again topiramate with very low dose and increase it very slowly, the patient was followed and she tolerated it well and didn't find ocular problem anymore. So it could be suggested that starting with low dose topiramate may prevent recurrence of myopia.

Keywords: *Topiramate, Acute myopia, Oral sulfamate*

Topiramate (Topamax; Ortho-McNeil Pharmaceutical, Raritan, NJ, USA) is an oral sulfamate medication used primarily for seizure treatment.

Acute myopia, a rare idiosyncratic reaction to sulfonamides, was first described in 1938 [1]. Drug induced myopia has been associated with sulfa drugs such as acetazolamide [2, 3], sulfamethoxazole/trimethoprim [2], indapamide, promethazine, spironolactone, isosorbide dinitrate, and bromocriptine [3]. Other drugs include tetracycline [2, 3], corticosteroids, hydrochlorothiazide, penicillamine, quinine, metronidazole, isotretinoin, and aspirin [3]. Recently, bilateral angle closure glaucoma with uveal effusions, forward rotation of the iris-lens diaphragm, transient myopia and secondary angle closure have been associated with topiramate [4-6].

REPORT OF A CASE

A 21-year-old girl was seen in our hospital with acute blurred vision in both eyes since 3 days before. She was a known case of epilepsy since childhood. Her seizure attacks were not on good control in spite of many anti-epileptic drug prescriptions. Topiramate had been added to her anti-epileptic regime therapy (Phenobarbital and carbamazepin) one week prior to visual blurring. Starting dose of topiramate was 50 mg per day. Her ophthalmologic history was unremarkable, she had never worn glasses, and there was no family history of ocular diseases.

On ocular examination, visual acuity was 20/20 OU with 3.5-diopter (D) myopic correction. Slit lamp examination revealed no conjunctival injection, relatively clear cornea, normal anterior chambers. Pupils were normal bilaterally and reactive to light. The lenses were clear. Intra ocular pressure measured were normal bilaterally. Fundoscopic examination findings were normal with cup-disc ratio of 0.2 on both sides.

The diagnosis of bilateral induced myopia was made. Topiramate was discontinued rapidly and the patient was followed. After two weeks, visual acuity of the patient returned to normal and she had no visual complaint anymore. Because seizure was not still under good control and topiramate was apparently appropriate choice to decrease her attacks, we decided to start topiramate again but with low doses and increasing it very slowly up to desired dose Topiramate is a sulfamate-substituted monosaccharide, used primarily as an antiepileptic medication. Topiramate is thought to possess a state-dependent sodium channel-blocking action. It also potentiates the activity of GABA (γ-aminobutyric acid) and antagonizes the ability of kainate to activate the kainate/AMPA (α-amino-3-hydroxy-5-methylisoxazole-4-propionic acid) subtype of excitatory amino acid receptor. Topiramate also has a weak carbonic anhydrase inhibition [7]. These mechanisms of action help explain the antiepileptic nature of the drug, though the mechanism of choroidal effusions remains unclear. The following are caused by topiramate therapy: abnormal vision, acute secondary angle-closure glaucoma, acute myopia, and suprachoroidal effusions. All findings are reversible if recognized early and if the drug is discontinued [8]. Uveal effusions with ciliary body swelling cause forward rotation of the lens-iris diaphragm, causing myopia and angle closure glaucoma. Spontaneous uveal effusions are most common in individuals with microphthalmic eyes or with abnormal sclera [9]. Topiramate does cross the blood-brain barrier and has also been detected in the vitreous [10].

Starting dose of topiramate was 12.5 mg. She was also followed up regularly with ocular examination.

Fortunately she didn't find any visual problem and she could tolerate topiramate without serious side effects.

COMMENT

In previous reports of acute myopia following use of topiramate [11, 12], the authors speculated that the mechanism was related to partial inhibition of carbonic anhydrase. Although controversy exists regarding the exact mechanism of acute myopia and angle-closure glaucoma after sulfonamide use, most authors have attributed this to ciliary body swelling [13].

The pathophysiology of the ciliary body swelling is unknown. Krieg and Schipper [14] questioned an acute hypersensitivity reaction based on the observation that rechallenging with the same medication failed to produce a second event. Since rechallenging at lower doses does not cause recurrence of myopia, as we also had the same experience, allergic hypersensitivity is unlikely. They speculate that drug-induced elevated prostaglandins contribute to the formation of edema within the ciliary body without evidence of a systemic allergic response.

So we suggest if the patient need topiramate and there is no better alternative, the drug should be started with low doses and be increased slowly with regular ocular examination.

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Address correspondence to: Massoud Mehrpour, MD; Department of Neurology, Iran University of Medical Sciences P.O. Box 14155-6183, Tehran, Iran.
E-mail: mehrpr@yahoo.com