

Topiramate Associated Bilateral Acute Angle Closure Glaucoma And Myopia

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Abstract

Topiramate, an oral sulphamate medication, is primarily licensed for treating epilepsy, though it is increasingly being used for treating migraine¹. Studies have also established its role in treating cocaine addiction². Topiramate is thought to potentiate the activity of GABA (γ-aminobutyric acid) neurotransmitter by blocking the glutamate receptors³. It also has a weak carbonic anhydrase inhibitory activity.

We describe a case of acute myopia and angle closure glaucoma secondary to topiramate, used for the treatment of migraine.

CASE REPORT

A 31-year-old white male was seen in our emergency department with a three days history of severe headaches, painful red eyes, bilateral blurring of vision with photophobia and pain on eye movement. He was a known sufferer of migraine but had no previous ocular problems and had never required glasses. There was no ocular disease of significance within the family. He had no history of drug allergy. Four days prior to the onset of his ocular problems, he was started on a daily dose of 25mg Topiramate for his migraine. On examination he had an unaided right visual acuity of 4/36 and unaided left visual acuity of 6/60, improving to 6/12 with pinhole in both eyes. There was bilateral circumciliary congestion, being more marked in right eye. There was mild corneal oedema with mid-dilated sluggishly reacting pupil of the right eye and normally reacting pupil in left eye. Anterior chambers were shallow peripherally and centrally with central anterior chamber depth of 1.7mm in both eyes. Intraocular pressure was 26 mm in right eye and 19 mm in the left eye. Fundus examination revealed healthy optic discs in both eyes. Although there were very fine choroidal folds in right macula, there was no other sign of choroidal effusion in either eye. Goniocopy revealed 360 degree appositional angle closure in both eyes. Auto-refraction revealed myopia of -4.0 Dsph in right and -3.0 Dsph in left eye. Diagnosis of acute angle closure secondary to topiramate was made.

The patient was treated with 500mg oral acetazolamide, topical pilocarpine 2% QID after initial intensive dose and prednisolone acetate 0.5% QID. Within next few days his unaided vision improved to 6/4 in both eyes, auto refraction showed emmetropia and intraocular pressure was maintained at low teens in both eyes and the angle remained open with normal anterior chamber depth. Two weeks later he was discharged from the clinic without medication

Topiramate is a recent addition to the list of drugs that can precipitate acute angle closure glaucoma^{3,4,5,6}. Although the mechanism for topiramate induced myopia is unknown, it may partly be due to its weak carbonic anhydrase inhibitor activity or a prostaglandin mediated effect⁷. Since rechallenging at lower doses does not cause recurrence of myopia⁸, allergic hypersensitivity is unlikely. Forward rotation of the lens-iris diaphragm resulting from ciliary body swelling due to uveal effusion may cause myopia and angle closure glaucoma and can be confirmed by high frequency anterior segment ultrasound. Increased thickness of the crystalline lens contributes only minimally (9%-16%) to anterior chamber shallowing⁹. Topiramate crosses the blood-brain barrier and has also been detected in the vitreous¹⁰. Though our patient was male, topiramate induced ocular changes has been reported mostly in young women^{3,4,5,6}.

Most of cases of topiramate induced angle closure glaucoma

are reported on American population. To the best of our knowledge this is only the second report from the United Kingdom of acute angle closure glaucoma and myopia secondary to topiramate.

With the increasing use of topiramate there is a possibility of further incidence of these events with patients attending for ophthalmic emergency treatment. Ophthalmologists involved in emergency eye care should be aware of this documented side effect of topiramate when presented with a young patient suffering with bilateral angle-closure glaucoma as stopping the medication is the mainstay of treatment. Two signs, which we noted in our patient, and can help in clinically distinguishing from acute angle closure glaucoma, was of the ciliary congestion, which was remarkably less than what is expected in a patient with very high intraocular pressure from angle closure and also the presence of reactive, though sluggish, pupil, contrary to the fixed dilated pupil as seen in acute angle closure glaucoma. Though we used acetazolamide in our patient, ideally it should be avoided as concurrent use with the topiramate might induce renal calculi formation and further ciliary body oedema.

Neurologist prescribing topiramate should rule out pre existing narrow angles and warn the patients of this possibility. Patients should be advised to stop the medication and seek ophthalmologist's assessment should they experience any ocular symptoms suggestive of angle closure

glaucoma.

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