Case Report

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Topiramate Induced Acute Angle Closure Glaucoma and High Myopia : A Case Report

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ABSTRACT

Purpose: To report a case of Topiramate induced acute angle closure Glaucoma and High Myopia.

Case Presentation: A 24 year old unmarried female complaints of sudden onset of severe dimness of vision 7 days prior to which she was taking tab Topiramate 25 mg twice daily for migraine prophylaxis. Visual acuity reduced to counting finger. Retinoscopy shows -8.00 diopter myopia and vision was improving to 6/6(p) with subjective refraction. Slit Lamp examination reveals severe shallowing of anterior chamber, forward movement of lens iris diaphragm . Intraocular pressure was 25 mm Hgm both eyes. Gonioscopy shows appositional angle closure. B-scan USG shows Choroidal effusion. UBM shows thickening of cilliary body. All those changes, both anatomical changes resolved, and refractive status improved to -1.00 D right eye, -1.5 D left eye; 3 days after discontinuation of the drug.

Conclusion: Neurologist, ENT specialist, Ophthalmologist or Internist whoever is prescribing Topiramate needs to be aware of the potential ocular side effects. Although relatively rare prompt diagnosis is key and urgent discontinuation of the drug is important to prevent permanent ocular damage and visual loss.

Keywords: Topiramates, angle closure glaucoma, myopia.

Introduction

Acute angle closure glaucoma (AACG) is an ocular emergency. Late diagnosis and treatment can cause irreversible optic nerve damage and visual field defect. Angle closure glaucoma may be primary or secondary. Among the causes of secondary angle closure glaucoma drug, induced AACG is one of the important cause though rare. Drug-induced AACG is an ophthalmic emergency which may lead to persistent visual loss if not treated urgently. Presenting symptoms include conjunctival hyperemia, acute onset of impaired vision, ocular, periocular pain, colored halos and headache[17]. On examination findings are elevated intraocular pressure (IOP) above 21 mm Hg, conjunctival and cilliary congestion, a mid-dilated and fixed pupil and a shallow anterior chamber[15,16]. The hallmark of angle closure is the apposition or adhesion of the peripheral iris to the trabecular meshwork, then drainage of the aqueous humour through the angle is reduced and IOP rises. Certain drugs like antidepressants, cold medications, or antihistamines can cause acute angle closure.

Topiramate (Topamax; Ortho- McNeil Pharmaceutical, Raritan, NJ, USA) is an oral sulfamate medication used primarily for seizure treatment, for migraine prophylaxis, depression, and neuropathic pain induced AACG within the first 2 weeks after starting with almost all cases are bilateral AACG[17]. The exact mechanism of action is unknown, however, four properties that may contribute to Topiramate anti-epileptic and

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anti-migraine efficacy include a blockage of voltage-dependent sodium channels, an augmentation of gamma amino- butyrate acid activity at some subtypes of the GABA-A receptors, antagonism of AMPA/ kinate subtype of the glutamate receptor, and inhibition of the carbonic anhydrase enzyme, particularly isozymes II and IV[18]. We report a case of Topiramate induced 2ndary ocular hypertension and sudden onset of severe myopia. In this case prompt discontinuation of the drug causes reversal of myopia and resolution of angle closure within 72 hrs and could prevent from developing angle closure glaucoma. Sulpha drugs may induce AACG without pupillary block. Mechanisms in this type of angle closure include lenticular swelling, retinal edema, Choroidal effusion, and secondary shallowing of the anterior chamber. Basic mechanism is blockage of Na±k pump and water retention and swelling of cilliary body, followed by anterior rotation of the cilliary body , forward movement of lens iris diaphragm, that leads to acute angle closure glaucoma and high myopic shift. Ecstasy, a synthetic amphetamine derivate, and marijuana induced recurrent bilateral AACG. Cocaine has indirect sympatho-mimetic activity and causes mydriasis. AACG has been reported following therapeutic or abuse intranasal application of cocaine[17].

Case Presentation:

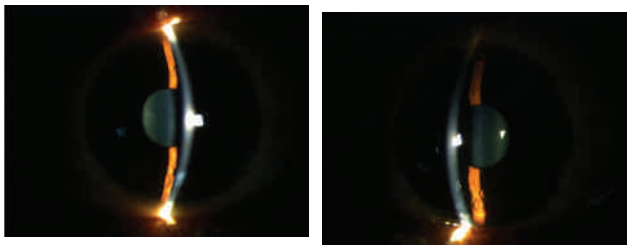
A 34 year old female complained of sudden severe dimness of vision. Her visual acuity on presentation was counting finger close to face. She gave history of taking Topiramate tab 25 mg twice daily as migraine prophylaxis prescribed by ENT specialist for 7 days prior to presentation.

Retinoscopy shows -9.5D RE and -8.5D LE myopia both the eyes. On subjective refraction visual acuity improved to 6/6 part with -8.50D RE, -8.0D LE. On slit lamp examination both eyes shows mild conjunctival and cilliary congestion, very shallow anterior chamber –lens iris diaphragm was coming forward to touch the cornea, mild AC reaction. Intraocular pressure was 25mm Hg in both eyes. Gonioscopy shows Shaffer grade 1 throughout 360◦.

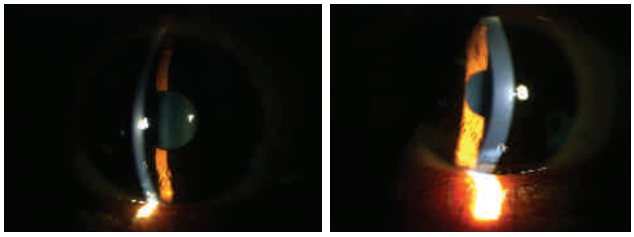
B scan shows mild Choroidal effusion. UBM shows cilliary body swelling. Few cells and flare and thin exudative membrane were there in anterior chamber of LE. Topiramate therapy has been discontinued immediately.

Brinzolamide eye drop 3 times a day, prescribed in both eyes. Neopred eye drop prescribed 6 times a day in LE. On following day morning her visual acuity improved to 6/6 with -5.5 D in RE and with-3.5 D in LE,. Anterior chamber started becoming deeper than before. Lens- iris diaphragm started moving backwards a bit. On the same day evening her visual acuity improved to 6/6 with -2D in RE and -1.75 D in LE.

On 3 days after follow up, her visual acuity becomes 6/6 unaided, Anterior chamber was formed and deep, exudative membrane was resolved in LE, IOP was reduced to 10 mmHg both eyes, Choroidal effusion has been resolved completely in B scan. One weeks later gonioscopy showed Shaffers grade 4 throughout 360^0 of the angle.

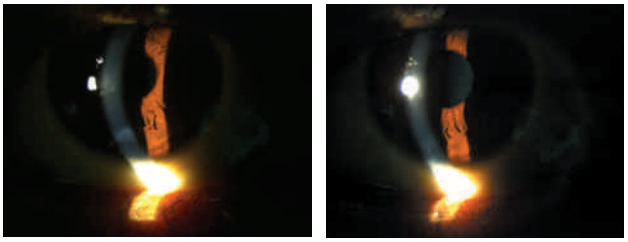


1st day of presentation: very shallow AC



1 day after stopping the drug

AC depth started increasing



1 week after

AC become normal depth completely

Ophthalmic Horizon
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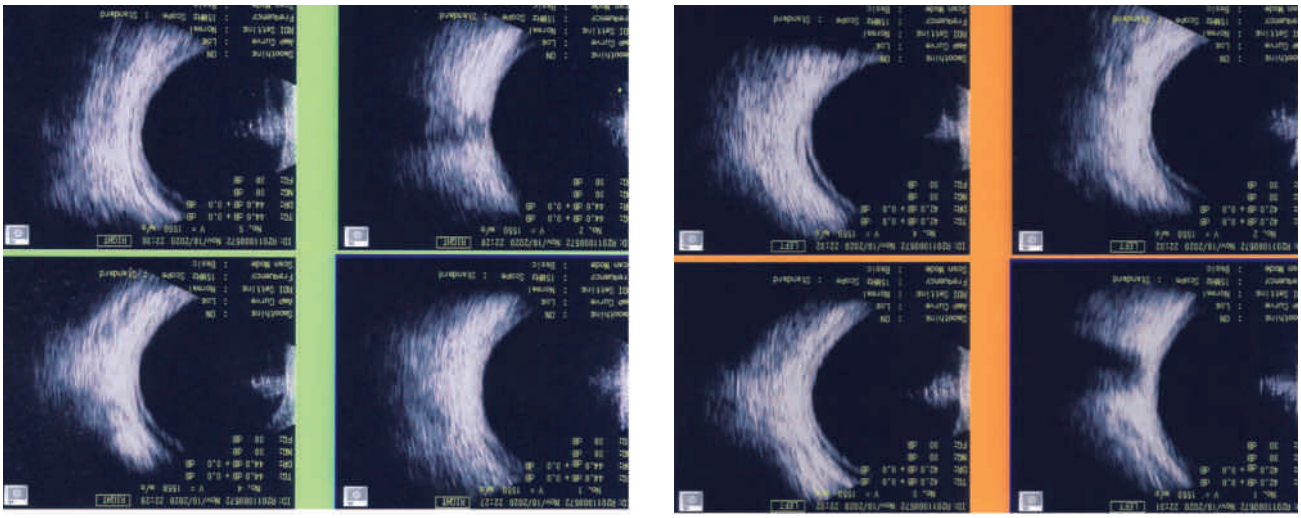

B scan USG showing Choroidal effusion that has resolved within 1 week time

Discussion

Topiramate is used for the treatment of epilepsy, as weight reduction agent in bipolar disorder, to treat depression and neuropathic pain and as prophylaxis for migraine. Case reports on ocular side effects of this drug was there date back to 2001[1,3]. In September 2001, Ortho-McNeil Pharmaceuticals sent out a Page 1 of 3 safety alert to healthcare professionals indicating 23 cases of secondary angle-closure glaucoma related to Topiramate use based on post-marketing experience in more than 825,000 patients. (Hulihan J: Important drug warning [letter]. Available at: http://www.fda.gov/medwatch/SAFETY/2001/topamax_deardoc. PDF). The majority of reported adverse events have occurred in female patients (up to 89%)[4]. Our case is a female patient. Ocular side effects have also been reported in children[5]. In the "certain" category of the World Health Organisation classification system adverse ocular side effects associated with topiramate include abnormal vision, acute IOP elevation, acute myopia (up to 8.75 dioptres), diplopia, nystagmus and shallow anterior chamber with angle-closure. "Probable/likely" include blepharospasm, myokymia, oculogyric crisis, suprachoroidal effusions and "possible" are congenital ocular abnormalities, periorbital oedema and scleritis[6]. High frequency ultrasound biomicroscopy, anterior segment ocular coherence tomography and B-scan ultrasound have helped establish and document the underlying mechanism of the myopia and angle-closure glaucoma[7,9] uveal effusions and ciliary body oedema result in antero-lateral rotation of the ciliary body, anterior displacement of the lens-iris diaphragm which contributes to the myopic shift, anterior chamber shallowing and secondary appositional angle closure. The effusion and oedema also lead to relaxation of the lens zonules resulting in thickening of the lens further narrowing the angle. Though the exact mechanism is unclear the fluid movement leading to effusions is thought to be related to drug induced changes in membrane potential[8]. In reported cases of angle-closure glaucoma topiramate doses varied from 50 mg or less to 100 mg or more, 5 reported cases were precipitated within hours after doubling the dose, 85% of cases occurred in the first 2 weeks of treatment with the drug[10]. Fraunfelder et al[10] advise the following management strategy for topiramate-associated angle-closure glaucoma: Stoppage of the drug in the first instance, the prescribing doctor should be consulted. Medical therapy such as oral

medications and aqueous suppressants should be given. Laser iridotomy or peripheral iridectomy are not helpful as topiramate angle closure is not pupil block related.

Topical miotics may be contraindicated as they could precipitate a relative pupil block.

Topical cycloplegic agents may be given as they possibly lower IOP by retracting ciliary processes. Care should be taken with acetazolamide as it is also a sulfa-based drug and has been reported to cause angle closure glaucoma in a similar manner to topiramate[11].

In this case the Topiramate induced anatomical changes has been reversed after stopping the drug immediately, before inducing angle closure glaucoma and any permanent ocular damage could prevented to happen. But the rapid onset of severe visual disturbance was much distressing to the patient. And as visual acuity was improved to 6/6 (p) with refraction, it was reasonable to counsel the patient about the better prognosis. As the half life of the drug is about 21-24 hrs[12], rapid visual recovery usually occurs although in some cases it can take several weeks[10]. If unrecognised as a drug-related event serious outcomes could occur (7 cases of permanent visual loss following angle-closure glaucoma have been reported)[13]. Ocular examination before starting Topiramate cannot identify eyes at risk[14].

Patients commencing Topiramate should therefore be advised to immediately report any symptoms of eye pain or blurred vision especially in the first few weeks of treatment.

Conclusion

Drug-induced AACG may be preventable if patients at risk are recognized earlier and properly treated. The causative drug should stop immediately. Delayed recognition and treatment may lead to permanent visual loss due to corneal decompression, optic nerve ischemia and retinal vein thrombosis due to high IOP. Both patients and treating physicians should be aware of the potential of the above drugs to cause AACG (AAO)[17].

Ophthalmologists, Neurologist, ENT specialist, Internist whoever is prescribing Topiramate need to be aware of the potential ocular side effects of it and counsel the patient about those side effects and to consult an ophthalmologist immediately if any symptoms of vision loss develops specially within initial few days. Although relatively rare prompt recognition is key. So appropriate management can be instituted and visual outcome can be regained and serious complications like permanent visual loss can be prevented.

Consent

The author obtained written informed consent from the patient for the publication of this case report.

Competing interests

The author declares that there are no competing interests.

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