



CASE REPORT

Topiramate Induced Acute Angle Closure and Myopia with Ciliochoroidal Effusion

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Abstract

Topiramate, a sulfamate substituted monosaccharide, is a widely used antiepileptic drug which has now gained widespread use for migraine prophylaxis and various other conditions. This drug has been associated with ocular side effects, of which secondary acute angle closure is a rare idiosyncratic reaction which can result in blindness if not identified and treated promptly. This case report demonstrates a middle-aged woman who presented with bilateral acute angle closure and myopia with suprachoroidal effusion. This serious ocular side effect was reversed by timely intervention. Hence it is important that the treating physician is aware of this rare condition and warn the patients while prescribing the drug.

Keywords

Topiramate, Acute Angle Closure, Myopia, Suprachoroidal Effusion

Introduction

Topiramate is a sulfamate-substituted monosaccharide which is widely used as an anti-epileptic and as prophylaxis for migraine. Various ocular side-effects like bilateral angle closure, acute myopia have been described with the use of topiramate.^[1,2]

The basic underlying mechanism seems to be the ciliary effusion causing ante-version of the ciliary body and anterior displacement of the iris-lens diaphragm which induces bilateral acute myopia, non-pupillary block angle closure and raised intraocular pressure.^[3]

We report a case wherein a middle-aged woman who was on tablet topiramate 50 mg for one week, presented

with bilateral acute angle closure and myopia with suprachoroidal effusion.

Case Report

A 40-year-old woman presented to emergency with complaints of headache, blurring of vision, nausea and eyelid swelling in both eyes. Conjunctival congestion developed after 5 hours of presentation. She has history of headache for which she was suspected to have migraine and was Started on tablet topiramate 50 mg, one week before she developed these symptoms. The patient had no complaints of ocular irritation, colored haloes, flashes, floaters, skin rash and vomiting. She has

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Fig 1. DAY 1: Slit lamp image showing shallow AC and B-Scan showing Suprachoroidal effusion

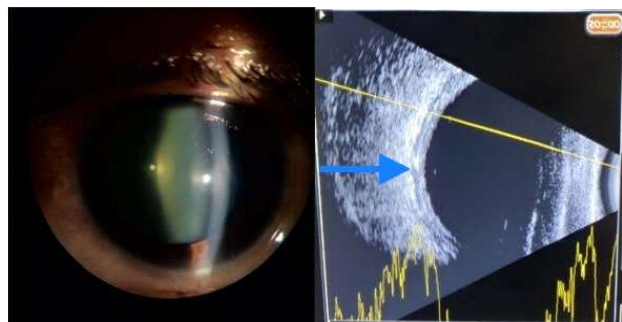


Fig. 3 1 WEEK FOLLOW UP

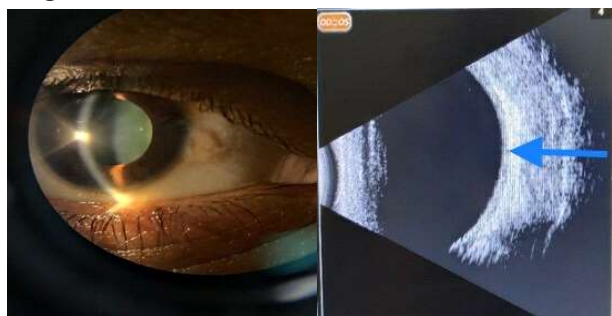


Fig.2 DAY 3: showing improvement in AC depth and resolving suprachoroidal effusion



Fig.4 3 WEEKS FOLLOW UP : AC normal in depth and B scan showing resolved suprachoroidal effusion.

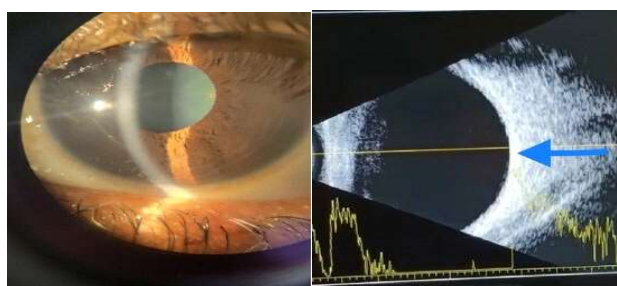


Table 1: Improvement in visual acuity (V/A), intra ocular pressure (IOP), central corneal thickness (CCT), anterior chamber depth after initiating the treatment.

Clinical course	DAY 1		DAY 2		DAY 3		DAY 4	
	OD	OS	OD	OS	OD	OS	OD	OS
Eye V/A	2/60	3/60	6/18(p)	6/18(p)	6/18	6/12	6/9(p)	6/6(p)
IOP (mmHg)	50	54	26	20	15.7	15.7	12	14.5
CCT (µm)	582	562	541	535	540	535	540	535
AC	Shallow	Shallow	Shallow	Shallow	Normal in depth. Gonio-grade 4	Normal in depth. Gonio-grade 4	Normal in depth. Gonio-grade 4	Normal in depth. Gonio-grade 4

history of using power glass for near vision only. She is a known diabetic and hypertensive and is under medication for the past one year.

On examination, bedside vision was - OD:2/60 and OS:3/60 at the initial presentation. Anterior segment of both eyes revealed lid edema, conjunctival chemosis, moderate corneal stromal edema, shallow anterior chamber,

anteriorly displaced iris, 3mm round pupils reacting to light, clear lens and free and full extra ocular movements. Fundus examination revealed hazy Media, Disc and vessels appeared to be normal, other details could not be appreciated.

B scan was performed which revealed 360 degree increased choroidal thickness with Supra choroidal

Table 2: Improvement in the induced myopia after initiating the treatment.

Manifest Refraction	DAY 2	DAY 3	DAY 4
OD	-3.00/-0.50 * 40 degree (6/6)	-2.00/-1.75 * 60 degree (6/6)	-0.50/-0.50 * 60 degree (6/6)
OS	-2.50/-1.50 * 65 degree (6/6)	-1.75/-0.75 * 70 degree (6/6)	+/- / -0.50 * 70 degree (6/6)

Table 3. Visual acuity (V/A), refraction, intraocular pressure (IOP), anterior chamber (AC) and gonioscopy at one week and three weeks follow-up.

	One week follow up	Three week follow up
V/A	OD: 6/6 OS: 6/6(blur)	OD: 6/6 OS: 6/6(blur)
Refraction	OD: +/- OS: +/- /-0.50 * 80 degree (6/6)	OD: +/- OS: +/- /-0.50 * 80 degree (6/6)
IOP	OD: 11.00 mmHg OS: 12.00 mmHg	OD: 15.00 mmHg OS: 15.00 mmHg
AC	Normal in depth	Normal in depth
Gonioscopy Treatment	Grade 3 angles OU Predforte 4 times per day for 1 week and then BD for 1 week Timolol BD	Grade 4 (wide open angles) OU Predforte OD for 1 week and then stop Timolol BD for 1 week

effusion (Image 1). Central corneal thickness was 582 µm in right eye and 562 µm in left eye. IOP by applanation tonometry was 50 mmHg in right eye and 54 mmHg in left eye. Gonioscopy revealed angle closure in three quadrants of both eyes except the inferior quadrant which showed very narrow angles (grade 1) as per ACA grading system.

A diagnosis of topiramate induced acute bilateral angle closure and myopia with ciliochoroidal effusion was made and patient was advised to discontinue topiramate and was started on Inj. Mannitol 100 ml slow i.v over 1 hour Stat, OU Timolol eyedrops BD, OU Cyclopentolate eyedrops TID and OU Predforte eyedrops 6 times/day tapered weekly.

Improvement in visual acuity, intra ocular pressure, central corneal thickness, anterior chamber depth was noted after initiating the treatment (table 1). The drug induced myopia

also improved with treatment (table 2). Patient was asked to review weekly for follow up and findings documented (table 3). Fig 2,3 and 4 shows the slit lamp and B-Scan images taken on day 3 of treatment, one week and 3 weeks follow up respectively. The slit lamp image shows improvement in AC depth and B-Scan shows improvement in suprachoroidal effusion. Visual acuity was stable and IOP maintained in the normal range subsequent to a stepwise withdrawal of all topical medications.

Discussion

Angle closure and acute myopia occurs due to ciliary effusion, an idiosyncratic reaction to topiramate.^[4] It was first reported in July 2001 by Banta *et al.* The widespread use of this drug will probably show an increasing frequency of such cases, and it is already showing in the literature as case reports.^[5,6,7] The exact mechanism of the ciliochoroidal effusion is unknown. But it is believed to



be due to its weak carbonic anhydrase activity and prostaglandin mediated effect.^{13,81} There occurs swelling and edema of ciliary body leading to relaxation of zonules and allowing the lens to thicken. Suprachoroidal effusion and anterolateral rotation of the ciliary body leads to anterior displacement of iris lens diaphragm resulting in myopic shift, shallowing of AC and secondary glaucoma. Ultrasound showing the presence of peripheral choroidal effusion can be used to confirm the diagnosis. First step in treatment is to stop topiramate. Topical and systemic antiglaucoma medications are prescribed to lower the IOP. Cycloplegics can help deepening the anterior chamber. Some authors suggest using systemic steroids considering that the ciliary body edema could be caused due to inflammation.¹⁹¹ There is a case report with lens-corneal touch which did not improve with topical and systemic treatment and they had to do a choroidal drainage for the resolution of the AACG. In this case the patient was treated initially with pilocarpine that worsened the clinical picture. Miotics are not helpful in this entity.¹¹⁰¹ Iridotomies may not be useful because there is no pupillary block. Ecography helps to rule out other conditions that can cause bilateral AACG like intraocular tumors or bilateral iris cyst in the periphery.

Conclusion

Proper drug history has to be elicited to solve the diagnostic dilemmas and avoid unnecessary complications, as this condition has to be differentiated from other causes of acute angle closure glaucoma. If not recognized as a drug related event, one can lose the vision permanently. It is important to alert physicians and patients using Topiramate for early recognition of signs and symptoms of this entity and prevent permanent damage.

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