Idiosyncratic Topiramate – Induced High Myopic Shift with Angle Closure Glaucoma

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Purpose: A case of idiosyncratic topiramate induced acute high myopic shift with bilateral choroidal effusion and angle closure glaucoma.

Material and Methods: A 24 year old female presented with severe bilateral visual loss after taking a single dose of Topiramate. Her visual acuity was counting finger and intraocular pressure was 32 mm Hg in both eyes, with conjunctival congestion, very shallow anterior chambers, forward displacement of iris – lens diaphragm and normal pupils. There was acute high myopic shift of up to – 12.00, which resolved completely and patient became 6/6 bilaterally 9 days after cessation of Topiramate.

Conclusion: Topiramate may induce acute myopia and angle closure glaucoma. Cession of treatment lead to complete resolution.

Topiramate (TPM) is an anti-epileptic drug which has recently gained popularity after being approved by Food and Drug Administration (FDA) for the use of prevention of migraine. Although the prophylactic role of TPM cannot be denied, the main predicament arises with its off-label use in treating migraine, leading to a number of side effects. The ocular side effects include angle closure glaucoma, myopic shift and suprachoroidal effusion. We report a case of TPM induced acute high myopia with bilateral choroidal effusion and angle closure glaucoma. The uniqueness of our case is that it has the highest myopic shift to be reported to date, with complete resolution in a short span of 9 days.

CASE REPORT
A 24 year old female presented to the emergency department with severe headache and sudden painful decreased vision in both eyes (OU) for few hours. Headache was continuous and was associated with nausea and vomiting while visual acuity (VA) was counting finger (CF) OU. Patient’s past history was significant for migraine. Previously there was no history of allergy or adverse drug reaction and she was emmetrope with no ocular disease.

On slit lamp examination (Table I), bilaterally there was conjunctival congestion, very shallow anterior chambers, forward displacement of iris–lens diaphragm, normal pupils and fundi with intraocular pressure (IOP) of 32 mm Hg (OU). She was given Mannitol 20% 500 ml intravenously over forty minutes and 500 mg acetazolamide orally stat but there was no effect on the IOP. To further manage the IOP, bilateral YAG (Yttrium aluminium garnet) laser peripheral iridotomies were performed and the IOP reduced to 22 mm Hg and 18 mm Hg, in right (OD) and left eye (OS) respectively. Patient was prescribed oral acetazolamide 250 mg, topical Pilocarpine 2%, Glantrim (Dorzolamide and Timolol Maleate, Atco Laboratories Limited, Pakistan), Maxidex (Dexamethasone, Alcon Scientific Service, Pakistan) and followed closely as a case of atypical narrow angle glaucoma.

On first two days refraction was not possible as there was error on autorefractometer (MR-3100, HUITZ, Korea). On fourth day (Table I) patient had a high myopic refractive error of -10.00 / -5.25 × 103° OD and -12.25 / -5.75 × 61° OS with VA of CF OU. She was followed carefully to evaluate her acute myopia and atypical narrow angle glaucoma. On further enquiry she revealed that she took a single dose of Topiramate 25 mg (Zopir, Gltiz Pharma, Pakistan) for her migraine, few hours before her presentation. She did not take any further dose after that. On the very
next day her myopia reduced to -6.50/-0.25 × 180° OD and -7.50/-0.50 × 112° OS with VA of 3/60 OD and 2/60 OS. The IOP was 14 mm Hg OD and 12 mm Hg OS thereafter all anti-glaucoma medications were stopped and she was kept under observation.

On the ninth day (Table I) patient recovered to emmetropia with mild astigmatism of +0.00/-0.50 × 179° OD and +0.00/+0.75 × 99° OS, and VA of 6/6 OU. Ocular examination was normal with slight iris displacement in the right eye (Figure 1A and 1B) while ultrasound biomicroscopy revealed forward displacement of iris in both eyes indicating that there was significant displacement in early stages of the event (Figure 1C and 1D). B-scan ultrasonography showed minor superior choroidal effusion remaining in the right eye while left eye was normal (Figure 2A and 2B).

On one month follow up patient had normal visual status. She was counseled on trigger control of migraine and was doing well without any medications.

**DISCUSSION**

Transient myopia can occur due to diabetes mellitus, ectopia lentis, contact lens wear and drug use. Our case did not have any of the aforementioned

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<th>Table 1: Progress of Patient on various days</th>
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<td><strong>VA</strong>: OD-OS <strong>Refractive Error</strong>: OD-OS <strong>IOP</strong>: OD-OS <strong>Anterior Chamber</strong>: OD-OS <strong>Iris Lens Diaphragm</strong>:</td>
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VA, Visual Acuity; IOP, Intraocular pressure mm Hg; OD, Right Eye, OS, Left Eye; CF Counting Finger; Fwd, Displaced Forward N, Normal
conditions except drug use. A number of drugs have been reported to cause transient acute myopia but most common are the sulfa group drugs including Topiramate (TPM)\textsuperscript{3}.

In literature, most of the cases of Topiramate induced myopic shift (TIMS) presented 8.59 ± 7.24 days after taking TPM, (4) while our case presented on first day after taking single dose of TPM. The exact mechanism of TIMS is not known but it has been hypothesized that the choroidal effusion leads to anterior rotation of ciliary body and forward displacement of the iris-lens diaphragm. The severity of symptoms is independent of the dose of TPM\textsuperscript{5} but over 70% patients were taking 50 mg or more.\textsuperscript{4} Our patient took just 25 mg of TPM, favoring the dose independence and idiosyncratic mechanism.

Topiramate induced myopic shift of up to 5.66 ± 1.57 has been reported in literature\textsuperscript{4}. The highest myopic shift reported so far is -10.00 in a 34 year old male with topiramate induced angle closure glaucoma\textsuperscript{6}. To our knowledge we are the first to report the TPM induced myopic shift of -12.00. The management typically involves discontinuing TPM nevertheless until a definite diagnosis is reached the IOP may be controlled with conventional therapy. Our patient had reversal to emmetropia and normal angle status within 9 days of stopping Topiramate.

CONCLUSION
This case highlights the significance of idiosyncratic ocular side effects of Topiramate. Cession of treatment leads to complete resolution of ocular side effects with retrieval of normal visual status.

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REFERENCES