Topiramate Induced Bilateral Angle-closure Glaucoma

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Abstract

Purpose: To describe a case of acute angle-closure glaucoma associated with oral topiramate (Topamax, Aria Daroo) therapy

Case report: Two weeks after initiation of oral topiramate therapy for epilepsy, a 35-year-old woman presented with blurred vision and headache. Intraocular pressure in both eyes was significantly elevated and her visual acuity was 20/30 Ocular Uterque (OU). Bilateral conjunctival chemosis, shallow anterior chamber and mild corneal edema were observed. Topiramate therapy was discontinued. Topical therapy was initiated in both eyes with betamethasone, atropine and timolol

Results: Symptoms and signs including vision accuracy, refraction and intraocular pressure resolved over the next 2 weeks.

Conclusion: Topiramate therapy may be associated with ciliochoroidal effusion resulting in angle-closure glaucoma; therefore, patients on such therapy should be carefully monitored.

Keywords: Topiramate, Angle-closure Glaucoma, Ciliochoroidal Effusion

Introduction

Topiramate (Topamax, Aria Daroo) is an oral sulfamate medication used mainly for seizure treatment and is also used in the management of migraine, depression and neuropathic pain.¹ Off-label, it has gained popularity as a weight reducing agent and a treatment for bipolar disorder.²

Topiramate may cause idiosyncratic ciliochoroidal detachments and ciliary body edema leading to anterior displacement of the lens-iris diaphragm, lens thickening, and acute angle-closure glaucoma.³

Herein, we present a case of acute angle-closure glaucoma, developed two weeks after starting oral topiramate therapy.

Case report

A 35-year-old woman was examined at the ophthalmology clinic of Tabriz University of medical sciences for a three day history of bilateral frontal headache and blurred vision. Her medical history was notable for epilepsy. Her ocular medical history was unremarkable. She had never worn glasses, and she did not recall any cases of ocular diseases within her family. Her medications included oral topiramate at 100 mg twice daily, which she had started 2 weeks before she came. At the time of presentation, her visual acuity was 20/30 Ocular Uterque (OU). Manifest refraction revealed -0.75 dioptres myopia in both eyes. In both eyes, slit-lamp examination revealed conjunctival chemosis and injection, mild corneal edema, and markedly shallow anterior chamber (Figure 1). Intraocular pressure (IOP) was measured as 29 mm Hg OD and 31 mm Hg OS. Gonioscopy revealed closed angles (Figure 2).

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Figure 1. Slit-lamp photograph of the right eye at the time of presentation, revealing shallowing of the anterior chamber

Figure 2. Appositional closure of the anterior chamber angle

Figure 3. Ultrasound biomicroscopy of the right eye obtained at initial examination showing ciliary body swelling (CB), ciliochoroidal effusion (*), and a closed angle (arrow)

Pupils were reactive with sluggish response. Hazy view of the optic nerve revealed normal cup: disc ratios of 0.3 in both eyes. Ultrasound biomicroscopy demonstrated a closed angle with a forward shift of the lens, and swollen ciliary processes (Figure 3). A-scan and B-scan ultrasonography revealed anterior chamber depth of 1.1, 1 mm in right and left eye respectively and 360° ciliochoroidal effusion in both eyes (Figure 4-A).

The diagnosis of bilateral angle-closure glaucoma without pupillary block was made. The patient was treated with 0.1% betamethasone, 1% atropine, 0.5% timolol and oral acetazolamide. After consultation with the patient's neurologist, topiramate was stopped and oral acetazolamide was tapered.

Figure 4–A. B-scan ultrasound of the right eye at the time of presentation, showing ciliochoroidal effusion

Figure 4–B. Resolution of ciliochoroidal effusion two weeks after cessation of topiramate

One week after presentation, her vision returned to 20/20 OU. Manifest refraction revealed emmetropia. Slit-lamp examination showed clear corneas and deep anterior

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chambers. Her IOPs were 13 mm Hg OD and 14 mm Hg OS. Gonioscopy revealed open angles without synechia in both eyes. Two weeks later the patient's eyes were back to normal state and IOP was 15 mm Hg OU without medication. B-scan ultrasound demonstrated resolution of ciliochoroidal effusion in both eyes (Figure 4-B).

Discussion

Topiramate is a sulfamate-substituted monosaccharide. Several mechanisms elicit its antiseizure effect; one of them is weak inhibition of carbonic anhydrase enzyme.

The following side effects have been reported with topiramate therapy: abnormal vision, acute secondary angle-closure glaucoma, acute myopia and suprachoroidal effusions.

Although controversies exists regarding the exact mechanism of acute myopia and angle-closure glaucoma after sulfonamide usage, most authors have attributed this to ciliary body swelling.

The pathophysiology of the ciliary body swelling is unknown. Krieg and Schipper noted that repeated use of the same drug produces a hypersensitivity reaction. They speculate that the drug-induced elevated prostaglandins contribute to formation of edema within the ciliary body without any evidence of a systemic allergic response.

In addition, topiramate's weak inhibition of carbonic anhydrase or an effect mediated by prostaglandin has also been suggested as causative mechanism by some authors.

Banta et al. first reported a case of uveal effusion and secondary angle-closure glaucoma associated with topiramate usage in July 2001. In September 2001, Ortho-McNeil Pharmaceuticals sent out a “Dear Healthcare Professional” letter, indicating that 21 cases of acute angle-closure glaucoma had been reported to their safety division, and physicians should be aware of this adverse drug reactions.

Rhee et al. described a 43-year-old woman with topiramate associated glaucoma that included high-frequency ultrasound evidence of ciliary process swelling and forward displacement of lens iris diaphragm.

Levy et al. reported a 35-year-old woman with topiramate-induced bilateral angle-closure glaucoma.

Craig et al. reported two women, 25 and 45 years old of age, who developed acute myopia after starting topiramate. One of them also developed bilateral angle-closure glaucoma.

The majority of reported adverse events have occurred in female patients (up to 89%) which is also the case for the patient presented in this paper.

Management of topiramate-related acute pressure elevation requires cessation of the drug after consultation with the prescribing physician because decreasing the dose by 50 mg per dose might exacerbate the preexisting systemic condition.

Topical cycloplegic agents probably lower the intraocular pressure by retracting the ciliary processes, along with topical β-blockers and oral pressure-lowering agents.

Because the mechanism of angle-closure does not involve pupillary block, pilocarpine and peripheral iridectomy are usually ineffective.

Conclusion

Topiramate can cause acute bilateral angle-closure glaucoma with ciliary body edema and ciliochoroidal detachment. Physicians should be aware of the possible ocular side effects after initiating topiramate use, principally during the first 2 weeks. It is also important to advise the patients about the possibility of blurred vision, eye pain, or headache and the need to seek immediate ophthalmic investigation in these events.

References