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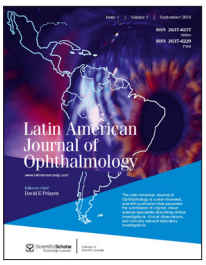

Glaucoma *Case Report*

Topiramate-induced acute angle-closure glaucoma: A case report and review of literature

Rishi Sharma[1], Jaya Kaushik[2]

[1]Department of Ophthalmology, Military Hospital, Secunderabad, Telangana, [2]Department of Ophthalmology, Command Hospital, Lucknow, Uttar Pradesh, India.

***Corresponding author:**
Rishi Sharma,
Department of Ophthalmology,
Military Hospital,
Secunderabad, Telangana,
India.

rishi4840@yahoo.co.in

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ABSTRACT

Drug-induced angle-closure glaucoma is an emergency associated with potentially vision threatening side effects. There are various classes of drugs which induce acute angle-closure glaucoma. Topiramate is an oral sulfa-based drug used for seizure, migraine, and neuropathic pain. There are published case reports of topiramate-induced angle-closure which can mimic acute angle-closure glaucoma. We present a case of a 32-year-old female who developed bilateral angle-closure glaucoma secondary to topiramate.

Keywords: Acute angle-closure glaucoma, Drug induced, Pupillary block, Topiramate, iris-lens diaphragm

INTRODUCTION

Drug-induced acute angle-closure glaucoma (AACG) has serious sight-threatening adverse effects. Therefore, it needs prompt recognition and immediate treatment. There are variety of drugs that can precipitate AACG which includes adrenergic agonists, anticholinergics, cholinergics, sulfonamides, and serotonergic medications.[1-3] Patients typically present with elevated intraocular pressure (IOP), headache, nausea, blurry vision, and halos around lights.[4] Our aim is to review the current literature regarding topiramate-induced AACG and provide ophthalmologists and physicians with the underlying mechanism and treatment protocol in such cases.

CASE REPORT

A 32-year-old female patient presented with the complaints of sudden onset painful diminution of vision in both eyes of 2 days duration with associated headache, nausea, and halos around lights. There was no history of trauma, previous history of uveitis, and spectacle usage. Her medical history revealed ongoing drug therapy for hypothyroidism and migraine. Her medications included sumatriptan, levothyroxine, and topiramate (for the past 1 month). Systemic examination was essentially within normal limits. Ocular examination revealed normal head posture with ocular movements full and free. Distant visual acuity was 6/60 unaided in both eyes correctable to 6/6 with −3.5 D Sph right eye and −3.0 D Sph left eye. Pupils were both 6 mm sized and sluggishly reacting pupils were present. IOP on arrival was 42 mmHg (RE) and 38 mmHg (LE). Slit-lamp evaluation revealed mild conjunctival chemosis with diffuse stromal

edema. Anterior chamber (AC) was very shallow in depth. Gonioscopy revealed closed angles in both eyes. Fundoscopy revealed normal optic discs with healthy neuroretinal rim and 0.3–0.4 cup to disc ratios in both eyes. No suprachoroidal effusion was detected on peripheral retinal examination and standard B-scan ultrasonography. She was diagnosed as a case of bilateral secondary AACG due to topiramate. Topiramate was stopped immediately. She was started on iv mannitol. Repeat IOP was 34 mmHg in the right eye and 32 mmHg in the left eye. She was started on topical aqueous suppressants (dorzolamide), oral hyperosmotic agents along with topical steroids, and cycloplegic agents. The patient was called for a follow-up the next day and IOP had come down to 28 mmHg (RE) and 24 mmHg (LE). After 1 week, the vision of the patient was 6/6 in both eyes unaided. The IOP was 20 mmHg (RE) and 18 mmHg (LE). The AC was normal in depth. She was continued on topical anti-glaucoma medications for 1 more week and then stopped. After 1 month, the recorded IOP was 17 mmHg in the right eye and 16 mmHg in the left eye [see table 1].

Table 1: IOP recordings.

DAY	IOP (mmHg)	
	RE	LE
On presentation	42 mmHg	38 mmHg
Post-mannitol	34 mmHg	32 mmHg
1st follow-up day	28 mmHg	24 mmHg
1 week follow-up	20 mmHg	18 mmHg
1 month follow-up	17 mmHg	16 mmHg

IOP: Intraocular pressure

DISCUSSION

There are various predisposing factors involved in the development of AACG. Anatomical risk factors for AACG include a shallow AC, short axial length, plateau iris configuration, and a thick and anteriorly positioned crystalline lens. The demographic risk factors include female sex, Asian ethnicity, family history, and advancing age.[5] Many pharmacological agents are associated with the development of AACG such as adrenergic agonists, anticholinergics, and sulfonamides. There are two key mechanisms involved in the pathophysiology of drug-induced AACG with different treatment strategies. The first is development of pupillary block and iridocorneal angle closure due to thickening of iris base with mydriasis. The second mechanism involves the anterior displacement of the lens-iris diaphragm secondary to mass effect such as misdirected aqueous, uveal effusion, or weakened zonules.[6,7] Sulfa-based drugs (acetazolamide, hydrochlorothiazide, cotrimoxazole, and topiramate) can cause AACG by ciliary body edema and anterior rotation of the iris-lens diaphragm. Topiramate is a sulfa derivative monosaccharide and is used in seizures and migraines. It has several mechanisms of action, such as blockage of voltage-gated sodium channels, leading to changes in the sodium and chloride movement. The post-synaptic gamma-aminobutyric acid receptor activity is enhanced and there is a mild inhibition of some carbonic anhydrase isoenzymes. This, in turn, results in alterations and fluctuations of ionic concentration in various tissues, including the ocular.[8] There are variety of ocular adverse effects attributable to topiramate which include AACG, ocular pain, headache, visual field defects, acute-onset myopia, and suprachoroidal effusions.[9] It is classically associated with AACG.https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7221246/- B32[10] Usually, the onset is around 2 weeks; however, cases have been reported to occur as early as 24 h–262 days. The underlying mechanism of topiramate-induced AACG is ciliary detachment and edema which causes relaxation of the zonules and thickening of the lens. Anterior displacement of the iris-lens diaphragm leads to AC shallowing and risk of angle closure.[11] One should be aware of the fact that it is the forward displacement of the iris-lens diaphragm and not the pupillary block which leads to angle closure.[12] Treatment involves discontinuation of topiramate, cycloplegia, topical corticosteroids, and mannitol.https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7221246/- B41[13] When the ciliary body gets relaxed, the zonular tension decreases and the iris-lens diaphragm is pulled, which leads to deepening of the AC and the reopening of the angle for aqueous drainage.[14] Since there is no pupillary block involvement, therefore, peripheral iridectomy and cholinergic agents (e.g., pilocarpine) have no role in management of such cases.

CONCLUSION

AACG glaucoma due to medications can have devastating effects on the eye; however, it is preventable. Physicians prescribing such medications should be aware of the potentially blinding adverse effects of such medications. The patient should be fully informed of such adverse effects and the warning symptoms. A meticulous history of drug medications used by the patient is the key in evaluation of AACG and its early management to prevent irreversible damage.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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