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Case Report

UNUSUAL PRESENTATION OF BILATERAL ACUTE ANGLE CLOSURE GLAUCOMA A CASE REPORT

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ABSTRACT

A 55-year-old male presented in emergency with sudden onset of bilateral blurred vision and headache. His visual acuity was 20/400 OD and 20/300 OS. Slit-lamp examination revealed bilateral corneal edema, shallow anterior chamber, forward displacement of lens-iris diaphragm, fixed dilated pupils. Intraocular pressures were 68 mm Hg OU. Keeping in mind bilateral presentation of acute angle closure glaucoma, we asked detailed history of patient which revealed treatment of deaddiction in the form of Topiramate drug which he had started 9 days back. So, diagnosis of topiramate induced bilateral angle closure glaucoma was made with underlying mechanism of uveal effusion syndrome. He was advised to discontinue topiramate and was administered maximum antiglaucoma medication, and cycloplegic drops. On second day, IOP returned to 29mmHg OD and 31mmHg OS. Corneal edema had decreased and AC had improved. The present case highlights unusual presentation of acute attack of ACG associated with Topiramate.

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INTRODUCTION

Angle closure glaucoma is a major cause of blindness worldwide. In angle closure glaucoma increased IOP is caused by impaired outflow facility secondary to appositional or synechial closure of anterior chamber angle. Various mechanisms can be responsible for this angle closure which either push iris from behind (Pupillary block, Plateau iris configuration, lens induced glaucoma, Malignant glaucoma, choroidal effusions) or pull iris forward (contraction of fibrovascular tissue as in NVG, ICE syndrome). Mostly these mechanisms lead to unilateral attack of ACG, Bilateral simultaneous acute angle-closure glaucoma is rare and has been reported due to drugs, general anesthesia, snake bite, microspherophakia and Vogt-Koyanagi-Harada syndrome.^[1] Various drugs which precipitate attack of bilateral angle closure glaucoma are Antipsychotics, Anticonvulsants (topiramate) Antidepressants, Antihistamines, Antispasmodics, Sympathomimetic agents.

Topiramate, a sulfamate-substituted monosaccharide, is generally prescribed as an antiepileptic and antidepressant medication. Treatment with topiramate is found to be useful in migraine, bipolar disorder, weight loss and neuropathic pain.^[2] Several cases of ocular adverse reaction related to topiramate administration have been published.^[3]

CASE HISTORY

A 55 year old healthy male presented in medical emergency department with severe headache, sudden onset of bilateral ocular pain and blurred vision. He was advised CT scan head and referred to ophthalmology department. On examination, his visual acuity with presenting glasses was 20/400 OD and 20/300 OS. Intraocular pressures were 68mmHg OU. Central corneal thickness was 610 μ m OD and 560 μ m OS. Slit-lamp examination revealed bilateral conjunctival chemosis, corneal edema, shallow anterior chamber (figures 1 and 2) and fixed dilated pupils.

On detailed history taking, he had history of undergoing treatment for deaddiction for last 6 months. He was taking many antipsychotic drugs like duloxetine, escitalopram, mirtazapine, quetiapine, lorazepam. He had started Topiramate 25 mg daily for headache and depression 9 days back. He had no other significant history and no family history of glaucoma. Diagnosis of Topiramate induced Bilateral acute ACG was made and patient was advised to discontinue topiramate and other antipsychotic drugs after psychiatric consultation. Patient was started on maximum antiglaucoma medication including intravenous mannitol, oral hyperosmotic (syrup Glycerol), topical antiglaucoma eye drops i.e timolol 0.5%, brimonidine 0.1% and steroid eye drops. Cycloplegic drops

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homatropine 2% were given considering the fact that mechanism of angle closure in such cases is not pupillary block but uveal effusion syndrome. The following day, conjunctival chemosis and shallow anterior chamber persisted with IOP decreasing to 29 and 31 mmHg respectively. Corneal edema started clearing. Gonioscopy showed closed angles OU. Fundus examination revealed normal retina and cup-disk ratio of 0.3 in both eyes.

um OD and 537 um OS.

Anterior chamber became within normal limits. (Figures 3 and 4) Patient was discharged on treatment with timolol 0.5% and homatropine 2% twice daily.

DISCUSSION

Acute angle closure glaucoma occurs in predisposed individuals (hypermetropia, narrow angle, thick lens) when

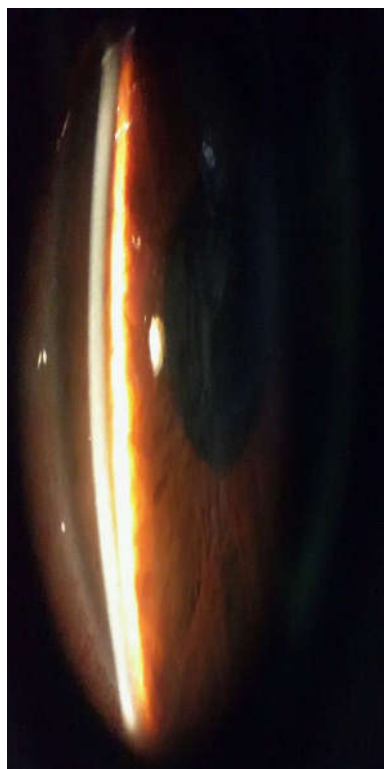


Figure 1

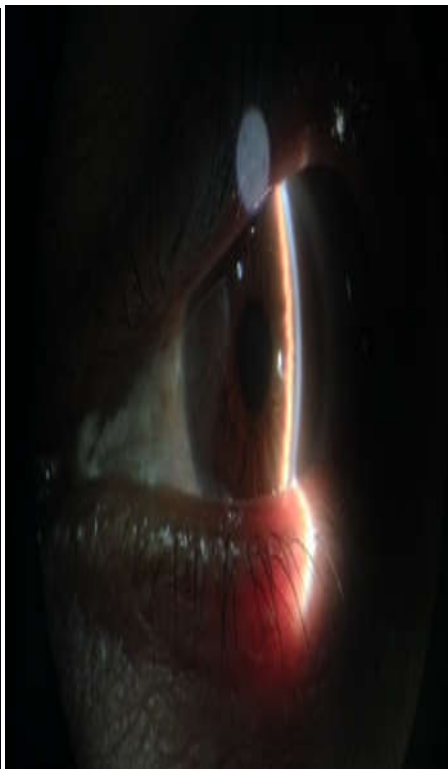


Figure 2

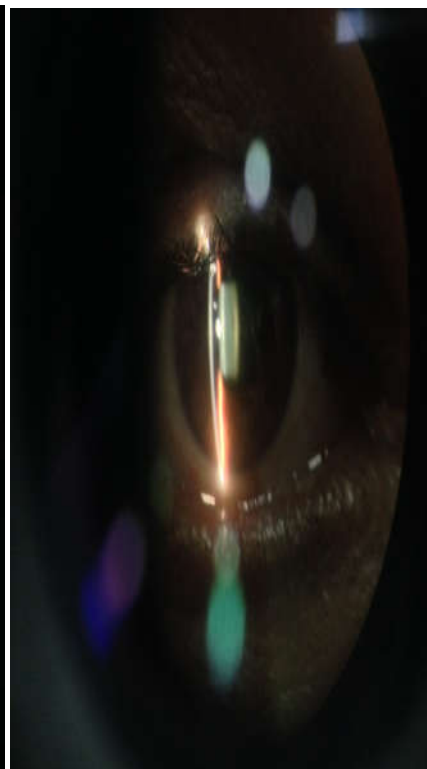


Figure 3



Figure 4

IOP started returning to normal limits with resolution of the corneal edema. Oral hyperosmotics were discontinued. His visual acuity improved to 20/20 OU with glasses. CCT was 528

pupil is mid dilated. At least one third of acute ACG cases are related to drugs. Among these, sulfonamide drug and its derivatives like topiramate have been documented to cause transient myopia, ciliary body edema, choroidal effusions and anterior rotation of the lens-iris diaphragm inducing secondary angle closure.^[4] Adrenergic agents and anticholinergic drugs cause mydriasis that can precipitate attack of acute ACG. Antidepressants, antihistamines and antianxiety drugs also have weak anticholinergic activity.

In this case, the differential diagnoses were primary angle-closure glaucoma (PACG), acute attack of chronic angle-closure glaucoma and drug-induced secondary angle-closure glaucoma. A few factors, such as no history of glaucoma, no peripheral anterior synechiae, no progressive cupping of the optic nerve head and no characteristic glaucomatous visual field loss, excluded the possibility of acute attack of chronic angle-closure glaucoma. PACG is unilateral most of times. Topiramate-induced bilateral acute angle-closure glaucoma and myopic shift had been reported in several literatures.

Topiramate can cause potentially serious ocular side effects including blurred vision, acute IOP elevation, acute myopia, diplopia, nystagmus and shallow anterior chamber with angle closure.^[5] In placebo-controlled trials, myopia was seen in 1% of children and abnormal vision in 13% of adults. Overall

ocular adverse effects >1% was noted from the literature review.^[6] These patients were predominantly female (89%) with a mean age of 34 years. Most cases have been reported to occur within the first 2 weeks (3-21 days) of starting topiramate administration, or within hours of doubling the doses.^[7] Few cases were presented as unilateral episodes.^[8]

The mechanism of topiramate-induced acute IOP elevation may be an idiosyncratic reaction and can occur in otherwise normal eyes with normal anterior chamber angles, resulting in ciliochoroidal effusions with ciliary body swelling inducing forward rotation of the iris-lens diaphragm, causing myopia and angle-closure glaucoma.^[4,5] The management of topiramate-related acute angle-closure glaucoma requires immediate cessation of topiramate and institution of oral and topical aqueous suppressants. Use of pilocarpine can lead to further narrowing of the angles and worsening of signs and symptoms. Traditional treatment for angle-closure glaucoma such as laser iridotomy or surgical peripheral iridectomy may not be of value as precipitating mechanism is not pupillary block. Topical cycloplegic agents can be given as they lower IOP by relaxing the ciliary muscle and deepening anterior chamber.^[4,5,6,7,8]

In our case, we administered intravenous mannitol, topical aqueous suppressants and topical steroids for inflammation, topical cycloplegics to relax ciliary muscle, in addition to topiramate cessation. Oral acetazolamide, a sulfa-derivative, was avoided as treatment for the elevated IOP, because it has been reported to cause secondary angle-closure glaucoma.^[9] In our opinion, any systemic or topical sulfa-derivative drugs should be avoided when clinical suspicion of topiramate-associated acute angle-closure glaucoma is encountered.^[9,10] Ophthalmologist should be aware of this potential complication. The quick identification of sulfa drugs and subsequent discontinuation can expedite resolution of angle closure. If causative medication is not recognized, persistent high IOP may result in permanent visual loss. Neurologist and psychiatrist prescribing topiramate to patients should be alert for side effects and be advised immediate referral to ophthalmologist under clinical suspicion.

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