Case Report

Thioridazine Induced Bilateral Angle Closure and Myopic Shift

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ABSTRACT

Drug induced angle closure is a sight threatening complication in ophthalmology which if not treated urgently can lead to perpetual visual loss. Identifying the cause of disease and timely intervention can prevent permanent damage to the eye. This case report presents a case and subsequent discussion on thioridazine induced angle closure along with transient myopia. . This case report highlights some of the key issues pertaining to drug induce angle closure and the potential approach to tackling it.

Keywords: Thioridazine, Secondary Angle Closure, Myopia.

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INTRODUCTION

Several oculars as well as non-ocular drugs are known to cause angle closure glaucoma as their side effect including sulfa drugs, tricyclic antidepressants, antipsychotics, benzodiazepines, anti-parkinsonians, anti-convulsant, few non-steroidal ant- inflammatory drugs, some antibiotics and H2 blockers. Thioridazine is known to cause idiosyncratic reactions and retinal toxicity but thioridazine induced angle closure glaucoma has never been reported. The mainstay of treatment is to immediately stop the drug and observe the patient as symptoms are usually transient.

Case Report

A 38-year-old gentleman presented with a complaint of bilateral sudden decrease in vision associated with severe ocular pain. History revealed that he had used one tablet of over-the-counter Diagesic-P (thioridazine, caffeine and paracetamol) for headache followed by symptoms of acute angle closure within 2 hours. He consulted an ophthalmologist for these symptoms who prescribed him topical pressure lowering agents including timolol, dorzolamide and brimonidine along with oral acetazolamide 250mg QID after checking intraocular pressures. He was referred to us for peripheral iridotomy.

On examination, his visual acuity in both eyes were counting finger at 1 meter(CF 1M) which did not improve with refraction or pinhole. He had a myopic shift in both eyes despite having no previous history of wearing glasses. Slit lamp examination revealed that he had conjunctival hyperemia, corneal clouding along with bilateral sluggishly reactive pupils. Furthermore, intraocular pressure measured by Goldmann applanation tonometer was 44 mmHg in both eyes. Gonioscopy revealed 360 degrees closed angle in both eyes. Fundus details were unremarkable. Patients advised B scan, Optical coherence tomograph of retinal nerve fiber layer and anterior segment. B scan and OCT RNFL were unremarkable.

Based on the above findings, a provisional diagnosis of bilateral drug induced angle closure was made. Oral and topical pressure lowering drugs were prescribed which included oral acetazolamide 250mg QID, topical timolol, dorzolamide and brimonidine. Tablet Diagesic-P was with-held.

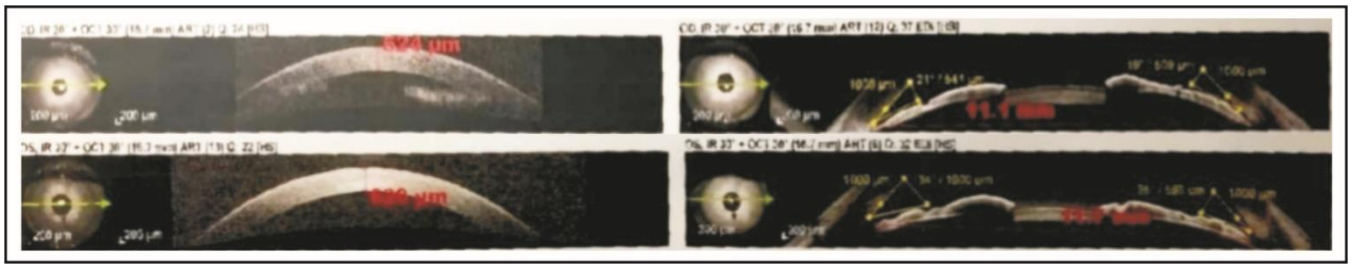

Figure 1: OCT anterior segment shows narrowing of angle with anterior displacement of lens vault and an increase in corneal thickness.

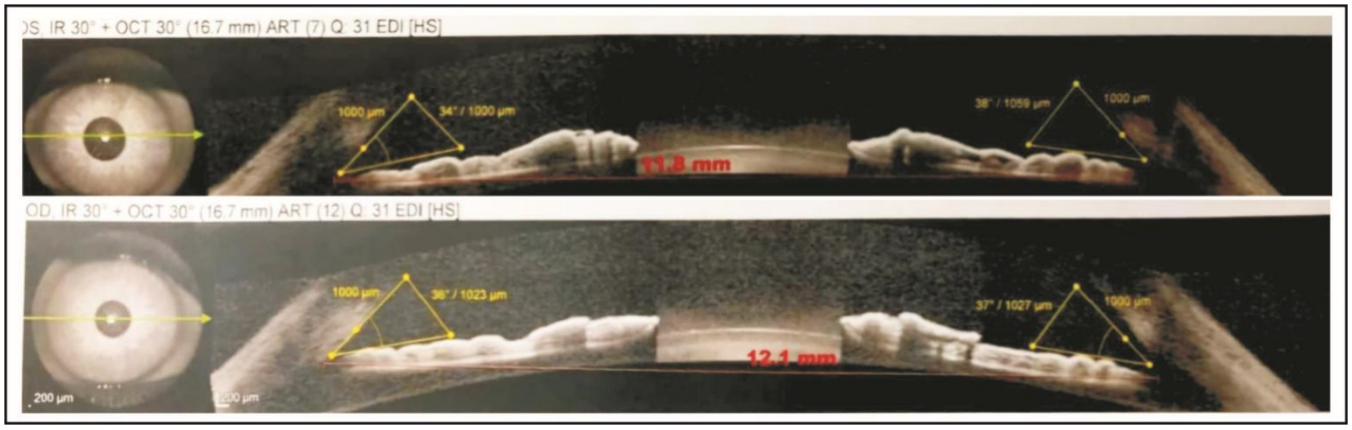

Figure 2: OCT anterior segment after discontinuation of thioridazine.

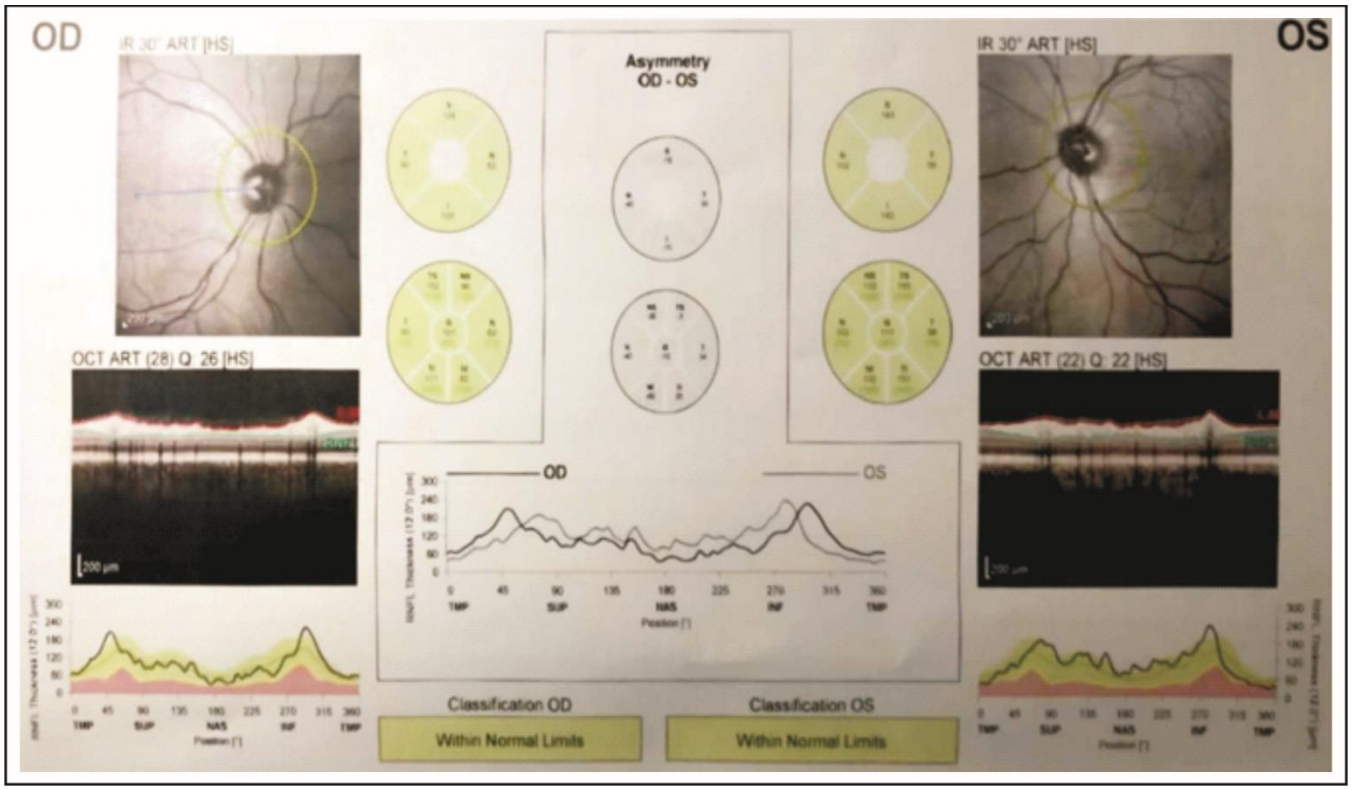

Figure 3:

Patient reported improvement in symptoms on next day and his visual acuity was 20/200 in both eyes. The intraocular pressure was 7mmHg in both eyes but gonioscopy still revealed grade 0 angle superiorly and grade 1 in nasal and temporal quadrant and grade 2 inferiorly. Patient was kept on the same treatment, and he was advised for the follow-up after 4 days.

The second follow-up revealed that the patient had significantly improved. His visual acuity was 20/20 in both eyes. Myopic shift was absent, corneal edema and conjunctival chemosis had resolved. Gonioscopy showed a bilateral grade 3 angle in all quadrants. Intraocular pressure with Goldmann tonometer were 10 and 8 mmHg in the right and left eyes respectively. Fundus examination was unremarkable with cupping 0f 0.4 and 0.5 and no disc pallor. Oral acetazolamide and topical brimonidine were discontinued and patient were kept on topical timolol and dorzolamide twice daily for 2 weeks. After the 3 weeks, the ocular examination was unremarkable and anti-glaucoma medications were discontinued.

DISCUSSION

Several psychotropic medications are known to induce angle closure glaucoma i.e. tricyclic antidepressants (TCAs), typical antipsychotics, atypical antipsychotics (olanzapine, ziprasidone) topiramate, selective serotonin and norepinephrine reuptake inhibitors (venlafaxine) and selective serotonin re-uptake inhibitors (citalopram).[1,2] Till date to our best knowledge, thioridazine induced angle closure has not been reported in published form.

Thioridazine belongs to a group of Phenothiazines which can cause irreversible hyperpigmentation of conjunctiva, cornea, lens, retina, choroid and macula[3]. It is known to cause idiosyncratic reactions which is postulated to be immune mediated. Idiosyncratic reactions are hypersensitivity reactions that occur rarely and unpredictably and these are also dose independent reaction. The proposed mechanism of idiosyncratic reaction of Thioridazine is that it is hydroxylated by myeloperoxidases which results in inhibition of retinal redox hemostasis and thus cell death.[4]

Various mechanisms which cause drug induced angle closure glaucoma are pupillary block with miosis, angle crowding with mydriasis, and disruption of the iridocorneal angle with ciliochoroidal effusion.[5] Like other psychotropic drugs, Thioridazine also caused angle closure due to disruption of iridocorneal angle with anterior displacement of iris-lens diaphragm. Another potential mechanism could be its anticholinergic activity leading to pupil dilation.[6]It has been hypothesized that rotation of ciliary processes due to ciliary body edema causes effacement of ciliary sulcus. It leads to forward and anterior rotation of lens iris-diaphragm along with shallow anterior chamber. Another theory is that idiosyncratic reaction can cause ciliochoroidal effusion.[7]

Desai et al, reported a patient who developed myopia as a side effect of Topiramate. To prevent angle closure glaucoma, they closely monitored the patient for ocular side effects related to the medication. They also recommended considering other possible diagnoses for drug-induced myopia and secondary angle closure, including accommodative spasm and posterior scleritis.[8]

Topical pilocarpine acts through pupillary constriction and opening the angle between iris and cornea. It facilitates aqueous humor drainage; thus, it is commonly used in angle closure glaucoma. However, in case of anterior displacement of iris-lens diaphragm it can further exacerbate the angle closure. Therefore, in cases of drug-induced angle-closure glaucoma, where the anterior chamber may already be compromised, pilocarpine should be avoided to prevent aggravating the condition.[9,10]

Drug induced angle closure is usually a transient, self-limited and reversible condition if the causative medication is identified and discontinued promptly. Given the rapid advancements in drug development, it is therefore important to thoroughly assess the patient's history and conduct a detailed examination. Furthermore, docking simulations can be used to study biomolecular interactions and predict idiosyncratic reaction to avoid any sight or life-threatening complications of new upcoming drug.

Patient's Consent: Researchers followed the guide lines set forth in the Declaration of Helsinki.

Conflict of Interest: Authors declared no conflict of interest.

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Authors Designation and Contribution

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