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Case Series

Drug induced secondary angle closure glaucoma- A case series

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ABSTRACT

Purpose: We report 3 cases with acute bilateral angle closure glaucoma following intake of oral chlorthalidone, mefenamic acid, topiramate.
Materials and Methods: Case series.
Results: All patients developed bilateral acute onset painful defective vision with shallow anterior chamber, raised intraocular pressure. Gonioscopy revealed bilateral occludable angle and ultrasonogram B scan showed shallow choroidal detachment, suggestive of drug induced non pupillary block secondary angle closure glaucoma. All patients were managed by discontinuation of causative drug and started on topical antiglaucoma medication and cycloplegic drug.
Conclusion: we would like to bring this potentially sight threatening and reversible ophthalmic condition to general physician, which can be managed effectively by timely referral and intervention.

1. Introduction

Drug induced acute angle closure glaucoma is a sight threatening ocular emergency condition, which can be present as acute painful diminution of vision, nausea and vomiting, headache, coloured halos following administration of topical or systemic medication. Here we report 3 patients developed bilateral drug induced non pupillary block secondary angle closure glaucoma following administration of oral chlorthalidone, mefenamic acid, topiramate. Clinically all patients showed bilateral simultaneous induced high myopia, shallow anterior chamber, occludable angles, choroidal effusion.

2. Case 1

60 year old male came with complaints of sudden onset painful diminution of vision in both eyes (BE) since1 day. He is known case of diabetic mellitus 5 years and systemic hypertension past 1 week, was on tablet metformin 500mg twice a day(BD) for 5 years and tablet telmesartan plus chlorthalidone (40/12.5mg) combination once daily for 1 week. On examination BCVA-RE 5/60 with -4.0 DS 6/6 LE-6/36 with -3.0 DS 6/6. Intraocular pressure (IOP) by using applanation tonometry (AT) showed RE-36mmHg LE -30mmHg. Gonioscopy revealed BE- grade1 appositional angle closure (shaffer's grading). Anterior segment examination showed BE- circumcorneal congestion, shallow anterior chamber (Van Hericks grade1), 5mm sluggishly reacting pupil, pseudophakia (Figure 1). Fundus examination was within

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normal limits. Ultrasonogram B scan BE showed shallow choroidal detachment all four quadrant. We arrived at the diagnosis of BE-chlorthalidone induced secondary angle closure glaucoma. Patient was advised to stop chlorthalodone, started on BE- Timolol eye drops twice a day and homatropine eye drops twice a day. Five days later patient was symptomatically better, BCVA-BE 6/12 with -0.75 DS 6/6. IOP was RE-14mmHg LE-10mmHg. Gonioscopy showed open angle in BE. Anterior and posterior segment examination was with in normal limits (Figure 2).

3. Case 2

45 year old male presented with complaints of painful defective vision BE since 3 days. Past five days, he was on tablet topiramate 25mg once daily for headache. On examination BCVA- BE 5/60 with -5.0 DS 6/6, IOP BE showed 26mmHG. Gonioscopy revealed BE- grade 2 appositional angle closure (shaffer's grading).Anterior segment examination showed bilateral circumcorneal congestion, shallow anterior chamber(Van Hericks grade 2), 5mm sluggishly reacting pupil, clear lens. Fundus examination was within normal limits. Ultrasonogram B scan showed bilateral shallow choroidal detachment all four quadrant. . We arrived at the diagnosis of BE- Topiramate induced secondary angle closure glaucoma. Patient was advised to stop tablet topiramate, started on timolol (0.5%) eye drops BD, homatropine (2%) TDS for 1 week. One week later patient was symptomatically better, BCVA-BE 6/6. IOP was RE-14mmHg LE-10mmHg. Gonioscopy showed open angle in BE. Anterior and posterior segment examination was with in normal limits.

4. Case 3

40 year old female presented with complaints of painful defective vision BE since 2 days. She was on tablet mefenamic acid 500mg BD for 3days for dysmenorrhea. On examination BCVA- BE 4/60 with -6.0 DS 6/9. IOP was BE- 32mmHg. Gonioscopy revealed BE- grade 1 appositional angle closure (shaffer's grading). Anterior segment examination showed bilateral circumcorneal congestion, shallow anterior chamber (Van Hericks grade 1), 5mm sluggishly reacting pupil, clear lens. Fundus examination was within normal limits. Ultrasonogram B scan showed bilateral shallow choroidal detachment all four quadrant. . We arrived at the diagnosis of BE- Mefenamic acid induced secondary angle closure glaucoma. Patient was advised to stop tablet mefenamic acid, started on timolol (0.5%) eye drops BD, homatropine (2%) TDS for 1 week. One week later patient was symptomatically better, BCVA-BE 6/6. IOP was BE-14mmHg. Gonioscopy showed open angle in BE. Anterior and posterior segment examination was with in normal limits.

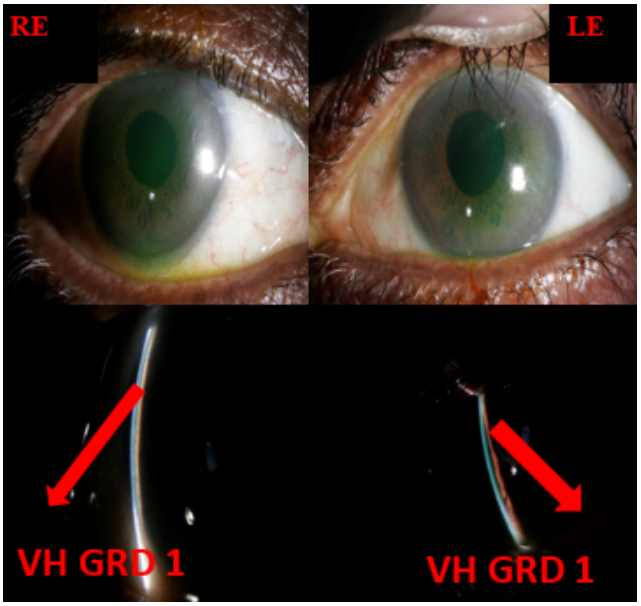

Fig. 1: Slit lamp photograph of 60 year old male showed bilateralshallow anterior chamber (Van Hericks grade1), 5mm sluggishly reacting pupil following administration of oral clorthalidone.

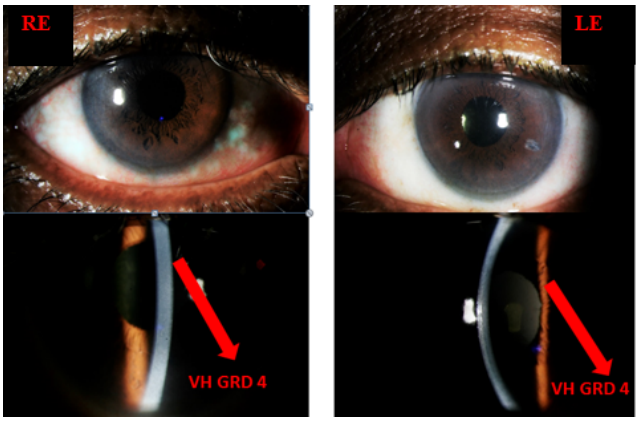

Fig. 2: Slit lamp photograph of 60 year old male showed bilateral normal depth anterior chamber(Van Hericks grade 4), 3mm normal reacting pupil, following withdrawal of oral clorthalidone.

5. Discussion

Drug induced angle closure glaucoma is a preventable cause of potential blindness. It caused by pupillary block and non-pupillary block mechanism.[1,2] In pupillary block angle closure, pupillary dilation increases iris-lens contact, which blocks aqueous drainage from posterior chamber, pushes peripheral iris against trabecular meshwork. In non-pupillary block angle closure, anterior rotation of ciliary body with or without choroidal effusion moves iris-lens diaphragm forward, which blocks trabecular meshwork. Some cases in non-pupillary block, cilary body swelling

Table 1: Drugs causing angle closure glaucoma through non-pupillary block mechanism

Cholinergics	**Other sulfa based drugs**
Pilocarpine	Topiramate
Carbachol	Sumitriptan
Anticholinesterase	Sotalol
Sulfonylureas	**Sulfa based diruetics**
Chlorpropamide	Acetazolamide
Glicazide	Furosemide
Glimepiride	Bumetanide
Tolbutamide	Chlorothiazide
	Chlorthalidone
Sulfa Based Antibiotics	Metolazone
Trimethoprim-sulfamethoxazole	Indapamide
Sulfadizine	
Dapsone	**Rheumatological Drugs**
	Sulfasalazine
Anti-Inflammatory	Probenecid
Mefenamic acid	Celecoxib
Anticoagulants	
Heparin	

causes zonule laxity, facilitates lens thickening lead to pushing iris against trabecular meshwork. Apart from gonioscopy and ultrasonogram, ultrasound biomicroscopy and anterior segment optical coherence tomography also useful tool for diagnosis. Since treatment modality differs based on underlying mechanism, it is important to know the mechanism. Several drugs have been documented, causing non-pupillary block angle closure glaucoma[3–9] (Table 1). Treatment modalities include, stop the causative drug and start anti glaucoma medications with topical cycloplegic drug. Laser peripheral iridotomy was ineffective, since non-pupillary block is the underlying mechanism.

6. Conclusion

We would like to bring this sight threatening ophthalmic condition to the general clinicians attention, since those drugs are commonly used in day to day practice. Once the diagnosis is suspected, urgent referral is required for appropriate and prompt management, which will be potentially sight saving.

7. Source of Funding

None.

8. Conflicts of Interest

There is no conflict of interest.

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