

GLAUCOMA

Occurrence of indapamide-induced acute angle closure in a predisposed patient calls for increased physician ophthalmologist collaboration: a case report

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Background: Drug-induced Acute Angle Closure (AAC) is a sight-threatening ophthalmic emergency that occurs more commonly in predisposed patients. Acute angle-closure glaucoma occurs when the circulation of aqueous humor from the posterior to the anterior chamber is blocked, causing the pressure inside the eye to rise rapidly.¹ This is an ocular emergency because if it is not treated quickly, it can lead to permanent loss of vision.² Risk factors include anatomical factors such as shallow anterior chamber (AC) depth, short axial length, and plateau iris configuration. Demographic factors include female sex, Asian ethnicity, family history, and advanced age. In addition, some medications known as trigger drugs include adrenergic agonists, anticholinergics, cholinergics, sulfonamides, supplements, and serotonergic medications.³

Objectives: The purpose of this case report is to draw the attention of ophthalmologists, physicians and general practitioners to the seriousness of drug-induced Acute Angle closure; to highlight the need to know the inciting classes of drugs and the need for physicians to collaborate with ophthalmologists to rule out predisposing eye conditions in patients before commencement of such medications.

Case Presentation: A 56-year-old male civil servant, a known hypertensive and diabetic, was recently placed on oral indapamide by his physician. He developed sudden, very severe ocular pain in his left eye (LE), soon after taking the first dose of oral indapamide 2.5mg. By the second dose, ocular pain became severe with profound diminution of vision, and seeing haloes around light.

Ocular examination revealed visual acuity (VA) of counting fingers, conjunctival congestion, corneal haziness, shallow anterior chamber and mid-dilated, unreactive pupil. The left eyeball was notably hard, and intraocular pressure (IOP) was greater than 70 mmHg.

He had recently been diagnosed with Primary Angle closure suspect based on shallow AC and occludable angles on gonioscopy in the same eye. He procrastinated the recommended urgent Laser peripheral iridectomy (LPI). He had previously undergone trabeculectomy in the right eye on account of primary angle-closure glaucoma.

He was immediately advised to stop taking the indapamide and was treated with intravenous 20% Mannitol 250mls over 20 mins, Eye drops Pilocarpine 4% thrice daily, Dorzolamide/timolol fixed drug combination twice daily and oral Acetazolamide 250mg thrice daily. He was scheduled for immediate LPI, but this was delayed due to persistent hazy cornea with IOP above 45 mmHg. The cornea eventually cleared after discontinuation of Acetazolamide. The LPI was subsequently performed with good outcome as VA improved to 6/9 while IOP reduced to 10 mmHg.

Discussion: This case report flags indapamide as a trigger for drug-induced AAC in a predisposed patient. Indapamide is a derivative of benzolsulphonamide, hence a sulphonamide.⁴ It is also noteworthy that whereas Diamox (acetazolamide) was indicated as an IOP-lowering agent to help this patient, it appeared to worsen his symptoms, also being a sulphonamide. Sulphonamide-induced angle closure glaucoma was first described in 1962.⁵

Indapamide is a very useful drug, growing in popularity as an effective antihypertensive treatment, especially among diabetic patients, probably because it is a thiazide-like diuretic without glucose or lipid disturbances.⁶ It is indicated for the treatment of hypertension and edema due to Congestive cardiac failure.⁴ However, like some other sulphonamides, it has been implicated as a trigger for drug-induced acute angle closure.

The proposed mechanisms for sulphonamide-induced angle closure glaucoma include:

- Osmotic Disturbance within the crystalline lens leading to hydration of the lens and subsequent thickening with anterior displacement of the lens-Iris diaphragm⁵
- Induction of ciliary body edema with subsequent supraciliary effusion resulting in forward rotation of the ciliary body and mechanical angle closure⁵
- Accommodative spasm of the ciliary muscles⁷

A case report of a multimodal imaging study of indapamide-induced choroidal effusion showed evidence supporting the second of the three mechanisms described above.⁷

Conclusion: Indapamide, an effective thiazide-like antihypertensive treatment among diabetics, has been flagged as a potential trigger for acute angle closure in a predisposed patient. It is therefore recommended that physicians and ophthalmologists should be aware of this potentially sight-threatening adverse effect of Indapamide and other medications that can precipitate Acute Angle closure and educate patients about the warning symptoms, and the need for early presentation to an ophthalmologist if symptoms occur. It is also recommended that physicians collaborate with ophthalmologists to rule out predisposing eye conditions before the prescription of such medications.

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Social media as a frontier for creating glaucoma awareness: strategies, prospects and challenges

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