ACETAZOLAMIDE INDUCED EFFECTS IN GLAUCOMA PATIENT–A CASE REPORT

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ABSTRACT
Glucoma is characterized by the slow, progressive degeneration of retinal ganglion cells (RGCs) and optic nerve axons. Those drugs that lower intra-ocular pressure and those drugs those offer the additional benefit of neuroprotection, independent of intra-ocular pressure reduction. Acetazolamide is indicated for adjunctive treatment of edema due to congestive heart failure, drug-induced edema, centrencephalic epilepsies and chronic simple (open-angle) glaucoma. Other therapeutic actions of ACZ include reduction in the number of bedtime arousals (apnea-associated hypoxemia) and improvement in the quality of sleep. In glaucoma, acetazolamide decreases the production of aqueous humor and hence lowers the intra-ocular pressure. Patients should be counselled with adequate information regarding the side effects of acetazolamide and advised to report back to physician if any noxious feeling is experienced.

KEYWORDS: Glaucoma, Intraocular pressure (IOP), Acetazolamide (ACZ), Primary open angle glaucoma (POAG).

INTRODUCTION
Glucoma is characterized by the slow, progressive degeneration of retinal ganglion cells (RGCs) and optic nerve axons.[1] Glucoma is a condition that involves distinctive changes in the optic nerve and visual field. It is marked by functional and structural abnormalities in the eye in which optic nerve damage can ordinarily be alleviated and inhibited by sufficiently reducing intraocular pressure (IOP).

Primary glaucoma is divided into primary open angle glaucoma (broad definition) (a disease concept that encompasses both conventional primary open-angle glaucoma and normal-tension glucoma) and primary angle-closure glucoma.[2] Primary open angle glucoma (POAG) is a leading cause of irreversible blindness world-wide.[1] Major risk factors for POAG include positive family history, thin central cornea, elevated intraocular pressure (IOP), and older age.[3]

Acetazolamide (ACZ) is a sulfonamide derivative that inhibits carbonic anhydrase (CA) and was approved by the Food and Drug Administration in 1953 for its use as a diuretic, anticonvulsant and antiglucoma agent. ACZ is rapidly absorbed, achieves a peak concentration at 2 to 4 hours, and is eliminated unchanged in the urine.[4]

Treatment of glaucoma can be either medical or surgical. In normo-tensive glucoma a reduction of 30% would be considered a reasonable initial target pressure after treatment. Sack (2000) outlined that drugs can be broadly broken into two groups. Those drugs that lower intra-ocular pressure and those drugs those offer the additional benefit of neuroprotection, independent of intra-ocular pressure reduction.[5]

CASE PRESENTATION
62 yr male presented with complaint of gradual decreased vision, especially in his left eye. Patient had no history of trauma to that both eye. There after he was progressively developed loose stools, nausea, vomiting, abdominal pain. Nobody in his family has had glucoma.

Past medical history
Known case of Glaucoma since 1 year. Past medication history: Tab. Acetazolamide 250mg BD.

On examination
Patient was found to be conscious, coherent febrile. On systemic examination pulse rate was 100 beats/min, BP was 100/70 mmHg, all other organ systems were normal.

Laboratory investigations
General Random Blood Sugar: 96gm/dL (80-120 mg/dL) Blood Urea (BU):18mg % (7–20mg/dl). Serum creatinine: 0.9mg % (0.6-1.2mg %). Serum electrolytes Sodium- 120mEq/L (135-145), Potassium- 4.3mEq/L (3.5-5.5), Chloride- 98mEq/L(95-105). RBC: 4.9 Cells/µm (4.2-5.4) PCV: 45.7% (42-50), MCV: 84.6FL (80-96). MCH: 29.9pg (27.5-33.2), MCHC: 38gm/dL

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(33.4-35.5), Platelets: 2.6 Lakhs/cu mm (1.5-4.0) and Hb: 12g/dL. WBC: 8,500 Cells/mm, (5000-10,000), Polymorphs: 59% (45-70), Lymphocytes: 38% (20-40), Eosinophils: 01% (1-4), Monocytes: 02% (2-10).

On examination his left eye revealed stromal edema and elevated IOP. He was diagnosed as a glaucoma for which his IOP was stabilized with mannitol eye drops. His visual acuity is normal in both eyes. He was found to have elevated IOP, in the mid-30 mmHg.

Gonioscopy of the angles reveals that in the right eye the trabecular meshwork is visible. Pigmentation/iris processes appear normal. The scleral spur is seen in only some directions of gaze. There are no peripheral anterior synchiae. Left eye looks similar. The intraocular pressures (IOP) are 22 mm right, 32 mm left at 3.30 pm. The optic discs: cup disc ratios (c/d) are 0.25 right and 0.95 left. Two weeks later IOPs are 22 and 28.

**Treatment**
Acetazolamide was stopped and Mannitol eye drops, IVF 1 pint NS and DNS, Inj. Ondansetron 4mg/BD, Tab. Sporolac/BD, Inj. Pantoprazole 40mg/OD, Inj. Ceftriaxone 1g/BD, Tab. Multivitamin/OD.

**DISCUSSION**
Acetazolamide is indicated for adjunctive treatment of edema due to congestive heart failure, drug-induced edema, centrencephalic epilepsies and chronic simple (open-angle) glaucoma. Other therapeutic actions of ACZ include reduction in the number of bedtime arousals (apnea-associated hypoxemia) and improvement in the quality of sleep.

In glaucoma, acetazolamide decreases the production of aqueous humor and hence lowers the intra-ocular pressure. Previous study was described a 33-year-old man who presented with hypotension and acute heart failure 30 minutes after acetazolamide assumption. Our patient had history of receiving Acetazolamide in the past but patient was experienced hypotension but not acute heart failure. There are several reports of calcium phosphate kidney stones apparently induced by the chronic use of ACZ. In 1975, one case of acute renal failure induced by acetazolamide therapy for glaucoma was reported. The patient had a full recovery after the administration of oral fluids and sodium bicarbonate. Recent papers have suggested that reducing the intraocular pressure by 30 percent does provide benefits. This amount of reduction might not be achieved with monotherapy using prostaglandin analogues only so combination therapy with drugs such as brimonidine or dorzolamide may be necessary.

**CONCLUSION**
Generally patients should counselled with adequate information regarding the side effects which are associated with acetazolamide and advised to report back to physician if any noxious feeling is experienced.

Betaxolol, a beta-1 selective agent thus reducing aqueous production and also thought to increase ocular blood flow to the ocular nerve head, due to vasodilatation. Studies suggest this may have a neuro-protective effect against visual field deterioration.

**REFERENCES**