Acetazolamide and Bilateral Uveal Effusion With Secondary Acute Angle-Closure Glaucoma

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CASE PRESENTATION
A 65-year-old white man presented with a bilateral acute onset of pain, blurred vision, and hyperemia. The patient had been under observation for diabetic retinopathy for 7 years. His only medication was insulin. The day before presentation, he received an intravitreal injection of bevacizumab in his right eye for the treatment of diabetic macular edema. He received a single oral dose of acetazolamide 250 mg to prevent a rise in IOP. His symptoms began in the early evening, hours after the injection.

Upon examination, the patient’s BCVA was 20/50 in his right eye and 20/40 in his left eye with corrections of -2.25 D and -1.25 D, respectively. A slit-lamp examination showed conjunctival congestion, no edema of the cornea, shallow anterior chambers, no rubeosis, and mild nuclear sclerosis in both eyes. The IOP measured 30 mm Hg in his right eye and 32 mm Hg in his left eye. Gonioscopy showed 360º of appositional angle closure with a convex iris configuration in both eyes. A fundus examination and B-scan ultrasonography revealed bilateral choroidal effusions (Figure 1). The anterior chamber depth was 2.21 mm in his right eye and 2.16 mm in his left eye. Ultrasound biomicroscopy revealed a 360º suprachoroidal effusion with anterior rotation of the ciliary processes in both eyes (Figure 2).

HOW WOULD YOU PROCEED?
- Would you administer a miotic for the angle closure?
- Would you perform a peripheral iridotomy?
- Would you prescribe treatment with aqueous humor suppressants such as a timolol or brimonidine?
CHALLENGING CASES

Would you treat with topical steroids?
How would you proceed surgically if medical treatment failed?

CLINICAL MANAGEMENT

We made a provisional diagnosis of acetazolamide-induced uveal effusions with secondary bilateral acute angle-closure glaucoma (ACG). The patient was treated with topical timolol 0.5%, brimonidine 0.2%, prednisolone acetate 1% q2h, and cyclopentolate HCl 1% b.i.d. On the fourth day of treatment, his choroidal effusions had resolved in both eyes. The anterior chamber depth was normal in both eyes, the angles were open, and the IOP measured 10 mm Hg in both eyes (Figure 3). Ultrasound biomicroscopy revealed a reattached ciliary body, with normally positioned ciliary processes (Figure 2). The patient’s BCVA was 20/50 OD and 20/40 OS with a refractive correction of +1.00 -1.25 X 90 and +1.00 D, respectively.

We stopped the antiglaucoma medications, tapered the topical steroid, and continued cycloplegia for 10 days. On the 14th day, his clinical findings were similar to the previous examination, and his IOPs were 14 mm Hg in his right eye and 15 mm Hg in his left eye. The anterior chamber depth was 3.40 mm in his right eye and 3.58 mm in his left eye, and the axial lengths were 23.72 mm and 23.84 mm, respectively.

OUTCOME

Two months after medical management, the patient’s BCVA was 20/40 bilaterally, which was limited by diabetic macular edema. His anterior segment revealed a deep anterior chamber, no rubeosis, and mild nuclear sclerosis in both eyes. Angles were open 360° in both eyes without neovascularization or synechiae. The IOP measured 16 mm Hg in his right eye and 13 mm Hg in his left eye without medication. His fundus showed no choroidal detachment in either eye.

DISCUSSION

Primary ACG is the most common form of acute ACG. Susceptible individuals have narrow angles, a shallow anterior chamber, and axial hyperopia.1 There are environmental drug exposures that can change the anatomy of the peripheral iris and trabecular meshwork. Such drugs include alpha-1 adrenergic agonists, cholinergic antagonists, antihistamines, antidepressants, and antianxiety drugs.2 Clinical findings include corneal edema, elevated IOP, a shallow anterior chamber, a central portion of the dilated iris apposed to the lens’ surface, and a
closed angle on gonioscopy. The goal of treatment is to lower IOP by performing laser iridotomy, paracentesis, or lensectomy, if there is a phacomorphic element. Glaucoma surgery may be necessary.

Other drugs, such as sulfa-based agents and anticoagulants, can induce acute ACG by ciliochoroidal effusions. The sulfa-based drugs, acetazolamide, hydrochlorothiazide, cotrimoxazole, furosemide, glipizid, and glimepirid, have been associated with acute ACG. Topiramate, a sulfamate-substituted monosaccharide has been reported to cause acute ACG, transient myopia, and uveal effusion.

An idiosyncratic reaction to certain drugs, such as acetazolamide, may cause uveal effusion and secondary acute ACG, which must be differentiated from primary ACG for appropriate management. A careful inquiry regarding the patient's current and past medications is critical. The onset of symptoms may occur hours following exposure with bilateral involvement, acquired myopia, anterior chamber shallowing, and choroidal effusion. Imaging with B-scan ultrasound and ultrasound biomicroscopy can confirm the diagnosis. Treatment is based on stopping the offending medication and therapy with aqueous suppressants, cycloplegia, and steroids. Because the management of acute ACG secondary to uveal effusion differs from that for pupillary block, unnecessary treatment of the pupillary block mechanism can be detrimental to the patient’s vision and quality of life.

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