

Angle-Closure Glaucoma: Primary, Secondary, or Both?

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CASE PRESENTATION

A 59-year-old white male presented to our emergency ward with a 3-month history of progressively blurred vision and intermittent pain without redness in his right eye. His medical history was significant only for erectile dysfunction, which was treated with sildenafil citrate. His last dose had been 2 to 3 days before his presentation. The patient's past ocular history was pertinent for hyperopia requiring spectacle correction. Many years earlier, he had also been informed that he had glaucoma, but he had not sought further care.

The patient had a BCVA of 20/20 OU (+3.50 D OD, +2.50 D OS). Trace conjunctival hyperemia and shallow anterior chambers were apparent in both eyes. His IOP was 48 mm Hg OD and 34 mm Hg OS. The C/D was 0.9 OD and 0.2 OS. Gonioscopy by the emergency ward resident revealed anterior trabecular meshwork for 90° OD and 180° OS, and the remainder of the angles were closed. He diagnosed bilateral angle-closure glaucoma (ACG) and administered timolol 0.5%, dorzolamide 2%, brimonidine 0.15%, latanaprost 0.005%, and 500 mg of oral acetazolamide. After the patient's IOP decreased to 12 mm Hg OD and 14 mm Hg OS, he was discharged with the aforementioned medical therapy.

We saw the patient in the glaucoma clinic on the following day (day 2). His examination was relatively unchanged, and his IOP was now 7 mm Hg OD and 6 mm Hg OS. We detected a right, relative, afferent pupillary defect. Repeat gonioscopy revealed some posterior trabecular meshwork and 180° of scleral spur in both eyes. The entire scleral spur was visualized by compression gonioscopy. There were no synechiae in either eye.

HOW WOULD YOU PROCEED?

1. How would you manage this patient?
2. Would ancillary testing be helpful?
3. What are the potential causes of angle closure in this patient?
4. Would laser peripheral iridotomy be of benefit?

CLINICAL COURSE

We proceeded with ultrasound investigation on day 2 (Table 1). B-scan revealed bilateral shallow inferior choroidal effusions (Figure 1) that were also observable anteriorly by ultrasound biomicroscopy (Figure 2). Small ciliary body cysts were noted at the 6- and 3-o'clock positions in the patient's right and left eyes, respectively (Figure 3).

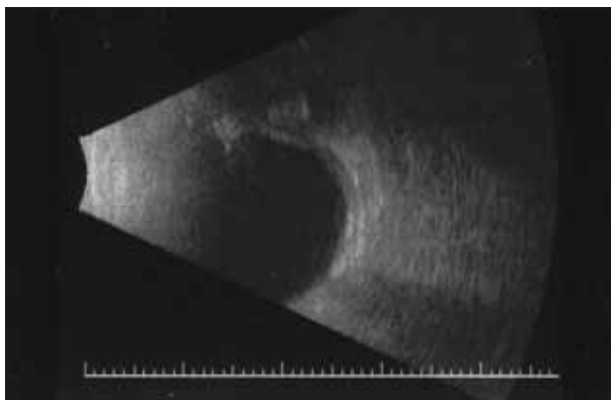


Figure 1. B-scan provides a long-axis view (6-o'clock meridian) of the patient's right eye and reveals shallow, inferior choroidal effusion. B-scan of his left eye yielded the same finding.

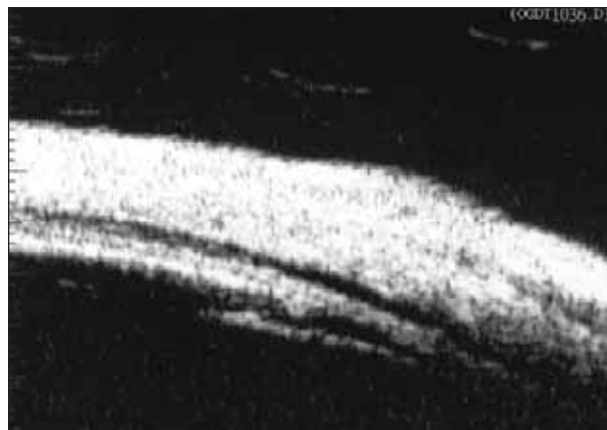


Figure 2. Ultrasound biomicroscopy of the patient's right eye also showed shallow choroidal effusion at the 6-o'clock meridian.

TABLE 1. A-SCAN MEASUREMENTS

	Day 2 (OD, OS)	Day 15 (OD, OS)
Axial Length (mm)	22.79, 22.47	22.86, 22.62
Anterior Chamber Depth (mm)	3.01, 3.01	2.82, 2.77
Lens Thickness (mm)	4.61, 4.82	4.92, 4.97

Our clinical impression was acute on subacute attacks of ACG, more prominent in the patient's right eye than his left. The ciliary body cysts did not appear sufficiently large to cause focal narrowing of the angle. Although the bilateral choroidal effusions were minimal, we instructed the patient to discontinue both the acetazolamide and sildenafil citrate in case either drug were a contributing factor.

On day 15, there were no significant changes in refraction, the slit-lamp examination, or gonioscopic examination. The patient's IOP was 11 mm Hg OU. B-scan revealed resolution of the choroidal effusions. A-scan measurements of the anterior segments were not conclusive for a drug-induced mechanism for ACG. We recommended laser peripheral iridotomies in both eyes, first the right and then the left.

A Humphrey visual field 24-2 (Carl Zeiss Meditec Inc., Dublin, CA) was consistent with advanced glaucomatous optic neuropathy in the patient's right eye, but his left eye was normal (Figure 4).

OUTCOME

After undergoing laser peripheral iridotomy in his right eye, the patient's anterior chamber deepened. More scleral spur was observable except for superiorly, where only the posterior trabecular meshwork was visible. Brimonidine was discontinued, and IOP was controlled bilaterally with latanoprost only. The patient was lost to follow-up before we could treat his left eye.

DISCUSSION

Differentiating between the various causes of ACG was challenging in this case. The pathophysiology of primary ACG is well known, and the disease usually occurs in smaller, hyperopic eyes with relative pupillary block, which increases as the lens enlarges with aging. The definitive treatment is a peripheral iridotomy, which was successful in deepening the angle of this patient's right eye. Other less traditional

treatments for ACG include cataract extraction, anterior goniosynechialysis, and argon laser iridoplasty.¹

Ancillary testing detected two potential contributing factors in this patient's ACG: possible drug-induced choroidal effusions and ciliary body cysts. Drug-induced choroidal effusions can cause secondary, bilateral ACG. This reaction is most commonly associated with sulfa-based medications.² The proposed mechanisms are an idiosyncratic reaction, carbonic anhydrase inhibitor activity, a prostaglandin-mediated effect, or, less likely, an allergic reaction.^{3,4} Uveal effusions with ciliary body swelling cause forward rotation of the lens-iris diaphragm and may result in a myopic shift and/or ACG. Some causative drugs include acetazolamide, hydrochlorothiazide, trimethoprim-sulfamethoxazole, indapamide, promethazine, spironolactone, isosorbide dinitrate, bromocriptine, tetracycline, corticosteroids, penicillamine, quinine, metronidazole, isotretinoin, aspirin, and topiramate.⁴ Treatment of drug-induced bilateral ACG is discontinuation of the causative drug. Medical treatment is often all that is needed. Laser iridotomy is usually not necessary.

Our patient was taking sildenafil citrate, which contains a sulfonamide moiety.⁵ Sildenafil citrate may also have a vasodilatory effect on the choroidal vasculature that can increase choroidal congestion diffusely.⁶ It is unclear whether this medication contributed to our patient's bilateral ACG. By the time we obtained ultrasound images, the choroidal effusions were minimal. The mechanism may have been in play, because the angle deepened from day 1 to day 2. This change may have indicated a decrease in the amount of choroidal effusions and a lessening of forward rotation of

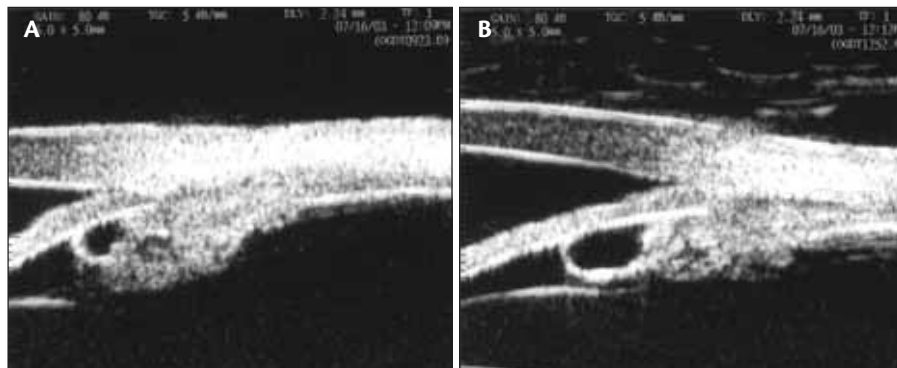


Figure 3. Ultrasound biomicroscopy showed ciliary body cysts at the 6-o'clock meridian OD (A) and at the 3-o'clock meridian OS (B).

the ciliary body. The acetazolamide and sudden decrease in IOP may also have caused the choroidal effusions. Although our patient did not have a history of glaucoma filtration surgery, individuals who have undergone filtration surgery and then received treatment with topical aqueous suppressants (eg, timolol 0.5% and dorzolamide hydrochloride) or oral acetazolamide may develop hypotony and ciliochoroidal effusions that resolve with discontinuation of the aqueous suppressants.^{7,8} Both sildenafil citrate and acetazolamide

were stopped in our patient.

Other mechanisms, not seen in our patient, that can cause choroidal effusion-related glaucoma include posterior scleritis, Vogt-Koyanagi-Harada syndrome, acquired immune deficiency syndrome, IgA nephropathy, and a tight scleral buckle or panretinal photocoagulation.⁹

Primary cysts of the iris or ciliary body may cause a secondary ACG when they are multiple.¹⁰ Multiple cysts can elevate the iris and produce narrowing or closure of the angle. When isolated, the cysts are often harmless and may be observed.¹¹ At the slit lamp, a "bumpy" or convex contour to the peripheral iris is visible. On gonioscopy, a focal narrowing of the angle may occur in locations where there are cysts. When the pupil is dilated, gonioscopy can often allow their direct visualization. Ultrasound biomicroscopy or ultra-high-resolution anterior segment optical coherence tomography can differentiate these cysts from a solid lesion.¹¹ Treatment may include laser or surgical incisional cyst puncture. Our patient's cysts were too small and too few to be considered a significant contributing factor in his ACG. □

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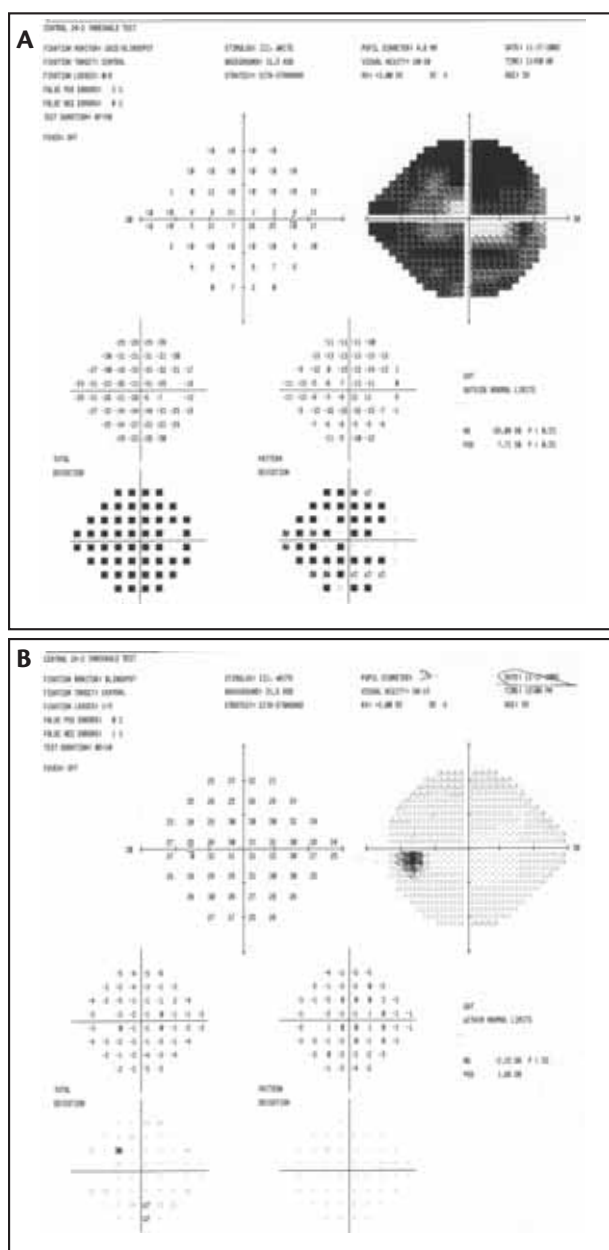


Figure 4. A Humphrey visual field 24-2 revealed severe constriction in the patient's right eye (A), whereas findings in his left eye were normal (B).

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