

BRIEF REPORTS

Dorzolamide-Induced Choroidal Detachment in a Surgically Untreated Eye

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PURPOSE: Choroidal detachment is a known complication of topical hypotensive agents when used to treat eyes sensitized by prior surgery. We document the abrupt development of an extensive choroidal detachment after initiation of dorzolamide therapy in a surgically untreated eye with primary open-angle glaucoma.

DESIGN: Observational case report.

METHODS: A 76-year-old woman with primary open-angle glaucoma and no history of ocular surgery developed a choroidal detachment 12 hours after initiation of therapy with dorzolamide eye drops. Choroidal detachment was diagnosed clinically and confirmed by echography.

RESULTS: Withdrawal of the drug and initiation of corticosteroid drops resulted in prompt resolution of the choroidal detachment.

CONCLUSIONS: Choroidal detachment can occur in surgically untreated eyes after use of a topical carbonic anhydrase inhibitor. (*Am J Ophthalmol* 2004;138:285–286. © 2004 by Elsevier Inc. All rights reserved.)

AQUEOUS SUPPRESSION-ASSOCIATED HYPOTONY IS A known phenomenon occurring in patients who have undergone surgery.^{1–3} In contrast, eyes that have not undergone surgery have not been reported to demonstrate this phenomenon. We report a patient with no prior history of ocular surgery who developed a marked choroidal detachment in one of her eyes following the initiation of dorzolamide therapy.

A 76-year-old woman with a 7-year history of open-angle glaucoma presented with distorted acuity following two doses of dorzolamide. The patient had no history of ocular surgery and no medical allergies. Her past medical history was significant for mild hypertension and mi-

graines. Systemic medications included verapamil and hydrochlorothiazide. Eye medications consisted of timolol 0.5% twice daily in both eyes, used for the past 7 years, and dorzolamide 2% twice daily in both eyes, which had been started on the previous day.

On initial examination, the patient's visual acuity was 20/25 in both eyes with use of her –7.00-diopter spectacles. Anterior segments were quiet and intraocular pressure measured 10 mm Hg in both eyes. Gonioscopy revealed open angles with no shallowing. Examination with a dilated pupil showed myopic changes, with staphylomas and tilted disks in both eyes. A peripheral choroidal detachment was found in the left eye, most pronounced inferotemporally, which was proved by echography to extend 360 degrees (Figure 1).

Given the association between aqueous suppressants and choroidal detachment, the timolol and dorzolamide were discontinued and treatment with topical prednisolone acetate 0.5% was initiated. The patient's systemic medications were continued. One week later the patient noted a significant improvement in her subjective distortion. There was complete resolution of the choroidal detachments, and intraocular pressure measured 16 mm Hg in both eyes. The corticosteroids were then tapered, and the patient remained asymptomatic.

Vela and Campbell¹ first described the phenomenon of aqueous suppression-associated hypotony in 1985. Patients were characterized by long-term aqueous suppression followed by trabeculectomy. When therapy with timolol or carbonic anhydrase inhibitors, or both, was later restarted, these patients developed hypotony and ciliochoroidal detachments. Discontinuation of therapy resulted in reattachment. The investigators hypothesized that preoperative use of aqueous suppression created a "hypersensitive" ciliary epithelium that later resulted in aqueous production shutdown.

A similar phenomenon has been reported with prostaglandins.² Hypotony has also been noted with the use of glaucoma drugs following nonglaucoma surgery, specifically cataract extraction.³ Furthermore, patients with underlying pathology resulting in elevated episcleral venous pressure, such as Sturge-Weber syndrome, appear to be predisposed to this phenomenon, even without prior surgical intervention.⁴

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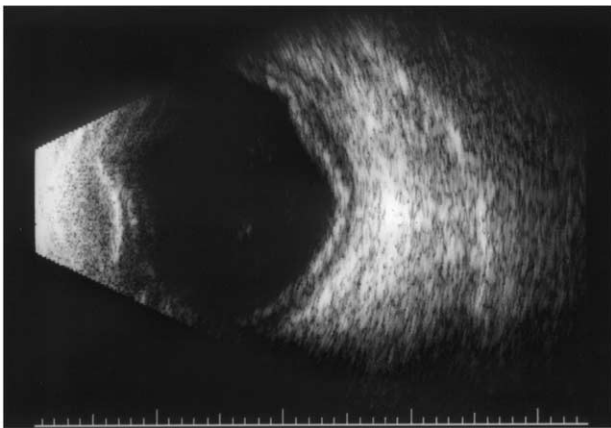
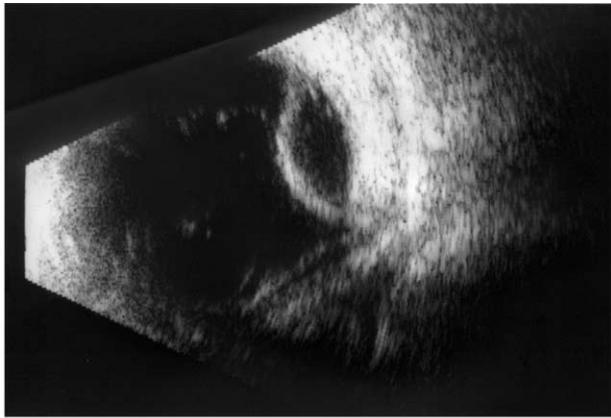


FIGURE 1. (Top) B-mode echography of the left eye showing a dome-shaped, inferotemporal choroidal detachment. (Bottom) Nasal view of the same eye demonstrating a shallow choroidal detachment.

In contrast, our patient developed choroidal detachments with the use of glaucoma therapy in a surgically untreated eye. The eye had not been “presensitized” by surgical intervention or underlying pathology. Nonetheless, the patient’s long-term use of low-dose systemic hydrochlorothiazide, started 5 years previously, might have been a “sensitizing factor.” This sulfa-containing drug was previously implicated in the development of choroidal effusions and angle-closure glaucoma.⁵ Unlike in the described reports, however, our patient recovered by removal of the aqueous suppressants alone, and her hydrochlorothiazide was continued. Furthermore, there was no shallowing of the angles or change in refraction.

In summary, we describe a case of abrupt choroidal detachment occurring in a surgically untreated eye after only two doses of dorzolamide. Possible mechanisms include hypotony or an idiosyncratic reaction to dorzolamide, a sulfa-containing topical agent.

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Early Rapid Rise in Intraocular Pressure After Intravitreal Triamcinolone Acetonide Injection

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PURPOSE: To report the occurrences of early rapid increases in intraocular (IOP) after intravitreal glucocorticoid injection.

DESIGN: Observational case series.

METHODS: We retrospectively reviewed the records of three patients seen and treated at Duke Eye Center.

RESULTS: In all three cases, a significant rise in IOP occurred within 1 week of intravitreal triamcinolone injection for refractory macular edema. In one patient, a white material was found in the angle on gonioscopy. All three cases required surgical intervention to reduce the IOP.

CONCLUSIONS: Considering the early rapid rise in IOP in these three cases, we suggest that clinicians closely monitor patients after intravitreal triamcinolone injections for the development of acute glaucoma. Additionally, it may be advisable to perform gonioscopic examinations to look for any abnormal accumulation of material in the angle. (*Am J Ophthalmol* 2004;138:286–287. © 2004 by Elsevier Inc. All rights reserved.)

INTRAVITREAL CORTICOSTEROIDS HAVE BEEN USED IN the past for the treatment of specific intraocular proliferations¹ and have recently gained popularity for the treatment of a wide range of retinal diseases, including

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