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Case report

Acute hypotony maculopathy following the initiation of a topical aqueous suppressant in a patient with a history of panuveitis without prior filtering surgery

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ABSTRACT

Purpose: To report a case of profound hypotony maculopathy as a complication of single-agent glaucoma therapy in a patient with a history of panuveitis without previous filtering surgery.

Observations: A 70-year old Hispanic male with a history of resolved bilateral panuveitis, chronic angle closure glaucoma, and pars plana vitrectomy was started on topical timolol 0.5% daily in the left eye for mildly elevated intraocular pressure (15 mmHg). The patient returned 1.5 weeks later with new onset hypotony (1 mmHg), chorioretinal folds, and cystoid macular edema in the same eye without associated signs of inflammation. The drop was discontinued. The patient returned 1 month later with normalized eye pressure and improved vision with near-resolution of chorioretinal changes on optical coherence tomography (OCT).

Conclusions and importance: Hypotony maculopathy is most commonly seen following glaucoma filtering surgery and ocular trauma. The development of hypotony maculopathy following the administration of topical glaucoma medication alone is rare. Our case is the first to our knowledge to describe the rapid onset of visually significant hypotony maculopathy characterized by profound OCT changes upon the administration of a single topical glaucoma agent in a patient without prior filtering surgery. Treatment with glaucoma medications in patients with complex ocular histories including uveitis and vitreoretinal surgery requires caution and close follow-up.

1. Introduction

Hypotony maculopathy, a term coined by Don Gass[1] in 1972, was first described by Dellaporta[2] in 1954 and represents the development of posterior fundus abnormalities including chorioretinal folds, vascular tortuosity, disc edema, and rarely cystoid macular edema in the setting of low intraocular eye pressure (IOP). Pederson[3] described “statistical” hypotony as an IOP less than 6.5 mmHg (3 standard deviations below the mean), whereas “clinically significant” hypotony is defined by an IOP below which results in visual loss. The pathogenesis of hypotony maculopathy, in particular, is deemed likely secondary to inward collapse of the scleral wall resulting in wrinkling and folding, especially of the thick perifoveal retina around the thin fovea. The subsequent photoreceptor distortion and decreased antero-posterior diameter of the eye leads to decreased visual acuity and relative hyperopia.[1] Hypotony maculopathy is most frequently seen following glaucoma filtering surgeries especially with the concurrent use of anti-fibrotic agents as well as in perforating eye injuries.[4,5] Other risk factors include young age, myopia, male gender, systemic illness, and elevated preoperative IOP.[6]

2. Case report

A 70- year old Hispanic male with a history of rheumatoid arthritis, resolved bilateral panuveitis, and chronic angle closure glaucoma presented for his first visit to our clinic for a routine exam. Examination of the right eye was unremarkable, but the left eye IOP was slightly above target at 15 mmHg. There were no signs of intraocular inflammation, and he was not taking any eye drops or systemic antihypertensive medications at the time. The patient was started on timolol 0.5% daily in the left eye and returned 1.5

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weeks later with an IOP of 1 mmHg and decreased vision to 20/70 from 20/40 in the same eye. Fundus exam was notable for wrinkling of the macula as well as new onset cystoid macular edema on optical coherence tomography (OCT) (Fig. 1). There were no signs of choroidal detachment, retinal detachment, or optic nerve abnormalities. Timolol was discontinued and the patient returned one month later with subjectively improved vision (visual acuity 20/60) and an IOP of 18 mmHg. OCT demonstrated near resolution of chorioretinal folds and cystoid macular edema (Fig. 2).

A thorough review of his clinical record spanning over the past 12 years under the care of different providers revealed a history of similar repeated episodes of hypotony secondary to glaucoma medications. The patient initially presented in 2003 with visual acuity of 20/400 in the right eye and 20/200 in the left eye with elevated IOPs of 35 mmHg and 37 mmHg, respectively, secondary to bilateral panuveitis and acute on chronic angle closure glaucoma. Of note, both eyes were pseudophakic with a small degree of hyperopia (+0.50 spherical equivalent) and axial length around 22.5 diopters. His panuveitis was deemed mostly likely related to his underlying rheumatoid arthritis and was eventually controlled with bilateral pars plana vitrectomy, intravitreal fluocinolone acetonide implants, systemic immunosuppression with methotrexate, and chronic low-dose topical steroid treatment. IOPs improved to the mid-teens following bilateral laser peripheral iridotomies, and topical glaucoma medications were tapered and ultimately discontinued.

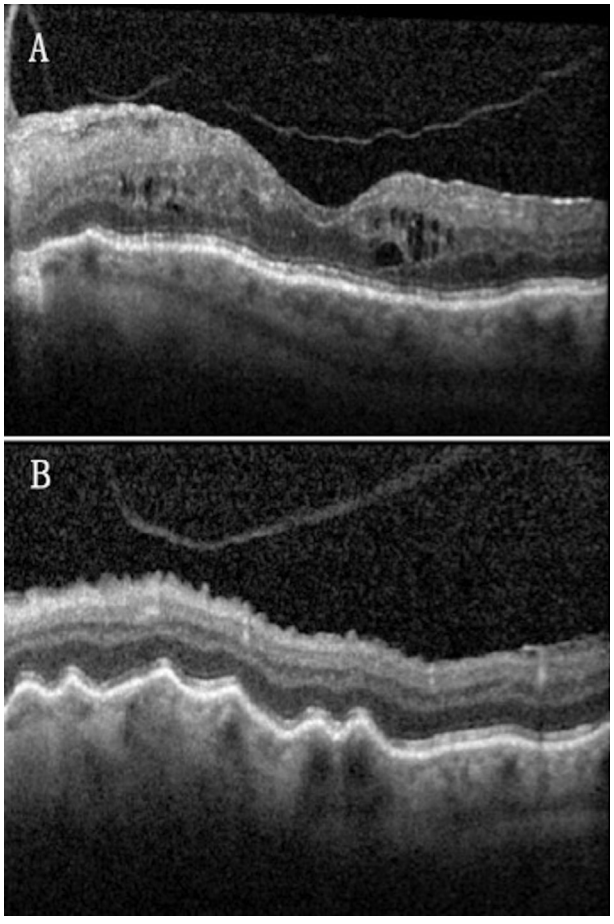

Fig. 1. Spectral domain-optical coherence tomography (SD-OCT) of the patient's central macula (A) and superior macula (B) demonstrating cystoid macular edema and extensive chorioretinal folds at 1.5 weeks following the initiation of timolol 0.5% daily in the left eye.

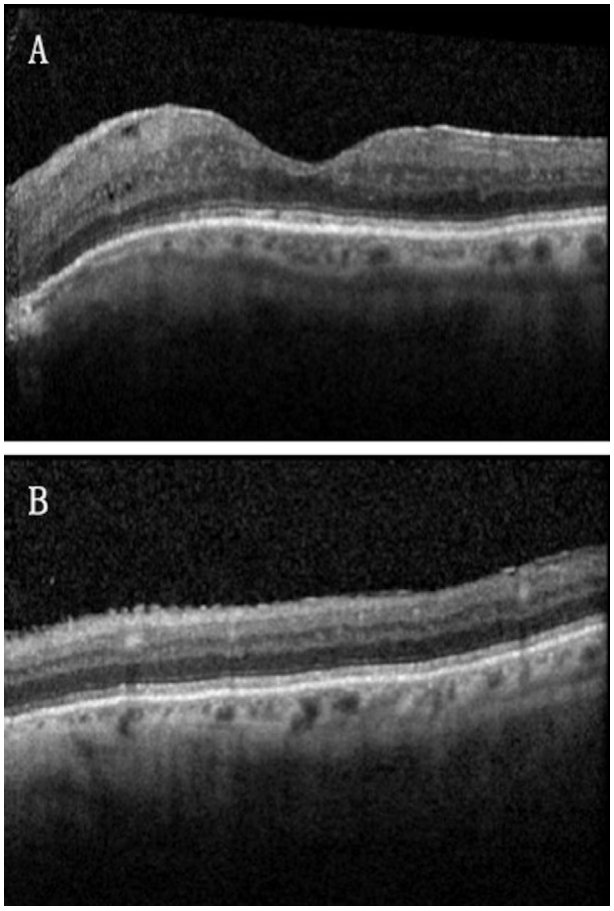

Fig. 2. Spectral domain-optical coherence tomography (SD-OCT) of the patient's central macula (A) and superior macula (B) demonstrating near resolution of cystoid macular edema and chorioretinal folds at 1 month following the cessation of timolol 0.5% daily in the left eye.

Nine years after his initial presentation, the patient was restarted on topical timolol 0.5% daily in the left eye for an elevated IOP of 22 mmHg at a routine visit. IOP of the right eye was acceptable at 11 mmHg. His next visit 3 months later revealed IOPs of 1 mmHg and 0 mmHg in the right and left eye, respectively. His vision remained unchanged at 20/60 and 20/50, and his fundus exam was normal with no intraocular inflammation noted. Gonioscopy was consistent with chronic angle closure. Timolol was discontinued, and the hypotony was treated with topical prednisolone acetate 1% three times daily in both eyes with improvement of his IOPs to 8 mmHg (right eye) and 16 mmHg (left eye) 1 month later. After a few years of relatively stable vision and IOPs on topical prednisolone acetate 1% twice daily, the patient presented with elevated IOPs of 18 mmHg and 31 mmHg in the right and left eye, respectively. He was treated with dorzolamide 2%/timolol 0.5% twice daily and latanoprost 0.005% nightly in both eyes. One month later, his IOPs had dropped to 2 mmHg and 1 mmHg, and the vision had decreased to 20/200 and 20/100 from his baseline of 20/60 and 20/40, respectively. Again no inflammation was seen, but macular striae were noted this time in both eyes. All glaucoma drops were again discontinued. He returned 3 months later with normalized IOP (9 mmHg and 15 mmHg), resolution of macular striae, and vision back to baseline.