Case report

A case of recurrent hypotony and choroidal effusion in a patient with previous filtration surgery

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1. Introduction

Hypotony is a known complication of glaucoma filtering surgery. Most cases result from an identifiable anatomic cause, such as a leaking bleb, over-filtration, inflammation, cyclodialysis cleft, or retinal detachment. Rarely, certain intraocular pressure (IOP)-lowering medications can be associated with hypotony.1-3 Here we report a case of recurrent hypotony with choroidal effusion thought to be induced by aqueous suppression therapy, in a patient with a history of pseudoexfoliation glaucoma and trabeculectomy surgery.

1.1. Case report

We present a case of a 70 year old male with advanced pseudoexfoliation glaucoma in both eyes with recurrent hypotony of the left eye following trabeculectomy. His surgical history was also significant for argon laser trabeculoplasty in both eyes, and failed trabeculectomy in the right eye, followed by Ahmed valve in the right eye. Other ocular history includes acute, recurrent bilateral anterior uveitis with negative lab work up, complex cataract extraction and anterior vitrectomy with sulcus intraocular lens placement in the right eye and uncomplicated cataract extraction with posterior chamber intraocular lens placement in the left eye.

Historically, the IOP in the right eye was well controlled without drops following trabeculectomy. However, his postoperative course following trabeculectomy in the left eye was complicated by hypotony with choroidal effusion due to overfiltrating bleb. He underwent revision of the trabeculectomy with compression sutures with resolution of the hypotony. IOP following bleb revision increased to 24 mmHg in the left eye, therefore brimonidine twice daily (BID) and later dorzolamide-timolol BID were added for better IOP control in this eye.

For the next three months, IOP in the left eye was well controlled between 8 and 11 mm Hg with brimonidine, and dorzolamide-timolol. At his four month follow up appointment, IOP was found to be 3 mm Hg and careful history revealed that the patient was using latanoprost despite prior instructions. On examination, vision was stable at 20/40 in the right eye and 20/50 in the left eye, and IOP in the right eye was 8 mm Hg. Slit lamp biomicroscopy demonstrated flat, seidel negative bleb with intact compression sutures. Anterior chamber was deep and quiet, and no choroidal effusion was present. There was no cyclodialysis cleft, anterior chamber reaction or retinal detachment. All IOP medications were stopped. He remained hypotensive without choroidal effusion for 1.5 months with confirmed adherence to medication discontinuation. The patient was then lost to follow up for six months. He re-presented with stable vision and an IOP of 9 mm Hg in the right eye and 23 mm Hg in the left eye off of all drops. Dorzolamide-timolol, and brimonidine were again restarted in the left eye at that time, and latanoprost was restarted 4 months later as IOP remained in the low twenties.

After 6 months of good IOP control on medications, the left eye...
became hypotonous once again with an IOP of 2 mm Hg. IOP in the right eye was stable at 15 mm Hg. On exam, visual acuity was stable, anterior chamber was deep and quiet, no corneal folds were present, and the bleb was flat and seidel negative. Choroidal effusions were seen on exam and confirmed with b-scan ultrasound. Once again, all drops were discontinued and hypotony resolved after 8 months. At this time, IOP was back in the mid-teens, therefore, latanoprost was initially restarted followed by both brimonidine and dorzolamide-timolol 5 weeks later.

Within 1 month of restarting dorzolamide-timolol and brimonidine, IOP in the left eye decreased to 3 mmHg and choroidal effusions were again noted. Visual acuity was 20/50. Hypotony improved and choroidal effusions resolved over two weeks with discontinuation of all of the IOP lowering medications. With each episode of hypotony, the bleb was flat without signs of over-filtration, leakage on seidel testing, or inflammation. There was also no evidence of cyclodialysis on gonioscopy or cell in the anterior chamber. Additionally, on the third presentation, ultrasound biomicroscopy was used to confirm absence of a cyclodialysis cleft. At this point, after 3 episodes of hypotony, with resolution after discontinuation of IOP lowering agents, and an otherwise unchanged clinical exam, hypotony and choroidal effusion due to aqueous suppression therapy was suspected. Therefore, only latanoprost was restarted in the left eye once IOP increased to 19 mm Hg. After 5 months of follow up, the vision improved to 20/50 in the right eye, 20/40 in the left eye, and IOP was stable at 14 mm Hg in the right eye and 13 mm Hg in the left eye while on latanoprost in both eyes. Pilocarpine 1% was later added in the left eye for additional IOP control, and aqueous suppressants were avoided. Refer to Table 1 for a summary for each episode of hypotony in the left eye.

Table 1 is showing each episode of hypotony in the left eye with the corresponding visual acuity and eye drops used followed by each intraocular pressure peak in the left eye with corresponding visual acuity and eye drops used to demonstrate the pattern of aqueous suppression.

2. Discussion

The patient’s recurrent hypotony of the left eye was thought to be due to the use of aqueous suppressant medications in the setting of dorzolamide-timolol use. According to Vela and Campbell, the first to describe this syndrome, the ciliary body beta-receptors become hypersensitized by the combined effect of long-term timolol treatment and filtration surgery with a period of postoperative hypotony. Therefore, restarting pharmacologic aqueous suppressants, specifically timolol, dorzolamide, and/or acetazolamide can lead to a more significant decrease in aqueous production from the sensitized ciliary epithelium resulting in hypotony. Hypotony and choroidal effusion or detachments are not permanent and resolve with medication discontinuation, which allows resumption of normal aqueous production. Sharma and colleagues and Callahan and colleagues have also shown hypotony and choroidal detachment with timolol and dorzolamide following trabeculectomy, with complete resolution when medications were discontinued. Another case report by Zarnowski et al. showed development of recurrent choroidal detachment soon after timolol was started to reduce IOP. These reports also demonstrated recurrence of hypotony on rechallenge with the same aqueous suppressant.

Our case further supports the literature on this topic, but differs by describing a patient with pseudoxefoliation glaucoma instead of primary open angle glaucoma. Additionally, our patient demonstrated similar findings with more rechallenges of aqueous suppressants than patients in previously published work. Latanoprost has more recently been reported in cases of hypotony and choroidal detachment following glaucoma filtering surgery, possibly due to a much greater increase in uveo-scleral outflow in these patients.

3. Conclusions

Hypotony with choroidal effusion induced by aqueous suppression therapy is rare, but should be considered in patients with hypotony of unclear etiology following a glaucoma filtering procedure. Aqueous suppressants should be discontinued and it is recommended that the glaucoma drop regimen be switched to non-aqueous suppressants in patients with these findings.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.ajoc.2018.05.004.

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