Development of late "in-the-bag" hyphema and UGH plus syndrome in a patient nine years post-op.

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Received: 10-February-2020, Manuscript No. OER-20-7179; **Editor assigned:** 13-February-2020, PreQC No. OER-20-7179; **Reviewed:** 27-February-2020, QC No. OER-20-7179; **Revised:** 08-August-2022, QI No. OER-20-7179, Manuscript No. OER-20-7179; **Published:** 05-September-2022, DOI: 10.35841/oer-6.6.137

Abstract

Uveitis-Glaucoma-Hyphema Syndrome (UGH Syndrome) is a rare complication of cataract surgery, typically presenting as blurry vision in the weeks to months post-op. Symptoms may include elevated Intra-Ocular Pressure (IOP), uveitis, and recurrent hyphema. We present a case of an unusual manifestation of UGH Syndrome with bleeding into the posterior capsule after an uneventful cataract surgery nine years prior.

Keywords: Uveitis-glaucoma-hyphema syndrome, Hyphema, Cataract surgery, Algae.

Introduction

Uveitis-Glaucoma-Hyphema Syndrome (UGHS) is a rare complication of cataract surgery, usually presenting as blurry vision in the weeks to months post-op. Symptoms may include elevated Intra-Ocular pressure (IOP), uveitis, and recurrent hyphema, in addition to pain and photophobial. A rare form of this complication is Incomplete Posterior UGH syndrome (IPUGH syndrome), which presents with hyphema in the posterior chamber on clinical examination, with or without elevated IOP, and no anterior inflammation. The addition of vitreous hemorrhage to these other signs is classified as UGH Plus syndrome. We present a case of IPUGH syndrome with acute bleeding into the posterior capsule and glaucoma after an uneventful cataract surgery nine years prior with progression to UGH Plus syndrome.

Case Presentation

A 68-year-old male presented to the ED at Bay Pines VA for evaluation of worsening blurry vision with a temporal shadow in his pseudophakic right eye onset one month prior. He otherwise denied pain, trauma, or any other concerns at that time. The patient was evaluated by optometry and ultimately referred to ophthalmology. His history included a previous cataract extraction in the right eye with Intra-Ocular Lens (IOL) placement in 2010 and myopia in the right eye prior to his surgery, in addition to several systemic conditions. Previous diagnoses include diabetes mellitus, obstructive sleep apnea, chronic obstructive lung disease, Hepatitis C, essential hypertension, coronary artery disease, aortic valve stenosis, and moderate stenosis of the carotid arteries. The patient is on seventeen medications to control these chronic conditions. Allergies were reported to penicillin and chlorpromazine [1].

The patient's uncorrected visual acuity was 20/80+2 OD and 20/20-2 OS. The patient's vision OD improved to 20/70 with pinhole. His intraocular pressures measured 30 OD and 18 OS *via* Tono-Pen. On examination of the right eye, the patient had

a posterior chamber IOL in place with bright red heme in the capsule posterior to the optic bisecting the pupil. There was evidence of heme staining the superior anterior capsule. The IOL was found to be fixed with no excessive mobility. Neovascularization of the iris was not present. On gonioscopy, there was presence of heme in the inferior angle with appearance of neovascularization of the angle underlying the heme. The patient also had evidence of pseudo-exfoliation syndrome of the affected eye. Dilated fundus examination and B-Scan of the right eye were negative for non-proliferative diabetic retinopathy, vitreous heme, retinal detachment, or mass [2].

Results

Carotid duplex study was ordered and found to be within normal limits. His diabetes was well-controlled at HbA1c of 6.0. The patient was placed on prednisone forte QID, dorzolamide BID, and brimonidine BID OD and advised to sleep with the head of the bed elevated to prevent the heme from staining the capsule (Table 1).

Table 1. Daily intake of drugs and its dosage intake by the patients.

Dosage form	Dose
Prednisone forte	QID (Daily four times)
Dorzolamide	BID (Daily two times)
Brimonidine	BID (Daily two times)

The patient was followed weekly to observe the progression of the hyphema and glaucoma. His one-week follow up revealed improved uncorrected visual acuity at 20/40 OD and 20/20 OS and intraocular pressures at 16 OD and 14 OS, though no significant improvement of hyphema on exam. At two weeks, the patient was found to have resolution of the hyphema with remaining heme and trace hemosiderin present at the inferior border of the pupil margin. No clotting of the heme was noted. Gonioscopy revealed pigment clumping and a fibrous band in the inferior angle with no gross appearance of

neovascularization of the angle. No obvious source for heme was appreciated. Anterior segment Optical Coherence Tomography (OCT) showed no indication of posterior iris or ciliary body mass. The patient's treatment plan was adjusted by decreasing prednisone forte to TID. Potential etiologies considered in the patient's workup included the following: pseudoexfoliation, UGH syndrome, neovascularization of the trabecular meshwork or of the anterior chamber angle, and infection. The diagnosis was determined to be IPUGH syndrome [3-5].

The patient returned three weeks later for re-evaluation. He reported subjective worsening in OD vision from the prior visit. However, his OD vision improved to 20/30 with an IOP of 14 OD and 16 OS. His prednisone forte was tapered down to BID. On slit lamp examination, the posterior chamber intraocular lens was visualized with bright red heme and trace hemosiderin as previously noted. There was heme staining of the superior anterior capsule. No pseudophakodonesis or shifting of heme on positional change was noted. The posterior capsule was unable to be visualized, which prevented use of a YAG laser to relieve the heme. On B-Scan, vitreous heme was present. The patient was advised to discontinue his Plavix and continue follow-up in two weeks. In his last visit at the clinic, the patient seemingly had progressed to UGH Plus syndromesigns of UGH syndrome in addition to vitreous hemorrhage. The hyphema in the posterior capsule with vitreous hemorrhage provided no view to the retina on exam. It was decided that the patient proceed with an anterior approach to blood extraction, in addition to an intraocular lens exchange if the capsule and zonular support were unable to withstand the manipulation. The patient was lost to follow-up after this visit. Attempts were made to contact him, but it is unclear if this was due to improvement of symptoms and thereby the perception that there was no need to return for evaluation or to other barriers that prohibited him from returning [6-10].

Discussion

In the case described, the patient presented with one month of worsening vision with a temporal shadow and was ultimately diagnosed with the rare complication post cataract surgery of incomplete posterior UGH syndrome. This was determined by the evidence of bleeding into the posterior capsule of the eye and elevated IOP on examination without anterior inflammation. This then progressed to UGH Plus syndrome with the development of vitreous haemorrhage [11-13].

There are currently two proposed mechanisms of "in-the-bag" UGH syndrome. Firstly, it is believed that posterior iris chafing may occur in patients with pseudoexfoliation syndrome *via* "subclinical phacodonesis of the haptic-capsule complex".3 Secondly, it is possible that focal capsular fibrosis creates areas of chafing on the posterior iris.4 Furthermore, there has been increasing awareness that "late in-the-bag single piece IOL dislocation [can occur] with incidence rates varying from 0.1 to 3.0%" with risk factors of pseudoexfoliation syndrome, high myopia, connective tissue disorders, and history of vitreoretinal surgery [14].

While no obvious mobility of the patient's IOL was evident on his exam, the mechanism of subclinical The patient additionally has two of the described risk factors for late inthe-bag IOL dislocation: A history of myopia in the affected eye as well as pseudoexfoliation syndrome [15].

Treatment of UGH syndrome typically involves management of glaucoma *via* aqueous suppressants, mydriasis drops, and corticosteroid drops for pain and inflammation relief. For the hyphema, management is based on measuring level of hyphema with consideration of IOP. Depending on severity and presentation, anterior capsule washout may be performed for prevention of corneal blood staining.

Conclusion

In conclusion, it is important to recognize UGH syndrome as a potential late complication of cataract surgery. This case reiterates the importance of a careful slit-lamp examination in post-operative patients to look for signs of posterior iris rubbing, pigment dispersion, anterior chamber inflammation, and elevation of IOP regardless of the time following the surgery. UGH syndrome can lead to many of its own complications, including corneal staining, optic nerve damage, and chronic inflammation. For this reason, appreciation of its signs and management course are crucial.

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