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Hypopyon following selective laser trabeculoplasty

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ABSTRACT

Purpose: To report a hypopyon following selective laser trabeculoplasty (SLT).

Observations: An 85-year-old woman with primary open-angle glaucoma underwent routine SLT. In the early post-procedural period, she presented with pain and decreased vision, and she was found to have hypopyon, trabeculitis, and corneal edema. The patient was treated with prednisolone acetate and empirically with valacyclovir due to the possibility of herpetic keratouveitis. Work-up for potential etiologies was unrevealing. Her symptoms resolved with treatment, and at eight months follow-up her visual acuity and intraocular pressure had stabilized to her baseline.

Conclusions: Though safe, SLT may be associated with rare adverse events requiring intervention. Hypopyon following SLT is extremely rare, and investigation for causes unrelated to the history of SLT should be undertaken as appropriate.

Importance: To the best of our knowledge, this is the first report of a hypopyon following SLT in a patient with no history of inflammatory intra-ocular disease.

1. Introduction

Selective laser trabeculoplasty (SLT) was approved by the FDA in 2001 to lower intraocular pressure (IOP) in patients with open-angle glaucoma (OAG).[1] SLT has been shown to be an effective first-line treatment of OAG and is generally considered well-tolerated with few significant adverse events (AEs).[1,2] Herein, we present the case of a patient with OAG treated with SLT who developed hypopyon, trabeculitis, and corneal edema in the early post-procedural period.

2. Case report

An 85-year-old woman with a history of primary open-angle glaucoma (POAG) was referred to our practice for further management of her intraocular pressure (IOP). Her past ocular history was notable for uncomplicated bilateral cataract extraction with in-the-bag posterior chamber lens implantation, severe dry eye disease, epithelial basement membrane dystrophy (EBMD), and bilateral dry age-related macular degeneration (AMD) with subfoveal geographic atrophy. Her past medical history included treated sinonasal carcinoma, asthma, and chronic obstructive pulmonary disease (COPD). She denied prior history of uveitis or systemic inflammation. She did report a possible history of herpetic labialis. Her review of systems was unremarkable for rheumatologic or inflammatory symptoms.

On initial examination, best-corrected visual acuity (BCVA) was 20/150 OD and 20/200 OS. The IOP was 17 mm Hg OU on bimatoprost 0.01% nightly OU and dorzolamide 2% three times daily OD. External examination revealed severe dorzolamide-related dermatitis of the lids OD. Slit lamp examination was remarkable for EBMD, trace haze in the corneal stroma OU, and posterior chamber intraocular lens implants OU, without plaque or deposit. Fundus examination revealed a cup-to-disc ratio of 0.75 OD and 0.45 OS, as well as bilateral geographic macular degeneration. Humphrey visual field testing showed generalized depression OU consistent with large areas of subfoveal geographic atrophy. Optical coherence tomography (OCT) of the retinal nerve fiber layer (RNFL) revealed glaucomatous cupping and inferior RNFL loss OD.

Review of prior records demonstrated a maximum IOP of 32 mm Hg OD. By report of the referring provider, serial OCTs had revealed progressive RNFL thinning despite maximally-tolerated topical therapy OD; the patient developed somnolence on brimonidine, and her severe COPD limited use of beta blockers. Thus, she was recommended to undergo selective laser trabeculoplasty (SLT) OD with a target IOP of 14 mm Hg.

On day six following unremarkable SLT (95 spots, 1 mJ/spot, 360°), the patient presented with worsening ocular pain and vision loss. Vision

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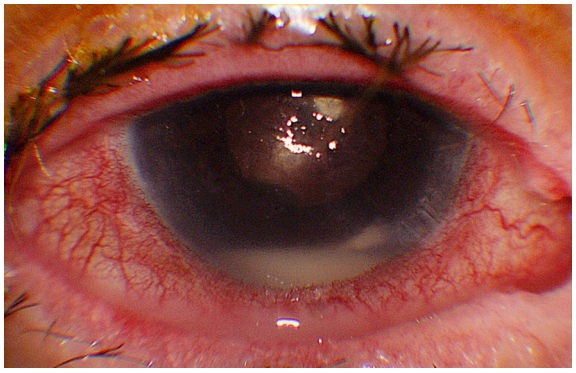

Fig. 1. External photograph of the right eye demonstrating marked conjunctival hyperemia and a residual hypopyon following anterior chamber paracentesis.

was hand motion with an IOP of 32 mm Hg OD. Examination revealed a 2 × 2 mm central epithelial defect with no corneal infiltrate, corneal edema, fibrinous material at the pupillary border, and a layered hypopyon OD (Fig. 1). Though the view of the posterior segment was limited, ultrasonography showed an attached retina without evidence of vitritis. The IOP was reduced to 25 mm Hg with topical therapy. She was discharged on cyclopentolate 1% twice daily, prednisolone acetate 1% hourly while awake, and moxifloxacin 0.5% six times daily as prophylaxis against bacterial keratitis.

The following day, she underwent anterior chamber paracentesis, and the aqueous fluid was found to be negative for varicella-zoster virus and herpes simplex virus 1 and 2 by polymerase chain reaction (PCR). Nonetheless, valacyclovir 1 gram three times daily was added due to the possibility of herpetic keratouveitis. Uveitis workup was initiated, including a complete blood count, blood culture, varicella-zoster virus serum and aqueous PCR, herpes simplex virus 1 and 2 aqueous PCR, angiotensin converting enzyme, lysozyme, Quantiferon-TB Gold, rapid plasminogen reagin, and perinuclear and cytoplasmic anti-neutrophil cytoplasmic antibodies, all of which were negative. She was found to have a positive 1:160 anti-nuclear antibody with a diffuse pattern. Human leukocyte antigen B27 (HLA-B27) was sent but was not resulted due to laboratory error. Due to concern for endogenous endophthalmitis, peripheral blood cultures were sent and were negative.

On day nine, the hypopyon had resolved, at which point a prednisolone acetate taper was initiated. At eight months follow up, uncorrected visual acuity measured 20/250 and IOP was 20 mm Hg on bimatoprost and dorzolamide.

3. Discussion

SLT has been shown to be an effective first-line treatment of OAG and is generally well-tolerated with few significant adverse events.[1,2] Though incompletely understood, SLT is thought to increase aqueous outflow through remodeling of the trabecular meshwork. Resultant cytokine production recruits macrophages and inflammatory mediators to the anterior chamber and trabecular outflow apparatus.[3] Thus, it is not surprising that anterior chamber inflammation is often observed clinically following SLT, though it tends to be mild and transient.[2,3]

The cause of this patient's observed anterior uveitis with hypopyon following SLT remains unknown. Given her history of possible herpetic labialis and the clinical findings of corneal edema, hypopyon, and trabeculitis, herpetic keratouveitis is highest on the differential diagnosis. Her use of a topical prostaglandin analog may be considered a risk factor in the pathogenesis of this disease, as prostaglandin analogs have been associated with HSV reactivation.[4] Although the sensitivity of aqueous PCR in infectious posterior uveitis and endophthalmitis has been reported to be high, the sensitivity of PCR in aqueous of patients with anterior uveitis from HSV is unknown.[5,6] The absence of viral detection by PCR analysis of aqueous does not exclude a herpetic etiology.[7-9] In rare instances, herpes reactivation following SLT has been reported. Clinicians should be aware of this possibility, which may inform selection and monitoring of patients undergoing SLT in the future.[7,10]

In addition to anterior chamber inflammation, the most common adverse events following SLT are IOP elevation, blurred vision, and discomfort.[2,3] Although rare, more serious adverse events have been reported, including the following: severe IOP spikes, hyphema, cystoid macular edema, foveal burns, and corneal edema, including eight cases of SLT-induced keratitis with hyperopic shift.[3,11]

4. Conclusions

To the best of our knowledge, this is the first report of hypopyon following SLT in a patient with no prior history of uveitic or inflammatory disease. Though SLT is widely considered a safe procedure, awareness of possible vision-threatening complications is important for patient selection and counseling.

Patient Consent

Consent to publish these case details was not obtained from the patient, as this case report does not contain details that would allow the identity of the patient to be determined.

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Intellectual property

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

Research ethics

We further confirm that any aspect of the work covered in this manuscript that has involved human patients has been conducted with the ethical approval of all relevant bodies and that such approvals are acknowledged within the manuscript.

Declaration of competing interest

No conflict of interest exists.

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All authors attest that they meet the current ICMJE criteria for Authorship.

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