DJCRO
DUKE JOURNAL OF CASE REPORTS IN OPHTHALMOLOGY

Duke Ophthalmology
Duke University School of Medicine

Delayed Onset Corneal Edema Following Selective Laser Trabeculoplasty

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DOI: 10.62856/djcro.v6.41

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Introduction

Selective laser trabeculoplasty (SLT) is a common procedure for reducing intraocular pressure (IOP) in patients with primary open angle glaucoma (POAG). The procedure involves delivering spots to the trabecular meshwork using a low, 532 nm frequency-doubled, Q-switched Nd:YAG laser.[1] SLT has become a more widely used treatment modality for POAG compared to argon laser trabeculoplasty due to its precision, which limits damage to surrounding tissues and has reduced complication rates.[2] Typically, the side effects of SLT are mild and resolve quickly with minimal or no intervention.

The most common side effects include anterior chamber inflammation, ocular pain or discomfort, transient conjunctival redness of the treated eye, and IOP elevation.[3] Corneal edema is a much more rarely reported complication of SLT.[4-9] This report describes a case of delayed onset corneal edema with subacute IOP elevation following SLT.

Case Report

A 64-year-old female was referred for glaucoma evaluation due to asymmetric optic nerve cupping and progressive thinning of the right optic disc on ocular coherence tomography (OCT) imaging. The patient did not have a history of ocular infections or inflammation and had not undergone any prior ophthalmic laser or surgical procedures. Refraction showed -2.0 diopter (D) spherical equivalent in the right eye and -1.0 D spherical equivalent in the left eye. On initial evaluation, the IOP was 20 mm Hg in the right eye and 18 mm Hg in the left eye. Prior records showed a maximum IOP of 26 mm Hg in the right eye and 20 mm Hg in the left eye. Pachymetry measurements found a central corneal thickness of 501 microns in the right eye and 503 microns in the left eye. Gonioscopy was open to scleral spur 360 degrees bilaterally with moderate pigmentation. Best

corrected visual acuity was 20/20 and 20/25 in the right and left eyes, respectively. There was increased cupping of the right optic disc compared to the left. OCT imaging showed inferotemporal thinning in the right eye which had progressed over the prior year. Humphrey visual field 24-2 testing showed no visual field deficits in either eye.

A diagnosis of mild stage primary open angle glaucoma of the right eye was made. SLT was performed in the right eye with an energy setting of 0.8 mJ using a 400 micron spot size. The patient was pre-treated with apraclonidine 0.5% solution 15 minutes prior to the procedure. In total, 105 spots were applied, and 360 degrees of trabecular meshwork was treated. The IOP was re-checked 30 minutes after the procedure and was unchanged from pre-treatment levels. No post-SLT eye drops were used.

Within 24 hours, the patient presented urgently with aching pain, photophobia, and difficulty opening the right eye. The anterior chamber was deep without evidence of pupillary block or acute angle closure. Examination showed conjunctival injection and diffuse microcystic corneal edema. Visual acuity was 20/60 in the right eye and IOP was 42 mm Hg. The IOP was consistently measured with either applanation or rebound tonometry. The patient was given acetazolamide 500mg orally and timolol maleate 0.5% and dorzolamide 2% eye drops. After 30 minutes, IOP improved to 19 mm Hg. The patient was sent home on brinzolamide/brimonidine tartrate 1%/0.2% (Simbrinza ®, Alcon) twice a day in the right eye and acetazolamide 500mg by mouth twice a day.

The following day, IOP was 15 mm Hg. Visual acuity had decreased to CF at 1 foot. Slit lamp examination showed resolution of the microcystic edema but diffuse stromal edema with prominent Descemet's folds. Topical prednisolone acetate 1% was started 4 times per day in the right eye. Two days later, the IOP decreased to 4 mm Hg in the right eye and visual acuity was 20/100. The low IOP measurement was attributed to presence of stromal edema rather than true hypotony. Acetazolamide was stopped and the prednisolone reduced to 2 times per day. Three days later (now about one week following SLT), IOP was 20 mm Hg, and visual acuity was 20/30 with mild stromal edema. Simbrinza was stopped and prednisolone was tapered in a stepwise fashion over the following 3 weeks. One month later, IOP was 16 mm Hg, and corneal edema resolved. Five months following SLT, the patient was off eye drops, IOP was 12 mm Hg, and visual acuity was 20/25.

Discussion

Corneal edema may develop following uneventful SLT. This case demonstrated a delayed onset of stromal edema that followed resolution of a subacute elevation in IOP. While the etiology of post-SLT corneal edema is not fully known, several possible causes have been suggested in the literature, including direct injury to the cornea, reactivation of latent Herpes simplex infection, or prominent anterior chamber inflammation.[9]

Herpetic reactivation was considered an underlying etiology in several reported cases.[7,8] Liu et al. reported three cases of postoperative edema successfully treated with oral antiviral therapy and topical steroids.[7] In one of those cases, endothelial deposits were present. Notably, none of the three cases had reported a prior history of recurrent eye infections or known herpetic disease. Chadha et al. reported two cases of corneal edema following SLT that both demonstrated epithelial keratitis (one with a dendritic lesion and the other with suspicious epithelial irregularity) and reduced corneal sensation.[8] Neither case demonstrated significant IOP elevation temporally associated with corneal edema.[7,8] Although prior reports of corneal edema suspected to be herpetic in etiology did not always present with a herpetic history, the lack of dendritic lesions and the rapid clearance of epithelial involvement makes this etiology less likely in our case.

Prior corneal endothelial damage has also been suggested as a possible trigger for post-operative edema.[9] Ozkok et al. described a case of corneal edema in a 64-year-old male with prior history of cataract and trabeculectomy

surgeries in the treated eye.[9] Corneal edema was noted two weeks after the SLT procedure, although the patient reported decreased visual acuity after approximately one week. This delayed onset is atypical compared to other reports.[4,5] The severity of edema was also atypical with a poor response to topical steroids and eventual progression to bullous keratopathy. Trabeculectomy surgery is known to cause corneal endothelial cell loss and may predispose to endothelial cell dysfunction.[10]

In our case, we suspect that a robust inflammatory response is the most likely etiology for the prolonged corneal edema. Our patient did not present with any other identifiable risk factors such as prior laser treatments or surgeries, corneal disease, herpetic infection, or uveitis. However, our case was notable for a relatively higher level of energy applied during the SLT. Guzey et al. has described an increase in free oxygen radicals in the aqueous humor of rabbits following SLT, which may be associated with corneal endothelial damage.[11] If sufficient, this inflammatory impact on the corneal endothelium may explain the corneal edema noted in our case. Paiva et. al. also noted that the interaction of the laser on the trabecular meshwork increases the release of cytokines and matrix metalloproteinases, and the degree of this response seems associated with the power level of laser energy applied.[12]

We suspect that the initial microcystic edema in our patient is consistent with the early IOP elevation; however the prolonged stromal edema persisting well beyond resolution of the IOP spike likely suggests a more ongoing inflammatory process. While IOP elevation can lead to the development of corneal edema, its initial presentation typically involves significant epithelial edema rather than stromal edema.[13] In a patient without other identifiable risk factors, we suggest that corneal edema following SLT is a rare but possible inflammatory-mediated response to higher energy SLT procedures.

Conclusion

Post-SLT corneal edema can occur in patients without any significant predisposing risk factors. Stromal edema can manifest a delayed onset and persist after normalization of IOP. Most cases resolve with topical steroid treatment, and long-term visual consequences are rare. While uncommon, ophthalmologists should be cognizant of this potential complication in the early post-SLT period.

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Statement of Ethics

This case report adheres to patient confidentiality and ethical principles in accordance with the guidelines of the Declaration of Helsinki and relevant local regulations. Consent was obtained from the patient for the publication of this case report.

Conflict of Interest Statement

The authors declare no conflicts of interest related to this topic.

Funding

This work received no funding or grant support.

Authorship

We attest that all authors contributed significantly to the creation of this manuscript, each having fulfilled the criteria as established by the ICMJE.