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Case report

Rare corneal complication following selective laser trabeculoplasty

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ABSTRACT

Purpose: This case report describes two glaucoma patients who underwent selective laser trabeculoplasty (SLT) and developed a rare post-procedure corneal complication and subsequent permanent corneal distortions and reduced acuity.

Observations: Both patients experienced early post-procedure corneal edema, reduced visual acuity, and pain. Each patient was treated with topical steroids with resolution of the edema, but each patient had irregular corneal astigmatism which was not present pre-operatively and did not resolve.

Conclusions and importance: There are very few reported cases of post-SLT corneal edema and these typically resolve without long-term complications. The following two cases represent an uncommon adverse event of irregular corneal astigmatism despite resolution of the corneal edema. The exact mechanism of these corneal effects is not known. Practitioners should inform patients of this rare, but possible complication following SLT and consider extra precautions in patients susceptible to corneal edema.

1. Introduction

Selective laser trabeculoplasty (SLT) was approved in 2001 by the US Food and Drug Administration as a treatment for elevated intraocular pressure (IOP). SLT uses a 532nm frequency-doubled, Q-switched Nd:YAG laser to treat the trabecular meshwork (TM) with a gonioscopic lens. The mechanism by which SLT lowers IOP is not completely understood.[1] The favored biological theory proposes that SLT works by increasing macrophage activity and inflammatory cytokines that remodel the extracellular matrix of the TM resulting in increased outflow.[1,2] The IOP lowering effect is similar to argon laser trabeculoplasty (ALT), but theoretically causes less collateral damage to the TM and Schlemm's canal as SLT uses only 1% of the energy compared to ALT.[3] Thus, SLT is generally considered more safe than ALT and is equally as effective. Post-operative SLT side effects include acute IOP spike, anterior segment inflammation, redness, and pain.[1,4] These side effects are infrequent, generally mild in severity, transient, and typically resolve within a few days without treatment. The following two cases describe an atypical corneal complication of SLT.

2. Findings

2.1. Case 1

A 69 year old Caucasian male presented with difficulty in reading in his left eye despite cataract surgery and YAG capsulotomy. His past medical history was significant for colon, bladder and testicular cancer, benign prostatic hyperplasia, and hearing loss. He had a positive family history of glaucoma in his mother and maternal grandfather.

His examination revealed best corrected visual acuity of 20/15 in the right eye and 20/25- in the left. Prior to cataract surgery, he had moderately high myopia (−6.00D right eye and −5.00D left eye). Post-operatively he was plano in the right eye and low myopia with astigmatism in the left eye. His intraocular pressures were 19 mmHg in the right eye and 22 mmHg in the left eye. His pupils revealed a mild afferent pupillary defect in the left eye. Pachymetry readings were 542 μm/544μm in right and left eyes, respectively. Gonioscopy was open to the ciliary body band in both eyes. His anterior segment findings showed bilateral clear corneas. The vertical cup:disc ratio of the optic nerve was 0.65 in the right eye and 0.85 in the left with a thin inferior rim.

Additional testing with the Humphrey visual field in his right eye showed good reliability and a few scattered points that did not match his optic nerve or retinal nerve fiber layer imaging. His left eye was reliable and showed a superior retinal nerve fiber layer type defect with a central defect, and a superior nasal step. The left eye field was repeated and these defects were confirmed. Optic nerve imaging with Spectralis OCT showed that his left optic nerve demonstrated borderline thinning of the inferior retinal nerve fiber layer, which corresponded with the visual field defect and the clinical appearance of the nerve.

Given these clinical findings and test results, the patient was diagnosed with normal tension glaucoma, mild stage in the right eye and

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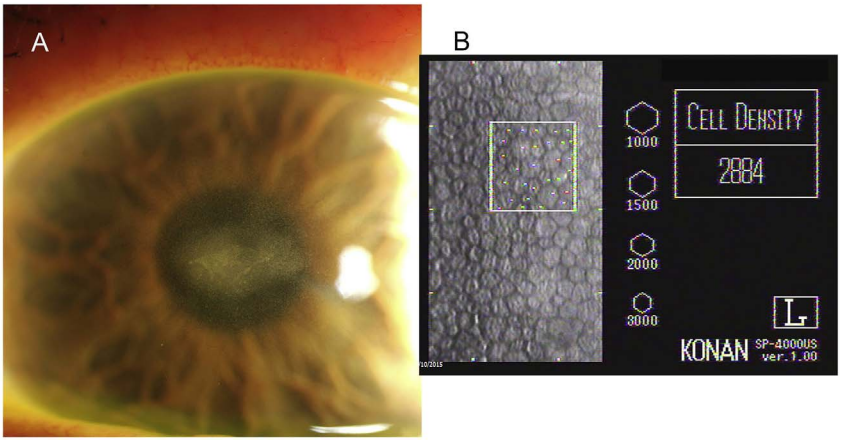

Fig. 1. A: Anterior segment image of the OS showing dense, central corneal edema. B: Specular microscopy of the OS showing normal morphology and number.

severe stage in the left. To reach the target IOP, the patient elected to have SLT in the left eye that same day. Proparacaine topical anesthetic was instilled, Goniovisc™ (HUB pharmaceuticals) was used with the gonioscopy lens, and SLT was performed with 100 spots treating 360° of the trabecular meshwork with 0.7 mJ, which falls within the typical number of spots and typical power range of 0.4–1.4 mJ[1]. Iopidine (aproclonidine) was instilled in the left eye before and after the procedure. Thirty minutes after the procedure, the cornea was clear and the IOP was 22 mmHg. The patient was instructed to use ketorolac four times a day for four days and to return in a week for a follow-up.

The patient returned on an urgent basis three days later with complaints of blurred vision, discomfort, tearing, and redness in the left eye which began at one day post-op. His IOP was 17 mmHg and acuity was reduced to 20/300 due to dense central stromal corneal edema (Fig. 1A) with an intact epithelium. View of the anterior chamber was difficult due to the corneal haze but mild cell was noted without presence of hypopyon. He was started on topical prednisolone four times a day and ketorolac was discontinued. Over the next three months, the corneal edema resolved without corneal scarring, but his vision remained at 20/100. During this period, he had an initial hyperopic shift and increase in astigmatism that ultimately shifted to be less hyperopic but the astigmatism remained.

At five month post-operative, he was referred to the cornea clinic due to persistent poor vision where his pachymetry readings were repeated which were stable and symmetric in both eyes. Specular microscopy and topography testing were performed and revealed normal endothelial cell density (Fig. 1B) and irregular astigmatism with steepening superiorly in his left eye (Fig. 2). At the 7 month visit, his vision remained at 20/80 and he was referred to optometry for a rigid gas permeable (RGP) lens fitting. Topography was repeated and was essentially stable. The refraction revealed 20/60 best corrected vision with a 1.25 diopter increase in regular astigmatism compared to baseline. With an RGP, he was able to achieve 20/30, which was essentially his baseline acuity before the SLT and was likely limited due to the central field loss from glaucoma.

2.2. Case 2

A 60 year old Caucasian female with untreated, moderate stage primary open-angle glaucoma and no family history of glaucoma was examined at Kellogg Eye Center. Her past medical history was significant for hypothyroidism treated with levoxyl. Her vision was 20/20 in both eyes with high myopia. Her vertical cup:disc ratio was 0.7 in the right eye and 0.6 in the left. Her angle in both eyes was open to ciliary body band with 3–4 + pigment. Her pachymetry readings were 566μm/560μm. The glaucoma specialist decided this patient required IOP lowering. Given her history of moderate-severe dry eye and contact lens wear, the patient preferred to avoid the use of topical glaucoma drops and elected for SLT in the left eye. SLT was performed (50 spots for 180°, 0.9–1.1 mJ/pulse). The patient was only treated 180° given the heavy pigmentation in the trabecular meshwork. Immediately after the procedure, there were no complications and the cornea and IOP were stable.

The patient presented after one week and noted pain and blurred

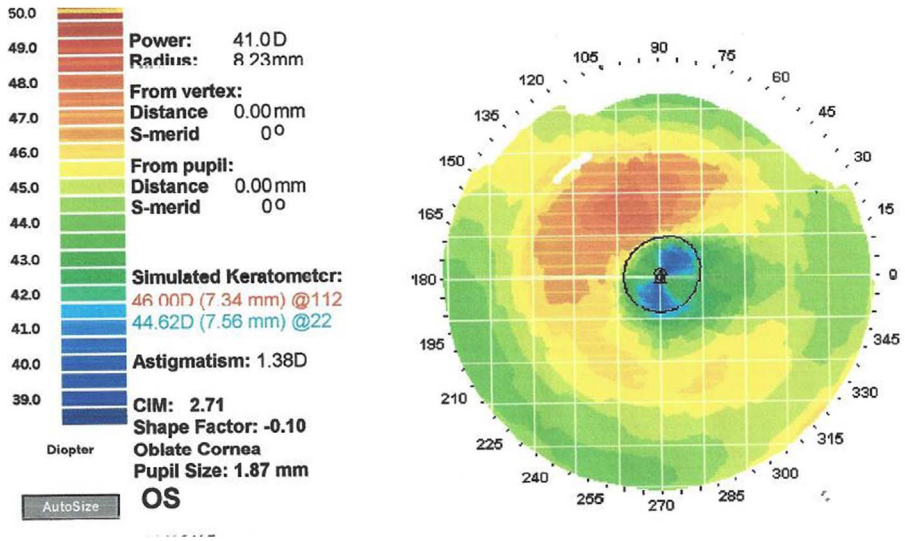

Fig. 2. Topography of the OS showing irregular astigmatism.

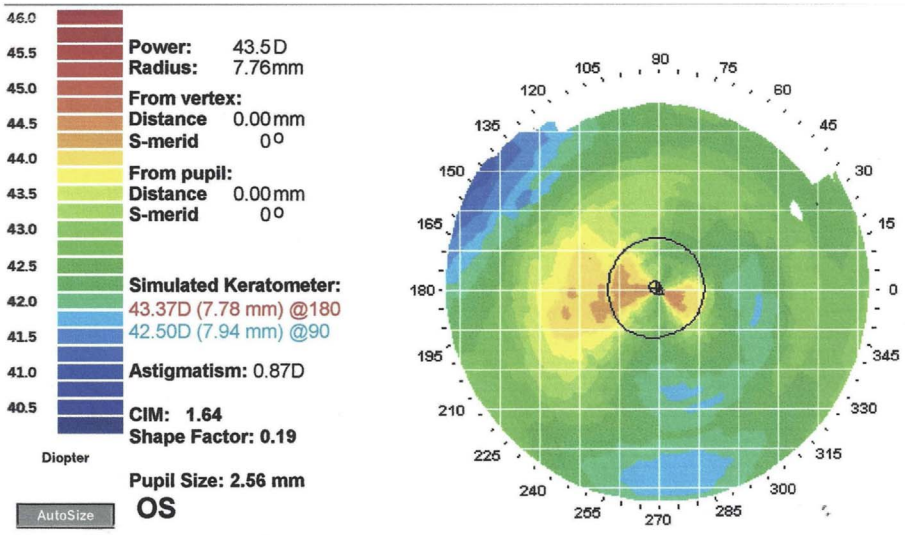

Fig. 3. Topography of the OS showing induced astigmatism.

vision OS. Her anterior stroma was moderately hazy centrally without infiltrate or defects and her vision was 20/40-. The IOP was stable and there was no inflammation in the anterior chamber. She was treated with prednisolone acetate four times a day for two weeks until the haze resolved and then tapered off the medication. Her vision remained 20/40 with her habitual prescription and improved to 20/25 with an increase of 3.75D of cylinder correction. Her prescription eventually stabilized at 1D of cylinder higher than pre-operative with an axis shift of 40° and vision remained at 20/25. Fig. 3 shows her topography after stabilization six months post-operatively.

3. Discussion

These two cases represent an adverse event following SLT with new irregular astigmatism despite the resolution of post-SLT corneal edema. Among the reported cases in the literature of post-SLT cornea edema, the onset of the corneal edema was between 1 and 7 days post-SLT following 360° of treatment.[5–7] The corneal edema resolved with topical steroid treatment between 3 weeks and 4 months. There is one case who did not resolve of the post-SLT corneal edema.[7] Thus, to the best of our knowledge, our two cases demonstrate a rare complication of irregular astigmatism following resolution of post-SLT corneal edema.

The pathogenic mechanism of corneal complications after SLT is not clearly understood, but several mechanisms have been proposed.[6] These mechanisms include: post-operative anterior chamber inflammation, alcohol residue on the gonioscopy lens used to clean the lens, a reactivation of the herpes simplex virus in the stroma, release of free radicals into the anterior chamber which causes damage to endothelial cells, direct laser damage to the cornea, or "champagne bubbles" contacting the cornea.[5–7] Each of these theories will be discussed specifically for our patient cases.

Post-operative anterior chamber inflammation is not uncommon in the first few days and could potentially cause the corneal edema. Neither patient had more than 1 + cell when they presented with corneal edema. High IOP could also cause corneal edema, but each patient did not have a post-SLT IOP spike.

Alcohol was used to clean the gonioscopy lens after each patient and then stored in the lens case. Thus, it is unlikely that unevaporated alcohol residue was on the lens. Furthermore, each patient was examined immediately after and at 30 minutes post-procedure and was clear with no epithelial staining or defects.

Inflammation in the anterior chamber from the procedure could cause a reactivation of herpes simplex virus in the stroma. Neither patient reported a history of ocular herpes simplex infection. In addition, the corneal edema resolved with topical steroid treatment without any development of epithelial dendrite. Also, neither patient was using a topical prostaglandin which can potentially increase the risk of herpes simplex virus reactivation.[8] A recent paper by E.T. Liu et al. discussed three cases where corneal edema occurred shortly after SLT and a reactivation of herpes simplex was suspected due to history of the disease, endothelial deposits, and/or endotheliitis.[9] The patients were treated with steroids and oral anti-virals. These cases appear to be different than ours based on the initial clinical presentation and none of them had long-term irregular astigmatism.

The release of free radicals into the anterior chamber during SLT could potential cause damage to endothelial cells. A study by Ong et al.[10] looked at 15 subjects with specular microscopy pre-SLT, immediately after, and at one month post-SLT. There were no differences in the endothelial cell counts at any time point. They found 12/15 subjects had either no difference or an insignificant difference between the three specular images immediately post-SLT. Three of the 15 subjects had greater than 5 dark spots on the immediate post-SLT endothelial image which resolved at the second post-SLT imaging test. These dark spots are believed to be areas where inflammatory cells temporarily attach to the endothelial cells. Three of these subjects had a shared pre-SLT finding of endothelial pigment. This is a small case series but perhaps the endothelium may be more susceptible to immediate post-operative changes if endothelial pigment is present; however, none of the subjects were reported to have post-SLT corneal edema. Neither of our two cases had endothelial pigment or any type of corneal abnormality with slit lamp examination.

Direct laser damage to the cornea could be a cause but it would seem that peripheral edema would be more common than central since this is the location where the laser is applied. Both of our patients had central edema.

During SLT, the appearance of "champagne bubbles" is an indication of appropriate amount of energy has been applied to the TM to get a therapeutic result. These bubbles when contacting the cornea could potentially cause edema. This is a possible etiology in our cases but it would seem the edema would be concentrated superiorly since the bubbles rise. Again, our subjects had central edema so this theory does not seem to fit.

The largest prospective study examining corneal changes after SLT was done by Lee et al.[3] There were 111 eyes that underwent 360° SLT among 66 subjects with OAG. Outcome measures included refractive error, central corneal thickness (CCT) measured by ultrasonic pachymetry, and endothelial cell count at time three points comparing pre-

SLT, 1 week post-SLT, and 1 month post-SLT. There was no change in refractive error at any point or corneal edema in any eyes. There were transient changes in the CCT and endothelial cell counts which significantly decreased by 1% and 4.5%, respectively at 1 week but were back to baseline at one month. Therefore, this study showed corneal effects after SLT but did not demonstrate any corneal edema or long-term corneal changes consistent with our two cases.

One observation that is common to both of our patients is their moderate to high myopia (greater than −5.00 diopters) which was also noted in the case reports by Knickelbein et al.,[8] Regina et al.,[6] and Song et al.,[11] all of their cases, 7 in total, with post-SLT edema had this same refractive error. However, the mechanism by which SLT causes post-SLT edema is not known.

4. Conclusions

Irregular astigmatism as a consequence of post-SLT corneal edema is a rare complication. Although these two cases showed no history of corneal compromise, it may be prudent to use caution in individuals who have pre-existing endothelial compromise, endothelial pigment, history of ophthalmic herpes simplex, or perhaps high myopia. Although specular microscopy is not standard of care to test prior to SLT, some precautions may include using lower energy, applying fewer spots during treatment, or use argon laser trabeculoplasty as an alternative since corneal changes are unreported after ALT. Although a rare adverse event, it is prudent to educate patients on the potential, yet rare, side effect of corneal edema and irregular astigmatism immediately following SLT.

Patient consent

Both patients gave written permission to publish their case. IRB approval was not indicated.

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Conflicts of interest

The following authors have no financial disclosures: SW, AE, SM.

Authorship

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

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.ajoc.2018.01.028.

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