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A study of endothelial cell count pre- and post-neodymium-doped yttrium aluminum garnet laser iridotomy in subacute angle closure using specular microscope

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Abstract:

AIM: The aim of this study was to study the effect of neodymium-doped yttrium aluminum garnet (Nd:YAG) laser iridotomy on corneal endothelial cell count in patients with subacute angle closure using specular microscope.

MATERIALS AND METHODS: In this prospective study, 50 cases of narrow-angle Grade 1 and Grade 2 (Shaffer gonioscopic grading) visiting the Regional Institute of Ophthalmology, Government Medical College, Amritsar underwent Nd:YAG laser peripheral iridotomy. After obtaining informed written consent, specular microscopy was performed before and after iridotomy at 1 week, 1 month, 3rd month, and at 6th-month follow-up visits. Central, nasal, and temporal endothelial cell counts were evaluated through noncontact specular microscopy.

RESULTS: The mean participant age was 51.52 ± 7.9 years, and majority of the participants were females (76%). The mean IOP before the laser was 19.25 ± 1.914 mmHg and it varied from 18.50 ± 1.647 to 18.25 ± 1.699 mmHg (day 1, $p = 0.06$ and at 6 months, $p = 0.04$) following laser procedure. The mean corneal endothelial cell count at superotemporal site before laser peripheral iridotomy was 2844 ± 260, and this value decreased to 2807 ± 263, 2699 ± 267, 2656 ± 270, and 2591 ± 275 cells/mm^2 at postiridotomy, 1, 3, and 6 months' follow-up visits, respectively; these differences were statistically significant ($p < 0.05$). The mean total energy required to produce iridotomy was 14.88 ± 6.71 mJ, ranging from 5 to 37 mJ. The linear regression analysis indicated no statistical correlation between change in endothelial cell count at the treated site and total mean energy used. No significant difference was found between preiridotomy and postiridotomy corneal thickness at any site.

CONCLUSION: This study demonstrated a significant endothelial cell loss at the treated site in 6 months' follow-up and suggested that Nd:YAG laser iridotomy may pose hazard to the corneal endothelium, although corneal decompensation at the treated site or as a whole was not seen.

Keywords:

Corneal endothelium, neodymium-doped yttrium aluminum garnet laser, specular microscope

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Introduction

About 60 million people are affected with glaucoma worldwide and out of these more than 20 million have primary angle closure glaucoma (PACG). Of these, more than 5 million with PACG are blind which is twice more than blindness caused by POAG.[1] It has been predicted that by the year 2020, 80 million people will have glaucoma, and approximately 11 million of

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them will be blind in both eyes. Half of this blindness will be the result of PACG.[2] The primary angle-closure must be effectively and promptly treated, to prevent permanent visual loss and irreversible optic nerve damage.[3]

Laser iridotomy, including argon laser iridotomy and neodymium-doped yttrium aluminum garnet (Nd:YAG) peripheral laser iridotomy (PI), has replaced surgical iridectomy as the preferred procedure for treating angle-closure glaucoma during the last decade. Laser iridotomy can be done entirely on an outpatient basis. Advantages of laser iridotomy, include the minimal risk of endophthalmitis, flat anterior chamber, or wound leakage (owing to it be an extraocular procedure), as may occur following intraocular surgery.[4] There is no surgical entry into the eye or need for injectable local or general anesthetic,[5] and also requires lesser patient preparation and procedure time.

In recent years, the Nd:YAG laser has become the laser of choice because of many advantages over the argon laser. The Nd:YAG laser has the ability to penetrate the irides of various hues (though it might be relatively more difficult to slice through dark irides and those having no crypts); requires fewer applications; has a higher success rate of penetration and long-term patency; and besides inducing lesser inflammatory response.[6]

To create a patent iridotomy, Nd:YAG laser generally requires fewer pulses and less energy than the argon laser (8–20 mJ for Nd:YAG compared to 5000–50,000 mJ for argon).[7] Early closure of the iridotomy by Nd:YAG laser is reported less than iridotomy created by argon laser.[8] Damage to the cornea in Nd:YAG iridotomy may occur as a result of local radiation effect, remote effect of the plasma formed by optical breakdown in the retrocorneal aqueous, or the effect of a shock wave generated at the retrocorneal focus.[9]

A complication, such as corneal decompensation, has been documented after argon and Nd:YAG laser iridotomy; however, the studies are very few and far in between, and hence, we deemed it necessary to investigate the effect of Nd:YAG iridotomy on corneal endothelium in subacute angle closure. It was important to determine, whether this prophylactic procedure was safe to endothelial cells, to prevent further ocular and especially corneal morbidity.

Materials and Methods

This prospective study was conducted at the Regional Institute of Ophthalmology, Government Medical College, Amritsar, from January 2016 to December 2016. The approval was obtained from the Ethics Review Board of the Institution. The study was conducted in accordance with the declaration of Helsinki. Every participant was informed of the study's objective in writing, and only those who gave written consent were enrolled in the study. The patients had the right to opt out of the study at any time during the course of the study without having to give reasons for doing so.

Each patient underwent slit-lamp examination, intraocular pressure (IOP) measurement through Goldmann applanation tonometry, and gonioscopy employing a Goldmann four-mirror gonioscope (Volk Surgicals, Ohio, USA) as well as stereoscopic evaluation of the optic disc using a +90 D noncontact lens (Volk Surgicals, Ohio, USA). All ophthalmic examinations and laser iridotomy procedures were performed by a single treating physician (AA).

Each eye eligible for inclusion in this study was clinically determined to have narrow and occludable angle (Shaffer gonioscopic Grade 1 or Grade 2). Specifically, subacute angle closure was defined as the eyes in which 180° or more of posterior trabecular meshwork was not visible in primary position with Goldmann goniolens with or without the presence of goniosynechiae or peripheral anterior synechiae in those locations on manipulative gonioscopy. All such eyes did not have specific optic disc changes attributive to glaucomatous optic neuropathy. Eyes with a history of intraocular surgery, retinal laser treatment, a history of acquired or genetic diseases of the cornea, any abnormality on specular microscopy (altered corneal endothelial cell morphology) before laser iridotomy, a history of ocular trauma or any retinal disease, and the presence of a systemic disease such as diabetes mellitus were excluded from the study. In addition, the eyes having an acute angle-closure attack were also excluded to remove the confounding effect of raised IOP, *per se*, affecting corneal endothelial cell count on specular microscopy.

Following participant selection, the corneal endothelium was photographed centrally and peripheral quadrants using a Topcon SP-3000P noncontact automated specular microscope (Topcon Corporation, Tokyo, Japan). This device autofocuses on the surface of the corneal endothelium and provides high magnification, good image quality and the potential to measure cell density, and perform morphometric evaluation. The device was used to focus corneal endothelium in three regions; superotemporal, central, and superonasal regions. Fifty cells in the selected square were selected and the software in the device then subsequently gave the specular count in that particular region. The software autofocussed on the specular image in the central cornea and subsequently the selection of the square to count

the cells in superotemporal and superonasal quadrants were done manually.

Subsequently, one drop of 2% pilocarpine was instilled every 15 min, 2 h before the procedure to induce miosis, and one drop of proparacaine hydrochloride 0.5% (Paracain, Sunways, India) was instilled into the eye. Laser peripheral iridotomy was performed on the peripheral superior temporal region of the iris of the selected eye by the treating physician using Nd:YAG laser SYL9000 (LightMed Corporation, Taipei, Taiwan). An Abraham goniolens (Bellevue, Washington, USA) with hydroxypropyl methylcellulose 2% (Occugel, Ophtechnics unlimited, India) as a coupling agent was used to focus the laser on the iris in all the cases. To reduce the postprocedure glare, the site of iridotomy was chosen as peripheral as possible avoiding the area of arcus senilis. Using appropriate settings, PI was performed. Laser shots between 3 and 8 mJ and bursts of 3 pulses/shot were aimed at the periphery between 10 'o clock and 1' o clock of iris onto the iris crypts or area of thin stroma. Endpoint of iridotomy completion was identified by sudden outpouring of melanotic pigments from iris pigment epithelium along with sudden gush of aqueous from posterior to anterior chamber and consequent deepening of the anterior chamber. Pulse energy, number of shots, and total energy used were precisely recorded. If additional pulses were required to enlarge or reopen the iridotomy site, especially in thicker irides, the second treatment was given 15 days after the initial treatment.

Following Nd:YAG laser iridotomy, the eyes were examined and photographed centrally, superior nasal, and superior temporal regions at different sessions: postiridotomy day 1 (D1), at day 7 (D7), 1 month (M1), 3 months (M3), and 6 months (M6). All specular microscopy measurements were documented by a single physician (NB) to reduce interobserver variability in statistical calculations and analyses. Even though measurements on a particular machine were carried by a single observer, there is an element of intraobserver variability which was minimized by taking three readings at every location and then averaging it.

Corneal thickness assessment at central, superotemporal, and superonasal quadrants was performed using an ultrasonic pachymeter Pacscan 300 P (Sonomed Escalon, Wayne, PA, USA). All pachymetric measurements were also performed by a single physician (SJ). Three readings at every location were taken on the machine and were then averaged to minimize intraobserver variation.

The postlaser regimen consisted of topical fourth-generation fluoroquinolone moxifloxacin 0.5% (Vigamox, Alcon, Fort Worth, TX, USA) four times daily along with prednisolone acetate 1% (Pred forte, Allergan, CA, USA) six times a day. The patients were also given topical timolol (Timolet OD, Sun Pharma, India) and systemic acetazolamide 250 mg sustained release (Iopar SR FDC, India) tablets twice daily for 1 week. The antiglaucoma medicines were stopped at day 7 whereas topical steroids were gradually tapered over 2–3 weeks.

Data analysis was performed using the Statistical Package for the Social Sciences version 17.0 for windows (IBM, New York, USA). The paired sample *t*-test was used to find the significance in endothelial cell count and corneal thickness postiridotomy at different time intervals in relation to preiridotomy value. $p < 0.05$ was accepted, as the level of statistical significance. The Pearson correlation test was used to find correlation between endothelial cell loss at the treated site and total mean energy used. The average effect size achieved with these three variables (endothelial cell count (ECC) at superotemporal, central, and superonasal regions) was 0.3403. Taking α error probability of 0.05, and $n = 100$, the power of the present study achieved was 90%.

Results

Out of 100 eyes, 50 patients were enrolled in the study. Out of 50 patients, 38 (76%) were female, and 12 (24%) were male. The mean age was 58 years. Out of 100 eyes, 55 (55%) eyes had angle with Shaffer Grade II, and 45 (45%) eyes had angle with Shaffer Grade I. All the eyes in the study had uniform brown and no heterochromia iridis or heterochromia iridium was noted. A total of 90 (90%) eyes underwent one laser sitting, and 10 (10%) eyes had to undergo two laser sittings. The mean energy required in first sitting was 13.07 ± 4.47 mJ and in second sitting energy, who underwent two sittings, was 18.1 ± 3.32 mJ. The mean total energy required to produce iridotomy was 14.88 ± 6.71 mJ, ranging from 5 to 37 mJ. The mean number of laser shots required was 4 ± 1 shots.

In the present study, the mean IOP before laser iridotomy was 19.25 ± 1.914 mmHg. There was no postlaser IOP spike on day 1 and 7 (18.50 ± 1.647 and 17.78 ± 1.460 mmHg) which might be attributed to the use of topical and systemic antiglaucoma medications. There was a statistical significant decrease in IOP at 3 and 6 months as compared to prelaser values (18.13 ± 1.916 and 18.25 ± 1.699 mmHg, $p = 0.035$ and $p = 0.04$, respectively) [Table 1]. The endothelial cell density, before iridotomy, in the treated superotemporal quadrant was 2844 ± 260 cells/mm^2. There was a significant endothelial cell loss in all-time sets when

compared to preiridotomy endothelial cell count. There was also a statistically significant loss of endothelial cells at 6 months when compared with endothelial cell count at 3 months ($p = 0.01$) [Table 2].

There was no statistical correlation between mean total energy used during treatment and change in the endothelial cell density during various time sets (on the 1st postoperative day, $p = 0.060$; at day 7, $p = 0.170$; 1st month, $p = 0.140$; 3rd month, $p = 0.145$; and 6th month, $p = 0.091$) [Figures 1-5].

There was no significant cell loss in untreated central and superonasal sites when compared to prelaser iridotomy endothelial cell count in any of the time sets ($p > 0.05$) [Tables 3 and 4].

No significant difference between preoperative and postoperative corneal thickness was observed at any of the treated superotemporal and nontreated central and superonasal sites [Tables 5-7].

Table 1: Progression of intraocular pressure pre and post neodymium: yttrium-aluminum-garnet peripheral iridotomy at various time points

Time period	Mean IOP±SD (mmHg)	Change in IOP (prelaser IOP -versus various time periods)	*p*
Prelaser	19.25±1.914		
Day 1	18.50±1.647	0.650±1.158	0.06
Day 7	17.78±1.460	1.470±1.291	0.025
Day 14	19.55±1.559	−0.300±0.560	0.25
Month 1	18.01±1.345	1.240±1.006	0.030
Month 3	18.13±1.916	1.120±0.769	0.035
Month 6	18.25±1.699	1.000±1.158	0.04

IOP=Intraocular pressure, SD=Standard deviation

Table 2: Endothelial cell count at superotemporal quadrant

Time period	Mean±SD		*p*
	Cell density (cells/mm²)	Change (preiridotomy-various time sets)	
Prelaser	2844±260	-	-
Postlaser day 1	2807±263	37±24	0.01
Day 7	2769±266	75±33	0.01
Day 28	2699±267	145±47	0.01
Month 3	2656±270	188±53	0.01
Month 6	2591±275	253±67	0.01

SD=Standard deviation

Discussion

Nd:YAG laser iridotomy causes tissue photodisruption. Damage to the cornea in Nd:YAG iridotomy may occur as a result of plasma formation propagating toward corneal endothelium and subsequently toward laser delivery source.[10]

In our study, the energy required to produce a patent iridotomy varied from 5 to 37 mJ with a mean energy of 14.88 ± 6.71 mJ. It is difficult to determine a formal energy setting because of the variety of factors in eyes such as peripheral anterior chamber depth (peripheral cornea-iris distance), corneal transparency, thickness of iris tissue, and presence or absence of iris crypts. Different eyes require varied energy settings, as response to a particular laser energy setting is different in different individuals.

We observed that there was a significant endothelial cell loss at the treated superotemporal site, although this loss was not statistically correlated with the total energy used

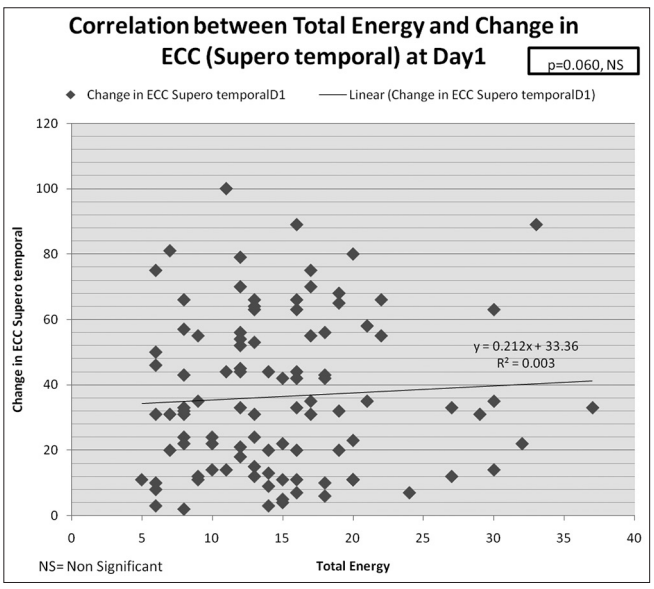

Figure 1: Correlation between total energy and change in endothelial cell count (ECC) superotemporal at D1

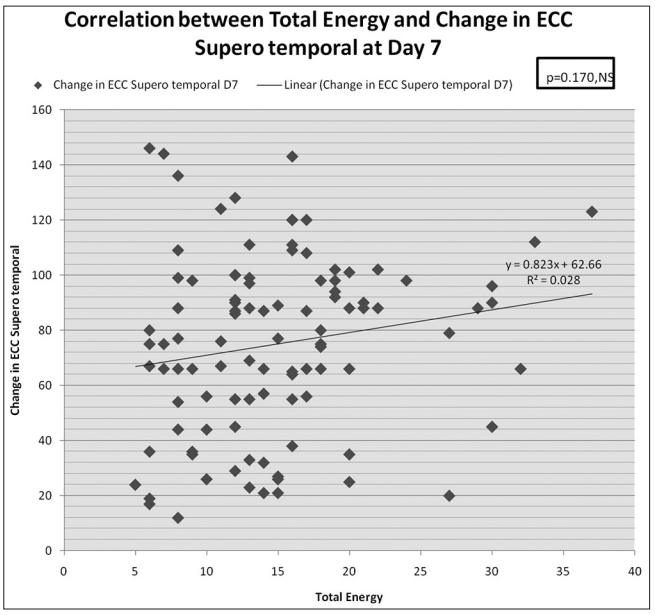

Figure 2: Correlation between total energy and change in endothelial cell count superotemporal at D7

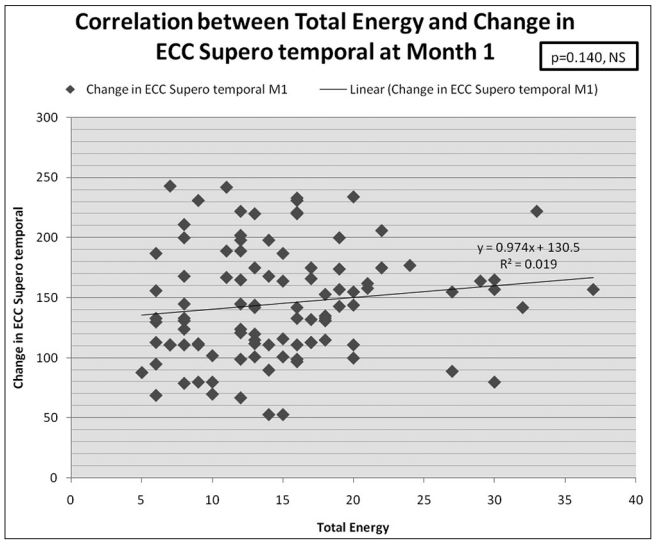

Figure 3: Correlation between total energy and change in endothelial cell count superotemporal at M1

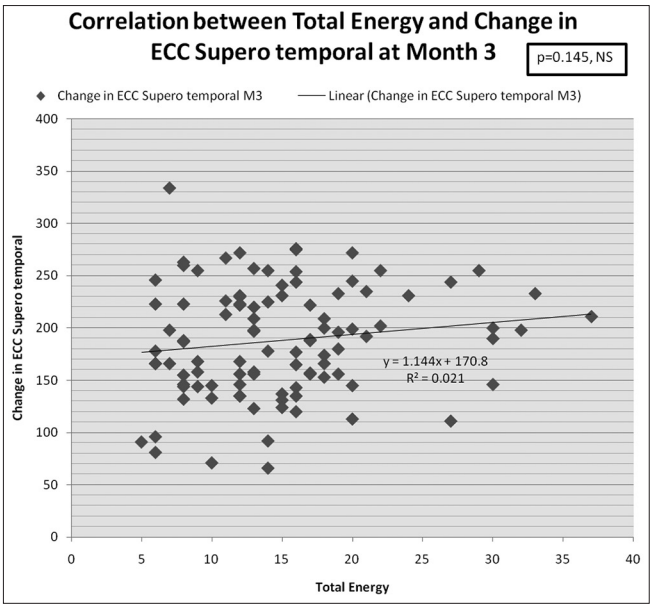

Figure 4: Correlation between total energy and change in endothelial cell count superotemporal at M3

Table 3: Endothelial cell count in untreated central quadrant

	Cell density, mean±SD (cells/mm²)	Change (preiridotomy -various time sets)	*p*
Prelaser	2784±268	-	-
Postlaser day 1	2783±267	2±6	0.47
Day 7	2779±268	5±13	0.44
Day 28	2777±269	7±14	0.42
Month 3	2775±268	8±15	0.41
Month 6	2774±267	9±135	0.40

SD=Standard deviation

Table 4: Endothelial cell count in untreated superonasal quadrant

	Cell density mean±SD (cells/mm²)	Change (preiridotomy-various time sets)	*p*
Prelaser	2830±256	-	-
Postlaser day 1	2828±257	2.0±5.0	0.47
Day 7	2825±254	5.0±27.0	0.44
Day 28	2824±257	6.0±10.0	0.44
Month 3	2822±361	8.0±31.0	0.43
Month 6	2820±255	10.0±22.0	0.39

SD=Standard deviation

Table 5: Corneal thickness in treated superotemporal quadrant

	Corneal thickness (mm), Mean±SD	*p*
Prelaser	0.560±0.001	-
Postlaser day 1	0.562±0.011	0.07
Day 7	0.559±0.009	0.09
Day 28	0.559±0.011	0.13
Month 3	0.558±0.017	0.14
Month 6	0.558±0.012	0.06

SD=Standard deviation

during iridotomy. There was no significant change in corneal thickness at any of the treated and nontreated sites.

Wu *et al*. in their study revealed significant cell density decreases at 1-year duration after Nd:YAG iridotomy, although no statistical correlation found between the change in endothelial cell density and the entire energy power used throughout the treatment procedure.[4] Marraffa *et al*. also found a substantial decrease in corneal endothelial cell density after Nd:YAG iridotomy specifically in cases of angle-closure glaucoma.[11] Canning *et al*. also found that majority of participants exhibited serious focal endothelial impairment following Nd:YAG laser iridotomy, although there was no evidence of persistent corneal injury based on short-term follow-up examinations.[12] Using prophylactic sequential laser iridotomy, Kumar *et al*. documented decline in corneal endothelial cell density and alteration in central corneal thickness over 3 years, although these changes were comparable to those in the fellow eye, which had not been treated.[13] However, they studied the central area of cornea alone, which is different from our reporting of 6-months follow-up in three different areas of cornea. Panek *et al*., in their series, have reported the results quite similar to ours, mainly a substantial decrease in corneal endothelial cell density following the Nd:YAG laser peripheral iridotomy but no significant difference between pre- and post-iridotomy corneal thickness at any of the treated superotemporal and nontreated central and superonasal sites.[14] However, there are studies available which have shown no significant decline corneal endothelial cell density and central corneal thickness following prophylactic laser iridotomy in primary angle closure suspect over 5 weeks to 1 year.[15-17]

According to studies by Meyer *et al*. and Martin *et al*., damage to corneal endothelium with Nd:YAG laser may be dependent on two factors-power level used and the distance

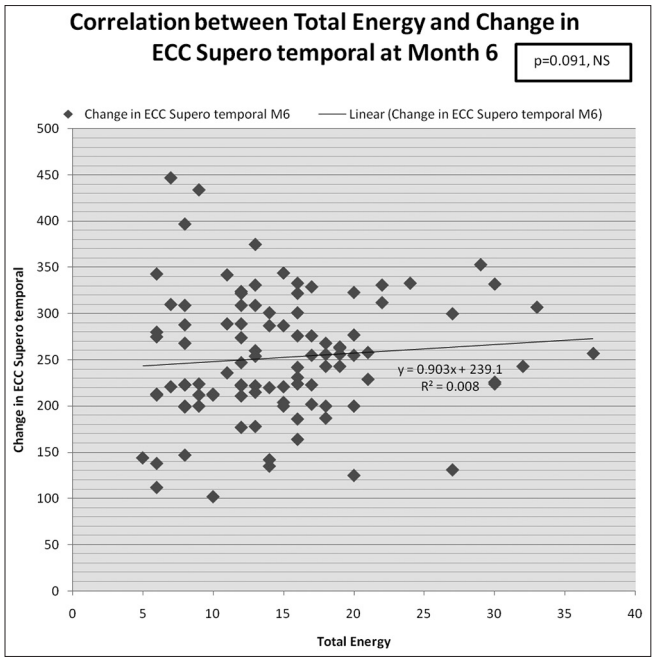

Figure 5: Correlation between total energy and change in endothelial cell count superotemporal at M6

Table 6: Corneal thickness in untreated central quadrant

	Corneal thickness (mm)	*p*
Prelaser	0.550±0.002	-
Postlaser day 1	0.549±0.009	0.12
Day 7	0.549±0.008	0.48
Day 28	0.549±0.007	0.32
Month 3	0.549±0.009	0.28
Month 6	0.549±0.010	0.09

Table 7: Corneal thickness in untreated superonasal quadrant

	Corneal thickness (mm)	*p*
Prelaser	0.561±0.005	-
Postlaser day 1	0.560±0.007	0.09
Day 7	0.559±0.010	0.06
Day 28	0.558±0.012	0.07
Month 3	0.558±0.011	0.06
Month 6	0.558±0.013	0.07

between the corneal endothelium and the iris surface.[10,18] Meyer *et al*. were first to propose that endothelium was focally denuded when laser shots were applied within 2 mm of rabbit corneal endothelium.[10] Martin *et al*. suggested a guideline of 1 mm from endothelium, to be safe retrocorneal distance threshold.[18]

The central depth of the anterior chamber in normal aging human eye is around 2.5 mm, and mid-peripheral depth is around 1.4 mm.[19] Ideally, we should choose midperiphery of the iris for laser iridotomy and avoid being too peripheral. However, in clinical practice, it is difficult to locate the midperiphery and determine a standard retrocorneal distance that is more than 1 mm.

Marraffa *et al*. reported by the use of ultrasound biomicroscopy (UBM) that the mean distance of the iridotomy from the corneal endothelium was 0.62 ± 0.23 mm, and the loss of endothelial cells was inversely proportional to the distance of the iridotomy from the endothelium.[11] This result might reflect the fact that in many cases, in routine clinical practice, the retrocorneal distance to the iridotomy site is <1 mm. Here, we also suppose that the retrocorneal distance was likely <1 mm and consider this factor to contribute toward superotemporal endothelial cell loss because in our study there was no significant correlation between the mean total energy delivered and corneal endothelial cell loss.

The mean IOP in our study returned to prelaser values at 2 weeks, once both topical and systemic antiglaucoma medications were stopped. There was a statistically significant decline in IOP at 3 and 6 months as compared to prelaser values.

The limitation of the present study was that we did not measure peripheral anterior chamber depth at the level of the iridotomy (which can be measured using Pentacam, UBM, or Anterior Segment Optical Coherence Tomography [ASOCT]). The thickness of the iris was also not measured (which can be measured by UBM and ASOCT).

The follow-up in the present study was also short (6 months) which precludes the assessment of long-term pattern of endothelial cell loss, if any.

Hex (percentage of hexagonal cells) and coefficient of variation were not measured, which signified parameters of pleomorphism and polymegathism, respectively, and could have picked up some early correlation (if any) between amount of laser energy used and subsequent corneal endothelial morphology change after laser iridotomy. This factor may be taken up for further subsequent studies with longer follow-up and more number of patients.

The time period between laser iridotomy and corneal decompensation has been stated to be up to 8 years.[6] In the present study, during the 6-month follow-up, none of our cases treated with Nd:YAG laser developed corneal decompensation; however, our results did reveal significant endothelial loss at the treated site.

Conclusion

Nd: YAG laser iridotomy may pose hazard to the corneal endothelium in the long term, therefore, analysis of both

corneal endothelium and thickness should be continued, even after six months, to pick up early endothelial decompensation, if it does arise.

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Conflicts of interest

The authors declare that there are no conflicts of interests of this paper.

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