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Effect of Neodymium Doped:Yttrium Aluminium Garnet Laser Capsulotomy on Central Corneal Thickness And Corneal Endothelial Cell Count In Type 2 Diabetes Patients

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ABSTRACT

Background: The most frequent delayed consequence after cataract surgery is PCO, and the most usual treatment for visual rehabilitation is posterior capsulotomy using a Nd:YAG laser. However, corneal endothelium cells may be impacted by the photodisruptive shock waves produced during the operation, particularly in diabetic individuals with decreased endothelial reserve.

Objective: To evaluate and contrast the changes in central corneal thickness and corneal endothelial density in individuals with type 2 diabetes and those without the disease, and to link these changes with total laser energy.

Methods: At a tertiary care facility, 150 pseudophakic patients with visibly significant posterior capsule opacification participated in an 18-month prospective comparison analysis. Groups of patients with and without diabetes were created.Endothelial cell density and central corneal thickness were measured using specular microscopy pre-procedure and at two hours, two weeks and four weeks following Nd:YAG capsulotomy. Laser parameters were recorded and statistical analysis performed.

Results: Diabetics had a greater central corneal thickness and a lower preoperative endothelial cell count. Following Nd:YAG both groups experienced endothelial cell loss, although the percentage of diabetic individuals was noticeably higher. Following laser, central corneal thickness temporarily rose, particularly in patients with diabetes. Endothelial loss was correlated with higher cumulative laser energy. Both groups' visual acuity significantly improved.

Conclusion: In diabetics, endothelial alterations were more noticeable following Nd:YAG. Careful endothelial monitoring and the prudent use of laser energy are stressed, especially in diabetics.

KEYWORDS: YAG Capsulotomy, Endothelial Cell Density, Posterior Capsule Opacification, Diabetes Mellitus, and Corneal Endothelium.

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INTRODUCTION

Worldwide, cataracts continue to be the leading cause of reversible blindness, and one of the most popular ophthalmological operations is cataract extraction. Advances in microsurgical instrumentation, IOL design, and surgical techniques have improved safety and visual outcomes. However, PCO continues to be the most common long-term consequence after cataract surgery and a major contributor to delayed vision loss. [1]

PCO develops due to proliferation and migration of residual lens epithelial cells over the posterior capsule, resulting in fibrous, Elschnig pearl, or Soemmering ring type opacification that interferes with the clarity of the visual axis. The incidence of PCO varies between 20% and 50% within a few years after surgery, depending on surgical technique, IOL material, and patient-related factors [2]. Diabetes mellitus is a significant systemic risk factor. Chronic hyperglycemia alters lens metabolism and promotes oxidative stress, inflammation, and advanced glycation end-product formation, which may enhance capsular fibrosis and influence postoperative ocular recovery [3].

Since it is non-invasive, provides a quick reversal of visual impairment, and is widely thought to be safe, Nd:YAG laser posterior capsulotomy is the obvious choice. It has been the cornerstone of treatment for visually significant PCO. Shock waves

produced by the photo disruptive process, however, have the potential to impact intraocular structures, such as the corneal endothelium. Loss of endothelial cells may impair corneal function since the corneal endothelium is essential for preserving corneal transparency and has a limited capacity for regeneration [4].

After intraocular surgeries, endothelial cell loss is widely known. Because diabetes individuals have a lower endothelium reserve and are more vulnerable to metabolic stress, this loss may be larger in these patients. Therefore, assessing endothelium alterations after Nd:YAG capsulotomy is therapeutically relevant, especially in diabetics. In this study, the impact of Nd:YAG laser capsulotomy on corneal endothelial cell density and central corneal thickness in individuals with and without diabetes will be evaluated, and the correlation between laser energy and endothelial changes will be investigated.

AIM OF THE STUDY

To investigate and contrast the effects of Nd YAG laser capsulotomy on corneal endothelial cell count and corneal thickness in individuals with type 2 diabetes and the general population

MATERIALS AND METHODS

A tertiary care centre based prospective comparative study was carried out in the Department of Ophthalmology, Integral Institute of Medical Sciences and Research,
Integral University, Lucknow, Uttar Pradesh, India.

Study participants – Participants in the study were all pseudophakic patients who were over 40 years old, had visibly significant posterior capsule opacification, had Type 2 diabetes mellitus or not, and were deemed fit for Nd:YAG laser posterior capsulotomy.

Design of study – The study's design is a prospective comparative analysis.

Total Duration of study- 18 months
Twelve months of data collecting (December 2023–December 2024)
Data collection, statistical analysis, andcompletion took six months, from January 2025 to June 2025.

Inclusion Criteria

1. **Age range:** forty to seventy.
2. Pseudophakic Diabetic patients with controlled blood sugar levels.
3. **Ocular Status:**Pseudophakic eyes with visually significant posterior capsular opacification, where Best Corrected Visual Acuity is ≤ 6/9 due to the effects of PCO.
4. **Time After Surgery:** Six months or more after cataract surgery.
5. **Consent:** Informed written consent to take part.

Exclusion Criteria

1. Any existing corneal pathology (e.g., dystrophies, infections, scars) that could interfere with the analysis.
2. History of any intraocular inflammation or ocular trauma.
3. Patients with RBS >300 mg/dL at procedure day (indicating poorly controlled diabetes).
4. Raised Intraocular pressure (IOP > 21 mmHg).
5. Patients who have received treatments or undergone surgeries that could alter the corneal endothelium, such as prolonged steroid use or corneal transplant surgery.
6. Patients who refuse to provide informed consent.

Included patients with visually significant posterior capsule opacification who were seen in the IIMSR Ophthalmology OPD. Included were pseudo-patients with managed type 2 diabetes and no history of diabetes. A thorough ocular examination, including a dilated fundus examination, intraocular pressure measurement, slit lamp examination, and visual acuity, was conducted along with a detailed history. Both groups' corneal endothelial characteristics, such as endothelial cell density and central corneal thickness, were evaluated using specular microscopy.

Every eligible patient had a posterior capsulotomy using a Nd:YAG laser. Laser parameters were noted, such as total laser energy, number of shots, and energy per pulse. Endothelial cell density and central corneal thickness were evaluated pre-procedure and at follow-up intervals of 2 hours, 2 weeks, and 4 weeks following capsulotomy.

Using the proper statistical techniques, all the data that was gathered was assembled and statistically examined. P-values below 0.05 were regarded as statistically significant.

RESULTS

Table 1. Baseline Demographic and Clinical Characteristics

Variable	Diabetic Group (n=75)	Non-diabetic Group (n=75)	P value
Age (years)	57.49 ± 6.82	58.48 ± 7.17	0.390
Male / Female	35 / 40	28 / 47	0.247*
Random Blood Sugar (mg/dL)	139.12 ± 24.01	117.41 ± 24.07	<0.001
Total Laser Energy (mJ)	80.77 ± 18.31	76.34 ± 17.90	0.136

The two groups were substantially comparable, as seen by the baseline comparison. There was no statistically significant variation in the distribution of age or gender, suggesting that demographic confounding was not present. Comparable procedural exposure was suggested by the fact that the total laser energy employed throughout the treatment was similar among the groups. Nonetheless, the diabetic cohort's random blood sugar was much higher, indicating a distinct metabolic divide between the groups. The diabetic cohort's mean duration of diabetes indicated a mild burden of the condition. When considered collectively, the baseline profile indicates that observed variations in endothelial end points are most likely due to metabolic and diabetic status rather than disparities in demographic inequality or procedural variables.

Figure 1. Baseline Demographic and Clinical Characteristics

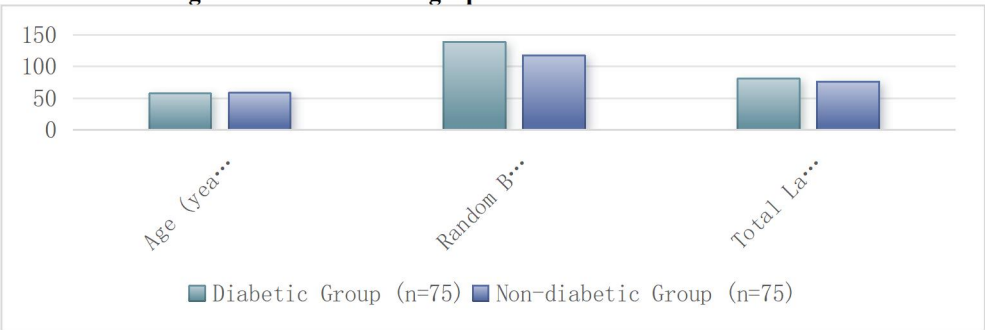


Table 2. Baseline Ocular Profile in Two Groups

Parameter	Diabetic Group (n=75)	Non-diabetic Group (n=75)	P value
Endothelial Cell Density	2287.55 ± 111.13	2413.43 ± 99.93	<0.001
Central Corneal Thickness	538.41 ± 16.32	527.17 ± 14.93	<0.001
BCVA (logMAR)	0.698 ± 0.213	0.659 ± 0.223	0.273
IOP (mmHg)	16.39 ± 2.44	16.93 ± 2.34	0.163

Baseline ocular assessment revealed significant pre-existing endothelial compromise in diabetic patients. Endothelial cell density was significantly lower in diabetics, indicating reduced endothelial reserve prior to laser exposure. Central corneal thickness was significantly higher in the diabetic group, possibly reflecting altered endothelial pump function or corneal hydration. In contrast, baseline visual acuity and intraocular pressure were comparable between groups, indicating similar functional and pressure status before intervention. These findings suggest that diabetic corneas may be intrinsically more vulnerable to additional endothelial stress, and baseline endothelial disadvantage likely contributes to the greater endothelial loss observed following Nd:YAG capsulotomy.

Figure 2- Baseline ECD

Figure 3- Baseline CCT

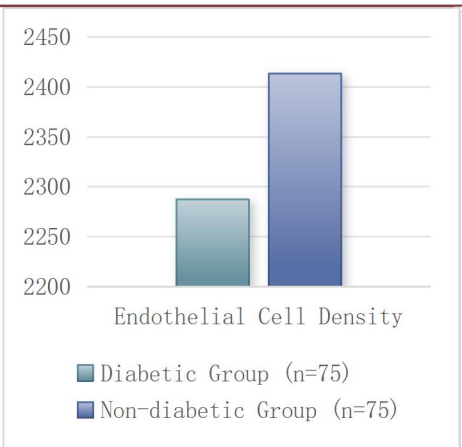


Figure 4- Baseline BCVA

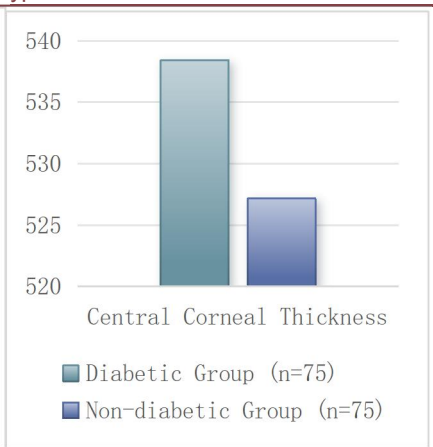
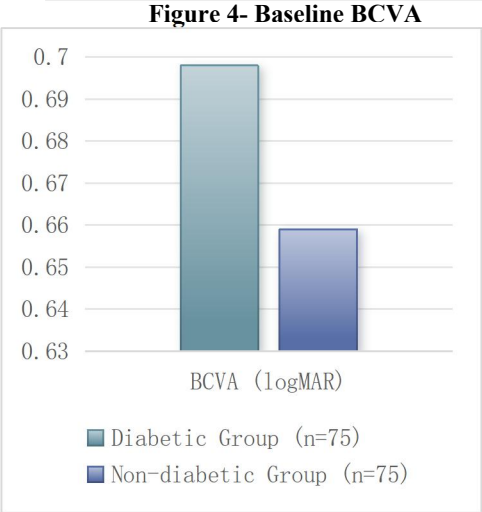


Figure 5- Baseline IOP



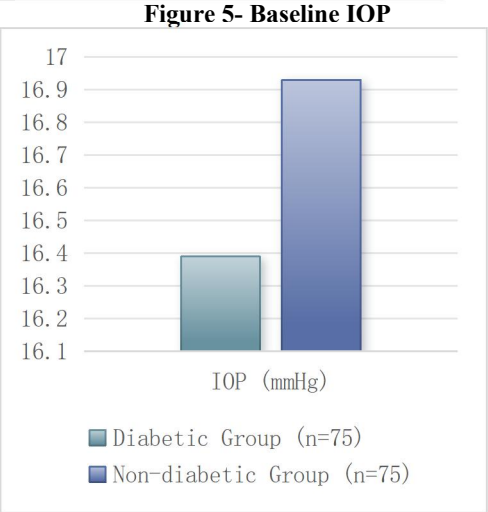


TABLE 3-Change In Endothelial Cell Density Over Time

Time Point	Diabetic Group (n=75)	Non-diabetic Group (n=75)	P value
Pre-laser	2287.55 ± 111.13	2413.43 ± 99.93	<0.001
2 hours	2266.57 ± 111.55	2399.59 ± 99.74	<0.001
2 weeks	2200.49 ± 108.35	2356.59 ± 101.09	<0.001
4 weeks	2164.68 ± 106.71	2331.77 ± 101.28	<0.001

Both groups demonstrated progressive endothelial cell loss following Nd:YAG capsulotomy; however, the magnitude of loss was consistently and significantly greater in diabetic patients at all time points. The difference persisted from the immediate postoperative period through four weeks, suggesting sustained endothelial vulnerability in diabetics. The continued decline indicates that endothelial injury may not be primarily due to acute laser effects but may reflect impaired endothelial recovery in diabetic corneas. These findings confirm that diabetes is associated with slight increased susceptibility to laser-induced endothelial damage, reinforcing the importance of cautious energy use and close postlaser monitoring in diabetic patients undergoing Nd:YAG capsulotomy.

Figure 6-Change In Endothelial Cell Density Over Time

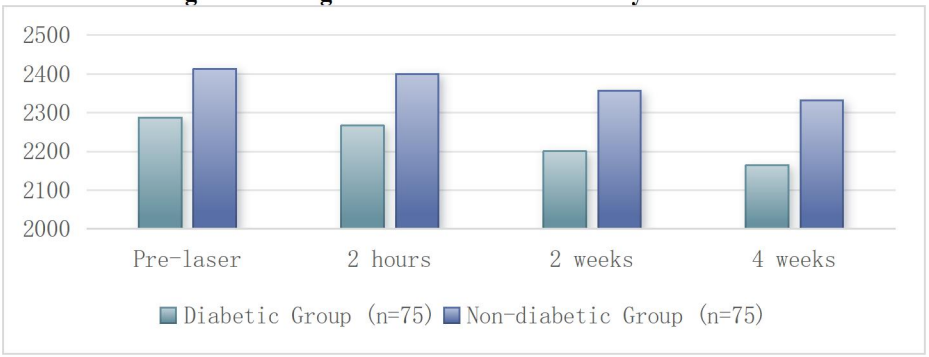


Table 4-Change In Central Corneal Thickness Over Time

Time Point	Diabetic Group (n=75)	Non-diabetic Group (n=75)	P value
Pre-laser	538.41 ± 16.32	527.17 ± 14.93	<0.001
2 hours	559.75 ± 18.36	549.52 ± 15.90	<0.001
2 weeks	540.69 ± 16.15	526.88 ± 15.28	<0.001
4 weeks	538.39 ± 16.29	527.35 ± 15.13	<0.001

Central corneal thickness increased transiently at two hours post-procedure in both groups, indicating temporary corneal edema following laser exposure. Thickness gradually returned toward baseline by two weeks and stabilized by four weeks, suggesting recovery of endothelial pump function. Despite similar recovery patterns, diabetics consistently exhibited significantly higher corneal thickness at all time points, reflecting underlying endothelial dysfunction. The absence of persistent edema despite significant endothelial loss suggests subclinical endothelial injury rather than overt decompensation. These findings support the concept that diabetic corneas have reduced functional reserve as seen from the baseline prelaser values and may demonstrate structural vulnerability even when clinical recovery appears satisfactory.

Figure 7- Change In Central Corneal Thickness Over Time

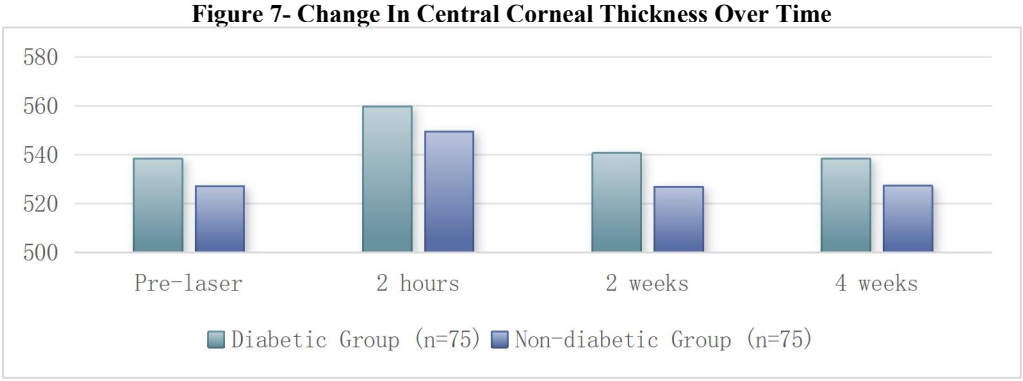
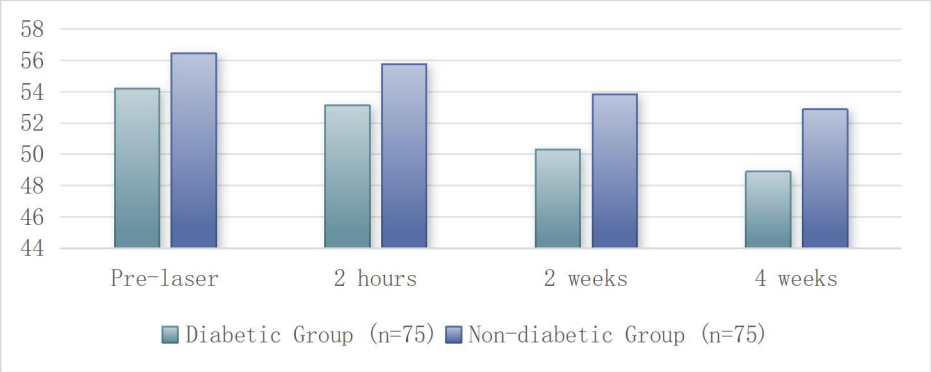


Table 5- Change In Endothelial Morphology (Hexagonality)

Time Point	Diabetic Group (n=75)	Non-diabetic Group (n=75)	P value
Pre-laser	54.22 ± 4.24	56.47 ± 4.28	0.0015
2 hours	53.14 ± 4.25	55.77 ± 4.28	0.00024
2 weeks	50.31 ± 4.25	53.85 ± 4.38	<0.001
4 weeks	48.89 ± 4.37	52.87 ± 4.48	<0.001

Hexagonality progressively declined in both groups, indicating increasing endothelial morphological stress after laser treatment. However, the reduction was significantly greater in diabetic patients at all time points as noted from prelaser to 4 weeks follow up, reflecting enhanced endothelial instability and polymegathism in diabetic corneas. The persistent morphological deterioration despite stabilization of corneal thickness suggests ongoing subclinical endothelial remodeling rather than reversible edema alone. These findings reinforce that diabetes affects not only endothelial cell density but also cell morphology, further compromising endothelial integrity. Morphological deterioration provides additional evidence that diabetic corneas are more susceptible to laser-induced endothelial injury and may require cautious procedural planning and closer follow-up.

Figure 8- Change In Endothelial Morphology (Hexagonality)



DISCUSSION

Posterior capsule opacification (PCO) remains the most common delayed complication after cataract surgery and a major cause of reduced visual acuity in pseudophakic patients. Nd:YAG laser posterior capsulotomy is the standard treatment for visually significant PCO, providing rapid visual rehabilitation. However, the photodisruptive mechanism produces plasma formation and shock waves that may affect the corneal endothelium, which has limited regenerative capacity. Endothelial safety is therefore clinically important, particularly in diabetic patients who have reduced endothelial reserve and altered corneal physiology[5].

In the present study, baseline endothelial cell density (ECD) was significantly lower in diabetic patients (2287.55 ± 111.13 cells/mm²) compared with non-diabetic patients (2413.43 ± 99.93 cells/mm²). Central corneal thickness (CCT) was higher in diabetics (538.41 ± 16.32 µm vs 527.17 ± 14.93 µm). Comparable findings were reported by El-Agamy et al., who documented similar endothelial compromise in diabetic corneas[5].
Following Nd:YAG capsulotomy, endothelial cell loss occurred in both groups. In the present study, diabetics showed a decline of 122.87 cells/mm² (5.37%), whereas non-diabetics showed a decline of 81.66 cells/mm² (3.38%) at 4 weeks. Similar endothelial loss after Nd:YAG capsulotomy has been reported in earlier studies[467]. Progressive endothelial decline after capsulotomy has also been described, suggesting ongoing endothelial remodeling rather than purely acute injury[8].

Central corneal thickness showed a transient postoperative increase, returning toward baseline by follow-up. Similar transient corneal thickening after capsulotomy has been reported previously[9].

Endothelial morphology showed deterioration in both groups, indicating structural endothelial stress. Comparable morphological changes following Nd:YAG capsulotomy have been documented in earlier studies[10].

Laser energy has been shown to influence endothelial response, with higher energy associated with greater endothelial changes[11]. In the present study, despite comparable energy levels, diabetics demonstrated greater endothelial loss, suggesting increased metabolic susceptibility.

Recent studies also report similar endothelial and pachymetric changes following Nd:YAG capsulotomy[121314]. Compared with these studies, endothelial loss in the present diabetic group was relatively greater, reinforcing the increased vulnerability of diabetic corneas.

Visual acuity improved significantly in both groups following capsulotomy. Previous clinical studies have similarly reported favorable visual outcomes with minimal clinically significant corneal complications[15].

Overall, the present study demonstrates measurable endothelial alterations following Nd:YAG capsulotomy, including progressive endothelial cell loss, transient corneal thickening, and morphological changes, with significantly greater impact in diabetic patients. These findings emphasize careful endothelial evaluation, judicious laser energy use, and close postoperative monitoring, particularly in diabetic individuals, to preserve long-term corneal health.

CONCLUSION

Nd:YAG laser posterior capsulotomy effectively restores visual clarity but is associated with measurable corneal endothelial alterations, particularly in diabetic patients. The present study demonstrated significantly greater endothelial cell loss and morphological deterioration in diabetics compared to non-diabetics, despite comparable visual recovery. Endothelial injury showed a dose–response relationship with laser energy and increased with higher PCO grade and fibrotic morphology. Central corneal thickness changes were transient, indicating predominantly subclinical endothelial damage. These findings highlight the importance of preoperative endothelial assessment, cautious energy titration, and close postoperative monitoring, especially in diabetic patients, to minimize long-term corneal risk while maintaining optimal visual outcomes following capsulotomy.

LIMITATIONS

Short follow-up limited long-term endothelial assessment.

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