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Corneal Endothelial and Central Corneal Thickness Changes Following Nd:YAG Laser Posterior Capsulotomy: Implications in Patients with Type 2 Diabetes Mellitus

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ABSTRACT:

Posterior capsule opacification is a common delayed complication of cataract surgery and is most effectively managed with neodymium-doped yttrium aluminum garnet laser posterior capsulotomy. Patients with type 2 diabetes mellitus have a higher incidence of posterior capsule opacification and often require earlier intervention than non-diabetic patients. Chronic hyperglycemia in diabetes induces structural and functional alterations of the corneal endothelium and increases central corneal thickness, potentially increasing susceptibility to laser-induced corneal injury. Nd:YAG capsulotomy involves controlled photodisruption of the posterior capsule, and corneal safety is influenced by technical parameters such as laser focus, pulse energy, total energy delivered, and capsulotomy size. Available evidence suggests that Nd:YAG capsulotomy causes measurable endothelial cell loss and transient corneal changes, which are energy-dependent and may be more pronounced in diabetic eyes with reduced endothelial reserve. While most eyes tolerate the procedure without persistent corneal edema, diabetic patients warrant individualized laser strategies and close postoperative monitoring. Further controlled studies are needed to refine procedural guidelines and optimize corneal safety in this population.

Introduction

Cataract surgery is one of the most frequently performed ophthalmic surgeries worldwide, generally yielding significant visual improvement for patients. However, a common delayed complication of modern extracapsular cataract extraction and phacoemulsification is posterior capsule opacification (PCO) [1]. PCO results from residual lens epithelial cells proliferating and migrating across the posterior capsule, leading to fibrous or pearl-like opacification behind the intraocular lens (IOL). Clinically, PCO can cause decline in visual acuity, glare, reduced contrast sensitivity, and other visual disturbances often reminiscent of the original cataract. When visually significant PCO occurs, intervention is required to restore the visual axis clarity [2].

The Nd:YAG laser posterior capsulotomy has become the treatment of choice for PCO due to its efficacy and non-invasive nature, boasting a success rate over 95% in opening the opacified capsule. The procedure uses a Q-switched Nd:YAG laser (wavelength 1064 nm) to create a focused plasma spark that disrupts the capsule. Compared to surgical capsulotomy, the laser approach is faster and avoids intraocular incisions, but it is not without risks. Complications such as corneal endothelial cell loss, IOL damage (pitting), transient intraocular pressure (IOP) spikes, cystoid macular edema, and

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retinal detachment have been documented [3]. In particular, corneal endothelial injury from the shockwaves or plasma generated by the laser is a pertinent safety concern, as is the possibility of corneal edema reflected by changes in central corneal thickness (CCT).

Patients with type 2 diabetes mellitus (T2DM) represent a subgroup of cataract patients who merit special attention in this context. Diabetes is an established risk factor for developing PCO more rapidly and to a greater severity than in non-diabetic individuals [4]. Diabetic patients not only tend to form PCO at higher rates, but their corneas are often physiologically different even before any secondary intervention. Chronic hyperglycemia induces subtle corneal alterations: diabetic corneas frequently exhibit a thicker stroma and basement membrane, lower endothelial cell density, and morphological changes in endothelial cells (polymegathism and pleomorphism) [5]. These changes stem from mechanisms such as accumulation of sorbitol and advanced glycation end-products in the cornea, oxidative stress, and impaired endothelial pump function [6]. The result is a cornea that may have less reserve to withstand additional stress or injury. Indeed, following routine cataract surgery, diabetic eyes have been shown to lose more endothelial cells on average than non-diabetic eyes. This raises concerns that an Nd:YAG laser capsulotomy – which delivers energy into the eye to disrupt the posterior capsule – could have a differential impact on diabetic corneas, potentially exacerbating endothelial cell loss or corneal thickness changes compared to non-diabetic eyes [7].

This review article provides a comprehensive overview of corneal endothelial and CCT changes after Nd:YAG laser posterior capsulotomy, with particular emphasis on patients with T2DM. We examine the technical aspects of the procedure that influence corneal outcomes, discuss the underlying pathophysiological differences in the diabetic cornea, compare clinical outcomes between diabetic and non-diabetic patients, and summarize key studies (including recent international research) on endothelial cell loss and CCT changes post-capsulotomy. The goal is to inform best practices and identify knowledge gaps to guide future research, ultimately improving the safety and efficacy of Nd:YAG capsulotomy in all patients, especially those with diabetes.

Posterior Capsule Opacification: Epidemiology and Impact

Posterior capsule opacification (PCO), often referred to as an “after-cataract,” remains the most common long-term complication following cataract surgery with intraocular lens implantation. Epidemiological studies report that approximately 20% of patients develop PCO within two years of surgery, with incidence increasing to nearly 28–30% at five years. The development of PCO is influenced by multiple factors, including patient age, intraocular lens material and edge design, surgical technique, and ocular comorbidities. Diabetes mellitus has consistently been identified as an important risk factor for PCO development [8].

In a landmark case–control study by Hayashi et al., diabetic patients demonstrated significantly greater PCO density over long-term follow-up compared with age-matched non-diabetic patients and were more likely to require Nd:YAG laser capsulotomy as time progressed. Notably, the severity of PCO in diabetic patients did not correlate with diabetes duration, glycemic control, or the presence of diabetic retinopathy, suggesting that diabetes itself acts as an independent risk factor, possibly through altered wound-healing responses and lens epithelial cell behavior [5].

Clinically, PCO produces a spectrum of visual symptoms, ranging from glare and halos in early stages to significant loss of visual acuity and contrast sensitivity in advanced cases. The visual impairment associated with PCO has important implications for patient quality of life and

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healthcare utilization, as Nd:YAG capsulotomy necessitates additional clinical visits and procedural costs [9,10]. In diabetic patients, even moderate PCO may impede adequate fundus visualization, complicating retinal evaluation and treatment. Consequently, Nd:YAG capsulotomy is often indicated earlier in diabetic individuals to maintain a clear visual axis for ongoing retinal surveillance.

Nd:YAG Laser Capsulotomy: Technical Overview

Nd:YAG laser posterior capsulotomy is performed by delivering focused laser energy to create an opening in the opacified posterior capsule, thereby restoring the visual axis. The procedure relies on photodisruption, in which a Q-switched Nd:YAG laser emitting at 1064 nm generates plasma at the focal point, producing shockwaves that mechanically disrupt the capsule. Because these shockwaves can affect adjacent structures, careful attention to technical parameters is essential to minimize complications such as corneal endothelial damage [11].

To optimize safety, the laser focus is typically positioned slightly posterior to the posterior capsule, usually by approximately 100–250 µm. This posterior offset ensures that plasma formation occurs just behind the capsule, directing the disruptive force forward while reducing the risk of intraocular lens (IOL) pitting and endothelial injury. Most modern laser systems allow precise adjustment of this offset, commonly set between 0.1 and 0.3 mm [12].

Energy per pulse and total energy delivery are critical determinants of tissue response. It is recommended to begin with the lowest effective energy, often 1.0–1.5 mJ per pulse for mild to moderate posterior capsule opacification. Energy can be increased incrementally if capsular disruption is inadequate. Dense fibrotic opacities may require higher pulse energies (2–5 mJ) and a greater number of pulses. Typically, 20–50 laser shots are delivered to achieve an adequate openingreviewofoptometry.com. Maintaining a low total cumulative energy—preferably below 50 mJ—is associated with fewer complications, while energies exceeding 80 mJ have been linked to increased risks such as intraocular pressure (IOP) elevation and retinal detachment [13].

Accurate focusing is achieved by ensuring precise convergence of the aiming beams on the posterior capsule. A contact capsulotomy lens, such as the Abraham lens, is commonly used to stabilize the eye, magnify the capsule, and reduce energy requirements. Capsulotomy openings are typically created using either a cruciate or circular pattern, with a final aperture of approximately 3.5–5.0 mm to balance visual improvement and safety.

Peri-procedural management includes prophylactic use of topical alpha-agonists to reduce IOP spikesreviewofoptometry.com, followed by post-procedure IOP monitoring and short-term anti-inflammatory therapy. Careful documentation of laser parameters and postoperative findings is essential for clinical follow-up and risk mitigation.

Table 1. Key Technical Parameters for Nd:YAG Laser Posterior Capsulotomy

Parameter	Recommended Practice	Clinical Rationale
Laser wavelength	1064 nm (Q-switched)	Enables photodisruption
Focus offset	100–250 µm posterior to capsule	Reduces IOL and endothelial damage reviewofoptometry.com
Initial energy	1.0–1.5 mJ per pulse	Minimizes collateral injury reviewofoptometry.com

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Typical pulse count	20–50 shots	Adequate opening with controlled energy reviewofoptometry.com
Total energy	Preferably <50 mJ	Lower complication rates reviewofoptometry.com
Capsulotomy size	3.5–5.0 mm	Visual improvement with reduced vitreous disturbance reviewofoptometry.com
Contact lens use	Recommended	Improves focus and reduces energy needs reviewofoptometry.com
IOP prophylaxis	Alpha-agonist pre/post	Prevents IOP spikes reviewofoptometry.com

Corneal Endothelium and Thickness in Diabetes

The cornea in patients with diabetes mellitus, particularly type 2 diabetes mellitus (T2DM), exhibits characteristic structural and functional alterations that distinguish it from non-diabetic corneas. These changes are clinically important when considering the corneal response to intraocular surgery or laser procedures. Two parameters are especially relevant: corneal endothelial cell density (ECD) and central corneal thickness (CCT), both of which reflect endothelial health and functional reserve [14].

Multiple studies have demonstrated that diabetic individuals have a significantly lower ECD compared with age-matched non-diabetic controls, even in the absence of prior ocular surgery. Reported mean ECD values in diabetics are consistently lower, and this quantitative loss is accompanied by qualitative morphological changes. Diabetic corneas show increased polymegathism, reflected by a higher coefficient of variation in cell size, and increased pleomorphism, indicated by a reduced proportion of hexagonal cells [15]. These features suggest chronic subclinical endothelial stress and reduced cellular stability in diabetes.

The pathophysiology underlying these changes is multifactorial. Chronic hyperglycemia leads to activation of the polyol pathway within corneal tissues, resulting in intracellular sorbitol accumulation and osmotic stress. In parallel, advanced glycation end-products accumulate in corneal proteins and extracellular matrix, potentially affecting Descemet's membrane and endothelial cell adhesion [16]. Diabetes is also associated with increased oxidative stress and a pro-inflammatory milieu in the aqueous humor, which further compromises endothelial cell survival and function.

Endothelial pump dysfunction represents a critical functional consequence of these structural changes. Reduced Na^+/K^+-ATPase activity has been demonstrated in diabetic endothelial cells, impairing fluid transport from the corneal stroma. As a result, diabetic corneas tend to retain more stromal water, leading to increased CCT. Numerous clinical studies have confirmed that mean CCT is significantly higher in diabetic patients than in non-diabetic controls, often by 20–30 μm or more. This increase is clinically relevant, as it influences intraocular pressure measurement and reflects reduced endothelial reserve [17].

Importantly, the severity of corneal changes correlates with systemic disease burden. Longer duration of diabetes, poorer glycemic control, and the presence of diabetic retinopathy have all been associated with lower ECD and higher CCT. Patients with advanced diabetic retinopathy often

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exhibit more pronounced endothelial compromise, indicating that corneal involvement parallels microvascular disease elsewhere in the eye.

Cataract surgery further stresses the corneal endothelium, typically causing 5–15% endothelial cell loss in non-diabetic eyes. In diabetic patients, postoperative endothelial loss is consistently greater. Studies have shown that diabetics experience significantly higher endothelial cell loss after cataract surgery, reinforcing the concept that diabetic corneas are intrinsically less resilient to intraocular trauma [14,15].

These findings have direct implications for Nd:YAG laser posterior capsulotomy. Diabetic pseudophakic patients begin with lower baseline ECD and increased CCT, placing them closer to the threshold of corneal decompensation. Consequently, a similar absolute endothelial cell loss following Nd:YAG capsulotomy may represent a disproportionately larger functional impact in diabetics compared with non-diabetics. In addition, transient inflammation or intraocular pressure spikes following laser treatment may be less well tolerated in diabetic corneas [18,19].

Overall, T2DM induces a fragile, pro-edematous corneal state characterized by endothelial cell loss, morphological abnormalities, and increased corneal thickness. These alterations underscore the importance of careful pre-procedural assessment, including specular microscopy and pachymetry, in diabetic patients undergoing Nd:YAG capsulotomy and form the biological basis for their potentially heightened susceptibility to laser-induced corneal injury.

Clinical Evidence of Nd:YAG Laser Impact in Diabetic vs Non-Diabetic Eyes

Clinical evidence evaluating the corneal effects of Nd:YAG laser posterior capsulotomy has largely focused on changes in corneal endothelial cell density (ECD) and, to a lesser extent, central corneal thickness (CCT). Early studies suggested minimal endothelial impact, but more recent prospective and energy-stratified investigations demonstrate that Nd:YAG capsulotomy induces a measurable, energy-dependent endothelial cell loss. Although direct comparative studies between diabetic and non-diabetic cohorts remain limited, available data and indirect evidence suggest that diabetes may amplify corneal vulnerability to laser-induced stress [20].

In non-diabetic or mixed populations, endothelial cell loss following Nd:YAG capsulotomy typically ranges from approximately 2% to 10%, with associated increases in cell size variability and reductions in hexagonality. Importantly, multiple studies demonstrate a clear relationship between higher per-pulse energy or total delivered energy and greater endothelial loss. Ultra–short-term analyses indicate that a substantial proportion of this loss occurs immediately or within hours of the procedure, supporting a direct mechanical or shockwave-mediated mechanism [21].

Timing of capsulotomy also appears relevant. Capsulotomy performed within the first postoperative year after cataract surgery is associated with greater endothelial loss than capsulotomy performed later, likely reflecting additive stress on an endothelium that has not fully stabilized following cataract surgery. This finding is particularly pertinent for diabetic patients, who tend to develop posterior capsule opacification earlier and therefore undergo Nd:YAG capsulotomy sooner than non-diabetics [22].

Changes in central corneal thickness following Nd:YAG capsulotomy are generally transient. Most studies report no sustained increase in CCT beyond the early postoperative period in eyes with adequate endothelial reserve. Transient thickening observed in some studies typically resolves within weeks,

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indicating preserved overall deturgescence function despite measurable endothelial loss.

Direct evidence comparing diabetic and non-diabetic patients remains sparse, as many studies exclude diabetics or do not analyze them as a separate subgroup. However, given that diabetic pseudophakic eyes begin with lower baseline ECD and higher CCT, an equivalent absolute endothelial loss may represent a greater proportional and functional impact [23]. Analogous data from cataract surgery studies consistently show greater endothelial loss in diabetics, supporting the hypothesis that diabetic corneas are less resilient to intraocular stress, including Nd:YAG capsulotomy. Emerging and ongoing case–control studies are expected to clarify these differences further.

Table 2. Endothelial Cell Changes After Nd:YAG Capsulotomy in General (Non-Diabetic or Mixed) Populations

Study	Population	Key Findings	Link
Slomovic et al., 1986	39 eyes	Mean ECD loss ~2.3%; no energy correlation	jamanetwork.com
Pathak et al., 2021	50 non-diabetic eyes	~6.7% ECD loss at 1 month; ↑ CV, ↓ hexagonality	jcdr.net
Agarwal et al., 2019	Energy-stratified groups	Higher energy → greater ECD loss (10.4% vs 13.0%)	ijsr.net
Mutaf et al., 2025	Short-term analysis	Immediate ~5.8% ECD loss; correlated with pulse energy	mdpi.com
Chen et al., 2022	Early vs late YAG	Early YAG (<1 yr) → greater ECD loss than late YAG	mdpi.com

Table 2. Central Corneal Thickness Changes After Nd:YAG Capsulotomy

Study	Population	CCT Outcome	Link
Eleiwa et al., 2021	Normal & mild Fuchs eyes	No sustained CCT increase	dovepress.com
Wróblewska-Czajka et al., 2008	Mixed eyes	Transient CCT rise, resolved by weeks	link.springer.com
Ultrasound biomicroscopy studies	Mixed	No significant CCT change at 1 week–1 month	link.springer.com

Table 3. Evidence Relevant to Diabetic vs Non-Diabetic Eyes

Study	Key Insight	Link
Hayashi et al., 2002	Diabetics require	pubmed.ncbi.nlm.nih.gov

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	earlier YAG due to faster PCO	
Kudva et al., 2020	Greater endothelial loss in diabetics after cataract surgery	pubmed.ncbi.nlm.nih.gov
Samir et al., 2022	YAG generally safe; diabetics not stratified	researchgate.net
Cakir et al., 2019	Slightly poorer visual outcomes in diabetics post-YAG	pmc.ncbi.nlm.nih.gov
Alshaikhsalama et al., 2025	YAG associated with retinopathy progression	researchgate.net

In absence of abundant direct comparative studies, Table 4 provides a summary of key studies on Nd:YAG capsulotomy and corneal changes, including any notes on diabetic populations when available:

Table 4: Summary of selected studies on corneal endothelial cell (ECD) loss and central corneal thickness (CCT) changes after Nd:YAG posterior capsulotomy.

Study (Year, Country)	**Patients (n)**	**Follow-up**	**Endothelial Cell Loss**	**CCT Change**	**Notes**
Slomovic *et al.* 1986 (USA) [24]	39 eyes (mixed; some aphakic)	1–3 months	~2.3% (42 cells/mm²) – minimal	None significant reported	Early study; no correlation with energy.
Agarwal *et al.* 2019 (India) [25]	40 eyes (2 groups by energy)	6 months	Group A (low energy): 10.4%; Group B (high energy): 12.97%	Not reported	Higher laser energy led to greater loss.
Pathak *et al.* 2021 (India) [26]	50 eyes (non-diabetic)	1 month	~6.7% (from 2357 to 2199 cells/mm²); CV ↑, hexagonality ↓	Not measured	Diabetics excluded; significant morphological changes.
Chen *et al.* 2022 (Taiwan) [27]	48 eyes (20 early vs 28 late YAG)	4 weeks	Early (≤1yr post-op): ~−9% at 1wk, recovered to −3% at 4wk; Late (>1yr):	No significant difference	Early capsulotomy caused significantly more loss than late. No corneal decomp

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			~−1.5%, ns		ensation noted.
Mutaf *et al.* 2025 (Turkey) [28]	32 eyes (mixed)	1 hour	~5.8% immediate loss (2184→2057 cells/mm²); avg cell area ↑ (polymegathism)	Not reported (AC parameters studied)	Strong correlation of ECD loss with energy per shot. Immediate changes demonstrate acute endothelial impact.
Eleiwa *et al.* 2021 (Egypt) [29]	50 eyes (25 normal, 25 FECD grade1-2)	3 months	**Normal:** significant drop (value not in snippet); **FECD:** significant drop; no difference between groups reported in drop magnitude	No significant increase in either group	Mild Fuchs dystrophy eyes tolerated YAG without persistent edema. Hyperopic shift noted in FECD due to IOL movement.
Samir *et al.*	60 eyes	"Short term	Not explicitly	Not mentione	No IOP rise seen.
2022 (Egypt) [7]	(post-ECCE, mixed diabetic status)	" (not specified)	quantified in abstract (likely measured but data in full text); reported as "safe" – implies minimal clinically significant loss	d (implied no edema)	Visual acuity improved ≥1 line in all, ≥2 lines in ~88%. Diabetic subset data not given in abstract.

FECD = Fuchs endothelial corneal dystrophy. ns = not significant.

Complications and Safety Considerations

Nd:YAG laser posterior capsulotomy is widely regarded as a safe and effective procedure; however, it carries a recognized spectrum of potential complications. In patients with type 2 diabetes mellitus (T2DM), these risks warrant particular attention because diabetes-related corneal and retinal vulnerabilities may amplify certain adverse outcomes.

Corneal endothelial injury represents the most direct corneal risk of Nd:YAG capsulotomy. While clinically significant corneal decompensation is rare in eyes with healthy endothelium, eyes with reduced endothelial reserve are more susceptible. Diabetic corneas, which typically exhibit lower endothelial cell density and impaired pump function, can be considered to have a narrower safety margin. In patients with advanced endothelial compromise—such as those with coexisting guttae, prior corneal surgery, or very low baseline endothelial counts—additional laser-induced cell

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loss may precipitate prolonged corneal edema. Transient corneal edema is occasionally observed after Nd:YAG capsulotomy and usually resolves with topical corticosteroids; however, more pronounced or persistent edema suggests significant endothelial stress. In diabetic patients, closer observation and adjunctive measures such as hypertonic saline drops may be warranted [30].

Intraocular pressure (IOP) elevation is a well-recognized early complication of Nd:YAG capsulotomy, attributed to inflammatory debris and capsular fragments obstructing aqueous outflow. Without prophylaxis, transient IOP rises have been reported in up to 15–30% of eyes. The routine use of topical alpha-agonists before and after the procedure significantly reduces this riskreviewofoptometry.com. Although diabetes itself does not fundamentally alter the IOP response, some diabetic patients may have compromised trabecular function or coexisting glaucoma, making vigilant IOP monitoring particularly important [31].

Inflammation and cystoid macular edema (CME) are of greater relevance in diabetic patients. Nd:YAG capsulotomy induces a low-grade intraocular inflammatory response that can disrupt the blood–retinal barrier. Diabetic eyes, especially those with existing diabetic retinopathy, are more prone to developing CME after capsulotomy. Several studies have identified diabetes mellitus as a risk factor for post-YAG CME. Prophylactic or extended use of topical nonsteroidal anti-inflammatory drugs, alone or in combination with corticosteroids, is often recommended in diabetic patients. Coordination with retinal care is advisable in patients with active diabetic macular edema [32].

Posterior segment complications, including retinal detachment and vitreous hemorrhage, are uncommon but potentially vision-threatening. Rhegmatogenous retinal detachment occurs in a small proportion of patients after Nd:YAG capsulotomy, particularly in high myopes or eyes with predisposing peripheral retinal pathology. Diabetes is not a classic risk factor for rhegmatogenous detachment; however, recent evidence suggests that Nd:YAG capsulotomy may be associated with progression of diabetic retinopathy and increased risk of tractional complications in susceptible eyesresearchgate.net. Accordingly, diabetic patients—especially those with moderate to severe non-proliferative retinopathy—should undergo careful retinal surveillance following capsulotomy [33].

IOL-related complications, most commonly IOL pitting, result from inadvertent laser impact on the lens optic. While small pits are frequent and usually visually insignificant, careful laser focusing and appropriate posterior offset minimize this risk. Diabetes does not independently increase susceptibility to IOL damage; however, rare cases of post-YAG calcification of hydrophilic acrylic IOLs have been reported [34].

Overall, Nd:YAG capsulotomy can be performed safely in patients with T2DM when appropriate precautions are taken. These include pre-procedural corneal and retinal assessment, use of minimal effective laser energy, routine IOP prophylaxis, aggressive control of postoperative inflammation, and closer follow-up. With such measures, the benefits of restoring visual clarity and facilitating retinal evaluation generally outweigh the risks, even in this higher-risk population.

Clinical Recommendations and Future Research

Based on current evidence, Nd:YAG laser capsulotomy should be performed using an individualized, risk-adapted approach, particularly in patients with type 2 diabetes mellitus (T2DM). Laser parameters should be tailored to ocular status: in diabetic patients or those with reduced endothelial reserve, operators should initiate treatment with lower energy settings and titrate gradually, favoring multiple low-energy pulses over

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fewer high-energy shots, given evidence that energy per pulse is a key determinant of endothelial stressmdpi.com. Use of a contact capsulotomy lens is advisable to improve focusing accuracy and minimize unintended energy dispersion. A conservative capsulotomy size may be appropriate initially, with staged enlargement if required. Perioperative management should be more stringent in diabetics, including routine use of IOP-lowering agents and extended anti-inflammatory prophylaxis. The addition of topical nonsteroidal anti-inflammatory drugs for several weeks post-procedure is recommended to mitigate the heightened risk of cystoid macular edema. Follow-up protocols should reflect the patient's risk profile: diabetic patients benefit from early and repeated IOP checks, assessment of corneal clarity, and retinal evaluation with optical coherence tomography at 4–6 weeks to detect subclinical macular changes. In patients with active diabetic retinopathy or macular edema, coordination with retinal management is essential, and in some cases retinal stabilization may precede or closely follow capsulotomy. Thorough patient counseling regarding symptoms of macular edema, retinal detachment, and the need for adherence to follow-up is critical.

Future research should focus on addressing current evidence gaps, particularly through prospective studies directly comparing diabetic and non-diabetic patients undergoing Nd:YAG capsulotomy, with standardized control of laser energy, timing after cataract surgery, and baseline endothelial status. Optimal laser protocols—such as ultra-low-energy, high-pulse strategies—should be formally evaluated, especially in diabetic eyes. Pharmacologic adjuncts aimed at endothelial protection, including antioxidants or osmoprotective agents, represent a promising but underexplored area. Emerging evidence that systemic therapies such as metformin may reduce posterior capsule opacification formationpmc.ncbi.nlm.nih.gov warrants further investigation, as delaying or preventing PCO could reduce the need for capsulotomy in diabetics. Long-term studies assessing corneal health years after Nd:YAG capsulotomy, stratified by diabetes status, are needed to determine whether early endothelial insults translate into late decompensation. Advances in laser technology, alternative approaches such as femtosecond laser capsulotomy, and AI-assisted endothelial risk stratification may further enhance procedural safety. Collectively, these efforts aim to refine a procedure that is already highly effective, ensuring optimal outcomes in an expanding diabetic population.

Conclusion:

Posterior capsule opacification is a common late complication of cataract surgery, and Nd:YAG laser capsulotomy remains an effective and widely used treatment. Although generally safe, the procedure can cause modest corneal endothelial cell loss and transient ocular effects. In patients with type 2 diabetes mellitus, who are predisposed to earlier PCO and have reduced endothelial reserve with increased corneal thickness, these effects may be relatively amplified. Evidence indicates that while persistent corneal edema is rare, careful technique using minimal effective laser energy, precise focusing, and appropriate perioperative management is essential in diabetic eyes. With individualized treatment and vigilant follow-up, Nd:YAG capsulotomy can restore visual clarity safely in diabetic patients, supporting optimal long-term ocular outcomes.

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