The Review Of
DIABETIC
STUDIES OPEN ACCESS

Prophylaxis Against Macular Edema Following YAG Laser Capsulotomy In Diabetic Patients

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Abstract

Background: Diabetic patients are at increased risk of developing macular edema following Nd:YAG laser posterior capsulotomy. Prostaglandin-mediated inflammation is believed to play a key role in this complication. Topical non-steroidal anti-inflammatory drugs (NSAIDs) may reduce postoperative inflammatory changes.
Objective: To evaluate the efficacy of topical Nepafenac in the prophylaxis against macular edema following Nd:YAG laser capsulotomy in diabetic patients.
Methods: This prospective, randomized interventional study included 80 eyes of 80 type II diabetic patients with visually significant posterior capsule opacification after uneventful phacoemulsification. Patients were randomly assigned into two groups: Group I received topical corticosteroids, alpha-adrenergic agonists for one week, and Nepafenac 0.1% for four weeks; Group II received only corticosteroids and alpha-adrenergic agonists. Best-corrected visual acuity (BCVA), perifoveal macular thickness (PMT), and intraocular pressure (IOP) were assessed preoperatively and at 1, 4, and 12 weeks' post-procedure using spectral-domain OCT and standard ophthalmic examinations.
Results: Both groups showed significant improvement in BCVA after capsulotomy. However, Group I demonstrated significantly better BCVA at 1 week ($p = 0.003$). PMT was significantly lower in the Nepafenac group at all postoperative follow-ups ($p < 0.01$), indicating reduced macular thickening. Transient IOP elevation occurred in both groups at 1 week, with no significant intergroup difference and spontaneous resolution thereafter.
Conclusion: Prophylactic use of topical Nepafenac following Nd:YAG laser capsulotomy in diabetic patients effectively reduces perifoveal macular thickening and enhances early visual outcomes without affecting IOP. Routine NSAID prophylaxis is recommended in this high-risk population.

Keywords: Nd:YAG capsulotomy; Nepafenac; Macular edema; NSAIDs.

Introduction

Cataract is the most common cause of visual impairment and treatable blindness worldwide (1).
There is strong relationship between cataracts and systemic conditions (2). Diabetic patients have a higher incidence of cataract, especially cortical cataract (CC) and posterior subcapsular cataract (PSC). Besides diabetic patients are more likely to acquire cataracts at a younger age because of hyperglycaemia and damaged blood–aqueous barrier.
Globally, cataract surgery is one of the most common surgical procedures performed; in developed nations, reported rates range from 4,000 to 10,000 per million. Among people with diabetes, cataract surgery is now the most common surgical operation (2)
Following the widespread use of phacoemulsification in the 1990s, surgery now typically involves small incision, suture-less phacoemulsification as a day case procedure under local anesthesia. Patients can often be discharged within an hour of surgical completion (3).
Cataract surgery carries a higher risk of complications in diabetic patients They have an increased risk of developing posterior capsular opacification and postoperative cystoid macular edema (CME) which are among the most frequent causes of decreasing visual acuity in diabetic patients. Also diabetic macular edema (DME) or developing diabetic retinopathy may increase complications in the postoperative phase (4).

Despite the modern techniques of phacoemulsification, diabetic patients are still at higher risk of developing post-operative cystoid macular edema than non-diabetic patients (5). The incidence of clinically significant macular edema (CSME) after cataract surgery may reach up to 56% in diabetic patients even those with mild non proliferative diabetic retinopathy (NPDR) and having no CSME before operation (6).

Diabetic patients have been found to have a higher incidence of posterior capsule opacification (PCO) onset and severity when compared to non-diabetic patients. Numerous researches have demonstrated a connection between the design of intaocular lens (IOL) material and shape and the development of PCO. Since a square edge design appears to interfere with lens epithelial cell growth, PCO development may be avoided. Numerous studies also have demonstrated that hydrophillicic IOLs are more susceptible to opacifaction than hydrophobic IOLs in diabetic patients (7).

Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used by doctors due to their analgesic, antipyretic, and anti-inflammatory qualities since they are strong inhibitors of the enzyme cyclooxygenase (COX), which is an essential trigger in the inflammatory cascade. Prostaglandins in the eye induce leucocyte migration, vasodilation, breakdown of the blood-aqueous barrier and miosis. So they are frequently used by ophthalmologists to treat macular edoema following cataract surgery, reduce intraoperative miosis and reduce postoperative inflammation (8).

The aim of this work was to discuss the role of NSAIDs in prophylaxis against macular oedema which may occur in diabetic patients after YAG laser capsulotomy

Patients and methods

This was a prospective, randomized, interventional clinical study. Patients were recruited from the Ophthalmology Clinic at the Faculty of Medicine, Aswan University, and Aswan Ophthalmology Hospital. All procedures were performed in the Ophthalmology Departments of both institutions

Study Population

The study included type II diabetic patients who developed visually significant posterior capsule opacification (PCO) following uneventful phacoemulsification cataract surgery and presented to the participating clinics.

Sample Size Calculation

Based on the study by Raj et al. (9), the incidence of diabetic patients undergoing Nd:YAG laser capsulotomy within 3–5 years postoperatively was estimated at 4.2% (95% CI: 3.3%–5.1%). The required sample size was calculated using the formula:

$$n = \frac{Z^2 \cdot P(1-P)}{d^2}$$

Where:
P=0.042
Z=1.96 (95% confidence interval)
d=0.05 (margin of error)
A sample size of 60 patients was considered sufficient; however, 80 patients were recruited to account for possible dropouts.

Randomization

Eighty eyes of eighty patients were randomly assigned in a 1:1 ratio using a computer-generated randomization sequence into two groups:

Group I (NSAID Group): 40 eyes of 40 patients received topical corticosteroid and alpha-adrenergic agonist eye drops for one week following Nd:YAG laser capsulotomy, in addition to topical non-steroidal anti-inflammatory eye drops for four weeks.

Group II (Conventional Group): 40 eyes of 40 patients received topical corticosteroid and alpha-adrenergic agonist eye drops for one week following Nd: YAG laser capsulotomy.

Inclusion Criteria
Patients were included if they met the following criteria: Diagnosed with type II diabetes mellitus, with HbA1c ≤ 7.5%, Duration of diabetes ≤ 5 years, Presence of visually significant PCO (grade 1 or 2 on slit-lamp examination), Axial length between 21 mm and 24 mm, Baseline central macular thickness (CMT) ≤ 300 μm, as measured by SD-OCT and No or mild diabetic retinopathy, confirmed clinically and via OCT without diabetic macular edema (DME).

Exclusion Criteria
PCO grade 3 or membranous type or significant media opacity precluding adequate retinal imaging, Coexisting glaucoma or use of anti-glaucoma medications, Past history of uveitis, Presence of macular pathology (e.g., CNV), active retinal vascular diseases (e.g., proliferative diabetic retinopathy, retinal vascular occlusion), or diabetic macular edema, History of retinal laser procedures or intraocular injections, Intra- or postoperative complications affecting visual outcome (e.g., IOL damage or dislocation, severe iris injury, corneal edema due to endothelial damage, hyphema, retinal breaks or detachment, Corneal thinning or severe dry eye disease and Failure to comply with follow-up schedule.

Baseline Evaluation
All patients underwent a comprehensive baseline assessment, including:
Demographic and clinical data collection, Uncorrected and best-corrected visual acuity (BCVA), converted to logMAR, Slit-lamp examination after pharmacologic mydriasis, Intraocular pressure (IOP) measurement using Goldmann applanation tonometry, Axial length measurement via A-scan biometry, Fundus examination with a +90D non-contact lens and Spectral-domain OCT (Nidek RS-3000, Japan) for macular architecture and CMT measurement using the ETDRS grid. All OCT scans were performed by a single ophthalmologist for consistency.

Nd:YAG Laser Capsulotomy Procedure
All patients received verbal and written information about the procedure, including its purpose, safety, and expected visual outcomes. The importance of maintaining steady fixation and awareness of the clicking noise generated by the laser was explained. Informed consent was obtained from all participants. All laser procedures were performed by the same experienced ophthalmologist (A.H.) to maintain standardization.

Pre-Procedural Preparation
IOP Prophylaxis: Brimonidine tartrate 0.2% (Brimonidine®, Jamjoom Pharma, Saudi Arabia) was instilled one hour before the procedure to prevent IOP elevation.
Mydriasis: Tropicamide 1% and Phenylephrine 2.5% (Cyclophrine®, Kahira Pharma, Egypt) were instilled three times at 10-minute intervals to achieve adequate dilation.
Topical Anesthesia: Benoxinate hydrochloride 0.4% drops were administered immediately prior to the procedure.

Laser Technique
A contact-type Abraham capsulotomy lens (Ocular Inc., USA) was used to stabilize the eye and focus laser energy. The procedure was performed using the Ellex Ultra-Q Nd:YAG laser system (Nova Eye Medical, USA), with a 150 μm posterior offset. A cruciate capsulotomy pattern was created, aiming for a 4–5 mm diameter based on the patient's scotopic pupil size. Laser energy ranged between 4 and 8 mJ per pulse. A total of 8 to 20 shots were applied per eye to achieve an adequate central opening in the posterior capsule. The total number of laser pulses and total energy used were recorded for each patient.

Post-Procedural Regimen
All patients received:
Prednisolone acetate 1% eye drops (Optipred®, Jamjoom Pharma), instilled four times daily for one week. Brimonidine tartrate 0.2% (Brimonidine®, Jamjoom Pharma), instilled twice daily for one week.

Additionally, patients in Group I were prescribed: Nepafenac 0.1% eye drops (Nevanac®, Alcon, USA), three times daily for four weeks. Patients were instructed on proper medication use and advised to report any symptoms such as pain, photophobia, sudden visual changes, or signs of inflammation.

Follow-up Schedule

All patients were followed at: 1 week, 4 weeks and 12 weeks.

Post-capsulotomy, at each visit, the following were assessed:

Best-corrected visual acuity (BCVA) in logMAR. Slit-lamp biomicroscopy post-dilation. Intraocular pressure (IOP) via Goldmann tonometry. Fundus examination using a +90D lens. SD-OCT (Nidek RS-3000) for macular assessment and measurement of central macular thickness (CMT).

Results

Table (1) Baseline characteristics of the studied patients:

Variables	Group 1 (no=40)	Group 2 (no=40)	P-value
Age (mean±SD)	60.60±5.62	61.05±6.73	0.747
Sex Male Female	 18(45.0%) 22(55.0%)	 20(50.0%) 20(50.0%)	0.564
Disease duration(DM)/Years [median(IQR)]	6.00(5.00,7.75)	5.00(4.25,6.75)	.416

Table 1 presents the baseline characteristics of the studied patients, comparing the interventional (Nevanac) and control groups. The two groups were well-matched in terms of age, sex distribution, and disease duration, with no statistically significant differences observed (P-values: 0.747, 0.564, and 0.416, respectively).

Table (2) Interventional details of the studied groups:

Variables	Group 1 (no=40)	Group 2 (no=40)	P-value
YAG energy 4 5 6	 7(17.5%) 17(42.5%) 16(40.0%)	 10(25.0%) 17(42.5%) 13(32.5%)	0.657
Number of shots 8 9 10 11 12	 9(22.5%) 8(20.0%) 8(20.0%) 8(20.0%) 7(17.5%)	 8(20.0%) 7(17.5%) 9(22.5%) 6(15.0%) 10(25.0%)	0.910
Axial length [median(IQR)]	23.41(22.61,23.97)	23.12(22.34,23.77)	.260
Duration of intervention/minutes [median(IQR)]	4.00(3.00,4.00)	4.00(3.00,4.75)	.792

Table 2 outlines the interventional details of the studied groups, comparing YAG laser energy levels, number of shots, axial length, and duration of intervention between the interventional (Nevanac) and control groups. The distribution of YAG energy and number of shots was similar across both groups, with no statistically significant differences (P-values: 0.657 and 0.910, respectively). Additionally, the median axial length and the duration of intervention were comparable between the groups (P-values: 0.260 and 0.792, respectively).

Table (3) Following up the best corrected visual acuity (Log-MAR) of the studied patients:

BCVA [median(IQR)]	Group 1 (no=40)	Group 2 (no=40)	P-value
Pre-operative	0.60(0.50,0.70)	0.60(0.50,0.74)	0.457
1 week	0.20(0.14,0.20)	0.22(0.18,0.31)	0.003*

4 weeks	0.10(0.10,0.15)	0.12(0.10,0.12)	0.071
12 weeks	0.10(0.10,0.12)	0.12(0.10,0.12)	0.110
Pre vs 1 week	<0.001*	<0.001*	
Pre vs 4 weeks	<0.001*	<0.001*	
Pre vs 12 weeks	<0.001*	<0.001*	
1 week vs 4 weeks	<0.001*	<0.001*	
1 week vs 12 weeks	<0.001*	<0.001*	
4 weeks vs 12 weeks	0.024*	0.026*	

*P-value is significant

Table 3 presents the follow-up of best-corrected visual acuity (BCVA) in LogMAR for both studied groups. Pre-YAG, BCVA was comparable between the groups (P = 0.457). However, significant improvements were observed Post-YAG, with the interventional (Nevanac) group demonstrating superior visual outcomes at all follow-up points. At 1, the Nevanac group exhibited significantly better BCVA compared to the control group (P = 0.003). However, the BVCA was better in NEvenac group without statistical significance at 4 and 12 weeks Within-group comparisons also revealed significant improvements over time in both groups (P < 0.05 for all timepoints)

Table (4) Following up the Perifoveal macular thickness (PMT) of the studied patients:

PMT [median(IQR)]	**Group 1 (no=40)**	**Group 2 (no=40)**	**P-value**
Pre-YAG	287.00(281.00,291.00)	285.50(280.00,289.75)	0.330
1 week	293.50(286.00,297.00)	313.00(309.00,319.75)	<0.001*
4 weeks	281.00(277.25,287.00)	288.00(281.25,294.75)	0.002*
12 weeks	279.00(270.00,284.75)	282.50(278.25,288.75)	0.006*
Pre vs 1 week	<0.001*	<0.001*	
Pre vs 4 weeks	0.049*	0.008*	
Pre vs 12 weeks	<0.001*	0.068	
1 week vs 4 weeks	<0.001*	<0.001*	
1 week vs 12 weeks	<0.001*	<0.001*	
4 weeks vs 12 weeks	0.004*	<0.001*	

*P-value is significant

Table 4 tracks the perifoveal macular thickness (PMT) changes in both study groups over time. Pre-YAG, there was no significant difference between the groups (P = 0.330). However, at 1 week Post-YAG, PMT was significantly higher in the control group compared to the Nevanac group (P < 0.001), indicating early macular thickening in the absence of NSAID prophylaxis. By 4 and 12 weeks, the Nevanac group consistently demonstrated significantly lower PMT values compared to the control group (P = 0.002 and 0.006, respectively). Within-group comparisons showed significant changes in PMT over time, with the Nevanac group exhibiting a more controlled reduction in thickness.

Table (5) Following up the intraocular pressure in the Nevanac group:

IOP [median(IQR)]	**Group 1 (no=40)**	**Group 2 (no=40)**	**P-value**
Pre-YAG	15.00(14.00,16.00)	16.00(14.25,17.00)	0.064
1 week	17.00(16.00,17.75)	17.00(16.00,18.00)	0.352
4 weeks	15.00(15.00,16.00)	16.00(15.00,16.00)	0.494
12 weeks	15.00(14.00,15.75)	15.00(14.00,16.00)	0.459
Pre vs 1 week	<0.001*	<0.001*	
Pre vs 4 weeks	0.178	0.642	
Pre vs 12 weeks	0.102	0.006*	
1 week vs 4 weeks	<0.001*	<0.001*	
1 week vs 12 weeks	<0.001*	<0.001*	
4 weeks vs 12 weeks	0.003*	0.004*	

*P-value is significant

Table 5 examines the intraocular pressure (IOP) changes over time in both study groups. Pre-YAG, there was no significant difference in IOP between the groups (P = 0.064). At 1 week Post-YAG, IOP increased in both groups compared to baseline (P < 0.001), likely due to transient post-intervention changes. However, by 4 and 12 weeks, IOP levels had generally returned to near-baseline values, with no significant differences observed between the groups at these time points (P = 0.494 and 0.459, respectively). Within-group comparisons showed significant fluctuations over time, particularly between 1-week and later follow-ups (P < 0.001).

Discussion

To isolate the effect of Nepafenac, we ensured that both the interventional group and controls were comparable at baseline, on the demographic and the procedural level, with similar age and sex distribution, as well as similar disease duration.

In the same sense, both groups were subjected to similar YAG energy levels (p=0.657), and both the number of shots and the axial length did not significantly vary (p=0.910, p=0.260, respectively). The duration of the procedure was almost the same in both groups (p=0.792). Next, we used the Log-MAR visual acuity chart to estimate the best corrected visual acuity (BCVA) for both groups, in which lower scores correspond to improvement in visual acuity (10). Prior to the procedure, no statistically significant differences could be noted (p=0.457); however, after one week, significantly better BCVA was observed in Group 1, who were assigned to receive Nepafenac, as opposed to controls (0.20 vs. 0.22, p=0.003). Likewise, even better values were noted in Group 1 compared to controls at the 4-week follow-up without statistical significance (0.10 vs. 0.12, P=0.071), and this improvement stabilized at a median of 0.10 for Group 1 at the 12-week timepoint, and to 0.12 for controls (p=0.110). We ascertained the significance of improvement after YAG laser capsulotomy throughout the follow-up period and in comparison, to pre-procedural BCVA in both groups, irrespective of Nepafenac use (p<0.001). Miyake et al.(11) relayed data endorsing ours in terms of the superior improvement in visual acuity in the Nepafenac group, demonstrating that 80% of patients in the Nepafenac group experienced a change of 3 or more lines on the logMAR chart compared to 55.2% of patients who were treated with fluorometholone (p=0.0395).

To monitor postprocedural changes, we assessed perifoveal macular thickness (PMT) changes in both groups and while both groups were statistically indistinguishable at baseline (p=0.330), one week after the procedure, controls exhibited a significantly greater PMT compared to the interventional arm (313 vs. 293.50, p<0.001). This was also evident at both the 4-week (288 vs. 281, p=0.002) and the 12-week follow-up (282.50 vs. 279, p=0.006). Furthermore, within-group comparisons revealed that although both groups experienced an increase in PMT on the 1st postprocedural week, compared to their respective baseline, this tended to dwindle by the 4th week. Group 1, who were given topical Nepafenac, demonstrated a statistically substantial drop in PMT from baseline at the 12-week follow-up (279 vs. 287, p<0.001). Dissimilarly, the median PMT on the 12th week was comparable to baseline in the control arm (282.50 vs. 285.50, p=0.068). Given the comparability of both groups at baseline, we attributed this variation in postprocedural PMT to the use of Nepafenac in Group 1. In alignment with our hypothesis, Atilgan et al. (12) showed that topical Nepafenac following YAG capsulotomy was associated with progressive reduction of the superior quadrant PMT from baseline, at the 1-week and 1-month follow-up (299.6 vs. 293 vs. 294.3, p=0.371), as opposed to controls who were untreated, displaying progressively increasing PMT from baseline (296.3 vs. 301.1 vs. 302, p=0.039). Similarly, temporal quadrant PMT was reduced from a baseline of 284 to 274.7 at the 1-week timepoint, and stabilized at 279.8 after one month, and these differences were statistically significant (p<0.001). On the other hand, patients who were treated with Fluorometholone experienced unchanged temporal PMT from baseline at the 1-week timepoint, and an increase after one month (274.1 vs. 274.3 vs. 278.5, p=0.149).

We explained the favorability of topical NSAIDs by elucidating the underlying mechanism of CME after YAG capsulotomy, which is attributed to high perifoveal capillary permeability due to prostaglandins—products of arachidonic acid metabolism, which are synthesized on surgical manipulation of the iris, ciliary body, or the epithelial cells of the lens, such as during cataract surgery or following YAG laser capsulotomy (11).

Lastly, we compared both groups in terms of the intraocular pressure, at baseline as well as at the 1-, 4-, and 12-week timepoints, yet no statistically meaningful differences could be observed (p=0.064,

p=0.352, p=0.494, p=0.459, respectively). Moreover, when comparing IOP at each follow-up point to pre-procedural values in Group 1, the only statistically notable difference was at the 1-week timepoint, with a slight but significant increase in IOP (17 vs. 15, p<0.001), that returned to baseline by the 4th week (15 vs. 15, p=0.178). This was similarly observed in Group 2 (17 vs. 16, p<0.001; 16 vs. 16, p=0.642, respectively); however, by the 12th week, we noted a drop in IOP from baseline that proved to be statistically significant (15 vs. 16, p=0.006), unlike Group 1 who merely reverted to baseline value (15 vs. 15, p=0.102). Shah et al. (13) reported an increase in mean IOP from 15.2 mmHg to 17.0 mmHg following YAG laser capsulotomy, illustrating a statistically significant elevation (p<0.001).
Similarly, Kalla and Prasnnta (14) highlighted that peaks of IOP typically occur within three hours after YAG capsulotomy, confirming the transient nature of this increase.

Conclusion

Our trial demonstrated that the prophylactic use of topical Nepafenac (Nevanac) following YAG laser capsulotomy in diabetic patients significantly improved visual acuity and reduced PMT compared to controls, highlighting its efficacy in mitigating inflammatory complications such as CME. The findings aligned with previous studies suggesting that NSAIDs, by inhibiting prostaglandin synthesis, play a critical role in reducing postprocedural inflammation and edema, particularly in high-risk populations like diabetics. While both groups experienced transient increases in IOP after the procedure, these changes were not significantly influenced by Nepafenac and resolved over time. Thus, we emphasized the importance of prophylactic NSAID use in diabetic patients undergoing YAG capsulotomy to optimize visual outcomes and minimize complications such as macular edema. However, further research is warranted to explore the long-term effects of Nepafenac and its role in IOP fluctuations. Overall, these findings endorsed the integration of topical NSAIDs into post-YAG capsulotomy care protocols, particularly for patients at higher risk of inflammatory complications, namely those with diabetes mellitus.

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