

Research Article

Pattern of Rise in Intraocular Pressure Following Nd:Yag Laser Posterior Capsulotomy

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ABSTRACT

Background: Posterior capsular opacification (PCO) remains the most common delayed cause of visual decline after cataract surgery. Nd:YAG laser posterior capsulotomy restores the visual axis but can precipitate transient intraocular pressure (IOP) elevation. The necessity and scope of prophylactic anti-glaucoma therapy in routine practice—particularly as a function of delivered laser energy—remain debated.

Methods: In a prospective observational study at a tertiary eye care center (November 2023–September 2025), 87 pseudophakic eyes with visually significant PCO underwent Nd:YAG capsulotomy; fellow eyes served as controls. No prophylactic anti-glaucoma medication was administered. IOP (non-contact tonometry) was recorded at baseline and 0.5, 1, 2, 3, 4, and 24 hours post-procedure. Total laser energy (mJ) was computed as pulse energy × number of pulses. “Significant IOP rise” was predefined as ≥ 6 mmHg from baseline in the treated eye or an inter-eye difference ≥ 6 mmHg. Associations with energy, age, and sex were analyzed (t-test, chi-square; $p < 0.05$).

Results: Significant IOP rise occurred in 16/87 eyes (18.4%). The mean onset of the spike was 1.4 hours (range 0.5–3.0), with 62.5% peaking at 1 hour. In the spike cohort, the mean magnitude was 7.5 mmHg (range 6–14); one eye reached 30.4 mmHg and received oral acetazolamide. By 24 hours, 87.5% of spikes had resolved; two eyes required observation up to 48 hours. Higher total energy correlated with IOP elevation ($t = 2.688$; $p = 0.009$). Incidence rose with energy strata: 8.3% (< 21 mJ), 25.0% (21–40 mJ), and 45.5% (> 40 mJ). Age and sex showed no significant associations.

Conclusion: Post-Nd: YAG capsulotomy IOP spikes are infrequent, early, and typically self-limited, but their risk increases with higher total energy. A pragmatic protocol is suggested: no prophylaxis for < 21 mJ; a 1-hour post-procedure IOP check for 21–40 mJ; and immediate anti-glaucoma treatment when > 40 mJ or if the measured IOP is elevated. Multi-session, low-energy strategies may further mitigate risk.

Keywords: Posterior Capsular Opacification, Nd:YAG Capsulotomy, Intraocular Pressure, Laser Energy, Glaucoma Prophylaxis, Pseudophakia.

INTRODUCTION

Posterior capsular opacification (PCO)—often called “after-cataract”—is a wound-healing response in which residual lens epithelial cells proliferate and transdifferentiate into myofibroblasts that migrate over the posterior capsule, culminating in Elschnig pearl formation and fibrotic opacity. Clinically, PCO degrades contrast, induces glare, and blurs vision; its cumulative incidence after extracapsular cataract extraction or phacoemulsification spans a wide range in the literature, with systematic estimates exceeding one quarter of patients at five years [1]. Management is either surgical capsulotomy (reserved for pediatric eyes or unusually thick fibrosis) or Nd:YAG laser

posterior capsulotomy, the contemporary standard in adults because it is rapid, non-incisional, and performed at the slit lamp. Q-switched Nd:YAG pulses photodisrupt the opacified capsule to clear the visual axis, typically achieving immediate visual rehabilitation [2]. Despite its efficiency, Nd:YAG capsulotomy is accompanied by short-term adverse events, notably transient IOP elevation. Mechanistically, pulses liberate capsular and pigment debris, lens epithelial cell remnants, and inflammatory mediators (e.g., prostaglandins) that can transiently obstruct the trabecular meshwork and reduce outflow facility [3]. The reported spike frequencies vary significantly between series because of

heterogeneity, as to patient selection, energy delivery, IOP measuring techniques and the application (or not) of prophylactic therapy (topical beta-blocker, alpha-agonist or oral carbonic anhydrase inhibitor) [4-5]. Energy dosing is a suitable (and clinical rule Pays) risk determinant. Studies have correlated increases of pulse energy with increases of cumulative energy with increases of IOP rises [6-7]. However, a large number of the patients may not need to receive blanket prophylactic treatment and it imposes cost, polypharmacy and side-effect charges on a large number of patients in whom glacialis may well add to symptoms. A data informed energy guided approach would be a good way to limit overtreatment but with protection of the vulnerable patient. We thus prospectively evaluated the temporal aspect and amplitude of IOP changes in the first 24-hour after Nd:YAG capsulotomy using paired fellow pulsely as intra-subject controls in an inter-relational fashion [8]. We investigated 3 important questions (1) What is the proportion of eyes with a clinically significant increase in IOP in early stages. (1) What is the relationship between cumulative energy of Nd:YAG and magnitude and probability of spike and (2) what is the relationship between patient factors including age and sex and IOP behavioral response. The answers are directed at the establishment of better post-capsulotomy surveillance and treatment regimens and the incorporation of efficient and yet safe practices in day-to-day practice.

MATERIALS AND METHODS

Study Design and Participants

Methods: It was a prospective observational study conducted in the tertiary OPD of Ophthalmology Department between November 2023-September 2025. Eighty-seven eyes that were pseudophakic and 85 adults in the treatment naive group with visually significant PCO are included. Eighty-seven pseudophakic eyes that were treated for the first time in treatment naive adults with visually significant are included. The fellow eye of each patient served as a control. Written informed consent was obtained before enrollment.

Inclusion Criteria: cooperative adults (≥ 18 years) with PCO causing ≥ 2 -line Snellen acuity loss and graded as Sellman–Lindstrom Grade 3 or 4 on slit-lamp biomicroscopy [6].

Exclusion Criteria: monocular status; pediatric patients; coexisting ocular disease

that could confound IOP (uncontrolled glaucoma, uveitis, retinal pathology); concurrent systemic/topical steroids.

Procedures

Baseline assessment recorded demographics, best-corrected visual acuity (Snellen), anterior segment evaluation, PCO grade, and fundus status (direct/indirect ophthalmoscopy). Baseline IOP in both eyes was measured using a calibrated non-contact tonometer. Pupils were dilated with 1% tropicamide (three doses, 10-minute intervals); proparacaine provided topical anesthesia.

Nd:YAG capsulotomy (Zeiss Visulas YAG III) created a 3–4 mm central posterior capsulotomy. The focal point was placed slightly posterior to the capsule to minimize IOL pitting; energy per pulse and the number of pulses were titrated to achieve a clean opening while keeping energy as low as reasonably achievable. Total energy (mJ) was recorded (pulse energy $\times$ pulses).

Post-procedure IOP was measured at 0.5, 1, 2, 3, 4, and 24 hours in both eyes. No routine anti-glaucoma prophylaxis was given. Topical ketorolac 0.5% was prescribed q.i.d. for one month. If IOP exceeded 30 mmHg at any time, measurements ceased and oral acetazolamide 250 mg was administered immediately. Patients were educated about warning symptoms (pain, halos, sudden blur).

Outcomes

The primary outcome was the proportion of eyes with significant IOP rise, defined a priori as either (a) ≥ 6 mmHg increase from baseline in the treated eye or (b) an inter-eye IOP difference ≥ 6 mmHg. Secondary outcomes included time-to-peak, magnitude, and duration of spikes; association of spikes with cumulative energy (mJ), and with age or sex.

Statistical analysis

Energy–IOP relationships were evaluated by independent-samples t-test; categorical associations (e.g., energy strata, age groups) used chi-square testing. Two-tailed $p < 0.05$ was significant. Analyses were performed on complete cases.

RESULTS

Eighty-seven treated eyes (study eyes) from 85 adults completed the 24-hour protocol, each paired to its fellow-eye control. Overall, 16/87 eyes (18.4%) met the prespecified criterion for a significant IOP rise (≥ 6 mmHg from baseline in the treated eye or an inter-eye difference ≥ 6

mmHg), while 71/87 (81.6%) did not. Spikes occurred early, with a mean onset of 1.4 hours (range, 0.5–3.0 h) and 62.5% peaking at 1 hour. Among spike eyes, the mean magnitude was 7.5 mmHg (range, 6–14 mmHg). One eye reached 30.4 mmHg, triggering protocol-mandated oral acetazolamide; all other spikes were observed without rescue therapy. By 24 hours, 14/16 (87.5%) had returned to within 6 mmHg of baseline; two eyes normalized by 48 hours with conservative care. Cumulative Nd:YAG energy (pulse energy × number of pulses) was higher in eyes with spikes (mean 32.94 mJ, SD 20.40) versus without spikes (mean 20.55 mJ, SD 15.74) (Table 1). The between-group difference (12.39 mJ) was statistically significant (independent-samples $t = 2.688$, $df = 85$, $p = 0.009$; Table 2) and

corresponded to a moderate effect size (Cohen's $d = 0.74$). Incidence rose monotonically across energy strata: 8.3% for <21 mJ, 25.0% for 21–40 mJ, and 45.5% for >40 mJ (Table 3). A five-bin view (1–20, 21–40, 41–60, 61–80, 81–100 mJ) showed the same dose–response pattern and an overall chi-square $p = 0.027$ (with small expected counts in the higher-energy tail), reinforcing energy as the main modifiable determinant of early IOP perturbation. Age distribution did not differ meaningfully by spike status (Table 4; $\chi^2 = 1.760$, $df = 4$, $p = 0.780$). No material differences by sex were detected (data not shown). Apart from the single high-magnitude spike (30.4 mmHg) that was promptly treated, no serious adverse events occurred. Visual axis clearance was achieved in all cases.

Table 1: Total Nd:Yag Energy By Spike Status (Independent-Samples T-Test)

Spike status	n (eyes)	Mean energy (mJ)	SD	SE
Significant IOP rise	16	32.94	20.401	5.100
No significant rise	71	20.55	15.737	1.868
Total	87	—	—	—
t (df), p	—	2.688 (85)	—	p = 0.009

Table 2: Incidence of Significant Iop Rise by Energy (3 Strata; Chi-Square)

Energy group (mJ)	Yes (n / %)	No (n / %)	Total	Row % with spike
<21	4 / 8.3%	44 / 91.7%	48	8.3%
21–40	7 / 25.0%	21 / 75.0%	28	25.0%
>40	5 / 45.5%	6 / 54.5%	11	45.5%
Total	16 / 18.4%	71 / 81.6%	87	18.4%
χ^2 (df), p	—	—	—	$\chi^2 = 8.86$ (2), p = 0.012*

Table 3: Incidence of Significant Iop Rise by Energy (5 Bins; Chi-Square)

Energy group (mJ)	Yes (n / %)	No (n / %)	Total	Row % with spike
1–20	4 / 8.3%	44 / 91.7%	48	8.3%
21–40	7 / 25.0%	21 / 75.0%	28	25.0%
41–60	4 / 50.0%	4 / 50.0%	8	50.0%
61–80	0 / 0.0%	1 / 100%	1	0.0%
81–100	1 / 50.0%	1 / 50.0%	2	50.0%
Total	16 / 18.4%	71 / 81.6%	87	18.4%
χ^2 (df), p	—	—	—	$\chi^2 = 10.93$ (4), p = 0.027 †

Table 4: Distribution of Significant Iop Rise by Age Group (Chi-Square)

Age (years)	Yes (n / %)	No (n / %)	Total	Row % with spike
37–47	2 / 33.3%	4 / 66.7%	6	33.3%
48–57	3 / 13.6%	19 / 86.4%	22	13.6%
58–67	5 / 17.9%	23 / 82.1%	28	17.9%
68–77	4 / 16.7%	20 / 83.3%	24	16.7%
78–87	2 / 28.6%	5 / 71.4%	7	28.6%
Total	16 / 18.4%	71 / 81.6%	87	18.4%
χ^2 (df), p	—	—	—	$\chi^2 = 1.760$ (4), p = 0.780

Figure 1: Temporal Profile Of Mean Δ Iop (Treated Vs Fellow Eye)

Figure 1. Temporal profile of mean Δ IOP (treated vs fellow eye)

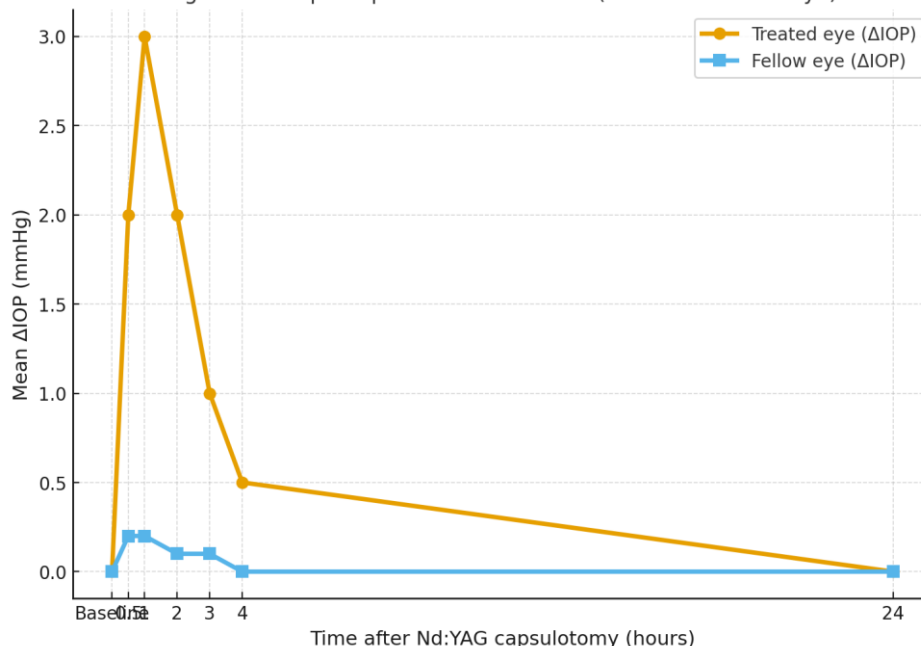
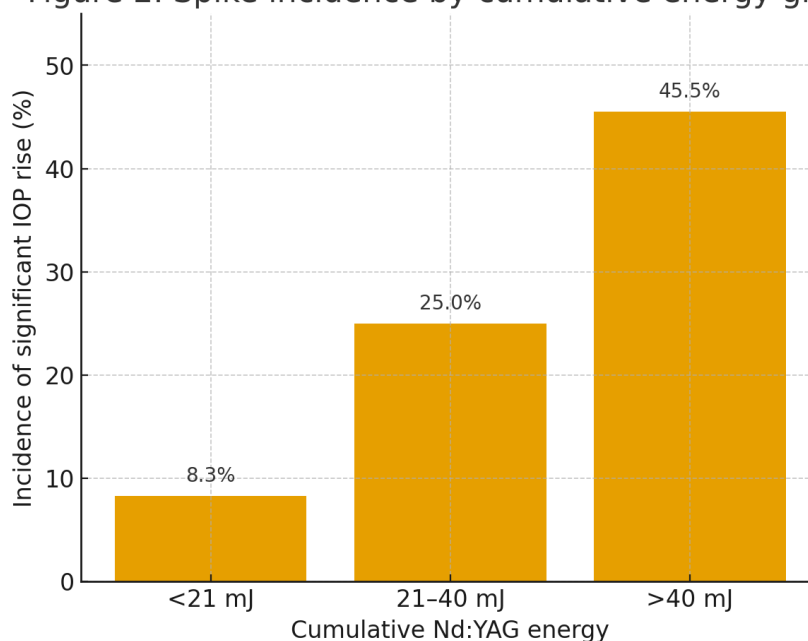


Figure 2: Spike Incidence by Cumulative Energy Group

Figure 2. Spike incidence by cumulative energy group



DISCUSSION

Our prospective series demonstrates that clinically meaningful IOP elevation after Nd:YAG posterior capsulotomy is infrequent (18.4%), early (mean 1.4 h), and self-limited in most cases (87.5% resolved by 24 h) when procedures are performed with judicious energy and without routine prophylaxis. The

distribution and timing align with the pathophysiology of transient outflow compromise due to capsular debris, pigment, and inflammatory mediators released by photodisruption. The single case requiring immediate systemic therapy underscores that while uncommon, high-magnitude spikes do

occur and are clinically meaningful. Development of Energy as Major Changeable Risk-Determinate Manductor Both the mean difference testing and the categoric analysis showed a very clear gradient such that readout of <21mJ showed an relatively low risk (8.3%), 21-40mJ shared a quarter of eyes show spikes and/or >40mJ moved towards about one in two eyes with spikes. These results are consistent with those in other reports supporting the hypothesis of a dose effect of pulse energetic and numbers on the IOP perturbation [9-11]. Mechanistically, this increased energy is most likely to be responsible for increased particulate load and in-flames within the anterior segment which is capable of transiently amplifying trabecular clearing. Energy saving technique is chosen through sharp and constant gradient and multi-session strategy (high burden division among sittings) is adopted for decreasing the exposure regarding energy per session. At 1 hour, our spike rate is lower as compared to other series reported in the literature [3; 11; 12], wherein Hassan reported 100% and Murtaza et al. 61.7% early iza weed spike, probably due to use of different energy delivery and selection of patients and the prophylaxis policy. The one literature closer to the studies in which the number of spikes has been considered rare in low-risk cohorts, some in a situation of a very low incidence and energy and in favorable conditions in the anterior segment [13, 14]. Importantly, compared to the current baseline of no prophylaxis, blanketed, pretreatment is not necessary for the majority of routine and low energy investment procedures and hence, does not suffer from the untoward side effects and costs of universal drug prophylaxis. Age, sex were not related to spike occurrence as expected since the parameters used to perform the IOP procedure are considered to be more important than demographic characteristics in the early stages of IOP dynamics. However, some anatomic predispositions (e.g. shallow chambers) and preexisting glaucoma could result in a higher risk; the numbers of glaucoma patients in our cohort were very low, which means that rigorous trials inhigh risk eyes are still warranted. The clinical implications are obvious and clear. Last of all, I believe that prophylaxis should not be more than 21 mJ. Second, for 21–40 mJ, perform a 1-hour IOP check and treat only if elevated. Third, for >40 mJ or if clinical assessment anticipates higher energy, administer prompt IOP-lowering therapy and consider staged capsulotomy. Finally, educate

patients to report pain or sudden blur immediately. Limitations include uneven representation of the highest energy bins (small n), reliance on non-contact tonometry, and follow-up limited largely to 24 hours (two cases extended to 48 hours, suggesting occasional prolonged dynamics). Future studies should incorporate Goldmann applanation, larger high-risk strata, and 1-week follow-up to capture delayed events.

CONCLUSION

In this prospective study of 87 pseudophakic eyes, significant IOP elevation after Nd:YAG posterior capsulotomy occurred in 18.4%, peaked early (~1.4 hours), and resolved by 24 hours in most cases. Cumulative energy was the key predictor: risk rose from 8.3% (<21 mJ) to 25.0% (21–40 mJ) and 45.5% (>40 mJ). A practical, energy-guided protocol—no prophylaxis <21 mJ; a 1-hour IOP check for 21–40 mJ; and immediate treatment when >40 mJ or if early IOP is high—balances safety with efficiency. Energy-sparing technique and staged procedures can further mitigate risk while preserving excellent visual outcomes.

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