

# Comparative Evaluation of Intraocular Pressure Fluctuations Post Laser Peripheral Iridotomy in PACD Spectrum

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## Abstract

**Background:** Laser peripheral iridotomy (LPI) is the standard intervention for relieving the pupillary block component of primary angle-closure disease (PACD). Transient intraocular pressure (IOP) elevation may occur following LPI, but comparative data across primary angle-closure suspect (PACS), primary angle closure (PAC), and primary angle-closure glaucoma (PACG) are limited. This study evaluated IOP fluctuations following LPI across the PACD spectrum.

**Methods:** This prospective observational comparative study was conducted at a tertiary eye-care centre. Patients with PACS, PAC, or PACG who underwent LPI were evaluated clinically and by gonioscopy. IOP was recorded before LPI, immediately after LPI, at 2 hours, and at 7 days. The primary outcome was change in IOP from baseline; secondary outcomes included mean IOP at each time point and the proportion of eyes with an IOP rise of  $\geq 5$  mmHg and  $\geq 8$  mmHg.

**Results:** A total of 166 eyes of 158 patients were included in the final analysis. Baseline mean IOP was  $13.97 \pm 3.04$  mmHg in PACS,  $14.26 \pm 4.57$  mmHg in PAC, and  $16.63 \pm 6.48$  mmHg in PACG ( $p = 0.081$ ). Immediately after LPI, mean IOP was  $14.29 \pm 4.37$ ,  $12.37 \pm 3.90$ , and  $15.15 \pm 5.35$  mmHg, respectively ( $p = 0.151$ ), and at 2 hours it was  $13.53 \pm 4.22$ ,  $12.68 \pm 4.12$ , and  $14.74 \pm 5.22$  mmHg ( $p = 0.357$ ). At 7 days, mean IOP increased to  $15.42 \pm 4.97$  mmHg in PACS,  $16.33 \pm 4.77$  mmHg in PAC, and  $22.00 \pm 7.70$  mmHg in PACG, with a statistically significant intergroup difference ( $p < 0.001$ ). The mean change from baseline to day 7 was  $+1.45$ ,  $+1.83$ , and  $+5.37$  mmHg in PACS, PAC, and PACG, respectively. Clinically significant immediate IOP elevation was uncommon, with an IOP rise of  $\geq 5$  mmHg reported in 6.0% of eyes and  $\geq 8$  mmHg in 1.2% of eyes in the study analysis. No vision-threatening complication due to acute IOP elevation was observed during follow-up.

**Conclusion:** LPI was associated with relatively stable immediate and 2-hour IOP measurements across the PACD spectrum, with clinically significant acute IOP spikes being uncommon. PACG eyes demonstrated a significantly higher IOP at 7 days and a greater mean increase from baseline, which may reflect underlying trabecular dysfunction and chronic impairment of aqueous outflow rather than a direct delayed laser effect. Closer post-LPI IOP monitoring and additional IOP-lowering treatment may be required in eyes with established PACG.

**Keywords:** Primary angle-closure disease; PACS; PAC; PACG; laser peripheral iridotomy; intraocular pressure; IOP spike; glaucoma.

## Introduction

Primary angle closure disease (PACD) contributes to the leading cause of irreversible visual impairment worldwide and is especially high in Asian populations. The disease spectrum encompasses primary angle closure suspect (PACS), primary angle closure (PAC), and primary angle closure glaucoma (PACG), as defined by the International Society of Geographical and Epidemiological Ophthalmology (ISGEO) classification proposed by Foster et al. [1]. Primary angle-closure disease (PACD) is an important cause of glaucoma-related visual morbidity worldwide. Although a single prevalence estimate for the entire PACD spectrum is not consistently available, a systematic review and meta-analysis of 37 population-based studies involving 144,354 participants reported a global pooled prevalence of primary angle-closure glaucoma (PACG) of 0.6% (95% CI, 0.5–0.8%), with a higher prevalence of 0.7% (95% CI, 0.6–1.0%) in Asian populations [2].

The pathophysiology of angle closure is multivariable, with pupillary block being the predominant mechanism in most of the eyes. The resistance to aqueous flow across the relative one way valve system results in a relative pressure difference between the posterior and chamber causing anterior bowing of the peripheral iris, which results in the obstruction of the trabecular meshwork [3]. Additional mechanisms such as plateau iris configuration, increased lens vault, pseudoplateau iris, intumescent and swollen lens, and anterior lens position may also contribute to angle closure [4].

Laser peripheral iridotomy (LPI) remains the first-line non surgical intervention for relieving pupillary block, thereby creating an alternative pathway for aqueous humor flow. LPI stabilizes the pressure between the posterior and anterior chamber, flattens iris convexity, widens the anterior chamber angle, and reduces the risk of attack of acute and chronic angle closure [5].

Over the last decade, pivotal studies have clarified the role of LPI in PACD. The Zhongshan Angle Closure Prevention (ZAP) Trial demonstrated that prophylactic LPI significantly reduced the risk of disease progression in PACS eyes, although the overall incidence of progression of PACS to PAC or PACG was low [5]. Similarly, the Singapore Asymptomatic Narrow Angles Laser Iridotomy Study (ANA-LIS) reported a lower risk of angle closure progression in treated eyes compared with untreated fellow eyes over long-term follow-up [6]. These studies proved the efficacy and safety of LPI in eyes at risk of angle closure.

Despite its established therapeutic benefits, laser peripheral iridotomy (LPI) may be associated with a transient elevation in intraocular pressure (IOP), with significant IOP rises reported in 9.8% of cases at 1 hour and 0.82% at 2 weeks following the procedure in primary angle-closure suspects. The known mechanisms behind this are pigment dispersion, inflammatory debris accumulation within the trabecular meshwork, release of iris tissue fragments, transient trabeculitis, and temporary compromise of aqueous outflow [6]. Such IOP elevations can lead to significant damage such as visual field loss in the eyes that have advanced glaucomatous damage.

Jiang et al. (2012) reported that approximately 10% of PACS eyes experienced a post-laser IOP rise of at least 8 mmHg one hour after LPI and identified higher laser energy levels and narrower angles as significant risk factors [7]. Lee et al. (2014) similarly demonstrated the occurrence of transient post-procedural IOP spikes despite sequential laser protocols and emphasized the need for postoperative monitoring [8].

Although several studies have examined immediate pressure changes after iridotomy, most have focused predominantly on PACS populations. There remains limited literature comparing post-LPI IOP fluctuations across the entire spectrum of PACD, particularly among PACS, PAC, and PACG eyes. Furthermore, it remains undetermined whether eyes with proven glaucomatous damage are at greater risk of pressure spikes post iridotomy than earlier stages of disease.

The current study was to investigate the incidence of IOP spikes post laser peripheral iridotomy among patients with PACS, PAC, and PACG.

## Methodology

### Study Design and Setting

This was a prospective observational comparative study conducted at a tertiary eye-care centre to evaluate changes in intraocular pressure (IOP) following laser peripheral iridotomy (LPI) in eyes with primary angle-closure disease (PACD).

### Study Population

Patients diagnosed with PACD were evaluated clinically and by gonioscopy and classified into three groups according to the stage of disease:

1. Primary angle-closure suspect (PACS)
2. Primary angle closure (PAC)
3. Primary angle-closure glaucoma (PACG)

A total of 83 eyes of 79 patients were included in the final analysis, comprising 38 PACS eyes, 18 PAC eyes, and 27 PACG eyes.

### Inclusion Criteria

Patients were included if they:

1. Were diagnosed with PACS, PAC, or PACG based on clinical examination and gonioscopic findings.
2. Were indicated for and underwent laser peripheral iridotomy.
3. Had a reliable baseline IOP measurement before LPI.
4. Were available for IOP assessment immediately after LPI, at 2 hours, and at 7 days.
5. Provided informed consent for participation.

### Exclusion Criteria

Patients were excluded if they had:

1. An acute primary angle-closure attack at presentation.
2. Previous laser peripheral iridotomy or other laser treatment in the study eye.
3. Previous intraocular surgery in the study eye.
4. Secondary causes of angle closure, including neovascular, uveitic, traumatic, or lens-induced angle closure.
5. Significant corneal pathology precluding reliable IOP measurement.
6. Inadequate follow-up or missing IOP measurements at the predefined study time points.

### Clinical Examination and Gonioscopy

All patients underwent a comprehensive ophthalmic examination, including visual acuity assessment, slit-

lamp examination, IOP measurement by Goldmann applanation tonometry, and goldmann zeiss 4 mirror gonioscopy. Gonioscopy was performed to assess the anterior chamber angle and confirm the diagnosis and stage of PACD. The findings were used to classify eyes into PACS, PAC, or PACG before undergoing LPI.

### Laser Peripheral Iridotomy and IOP Assessment

All eligible eyes underwent LPI as part of the management of PACD. IOP was measured at four predefined time points: before LPI (baseline), immediately after LPI, 2 hours after LPI, and 7 days after LPI. The change in IOP at each postoperative time point was calculated relative to the baseline IOP.

### Outcome Measures

The primary outcome was the change in IOP following LPI among the PACS, PAC, and PACG groups. Secondary outcomes included mean IOP at each assessment point and the proportion of eyes demonstrating an IOP increase of  $\geq 5$  mmHg and  $\geq 8$  mmHg from baseline. The frequency of IOP elevation was assessed immediately after LPI, at 2 hours, and at 7 days.

### Statistical Analysis

Continuous variables were expressed as mean  $\pm$  standard deviation. Mean IOP and changes in IOP from baseline were compared among the three disease groups using one-way analysis of variance (ANOVA). The proportion of eyes showing an IOP rise of  $\geq 5$  mmHg and  $\geq 8$  mmHg was compared between groups using an appropriate categorical-data test. A P value  $< 0.05$  was considered statistically significant.

### Results

A total of 83 eyes of 79 patients were included in the final analysis. 38 (45.8%) were classified as primary angle closure suspects (PACS), 18 eyes (21.7%) as primary angle closure (PAC), and 27 eyes (32.5%) as primary angle closure glaucoma (PACG).

The mean pre-procedure IOP was  $13.97 \pm 3.04$  mmHg in the PACS group,  $14.26 \pm 4.57$  mmHg in the PAC group, and  $16.63 \pm 6.48$  mmHg in the PACG group. There was no statistically significant difference in baseline IOP among the three groups (ANOVA,  $p = 0.081$ ).

Immediately following peripheral iridotomy, mean IOP values were  $14.29 \pm 4.37$  mmHg,  $12.37 \pm 3.90$  mmHg, and  $15.15 \pm 5.35$  mmHg in the PACS, PAC, and PACG groups, respectively ( $p = 0.151$ ). At 2 hours, the corresponding mean IOP values were  $13.53 \pm 4.22$  mmHg,  $12.68 \pm 4.12$  mmHg, and  $14.74 \pm 5.22$  mmHg, with no significant intergroup difference ( $p = 0.357$ ).

At 7 days, mean IOP values increased to  $15.42 \pm 4.97$  mmHg in PACS eyes,  $16.33 \pm 4.77$  mmHg in PAC eyes, and  $22.00 \pm 7.70$  mmHg in PACG eyes. This difference was statistically significant (ANOVA,  $p < 0.001$ ).

The mean change in IOP from baseline to day 7 was  $+1.45$  mmHg in PACS eyes,  $+1.83$  mmHg in PAC eyes, and  $+5.37$  mmHg in PACG eyes, indicating greater postoperative IOP variability among eyes with established glaucomatous damage.

Using a threshold of  $\geq 5$  mmHg rise from baseline, significant IOP spikes were observed in 5 eyes (6.0%). When a more firm threshold of  $\geq 8$  mmHg was applied, only one eye (1.2%) demonstrated a significant pressure spike. No patient developed vision-threatening complications due to acute IOP elevation during the follow-up period.

**Table 1: Comparison of Intraocular Pressure Parameters Among PACS, PAC and PACG Eyes Following Laser Peripheral Iridotomy**

| Parameter                                                   | PACS (n=38)      | PAC (n=18)       | PACG (n=27)      | P value |
|-------------------------------------------------------------|------------------|------------------|------------------|---------|
| Pre-PI IOP (mmHg), mean $\pm$ SD                            | 13.97 $\pm$ 3.04 | 14.26 $\pm$ 4.57 | 16.63 $\pm$ 6.48 | 0.081   |
| Immediate Post-PI IOP (mmHg), mean $\pm$ SD                 | 14.29 $\pm$ 4.37 | 12.37 $\pm$ 3.90 | 15.15 $\pm$ 5.35 | 0.151   |
| 2-Hour IOP (mmHg), mean $\pm$ SD                            | 13.53 $\pm$ 4.22 | 12.68 $\pm$ 4.12 | 14.74 $\pm$ 5.22 | 0.357   |
| 7-Day IOP (mmHg), mean $\pm$ SD                             | 15.42 $\pm$ 4.97 | 16.33 $\pm$ 4.77 | 22.00 $\pm$ 7.70 | <0.001* |
| Change from baseline to Immediate Post-PI (mmHg)            | 0.32             | -2.11            | -1.48            | —       |
| Change from baseline to 2 Hours (mmHg)                      | -0.45            | -1.67            | -1.89            | —       |
| Change from baseline to 7 Days (mmHg)                       | 1.45             | 1.83             | 5.37             | —       |
| Eyes with IOP rise $\geq$ 5 mmHg, n (%) [immediate post PI] | 3 (7.9%)         | 1 (5.6%)         | 1 (3.7%)         | —       |
| Eyes with IOP rise $\geq$ 8 mmHg, n (%) [immediate post PI] | 1 (2.6%)         | 0                | 0                | —       |
| Eyes with IOP rise $\geq$ 5 mmHg, n (%) [2hrs post PI]      | 0 (0%)           | 3 (7.9%)         | 0 (0%)           | —       |
| Eyes with IOP rise $\geq$ 8 mmHg, n (%) [2hrs post PI]      | 0 (0%)           | 1 (2.6%)         | 0 (0%)           | —       |
| Eyes with IOP rise $\geq$ 5 mmHg, n (%) [7day post PI]      | 4 (22.2%)        | 7 (18.4%)        | 10 (37.0%)       | —       |
| Eyes with IOP rise $\geq$ 8 mmHg, n (%) [7 day post PI]     | 1 (5.6%)         | 3 (7.9%)         | 8 (29.6%)        | —       |
| 95% CI of 7-Day IOP                                         | 13.79–17.05      | 14.01–18.66      | 18.95–25.05      | —       |

\*Statistically significant (ANOVA,  $p < 0.05$ )

**Table 2: Frequency of Intraocular Pressure Elevation Among Diagnoses**

| Diagnosis    | No. of Eyes | IOP Rise >5 mmHg  | IOP Rise >8 mmHg  |
|--------------|-------------|-------------------|-------------------|
| <b>PAC</b>   | 18          | 8 (22.2%)         | 2 (5.6%)          |
| <b>PACS</b>  | 38          | 14 (18.4%)        | 6 (7.9%)          |
| <b>PACG</b>  | 27          | 20 (37.0%)        | 16 (29.6%)        |
| <b>Total</b> | 83          | <b>42 (25.3%)</b> | <b>24 (14.5%)</b> |

## Discussion

Laser peripheral iridotomy (LPI) remains the cornerstone of treatment for primary angle closure disease by relieving the pupillary block component and improving aqueous humor outflow between the posterior and anterior chambers. Although LPI is generally considered a safe procedure, a temporary rise in intraocular pressure (IOP) remains a well-known postoperative complication, particularly in eyes with proven glaucomatous damage.

In the present study, significant immediate postoperative IOP spikes were uncommon. The mean IOP remained relatively stable immediately after LPI and at 2 hours in all three groups. An IOP rise of  $\geq 5$  mmHg was observed in 6.0% of eyes, while only 1.2% demonstrated a rise of  $\geq 8$  mmHg. These findings indicate that clinically significant acute IOP elevation following LPI was uncommon in our study.

Our findings are comparable with previous studies describing the occurrence of transient IOP rise post LPI. Lee et al. (2014) reported an IOP elevation of  $\geq 8$  mmHg in 10.7% of eyes following consecutive laser peripheral iridotomy follow up [8]. In comparison, only 1.2% of eyes in our study had an IOP rise of  $\geq 8$  mmHg. This diversity may be related to variations in patient characteristics, laser parameters, perioperative IOP-lowering treatment, and the timing of postoperative IOP assessment. However, both studies support the observation that clinically significant acute IOP elevation following LPI is relatively unusual.

A literature review by Rozon and Des Marchais reported that IOP spikes one hour after LPI occurred in approximately 6 to 9.8% of treated eyes, while less than 1% developed an immediate post-LPI IOP of  $\geq 30$  mmHg. The authors emphasized that although IOP spikes may occur post-LPI, most are not severe, and post-procedural IOP measurement is particularly important in patients with risk factors or pre-existing advanced optic nerve damage. These findings are similar to the low frequency of immediate IOP elevation observed in our study.

An important finding of the present study was the significantly higher IOP observed in PACG eyes at the 7-day follow-up. The mean IOP at 7 days was  $22.00 \pm 7.70$  mmHg in PACG eyes compared with  $15.42 \pm 4.97$  mmHg in PACS and  $16.33 \pm 4.77$  mmHg in PAC eyes. The mean change from baseline was +5.37 mmHg in PACG eyes, compared with +1.45 mmHg and +1.83 mmHg in PACS and PAC eyes, respectively. This difference was statistically significant and represents the most clinically relevant finding of our study.

The higher IOP observed in PACG eyes at 7 days should, however, be interpreted as late post procedural IOP elevation rather than a conventional acute laser-induced IOP spike. Since IOP was measured immediately, at 2 hours, and again at 7 days, the study design does not establish the exact timing or mechanism of the pressure increase between these time points. Therefore, the observed rise in IOP may

reflect the basic outflow dysfunction associated with proven glaucomatous damage rather than a direct delayed effect of the laser procedure.

Unlike PACS eyes, which generally have relatively pristine trabecular function, PACG eyes may have chronic trabecular meshwork damage, peripheral anterior synechiae, and chronic impairment of aqueous outflow. Although LPI effectively excludes the pupillary block component, it does not necessarily reverse earlier trabecular dysfunction. The American Academy of Ophthalmology review reported that persistent angle closure after LPI can occur across the spectrum of primary angle closure disease and that further IOP-lowering treatment was required in 83–100% of PACG eyes in the studies reviewed, compared with 0–8% of PACS eyes and 42–67% of PAC eyes. This reinforces the concept that LPI alone may be inadequate to achieve target IOP control in eyes with advanced PACG.

The findings of our study therefore suggest that the post-procedural IOP behaviour may vary according to the stage of primary angle closure disease. PACS and PAC eyes demonstrated relatively stable IOP following LPI, whereas PACG eyes showed a clinically and statistically significant elevation in IOP at 7 days. This result may reflect the greater degree of pre-existing trabecular damage and compromised aqueous outflow in PACG rather than an acute adverse effect of the laser procedure.

These findings have important clinical implications. Although immediate clinically significant IOP spikes were unusual in our cohort, patients with PACG may remain sensitive to raised IOP beyond the immediate post-procedure period. Therefore, post procedural monitoring should not necessarily be limited to the immediate period following LPI, particularly in eyes with proven glaucomatous damage. Additionally, IOP-lowering therapy is required depending on the post-LPI IOP spike and the presence of degree of glaucomatous damage.

The present study has several strengths. It directly compares postoperative IOP behavior among PACS, PAC, and PACG eyes within the same cohort and assesses IOP at multiple post procedural time intervals. Comparative data evaluating the post procedural IOP behaviour across the complete range of primary angle closure disease remain relatively limited.

There are certain limitation in our study. The study was conducted at a single tertiary care center and follow-up was limited to 7 days, preventing discussion of long-term IOP assessment. Furthermore, laser energy, gonioscopy, stage of PACG, baseline antiglaucoma medication burden, and other risk factors for post-procedural IOP spike were not analyzed. Because IOP was not measured continuously between 2 hours and 7 days, the exact timing and mechanism of the late IOP spike observed in PACG cannot be assessed; additionally, no comment is made on diurnal variation in IOP. Future prospective studies with larger cohorts, standardized laser parameters, and longer follow-up may help to clarify the factors of post procedural IOP spike in PACG.

## Conclusion

Laser peripheral iridotomy is a safe and effective procedure for the management of primary angle closure disease, with a low incidence of significant immediate post procedural IOP spikes. While PACS and PAC eyes had stable IOP profiles following the procedure, PACG eyes had a significantly higher IOP at one week of follow-up. This finding likely reflects underlying trabecular dysfunction and chronic outflow impairment rather than a direct laser-induced effect. The results suggest that although LPI effectively relieves pupillary block across all stages of angle closure disease, patients with known cases of PACG require closer monitoring and may need additional IOP-lowering therapy to achieve target pressure control.

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