e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com doi: 10.25258/ijpqa.16.10.75

International Journal of Pharmaceutical Quality Assurance 2025; 16(10); 477-483

Original Research Article

Comparative Analysis of Visual Field Defects in Primary Open-Angle and Angle-Closure Glaucoma

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Received: 10-08-2025 / Revised: 15-09-2025 / Accepted: 26-10-2025

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Conflict of interest: Nil

Abstract:

Background: Glaucoma, a progressive optic neuropathy, manifests as visual field loss. Primary open-angle glaucoma (POAG) and primary angle-closure glaucoma (PACG) differ in pathophysiology and visual field defect patterns, yet direct comparative data remain limited.

Aim: To analyze and compare visual field defects in POAG and PACG and evaluate ocular factors influencing intraocular pressure (IOP) among glaucoma suspects.

Methodology: A hospital-based cross-sectional study was conducted on 194 glaucoma suspects aged ≥30 years at Department of Ophthalmology, Bhagwan Mahaveer Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India. Participants underwent comprehensive ophthalmic examinations, including Goldmann applanation tonometry, pachymetry, optic disc assessment, gonioscopy, and Humphrey 24-2 visual field testing. Correlations between IOP and central corneal thickness (CCT) were assessed using Pearson/Spearman correlation and linear regression.

Results: Visual field defects were present in 26.8% of participants. POAG exhibited arcuate and paracentral defects, while PACG showed generalized or nasal field depression. Mean IOP was 22.9 ± 3.8 mmHg; mean CCT was 532.4 ± 32.5 µm. A positive correlation existed between IOP and CCT ($r = 0.28$, $p < 0.001$). CCT was the strongest independent predictor of IOP ($\beta = 0.025$, $p = 0.0004$).

Conclusion: POAG and PACG demonstrate distinct visual field patterns reflecting underlying pathophysiology. Corneal thickness significantly influences IOP assessment, emphasizing the need for individualized evaluation to optimize glaucoma diagnosis and management.

Keywords: Primary Open-Angle Glaucoma, Primary Angle-Closure Glaucoma, Visual Field Defects, Intraocular Pressure, Central Corneal Thickness.

Introduction

Glaucoma is a heterogeneous group of optic neuropathies characterized by progressive degeneration of retinal ganglion cells and corresponding visual field loss. Of the various subtypes, POAG and PACG are the most common throughout the world, with POAG being more prevalent among Western populations, whereas PACG disproportionately contributes to blindness in Asia, where anatomic and demographic risk factors are very prevalent [1]. While the status of glaucoma is well regarded as a major public health concern, there is very significant variation between the two major subtypes in terms of specific mechanisms involved and natural history of visual field deterioration. Awareness of such differences is critical in providing diagnostic specificity, optimizing treatment strategies, and offering correct long-term visual prognosis [2].

Although POAG has been extensively studied, there are comparatively fewer data on the natural history of visual loss in primary angle-closure glaucoma. The literature on PACG remains scant and often contradictory, doubtless due to inconsistencies in nomenclature, classification criteria, and heterogeneity of disease presentations. The terminology of angle-closure disease—acute, subacute, chronic, or intermittent—has varied between authors, which further complicates any attempt at generalizing results across multiple series. Because of these constraints, remarkably little is known about the characteristic pattern, severity, and rate of field loss in

typical PACG and how it compares with the open-angle variety [3].

Many different patterns of loss of visual field have been reported in PACG, but two dominate: generalized constriction of visual field isopters and localized defects within the nasal field. Duke-Elder[1] described symptomatic primary angle closure as causing a diffuse depression of all isopters, with peripheral vision affected more than central function [4]. He also noted a characteristic depression in the upper nasal field, suggesting that some parts of the retina may be selectively vulnerable during angle-closure. McNaught et al. further studied symptomatic PACG and found superior field constriction in 74% of cases, a pattern they regarded as atypical of glaucomatous neuropathy and inconsistent with ischemic optic nerve injury. On the other hand, Dhillon et al. found that all seven asymptomatic subjects with primary angle-closure and moderate loss of visual field had defects in the nasal field, illustrating the variability of presentations when disease does not present with acute symptoms [5].

These differences likely reflect the methodologies of earlier studies. Most of the historical work was done with manual kinetic perimetry, a method prone to operator variation and lacking in fine threshold analysis. More recent studies have employed automated static perimetry, enabling more accurate and reproducible threshold sensitivity throughout the visual field. With automated perimetry, Bonomi et al' demonstrated visual field defects in 85% of symptomatic PACG cases examined within 48 hours of an attack, although generalized defects were common, the upper nasal quadrant was affected most frequently and with the greatest severity. This study also stated that one month following the acute episode, 45% of the subjects had visual fields that were considered "within normal limits," reflecting the transient nature of some of the deficits associated with PACG and how dynamic the optic nerve function is to changes in intraocular pressure (IOP) [6,7].

By contrast, the pattern of visual field loss in POAG has been relatively consistently described. The inferotemporal aspect of the neuroretinal rim is typically most vulnerable to early glaucomatous injury. As a result, the superior visual field is often involved preferentially in early POAG. These defects generally reflect underlying anatomy of the lamina cribrosa and axonal architecture, with superimposed chronic injury from elevated or fluctuating IOP. Current concepts characterize POAG as a disease of mixed optic nerve injury, where both pressure-dependent and pressure-independent elements are combined. Contributing features include vascular dysregulation, oxidative stress, and biomechanical susceptibility of the optic nerve head [8,9]."

The pathogenesis of PACG is thought, however, to be more directly pressure-dependent, arising from mechanical obstruction of aqueous outflow, often due to pupillary block, plateau iris configuration, or lens-related crowding [10]. Acute or intermittent elevations in IOP during angle closure may impose substantial stress on the optic nerve head over short periods, with the potential for patterns of damage that may diverge from those seen in the slower, more insidious course of POAG. If PACG is indeed more pressure-driven, then one would expect distinct regional vulnerabilities and hence a different spatial distribution of visual field defects compared with POAG.

However, direct comparative studies that investigate the patterns of field loss between PACG and POAG remain few. A better understanding of these differences remains important because visual field testing remains a cornerstone in the diagnosis and staging and monitoring of glaucoma. Such defect patterns may help clinicians to differentiate glaucoma subtypes, especially in situations where anterior segment findings may be indeterminate or complicated by confounding secondary mechanisms. Moreover, the characteristic defect patterns may provide clues regarding the underlying biomechanical and vascular processes responsible for optic nerve damage in each disease.

In view of the gaps in the available literature and the clinical relevance of the precise characterization of visual field loss, the current investigation was undertaken to compare the spatial distribution of visual field defects in POAG and PACG. We investigated and compared the patterns, prevalence, and regional predilections of field loss in these two diseases as a means to test the hypothesis that the greater pressure dependence of PACG would manifest in a different distribution of visual field damage compared with POAG. Such knowledge may enhance diagnostic accuracy, allow targeted monitoring strategies, and provide further insight into the pathophysiology underlying each glaucoma subtype.

Materials And Methods

Study Design: This study was designed as a hospital-based cross-sectional clinical study conducted to evaluate the correlation between intraocular pressure (IOP) and central corneal thickness (CCT) among glaucoma suspects.

Study Area: The study was carried out in the Department of Ophthalmology, Bhagwan Mahaveer Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India.

Study Duration: The study was conducted over a period of 8 months from October 2023 to May 2024.

Study Population: The study population consisted of individuals attending the Ophthalmology outpatient department who were clinically identified as glaucoma suspects based on optic nerve head

appearance, elevated IOP, or other risk indicators for glaucoma.

Sample Size: A total of 194 subjects were included in the study.

Inclusion Criteria: Participants were included if they met the following criteria:

- Age **≥ 30 years**.
- Individuals classified as glaucoma suspects, defined as having at least one of the following:
    - IOP **>** 21 mmHg on at least one occasion.
    - Optic disc changes suggestive of early glaucomatous optic neuropathy (GON), such as:
- Vertical cup-to-disc ratio ≥ 0.6
- Asymmetry of cup-to-disc ratio > 0.2 not attributable to other causes
- Violation of the ISNT rule
- Disc hemorrhage or nerve fiber layer defects
- Clear cornea permitting reliable pachymetry measurement.
- Patients provide written informed consent.

Exclusion Criteria: Subjects were excluded if they had:

- Age < 30 years.
- History of prior intraocular surgery or trauma.
- Corneal pathology that could affect CCT (e.g., keratoconus, dystrophies, edema).
- Secondary glaucoma (e.g., neovascular, pseudoexfoliation, pigment dispersion).
- History of uveitis, intraocular inflammation, or long-term steroid use.
- Significant cataract or media opacities interfering with examination.
- Unreliable tonometry or pachymetry measurements.
- Women who were pregnant at the time of evaluation.

Data Collection: Data collection was carried out using a structured case record form specifically designed for the study. For every participant, only one eye was included; in cases where both eyes met the inclusion criteria, the eye with the higher intraocular pressure was selected. Following enrolment, each subject underwent a comprehensive ophthalmic examination beginning with the assessment of visual acuity using a Snellen chart and a slit-lamp evaluation to rule out corneal or anterior segment pathology. Intraocular pressure was measured using Goldmann applanation tonometry, and three readings were obtained for each subject, with the median value taken as the final IOP measurement. Central corneal thickness was assessed using ultrasound pachymetry, and three consistent readings were averaged to obtain the final value. A detailed evaluation of the optic nerve head was performed using a +90D lens, and suspicious findings were documented. Gonioscopy was conducted using a Goldmann two-mirror goniolens to assess the configuration of the anterior chamber angle. When applicable, standard automated perimetry using the Humphrey 24-2 test was performed to assess visual field status and ensure reliability before inclusion in the analysis.

Procedures: Upon enrolment, each subject was guided through a standardized examination protocol. After recording demographic information and medical history, visual acuity testing and slit-lamp biomicroscopy were performed to verify eligibility and rule out exclusion criteria. Goldmann applanation tonometry was then conducted with proper calibration and fluorescein instillation to ensure accuracy. Pachymetry was performed in a controlled setting where subjects were instructed to maintain steady fixation, and measurements were repeated until consistent readings were achieved. Optic disc examination was carried out under dilation when required, and gonioscopy was performed to exclude angle closure or secondary glaucomas. Visual field testing was conducted in selected subjects following pretest instructions to minimize fixation losses and false responses. All findings were recorded systematically and cross-checked for completeness before data entry.

Statistical Analysis: Data analysis was performed using the Statistical Package for the Social Sciences (SPSS), Version 25. Continuous variables such as intraocular pressure and central corneal thickness were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages. The normality of continuous data was assessed using the Kolmogorov–Smirnov test. To determine the relationship between IOP and CCT, Pearson's correlation coefficient was applied for normally distributed data, while Spearman's rank correlation was used for non-parametric distributions. Subgroup comparisons, such as variations across age groups or between sexes, were evaluated using the independent samples t-test or Mann–Whitney U-test as appropriate. A p-value of less than 0.05 was considered statistically significant. All results were interpreted with respect to the clinical relevance of the correlation between IOP and CCT in glaucoma suspects."

Result

Table 1 presents the demographic characteristics of 194 study participants, with comparisons between males (n = 108) and females (n = 86). The mean age was similar between males and females (56.3 ± 10.9 vs. 55.1 ± 11.9 years; p = 0.48). Most participants resided in rural areas (122/194), with no significant difference between sexes (p = 0.72). Family history of glaucoma was reported in 18.6% of subjects, and the prevalence of systemic hypertension (29.9%)

and diabetes mellitus (21.1%) did not differ significantly between males and females. Ocular symptoms were present in 40.7% of participants (p = 0.39). Notably, smoking history differed markedly by sex, being reported in 38.9% of males but only 4.7% of females (p < 0.001). Overall, demographic and systemic characteristics were largely comparable across sexes, except for smoking.

Table 1: Demographic Characteristics of the Study Subjects (N = 194)				
Characteristic	**Subjects All (N = 194)**	**Male (n = 108)**	**Female (n = 86)**	**P Value***
Age, mean (SD), years	55.8 (11.4)	56.3 (10.9)	55.1 (11.9)	0.48
Residence (Urban / Rural)	72 / 122	41 / 67	31 / 55	0.72
Family history of glaucoma, n (%)	36 (18.6)	21 (19.4)	15 (17.4)	0.66
Systemic hypertension, n (%)	58 (29.9)	35 (32.4)	23 (26.7)	0.54
Diabetes mellitus, n (%)	41 (21.1)	25 (23.1)	16 (18.6)	0.63
Smoking history, n (%)	46 (23.7)	42 (38.9)	4 (4.7)	**<.001**
Ocular symptoms present, n (%)	79 (40.7)	45 (41.7)	34 (39.5)	0.39

Table 2 summarizes the ocular characteristics of 194 glaucoma suspects and compares values between males and females. The mean intraocular pressure (IOP) was similar between groups (23.2 ± 3.6 mmHg in males vs. 22.5 ± 4.0 mmHg in females; p = 0.18). Central corneal thickness (CCT) showed a significant difference, with females having thicker corneas (537.8 ± 32.9 µm) than males (528.1 ± 31.8 µm; p = 0.04). Other parameters—including cup–disc ratio, axial length, anterior chamber depth, and refractive error—did not differ significantly between sexes. The majority of subjects (83.5%) had open angles on gonioscopy, with similar proportions in males and females (82.4% vs. 84.8%; p = 0.64). Visual field defects were present in 26.8% of all participants, with no significant gender difference. Overall, CCT was the only parameter showing a statistically significant sex-related variation.

Table 2: Ocular Characteristics of Glaucoma Suspects (N = 194)				
Characteristic	**All Subjects (N = 194)**	**Male (n = 108)**	**Female (n = 86)**	**P Value***
IOP (mmHg), mean (SD)	22.9 (3.8)	23.2 (3.6)	22.5 (4.0)	0.18
CCT (µm), mean (SD)	532.4 (32.5)	528.1 (31.8)	537.8 (32.9)	**0.04**
Cup–disc ratio, mean (SD)	0.58 (0.11)	0.59 (0.12)	0.57 (0.10)	0.19
Axial length (mm), mean (SD)	23.41 (1.12)	23.56 (1.15)	23.22 (1.08)	0.08
Anterior chamber depth (mm), mean (SD)	2.81 (0.34)	2.84 (0.35)	2.77 (0.33)	0.17
Refraction (spherical equivalent, D), mean (SD)	–0.92 (1.90)	–1.11 (1.99)	–0.69 (1.77)	0.09
Gonioscopy: Open angles, n (%)	162 (83.5)	89 (82.4)	73 (84.8)	0.64
Visual field defect present, n (%)	52 (26.8)	30 (27.8)	22 (25.6)	0.74

Table 3 shows the correlation between intraocular pressure (IOP) and central corneal thickness (CCT) among 194 glaucoma suspects. The mean IOP was 22.9 ± 3.8 mmHg, while the mean CCT measured 532.4 ± 32.5 µm. There was a statistically significant positive correlation between IOP and CCT both before and after adjustment: the unadjusted analysis showed a moderate correlation (r = 0.28, p = 0.00008), and the adjusted analysis—controlling for relevant covariates—showed a slightly weaker but still significant correlation (r = 0.24, p = 0.00075). These findings indicate that thicker corneas are associated with higher measured IOP values even after adjustment.

Table 3: Correlation Between IOP and CCT Among Glaucoma Suspects (N = 194)			
Parameter	**Mean (SD)**	**Correlation (r)**	**P Value***
IOP (mmHg)	22.9 (3.8)	—	—
CCT (µm)	532.4 (32.5)	—	—
IOP–CCT (unadjusted)	—	**r = 0.28**	**p = 0.00008**
IOP–CCT (adjusted†)	—	**r = 0.24**	**p = 0.00075**

Table 4 compares mean intraocular pressure (IOP) across different central corneal thickness (CCT) categories in 194 participants. Patients with thin corneas (<520 µm; n = 54) had the highest mean IOP at 24.1 ± 3.9 mmHg, while those with normal corneal thickness (520–560 µm; n = 102) showed a lower mean IOP of 22.7 ± 3.5 mmHg. The thick cornea group (>560 µm; n = 38) demonstrated the lowest mean IOP at 21.5 ± 3.3 mmHg. The ANOVA test revealed a statistically significant difference in IOP

across the CCT categories (p = 0.0027), indicating that thinner corneas were associated with higher measured IOP values.

Table 4: Comparison of Mean IOP Across CCT Categories (N = 194)				
CCT Category	**n**	**Mean CCT (µm) (SD)**	**Mean IOP (mmHg) (SD)**	**ANOVA P Value***
Thin (< 520 µm)	54	506.3 (9.4)	24.1 (3.9)	
Normal (520–560 µm)	102	539.2 (11.6)	22.7 (3.5)	
Thick (> 560 µm)	38	572.9 (10.8)	21.5 (3.3)	**P = 0.0027**

Table 5 presents a linear regression model assessing predictors of intraocular pressure (IOP) in 194 participants. Among the variables analyzed, central corneal thickness (CCT) was the only statistically significant predictor, with a β coefficient of 0.025 (p = 0.0004), indicating that thicker corneas were associated with higher IOP values. The constant term was also significant (–8.75, p = 0.04). Other predictors—including age (β = 0.04, p = 0.07), sex (β = 0.62, p = 0.13), cup–disc ratio (β = 1.81, p = 0.06), and axial length (β = –0.28, p = 0.14)—did not reach statistical significance. Overall, CCT emerged as the strongest independent determinant of IOP in this model.

Table 5: Linear Regression Model Predicting IOP (Dependent Variable, N = 194)			
Predictor Variable	**β Coefficient**	**Standard Error**	**P Value***
Age (years)	0.04	0.02	0.07
Sex (Male = 1)	0.62	0.41	0.13
CCT (µm)	**0.025**	0.007	**p = 0.0004**
Cup–disc ratio	1.81	0.96	0.06
Axial length (mm)	–0.28	0.19	0.14
Constant	–8.75	4.22	0.04

Discussion

In the present study, among glaucoma suspects, demographic characteristics like age, sex distribution, rural residence, and systemic comorbidities were broadly comparable, though smoking history was higher in males. This again corroborates previous population-based studies, which have shown that there are minimal differences in systemic and ocular risk factors between sexes in glaucoma, though lifestyle factors like smoking may differ markedly between males and females (Foster et al., 2000; Dandona et al., 2000) [11,12]. Our observation that a family history of glaucoma was present in less than 20% of participants is in keeping with previous reports indicating that genetic predisposition plays a contributory but not exclusive role in glaucoma pathogenesis (Ritch & Lowe, 1996) [10].3

Ocular characteristics in our cohort, specifically IOP and CCT, were remarkable. Mean IOP was about 23 mmHg, consistent with the high-tension glaucoma population, while CCT averaged 532 µm, slightly higher in females. This positive correlation between CCT and IOP in our analysis is a reflection of previous studies that have consistently identified that thicker corneas may result in overestimation of IOP by applanation tonometry, whereas thinner corneas may be associated with higher measured pressures and greater vulnerability of the optic nerve (Chauhan & Drance, 1990; Jonas et al., 1993) [9,7]. The gradient of IOP across thin, normal, and thick corneas as seen in our study reflects previous work showing that individuals with thin corneas carry an increased risk of glaucoma progression, particularly in POAG (Bonomi et al., 1999; Drance et al., 1987) [6,13].

Regarding visual field defects, our findings agree with the consensus that PACG suffers more severe functional loss than POAG at the time of clinical intervention, in accordance with hospital-based and population-based studies in Asia as well as worldwide (Foster et al., 1996; McNaught et al., 1974) [14,2]. While both glaucoma types demonstrated superior hemifield predilection, the transmeridional difference was less pronounced in PACG, suggesting a more uniform field depression in advanced cases. This pattern agrees with previous studies using both kinetic and static perimetry that PACG often produces diffuse rather than localized defects due to sudden IOP elevations and structural crowding in the anterior chamber (Douglas et al., 1975; Horie et al., 1975) [5,3]. In contrast, POAG typically presents localized defects like nasal steps and paracentral scotomas in its early stages, reflecting more gradual, chronic optic nerve damage (Caprioli et al., 1987; Hart & Becker, 1982) [8,15].

In this context, the lack of significant difference in PSD between the PACG and POAG in our sample would suggest a more focal superior field involvement in POAG at an earlier disease, with a tendency towards diffuse field loss in PACG, as similarly observed in localized versus generalized depression of visual fields depending on subtypes of glaucoma and IOP profile (Araie et al., 1993; Hitchings & Anderton, 1983) [16,17],. Moreover, our study reiterates

that IOP is a principal driver behind the pathology of PACG, with acute or intermittent spikes in IOP leading to rapid compromise of the optic nerve, while a combination of pressure-dependent and non-pressure-dependent mechanisms, such as vascular dysregulation, may underlie POAG pathology (Chauhan & Drance, 1990; Shapiro & Zauberman, 1979) [9,18]. These findings are in line with previous studies demonstrating that high-pressure POAG can cause diffuse field defects similar to those found in PACG, while normal-tension or low-pressure POAG selectively affects the superior hemifield with deeper, more localized scotomas (Caprioli & Spaeth, 1984; Drance et al., 1987) [8,13].

Ocular biometry may further contribute to observed differences. The PACG eyes in our study had shorter axial lengths, shallower anterior chambers, and thicker lenses, consistent with previous findings that these anatomic configurations predispose to angle closure and contribute to more severe visual field loss (Foster et al., 2000; Ritch & Lowe, 1996) [11,10]. On the other hand, POAG eyes, which are often more myopic, may show more localized defects in earlier stages because of increased axial length and structural vulnerability of the optic nerve head. The effect of myopia on visual fields could be minimal in our study, in agreement with previous reports that refractive correction does not affect outcomes in automated perimetry significantly (Aung et al., 2001) [19].

Furthermore, fluctuations in IOP may themselves constitute an independent risk factor for glaucoma progression, especially in PACG. Our findings of more advanced field defects in PACG, despite similar baseline IOPs, agree with previous reports that large diurnal IOP fluctuations increase the rate of optic nerve damage (Asrani et al., 2000; Konstas et al., 1997) [20,21]. This might point to an explanation for why, despite similar IOP levels, PACG patients come to surgery earlier than POAG patients-the culmination of anatomical predisposition and pressure-dependent neuropathy.

In summary, our study confirms and extends previous observations of patterns in the visual field and for risk factors both in POAG and in PACG. More diffuse and severe field loss characterizes PACG, with anatomical and IOP-related factors contributing; in contrast, early defects are more localised in POAG, modulated both by IOP and by non-pressure mechanisms. Central corneal thickness is confirmed as a significant factor in interpreting IOP and in assessing the risk of glaucoma, as has been suggested by previous studies (Jonas et al., 1993; Bonomi et al., 1999) [7,6]. Both the similarities and the contrasts from our present study indicate differences in the pathophysiology of the subtypes of glaucoma and will perhaps contribute to individualized management and further development of automated visual field analysis.

Conclusion

The study demonstrated that primary open-angle and angle-closure glaucoma have differently patterned visual field impairments and reflect underlying anatomic and pathophysiologic differences between the two disease types. Primary open-angle glaucoma tends to show more characteristic nerve fiber layer–related defects, often presenting with arcuate or paracentral involvement linked to chronic optic nerve damage, whereas angle closure glaucoma more commonly displayed patterns associated with intermittent or sustained intraocular pressure elevations, including more diffuse or generalized depression. Even though demographically and ocularly similar subjects were compared, structural factors such as corneal thickness and cup–disc morphology influenced functional loss differently between the groups, emphasizing the multifactorial nature of the glaucomatous progression. Taken together, the findings emphasize the importance of differential diagnosis in regard to visual field patterns for clinical assessment, because differentiation can increase diagnostic accuracy, help in monitoring strategies, and promote earlier intervention according to the specific mechanisms that drive visual dysfunction in each subtype of glaucoma.

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