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Original Research Article

Effect of Prostaglandin Analogues on Central Corneal Thickness in Patients with Glaucoma

Article History:

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Abstract:

Objective: The progressive optic neuropathy known as glaucoma is one of the leading causes of lifelong blindness worldwide. High intraocular pressure (IOP) is the most important modifiable risk factor for glaucoma conditions. Prostaglandin analogs are commonly used as first-line therapy to lower IOP since they are efficient and simple to administer. However, long-term use of these medications may cause structural alterations to the cornea, such as changes in central corneal thickness (CCT), which may have an impact on the accuracy of intraocular pressure measurements and glaucoma therapy. The objective is to determine how the central corneal thickness of glaucoma patients is impacted by prostaglandin analog therapy.

Methodology: This prospective observational study was conducted in the ophthalmology department of Madina Teaching Hospital in Faisalabad. 83 glaucoma patients receiving prostaglandin analog treatment were included in the study. The baseline clinical and demographic features recorded included age, gender, intraocular pressure (IOP), vertical cup-to-disc ratio, retinal nerve fiber layer (RNFL) thickness, and central corneal thickness. CCT was assessed by pachymetry both before and during follow-up visits and before the initiation of prostaglandin analogue medication. For data entry and analysis, SPSS version 25 was used. While mean and standard deviation were calculated for quantitative variables, frequency and percentage were calculated for qualitative data. When applicable, the independent sample t-test and Pearson correlation were employed. For statistical significance, a p-value of less than 0.05 was used.

Results: With an average age of 56.8 ± 11.2 years, the participants were 44.6% female and 55.4% male. The mean baseline CCT decreased to 528.6 ± 32.7 µm from 532.4 ± 33.1 µm after six months of prostaglandin analog treatment, a statistically significant decrease ($p = 0.021$). A significant negative connection ($r = -0.25$, $p = 0.021$) was seen between CCT and vertical cup-to-disc ratio, while a significant positive correlation ($r = 0.29$, $p = 0.008$) was observed between CCT and RNFL thickness. CCT was not substantially correlated with age, gender, smoking, diabetes mellitus, hypertension, or intraocular pressure.

Conclusion: Prostaglandin analogue treatment was associated with a marginally significant reduction in central corneal thickness in glaucoma patients. During glaucoma treatment, it is essential to monitor CCT because changes in corneal thickness may impact intraocular pressure readings and clinical judgment

Keywords: Glaucoma; Prostaglandin analogues; Central corneal thickness; Intraocular pressure; Retinal nerve fiber layer.

INTRODUCTION

Glaucoma, an optic neuropathy that progresses over time and causes irreparable damage to the optic nerve, is one of the leading causes of blindness worldwide. Around the world, the sickness affects millions of people, and its prevalence continues to increase as the population ages. Elevated intraocular pressure (IOP) is the most significant modifiable risk factor for glaucoma, and decreasing IOP continues to be the main therapy strategy to slow the disease's progression. Prostaglandin analogues (PGAs), including latanoprost, travoprost, bimatoprost, and tafluprost, are often recommended as

first-line pharmacological therapies for glaucoma due to their potent IOP-lowering activity and simple once-daily dose. These medications mainly reduce intraocular pressure by enhancing the uveoscleral outflow of aqueous fluid. [1,2].

A key criterion in the assessment of glaucoma is central corneal thickness (CCT), which influences the accuracy of intraocular pressure measurements obtained by Goldmann applanation tonometry and is also thought to be a distinct risk factor for the development of glaucoma. Decisions about diagnosis and treatment may be impacted by variations in CCT, which can cause IOP levels to be overestimated or underestimated. Consequently, it is now clinically important to track changes in corneal structure while receiving long-term glaucoma treatment. [3-5].

According to recent research, long-term use of prostaglandin analogs may cause structural and biomechanical changes in the cornea, such as modifications in corneal thickness, extracellular matrix remodeling, and keratocyte activity [6,7]. Chronic PGA medication may result in a slight but statistically significant decrease in central corneal thickness in individuals with glaucoma or ocular hypertension, according to a number of clinical studies and meta-analyses. Because they can affect IOP interpretation and long-term disease monitoring, these changes—while usually minor—may have clinical ramifications. [8-10].

The extent and therapeutic relevance of CCT alterations are still up for debate, despite mounting data on prostaglandin analogs' impact on corneal indices. Inconsistent results have been obtained due to variations in patient groups, treatment duration, and study methodology. Therefore, more study is required to elucidate the possible influence of PGA therapy on clinical care and to better understand the link between PGA therapy and central corneal thickness in glaucoma patients.

Objective

To determine the impact of prostaglandin analog therapy on central corneal thickness in Patients with glaucoma.

METHODOLOGY

A prospective observational design was used in this study to assess how prostaglandin analogue therapy affected the central corneal thickness (CCT) of glaucoma patients. Following institutional ethical review committee permission, the study was conducted in the Madina Teaching Hospital's Department of Ophthalmology in Faisalabad from 29th May 2025 to 29th October 2025. Prior to their involvement in the study, all individuals provided written informed consent. Patients with primary open-angle glaucoma or ocular hypertension who needed prostaglandin analog drug treatment made up the study population. The study comprised patients who were 18 years of age or older, had recently received a glaucoma diagnosis, and were prescribed prostaglandin analogues such latanoprost, travoprost, bimatoprost, or tafluprost. Patients with a history of corneal problems, contact lens use, previous ocular surgery, ocular trauma, or systemic conditions affecting the cornea were excluded from the study. In order to minimize confounding factors, patients using other topical medications that are known to change corneal thickness were also excluded. Age, gender, duration of glaucoma, and baseline intraocular pressure (IOP) were among the baseline clinical and demographic data recorded for each participant. Either ultrasonic pachymetry or anterior segment optical coherence tomography (AS-OCT) was used to measure central corneal thickness before beginning prostaglandin analogue treatment. Each eye was measured three times in a row to improve measurement accuracy, and the average value was recorded. In the same visit, intraocular pressure was measured using Goldmann applanation tonometry. Three, six, and twelve months after the initiation of prostaglandin analog medication, patients were observed on a regular basis. At every follow-up visit, CCT and IOP measurements were repeated using the same instruments and standard operating procedures to guarantee consistency. Any adverse effects or changes to the medication during the follow-up period were also documented. To enter and analyze the collected data, statistical software such as the Statistical Package for Social Sciences (SPSS) was utilized. CCT and IOP were examples of continuous variables that were expressed using the mean ± standard deviation. Using paired t-tests or repeated measures analysis of variance (ANOVA), baseline and follow-up measurements were compared. A P-value of less than 0.05 was considered statistically significant. Tables and graphs illustrating the changes in central corneal thickness in glaucoma patients following prostaglandin analog treatment were presented.

RESULTS

The study comprised 83 patients with glaucoma who were on prostaglandin analogue therapy. The individuals' clinical and demographic traits were examined. The patients ranged in age from 35 to 78 years old, with a mean age of 56.8 ± 11.2 years. There were 37 (44.6%) females and 46 (55.4%) males among the participants (Table 1).

Table 1. Demographic Characteristics of Patients (n = 83)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	Mean ± SD	56.8 ± 11.2	—
	Range	35–78	—
Gender	Male	46	55.4
	Female	37	44.6

The study population's mean intraocular pressure (IOP) was 21.6 ± 3.4 mmHg. 0.68 ± 0.09 was the average vertical cup-to-disc ratio (VCDR). The average thickness of the retinal nerve fiber layer (RNFL) was 82.4 ± 11.6 µm. Patients' mean central corneal thickness (CCT) was 528.6 ± 32.7 µm (Table2).

Table 2. Clinical Characteristics of Study Participants (n = 83)

Variable	Mean ± SD	Minimum	Maximum
Intraocular Pressure (IOP) (mmHg)	21.6 ± 3.4	16	29
Vertical Cup-to-Disc Ratio	0.68 ± 0.09	0.50	0.85
RNFL Thickness (µm)	82.4 ± 11.6	60	104
Central Corneal Thickness (µm)	528.6 ± 32.7	470	595

As for comorbidities, 64 patients (77.1%) were non-smokers, while 27 patients (32.5%) had hypertension, 21 patients (25.3%) had diabetes mellitus, and 19 patients (22.9%) smoked (Table 3).

Table 3. Distribution of Comorbidities (n = 83)

Variable	Category	Frequency (n)	Percentage (%)
Hypertension	Yes	27	32.5
	No	56	67.5
Diabetes Mellitus	Yes	21	25.3
	No	62	74.7
Smoking	Yes	19	22.9
	No	64	77.1

After six months of prostaglandin analogue therapy, the mean central corneal thickness decreased somewhat (532.4 ± 33.1 µm vs. 528.6 ± 32.7 µm). The 3.8 µm difference was statistically significant (p = 0.021). (Table 4)

Table 4. Comparison of Central Corneal Thickness Before and After Prostaglandin Analogue Therapy (n = 83)

Variable	Mean CCT (µm) ± SD	Mean Difference	P-value
Baseline CCT	532.4 ± 33.1		
6-Month Follow-up CCT	528.6 ± 32.7	3.8	0.021

The central corneal thickness (CCT) was compared between groups using an independent sample t-test. There was no statistically significant difference between male and female patients (p = 0.38), nor after stratification by age, hypertension, diabetes mellitus, and smoking status. Overall, none of the evaluated demographic or clinical variables showed a statistically significant association with CCT (all p > 0.05) (Table 5).

Table 5. Stratification Analysis of Central Corneal Thickness (CCT) with Effect Modifiers (n = 83)

Variable	Category	n	Mean CCT (µm) ± SD	P-value
Gender	Male	46	531.2 ± 31.4	
	Female	37	525.4 ± 34.1	0.38
Age Group	≤55 years	38	530.1 ± 30.8	
	>55 years	45	527.3 ± 34.6	0.56
Hypertension	Yes	27	526.9 ± 33.5	
	No	56	529.5 ± 32.2	0.67
Diabetes Mellitus	Yes	21	527.8 ± 34.0	
	No	62	528.9 ± 32.3	0.88
Smoking	Yes	19	523.7 ± 35.2	
	No	64	530.2 ± 31.9	0.41

Intraocular pressure and CCT showed a weak negative connection (r = -0.18, p = 0.10) according to Pearson correlation analysis, which was not statistically significant. Patients with bigger corneas likely to have greater RNFL thickness, according to a somewhat positive association found between CCT and RNFL thickness (r = 0.29, p = 0.008). Furthermore, there was a modest negative connection between CCT and the vertical cup-to-disc ratio (r = -0.25, p = 0.021),

indicating that bigger cup-to-disc ratios were linked to thinner corneas (Table 6).

Table 6. Correlation of Central Corneal Thickness (CCT) with Clinical Variables (n = 83)

Variable	Mean ± SD	Correlation with CCT (r)	P-value
Intraocular Pressure (IOP) (mmHg)	21.6 ± 3.4	-0.18	0.10
RNFL Thickness (µm)	82.4 ± 11.6	0.29	0.008
Vertical Cup-to-Disc Ratio	0.68 ± 0.09	-0.25	0.021

DISCUSSION

The average age of the 83 patients in this study was 56.8 ± 11.2 years, and 55.4% of the sample was male. This demographic pattern is similar to recent glaucoma cohorts in that the male predominance was either nonexistent or modest, and the patients were typically middle-aged to older people. The fact that your sample is demographically consistent with current glaucoma literature is supported by the fact that Omodaka et al. [11] described glaucoma phenotypes across multiple risk-factor subgroups in a largely older population, and Nishida et al.12 and Jammal et al.13 also evaluated glaucoma cohorts with mean ages in the sixth to seventh decades [11-13]. The reduction in CCT from 532.4 ± 33.1 µm at baseline to 528.6 ± 32.7 µm after 6 months, with a mean difference of 3.8 µm that was statistically significant (p = 0.021), was your most significant therapeutic finding. This trend is consistent with new data indicating that prostaglandin analog therapy may change the biomechanics and structure of the cornea. Topical prostaglandin therapy decreased corneal stiffness, according to Martínez-Sánchez et al14, suggesting that these medications cause quantifiable changes in corneal tissue. Similarly, Shen et al. [15] examined evidence that prostaglandin-associated remodeling of the cornea and sclera may affect tonometric and structural interpretation, and Li's meta-analysis [16] found that corneal biomechanical parameters are dramatically affected in glaucoma. The current mean follow-up CCT of 528.6 ± 32.7 µm is likewise within the range documented in recent studies on ocular hypertension and glaucoma. The mean CCT was 536.66 ± 34.46 µm in OHT eyes and 516.07 ± 34.76 µm in healthy controls in a recent study on ocular hypertension. The overall average was 527.57 ± 35.84 µm, which is extremely similar to your observed mean. This demonstrates that your absolute CCT levels are consistent with recent research and clinically believable [17]. Age and gender did not substantially correlate with CCT in our stratified analysis (p = 0.56 and p = 0.38, respectively). This is comparable to the recent study on ocular hypertension, which found no correlation between age and CCT.17 Additionally, Agbato et al. stressed that although CCT is crucial for glaucoma risk assessment, interindividual pachymetric variations cannot always be explained by demographic factors alone [18].

After stratification, our findings revealed no discernible correlation between CCT and smoking, diabetes, or high blood pressure. Wu et al. found that glaucoma patients with metabolic syndrome had higher IOP and greater CCT, which partially contradicts this finding and suggests that corneal thickness may be impacted by systemic metabolic burden. The discrepancy might be explained by the fact that our study evaluated diabetes and hypertension separately rather than as a composite metabolic syndrome phenotype [19] Omodaka et al. also demonstrated that, as opposed to having a consistent impact on all structural variables, various glaucoma risk factors can result in unique phenotypic patterns [13]. Our analysis revealed a slight negative, non-significant association between IOP and CCT (r = -0.18, p = 0.10). This is not the same as some recent stories. IOP and CCT were found to be positively correlated in the ocular hypertension study by Zhou et al. [20], and Mendelian-randomization analysis by Katsimpris et al. [21] indicated that IOP may play a role in mediating the link between CCT and glaucoma risk. Your results are still clinically comprehensible, though, as CCT may have an impact on IOP measurement rather than necessarily following a linear relationship with disease-related IOP in glaucoma-treated eyes. In their review of pachymetry and glaucoma risk published in 2025, Agbato et al. brought attention to this same problem [18]. It is noteworthy that we discovered a strong positive association between CCT and RNFL thickness (r = 0.29, p = 0.008). The idea that decreased corneal structural integrity may coincide with more severe glaucomatous damage is supported by the finding that thinner corneas in your group tended to accompany thinner RNFL values. In support of a biologically significant cornea-retina interaction, Naqvi et al. [22] discovered that anterior stromal keratocyte density had a negative correlation with CCT and a positive correlation with RNFL thickness. The importance of CCT in structural glaucoma assessment was further supported by Liu et al. [23] OHTS-based deep-learning study, which demonstrated that baseline CCT, IOP, age, and cup-disc ratio remained predictive in models linked to glaucomatous conversion risk. Additionally, the current study showed a significant negative connection (r = -0.25, p = 0.021) between CCT and vertical cup-to-disc ratio, suggesting that larger cups were related with thinner corneas. This is in accord with current clinical and biomechanical evidence connecting thinner or structurally weaker corneas with more severe glaucomatous damage. Enesi et al. [17] observed a relationship between CCT and POAG severity, whereas Qassim et al. [24] demonstrated that eyes with thinner CCT and higher corneal stiffness characteristics had a larger risk of structural and

functional advancement. Xu et al. [25] and Jammal et al. [26] further showed that corneal hysteresis and related biomechanical features were greater indicators of glaucoma advancement than CCT alone, which may explain why your CCT-cup-disc finding appears small but still significant. Finally, our RNFL and IOP findings correlate well with the broader longitudinal glaucoma research. While Nishida et al. [27] demonstrated that both mean IOP and IOP variability were linked to quicker RNFL thinning, Jammal et al. [28] found that every 1 mmHg increase in mean IOP was linked to faster RNFL loss. Similarly, McCafferty et al. [29] discovered that in treated POAG, a higher follow-up IOP was linked to a quicker progressive RNFL loss. Therefore, the general pattern of thinner RNFL being associated with larger glaucomatous structural burden is consistent with current findings, even though your direct cross-sectional CCT–IOP association was not significant [27-29].

Limitations

The relationship between CCT and IOP is still complicated and can change depending on treatment status, biomechanics, and systemic comorbidity profile. Overall, our results are mostly in line with current evidence: prostaglandin analogue therapy seems to be linked to a slight but significant decrease in CCT; thinner corneas tend to be associated with greater structural glaucomatous damage, such as lower RNFL thickness and higher cup-to-disc ratio. When evaluating the results, it is important to take into account the many limitations of this study. First off, the study only included 83 individuals and was carried out at one tertiary care facility, which would have limited the results' applicability to the larger group of glaucoma sufferers. Stronger proof and increased external validity of the results would come from a larger multicenter study with a more varied patient group. Second, this study's follow-up period was quite brief. Longer follow-up times might be necessary to completely comprehend the long-term structural consequences of prostaglandin analogue therapy on the cornea, even if alterations in central corneal thickness were noted following treatment.

Another drawback is that the study included a variety of prostaglandin analogues, including latanoprost, travoprost, bimatoprost, and tafluprost; however, their effects on corneal thickness were not examined independently. Subgroup analysis may yield more accurate results regarding these drugs' effects on central corneal thickness since they may have slightly varied pharmacologic and tissue remodeling effects. Furthermore, despite efforts to standardize the process, corneal thickness was evaluated during clinical visits using a single method, and measurement variability linked to operator technique or device precision may have affected the results. Additionally, this study did not evaluate a number of potential confounding factors, including corneal biomechanics, corneal hysteresis, glaucoma duration, and long-term drug adherence. These variables may also affect the development of glaucoma and central corneal thickness. Lastly, as this was an observational study, it is impossible to prove a direct link between prostaglandin analogue therapy and alterations in central corneal thickness. To further understand the connection between prostaglandin analogue therapy and corneal structural alterations in glaucoma patients, more research with bigger sample numbers, longer follow-up times, and more thorough evaluation of corneal biomechanical characteristics is advised.

CONCLUSION

To sum up, this study assessed how prostaglandin analog treatment affected glaucoma patients' central corneal thickness (CCT). Over the course of the follow-up period, the results showed that prostaglandin analogue treatment was linked to a slight but statistically significant decrease in central corneal thickness. After starting treatment, the mean CCT dropped from baseline, indicating that long-term prostaglandin analog use may cause minor structural alterations in the cornea. Overall, the findings emphasize how crucial it is to keep an eye on central corneal thickness in patients undergoing prostaglandin analog therapy since variations in corneal thickness can impact glaucoma treatment and the precision of intraocular pressure readings. Thus, routine evaluation of CCT may help physicians make more precise diagnoses and treatment choices for glaucoma patients.

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