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ORIGINAL PAPER

Evaluation of corneal and anterior segment alterations following short-term use of topical latanoprostene bunod

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Abstract

Purpose This study aimed to evaluate the short-term effects of topical latanoprostene bunod on corneal and anterior segment parameters in patients with primary open-angle glaucoma (POAG).

Methods A prospective, cross-sectional study was conducted on 30 eyes of 30 patients with POAG. All participants received topical latanoprostene bunod monotherapy. Corneal and anterior segment parameters, including keratometry, corneal thickness, endothelial cell characteristics, and corneal densitometry, were measured before and approximately after one month of treatment using the Pentacam HR and non-contact specular microscopy. Statistical analysis was performed to compare pre- and post-treatment measurements.

Results No significant changes were observed in corneal keratometry, endothelial cell density, hexagonality, or anterior chamber parameters including anterior chamber angle, depth, and volume following treatment ($p > 0.05$ for all). Although reductions in central corneal thickness and thinnest corneal thickness were noted, these changes were not statistically significant ($p > 0.05$ for both). A significant decrease in densitometric parameters was observed in most corneal zones after treatment, including anterior, central, posterior, and total thickness measurements ($p < 0.05$ for all). In contrast, no significant change was found in the peripheral 10–12 mm zones ($p > 0.05$ for all).

Conclusion Short-term use of latanoprostene bunod reduced corneal densitometry values, suggesting improved corneal transparency without significantly affecting corneal thickness, endothelial cell characteristics, or anterior segment parameters. Further long-term studies with larger sample sizes are needed to evaluate its prolonged effects on corneal health.

Keywords Corneal endothelium · Corneal transparency · Nitric oxide · Primary open-angle glaucoma

Introductıon

Topical antiglaucoma medications are indispensable in the management of elevated intraocular pressure (IOP), particularly seen in glaucoma and ocular hypertension. These agents are critical in preventing progressive optic nerve damage and preserving visual function. However, their use is frequently associated with adverse effects on the ocular surface and anterior segment, which may compromise patient adherence and overall treatment outcomes. Reported side effects range from mild discomfort, such as dryness or irritation, to significant structural and functional

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changes, including dermatitis, tear film instability, conjunctival inflammation, and corneal surface alterations [1].

Latanoprostene bunod, a nitric oxide (NO)-donating prostaglandin analog, is an effective treatment for reducing IOP. Although its efficacy in lowering IOP has been well-documented in clinical trials, there is a paucity of data regarding its impact on the cornea and anterior segment [2]. Preliminary reports have indicated mild ocular side effects, including punctate keratitis and ocular hyperemia; however, further investigation utilizing objective and quantitative methods is necessary to comprehensively evaluate the anterior segment alterations following topical latanoprostene bunod therapy[3].

The Pentacam, a rotating Scheimpflug imaging system, is a highly accurate and objective instrument for assessing corneal and anterior segment morphology. Its quantitative capabilities facilitate precise measurements of corneal structure and transparency, yielding reliable and reproducible data [4]. Utilizing corneal densitometry analysis to assess transparency and visualize corneal morphology, the Pentacam can identify subtle changes that may not be clinically evident and is increasingly recognized as a valuable tool in diagnosing and evaluating eye diseases and surgical outcomes[5].

Considering the well-documented ocular surface complications associated with long-term use of antiglaucoma medications, including central corneal thickness (CCT) alterations and corneal toxicity [6, 7], it is essential to assess the effects of latanoprostene bunod on the cornea and anterior segment. This study aims to evaluate corneal and anterior segment changes induced by short-term administration of latanoprostene bunod, utilizing the Pentacam HR.

Materıals and methods

This prospective, cross-sectional study was conducted at the Ophthalmology Clinic of a tertiary hospital. Ethical approval was granted by the hospital's ethics committee in accordance with the principles outlined in the Declaration of Helsinki. Prior to participation, written informed consent was obtained from all participants, ensuring their voluntary involvement and understanding of the study procedures.

Each participant underwent a comprehensive ophthalmological assessment, which included the measurement of best-corrected visual acuity (BCVA) using Snellen charts, IOP via non-contact tonometry, and evaluation of the anterior segment and fundus through biomicroscopy. Refractive error was determined using an RK-F2 automatic refractor-keratometer (Canon Inc., Tokyo, Japan). Additionally, any side effects resulting from the treatment were subsequently recorded.

The exclusion criteria for this study were established to ensure the selection of participants with optimal corneal and ocular health, thereby minimizing confounding variables. The study included only individuals newly diagnosed with primary open-angle glaucoma (POAG), specifically those with IOP $\leq$ 24 mmHg (as high IOP can induce corneal edema) and a clinically clear cornea. *Exclusion criteria included a history of ocular trauma or any prior ocular surgeries, except for cases of uncomplicated cataract surgery performed at least six months before the study.* Additionally, participants with pre-existing ocular conditions, such as corneal scars, keratoconus, or keratitis, were excluded. Patients with a spherical equivalent refractive error exceeding $\pm$2.00 diopters were also excluded. Participants receiving anti-glaucomatous therapy other than latanoprostene bunod and any topical treatment were excluded from the study. The study also excluded individuals with a fluorescein-tear breakup time (FTBUT) of less than 10 s before and after treatment, as a low FTBUT indicates potential tear film instability, which could affect corneal health and imaging results. Participants who were pregnant, and those with systemic diseases affecting the ocular system (such as diabetes or autoimmune disorders), were also excluded due to their potential influence on ocular health.

Corneal imaging was performed using the Pentacam®HR Scheimpflug tomography system (Oculus Optikgeräte GmbH, Wetzlar, Germany) and non-contact specular microscopy (EM-4000; Tomey Corp., Nagoya, Japan). All imaging was conducted by a single technician between 9:30 and 10:30 AM to minimize potential variations related to diurnal changes in the cornea. We evaluated the agreement between measurements with Intraclass Correlation Coefficient. The ICCs for all values (densitometric, keratometric, pachymetric and anterior segment

parameters) were above 0.90. A corneal specialist (P.K.) reviewed the obtained images to assess for any distortions or signs of corneal pathology. Corneal measurements were obtained prior to the initiation of treatment and approximately one month thereafter.

Corneal densitometry was performed using the Pentacam®HR, which evaluates corneal light scatter across a 12 mm diameter region, subdivided into four concentric zones: 0–2 mm, 2–6 mm, 6–10 mm, and 10–12 mm. Densitometric values were recorded at three distinct corneal depths: the anterior 120 µm, the posterior 60 µm, and the mid-stromal layer. These values were expressed in greyscale units (GSUs), with a range from 0 (indicating maximum transparency) to 100 (indicating complete opacity), reflecting the degree of light scattering within the cornea. Furthermore, keratometric values (flat keratometry (K1), steep keratometry (K2), and mean keratometry (Kmean) for the central 3.0 mm of both the anterior and posterior corneal surfaces), pachymetric measurements (thinnest corneal thickness (CTmin), corneal volume), and anterior segment parameters (anterior chamber depth (ACD), anterior chamber volume (ACV), and anterior chamber angle (ACA)) were assessed.

The recorded parameters of non-contact specular microscopy included cell density (CD), the percentage of hexagonal cells (HEX), as well as the average (AVG), standard deviation (SD), coefficient of variation (CV), maximum (MAX), and minimum (MIN) cell area values. CD represents the total number of endothelial cells, HEX reflects the proportion of hexagonal-shaped cells, and CV indicates the degree of variation in cell size.

Statistical analysis

The analysis of patients' data was conducted using SPSS version 24.0 (Statistical Package for Social Sciences). To evaluate the data distribution, the Kolmogorov–Smirnov test was applied. Data with a normal distribution were expressed as mean ± standard deviation, whereas non-normally distributed data were reported as median (minimum–maximum). Statistical comparisons included the paired-samples t-test for normally distributed variables and the Wilcoxon test for variables with non-normal distribution. The Spearman correlation coefficient was used to evaluate the relationship between variables. A significance level of $p < 0.05$ was set for all analyses.

Results

Forty-three newly diagnosed POAG patients were initially assessed. Five patients were excluded due to poor treatment compliance or irregular follow-up, and eight were excluded because their post-treatment FBUT was below 10 s. Finally, 30 eyes of 30 patients (14 female, 16 male) were included in the study. The mean age was 69.0 ± 12.3 (35.0–88.0) years. The mean follow-up duration between measurements was 1.2 ± 0.3 months. The median spherical equivalent refractive error was 0.75 D (–2.00 to + 2.00). The mean pre-treatment BCVA was 0.87 ± 0.15, while the post-treatment BCVA was 0.85 ± 0.18, with no statistically significant difference between the two (p = 0.662). Mean pre-treatment and post-treatment IOPs were 21.3 ± 3.4 mmHg and 16.0 ± 3.4 mmHg, respectively ($p < 0.001$). The mean pre-treatment FBUT was 11.9 ± 1.3 s, while the post-treatment FBUT was 11.4 ± 1.1 s. One patient reported a foreign body sensation ($n = 1$ [3.3%]), and another patient reported eye pruritus ($n = 1$ [3.3%]). Following treatment, no patients, aside from these issues, reported symptoms of dry eye.

Tedavi öncesi ortalama FBUT değerleri 11.9 ± 1.3 (10–15) iken, tedavi sonrası 11.4 ± 1.1 (10–14) olarak hesaplandı.

The anterior segment parameters were evaluated before and after treatment (Table 1). No significant changes were observed in the anterior corneal surface parameters, including K_1 ($p = 0.133$), K_2 ($p = 0.877$), and K_{mean} ($p = 0.932$) values. Similarly, the posterior corneal surface parameters, including K_1 ($p = 0.700$), K_2 ($p = 0.336$), and K_{mean} ($p = 0.427$) values, remained stable following treatment. Although a reduction in CCT and the CTmin values was observed, these changes were not statistically significant ($p > 0.05$ for both). Corneal volume showed a statistically insignificant decrease from 59.2 ± 3.5 mm^3 to 58.7 ± 3.4 mm^3 ($p = 0.101$). There were no statistically significant changes in the ACD, ACV, and ACA values ($p = 0.111$, $p = 0.275$, and $p = 0.338$, respectively).

Table 1 Comparison of corneal and anterior chamber parameters before and after topical latanoprostene bunod therapy

	Pre-treatment ($n=30$)	Post-treatment ($n=30$)	p
Anterior corneal surface			
K_1, *D*	43.4 (39.1–47.1)	43.2 (39.4–46.7)	0.133*
K_2, *D*	44.0 ± 2.0	44.0 ± 1.9	0.877†
K_{mean}, *D*	43.6 ± 1.9	43.5 ± 1.7	0.932†
Posterior corneal surface			
K_1, *D*	− 6.0 ± 0.3	− 6.1 ± 0.2	0.700†
K_2, *D*	− 6.4 (-6.9-[-5.6])	− 6.3 (-7.1-[-5.6])	0.336*
K_{mean}, *D*	− 6.2 ± 0.3	− 6.1 ± 0.8	0.427†
CCT, *μm*	556.0 (497.0–652.0)	550.0 (454.0–621.0)	0.076*
Thinnest corneal thickness, *μm*	552.0 (479.0–645.0)	541.0 (469.0–610.0)	0.144*
Corneal volume, mm^3	59.2 ± 3.5	58.7 ± 3.4	0.101†
ACD, *mm*	2.6 (1.9–4.8)	2.6 (1.9–4.7)	0.111*
Anterior chamber volume, mm^3	136.6 ± 45.0	136.8 ± 44.3	0.275†
Anterior chamber angle, *degree*	30.7 (16.1–53.1)	31.0 (16.2–51.4))	0.338*

Non-normally distributed variables are presented as median (min–max), normally distributed variables are presented as mean ± standard deviation. Significant p values are in bold

D, dioptre; *CCT*, central corneal thickness; *WTW*, white-to-white; *ACD*, anterior chamber depth

* Wilcoxon Signed Ranks Test, † Paired-Samples T-Test

Table 2 Comparison of the corneal endothelial cell characteristics between the study groups

	Pre-treatment (n = 30) Median (Min–Max)	Post-treatment (n = 30) Median (Min–Max)	p *
CD, *cell/mm²*	2346.0 (1080.0–2907.0)	2308.0 (1182.0–2986.0)	0.830
AVG, *μm²*	426.0 (344.0–1038.0)	433.0 (335.0–846.0)	0.789
SD, *μm²*	160 (119.0–430.0)	164.0 (113.0–410.0)	0.667
MAX, *μm²*	1023 (669.0–2892.0)	1085.0 (705.0–3102)	0.671
MIN, *μm²*	103.0 (69.0–311.0)	104.0 (72.0–290.0)	0.314
CV, *(%)*	38.0 (28.0–69.0)	40.0 (30.0–96.0)	0.904
HEX, *(%)*	45.0 (22.0–56.0)	47.0 (21.0–55.0)	0.625

CD, Cell density; *AVG*, Average cell area; *SD*, Standard deviation of cell area; *MAX*, Maximum cell area; *MIN*, Minimum cell area; *CV*, Coefficient of variation; *HEX*, Variability in hexagonal shape

*Wilcoxon signed rank test. Significant p values are in bold

A comparison of endothelial cell characteristics between pre-treatment and post-treatment groups is presented in Table 2. The CD showed a median value of 2346.0 cells/mm^2 in the pre-treatment group and 2308.0 cells/mm^2 in the post-treatment group, with no statistically significant difference ($p=0.830$). There was also no statistically significant changes in the AVG, SD, MAX, and MIN results ($p>0.05$ for all). The CV was 38.0% pre-treatment and 40.0% post-treatment, with no significant difference ($p=0.904$). The HEX was 45.0% pre-treatment and 47.0% post-treatment, showing no significant change ($p=0.625$).

A comparison of corneal densitometry measurements between the pre-treatment and post-treatment groups is presented in Table 3. For the anterior 120μ, significant reductions were observed in the 0–2 mm ($p=0.029$), 2–6 mm ($p=0.008$), and 6–10 mm

Table 3 Comparison of the corneal densitometry measurements between the pre- and post-treatment groups

	Pre-treatment (n = 30)	Post-treatment (n = 30)	*p*
Anterior 120µ (GSUs)			
0–2 mm	28.7 (22.9–97.9)	27.1 (22.5–62.5)	**0.029***
2–6 mm	27.4 (22.5–90.1)	25.1 (19.7–54.3)	**0.008***
6–10 mm	41.6 (22.3–78.0)	36.7 (19.6–62.9)	**0.003***
10–12 mm	48.0 ± 13.2	47.0 ± 13.7	0.404†
Total diameter	39.4 ± 13.4	35.5 ± 9.2	**0.011**†
Central (GSUs)			
0–2 mm	17.5 (13.2–31.2)	16.5 (14.4–31.5)	**0.002***
2–6 mm	16.8 (12.7–23.7)	15.9 (12.9–25.4)	**0.001***
6–10 mm	25.0 (14.8–39.9)	23.1 (13.3–38.9)	**< 0.001***
10–12 mm	26.4 ± 6.9	25.9 ± 6.7	0.292†
Total diameter	21.1 ± 3.6	20.2 ± 3.4	**< 0.001**†
Posterior 60µ (GSUs)			
0–2 mm	10.9 (8.0–21.7)	10.6 (8.4–20.4)	0.166*
2–6 mm	11.5 (8.4–17.8)	10.9 (8.0–17.2)	**0.047***
6–10 mm	17.0 (10.6–24.5)	16.2 (9.9–24.8)	**0.016***
10–12 mm	20.1 ± 5.0	21.8 ± 12.8	0.361†
Total diameter	14.7 ± 2.7	14.2 ± 2.5	**0.042**†
Total thickness (GSUs)			
0–2 mm	20.2 (16.3–43.1)	18.9 (15.3–29.6)	**0.015***
2–6 mm	19.1 (15.1–39.7)	17.8 (13.6–27.4)	**0.001***
6–10 mm	28.2 (15.9–43.8)	26.2 (14.3–40.5)	**< 0.001***
10–12 mm	31.5 ± 7.8	30.9 ± 8.0	0.379†
Total diameter	25.1 ± 5.3	23.3 ± 4.5	**0.001**†

GSU, Grayscale units. **Wilcoxon Signed Ranks Test,* † *Paired-Samples T-Test.* Non-normally distributed variables are presented as median (min–max), normally distributed variables are presented as mean ± standard deviation. Significant *p* values are in bold

($p = 0.003$) regions, while no significant change was seen in the 10–12 mm region ($p = 0.404$). The total diameter also showed a significant decrease ($p = 0.011$). Regarding the central region, significant reductions were observed in the 0–2 mm ($p = 0.002$), 2–6 mm ($p = 0.001$), and 6–10 mm ($p < 0.001$) regions, while the 10–12 mm region showed no significant change ($p = 0.292$). The total diameter of the central region decreased significantly ($p < 0.001$). In the posterior 60µ region, a significant reduction was found in the 2–6 mm ($p = 0.047$) and 6–10 mm ($p = 0.016$) regions, with no significant change in the 0–2 mm ($p = 0.166$) and 10–12 mm ($p = 0.361$) regions. The total diameter in the posterior region also showed a significant reduction ($p = 0.042$). Lastly, in total thickness measurements, significant reductions were observed in the 0–2 mm ($p = 0.015$), 2–6 mm ($p = 0.001$), and 6–10 mm ($p < 0.001$) regions, with no significant change in the 10–12 mm region ($p = 0.379$). The total diameter also decreased significantly ($p = 0.001$).

At the time of diagnosis, no significant correlations were observed between IOP and corneal densitometry values ($p > 0.05$ for all). After treatment, this lack of significant correlation between changes in corneal densitometry parameters and variations in IOP persisted ($p > 0.05$ for all).

Discussion

This study investigated the effects of latanoprostene bunod treatment on corneal morphology as assessed by corneal tomography and specular microscopy. The findings indicated an increase in corneal transparency following treatment, while no significant changes were observed in the corneal endothelium, keratometric measurements, or anterior chamber parameters.

NO is a highly reactive signaling molecule synthesized from L-arginine by nitric oxide synthase (NOS) enzymes, which exist in three isoforms: neuronal (nNOS), inducible (iNOS), and endothelial (eNOS) [8]. eNOS, predominantly found in vascular endothelial cells, contributes to maintaining vascular homeostasis and may influence aqueous humor dynamics, potentially impacting IOP [9, 10]. Additionally, NO plays a crucial role in cellular proliferation, migration, and adhesion, all of which are essential processes for tissue repair, including corneal wound healing [11, 12]. However, its effects are concentration-dependent; lower levels promote cell proliferation, while excessive NO production can lead to cytotoxicity and tissue damage. [8, 13, 14]

In the cornea, NO has been implicated in both physiological and pathological processes. It contributes to corneal wound healing by regulating the activity of cytokines, growth factors, and proteinases, thereby influencing epithelial cell proliferation, migration, and differentiation [12, 15, 16]. NO is believed to support corneal deturgescence, helping to maintain proper hydration levels and transparency [17]. However, in pathological conditions such as uveitis and inflammatory corneal edema, excessive

NO production can contribute to tissue damage and endothelial dysfunction, leading to increased corneal thickness and opacity [9]. The dual nature of NO in corneal physiology underscores its significance as both a regulatory and potentially damaging factor, necessitating a balanced modulation of its production for optimal corneal health.

Latanoprostene bunod is a pharmacological agent used in the treatment of glaucoma, metabolizing into latanoprost acid and the NO-releasing component, butanediol mononitrate [18]. It effectively reduces IOP through a dual mechanism, facilitating both uveoscleral and trabecular outflow [19]. NO contributes to this process by inducing relaxation of the trabecular meshwork and Schlemm's canal, thereby enhancing aqueous humor drainage [19]. Randomized controlled trials have evaluated the efficacy of latanoprostene bunod in lowering IOP in individuals with open-angle glaucoma and ocular hypertension [20, 21]. However, previous studies have reported ocular side effects associated with topical latanoprostene bunod, including conjunctival hyperemia, eye irritation, eye pruritus, and foreign body sensation, which are comparable to those observed with other topical anti-glaucomatous medications [22]. Consistent with these findings, foreign body sensation and eye pruritus were noted as adverse effects in the present study. However, it is important to recognize that the limited duration of the present study restricts the available data on the side effects of latanoprostene bunod. Although the side effect profile of latanoprostene bunod has been well-documented, the effects of this treatment on corneal and anterior segment parameters have not been previously studied. Therefore, this study primarily aimed to evaluate the short-term effects of latanoprostene bunod treatment on corneal and anterior chamber parameters, which were assessed using quantitative and objective methods.

This study found no significant differences in corneal keratometry readings of the anterior and posterior corneal surfaces between the pre-treatment and post-treatment groups. While reductions in CCT, CTmin, and corneal volume were observed, these changes did not reach statistical significance. Similarly, alterations in ACD, ACV, and ACA following latanoprostene bunod treatment were not statistically significant. The comparison of endothelial cell characteristics demonstrated no statistically significant differences between the pre-treatment and post-treatment groups. Despite the absence of significant differences in corneal endothelial parameters, this study observed a reduction in corneal densitometry values across nearly all corneal layers, with the exception of the anterior, central, posterior, and total 10–12 mm zones, as well as the posterior 0–2 mm zone, suggesting an increase in corneal transparency.

These findings suggest that NO may play a role in maintaining corneal transparency. The results of the present study align with previous research demonstrating that NO contributes to corneal wound healing, the maintenance of corneal thickness, and the reduction of corneal opacity [12, 17]. Yanagiya et al. [17] investigated the effects of NO on the rabbit cornea and demonstrated that inhibition of NO synthase led to an increase in corneal thickness, indicating corneal edema. Additionally, Park et al. [12] examined the effects of exogenous topical NO on corneal wound healing in human corneal epithelial cells and corneal opacity in murine corneas subjected to alkali burn-induced damage. The findings of this study indicated that an exogenous NO donor-supported corneal epithelial healing and reduced corneal opacity [12]. However, the effects of NO were also found to be dose-dependent. In alignment with previous research [14], lower concentrations of sodium nitrite ($NaNO_2$) enhanced the viability of human corneal epithelial cells and facilitated corneal wound healing, whereas higher concentrations exhibited cytotoxic effects on them. Therefore, long-term studies with a larger participant cohort are required to evaluate the effects of chronic latanoprostene bunod use on corneal transparency and thickness, as this treatment is typically intended for prolonged use.

Decreased corneal densitometry values in the present study may also be attributed to lower IOP, reduced corneal thickness, and the effects of topical latanoprost treatment. Although the differences in IOP measurements between the pre- and post-treatment groups were statistically significant, it is unlikely that the decrease in IOP influenced corneal densitometry results, as the study included only patients with an IOP lower than 24 mmHg and no statistically significant correlations were found between corneal densitometry values and IOP measurements. It can be inferred that the observed decrease in corneal densitometry may be linked to the reduction in corneal thickness. However, in our

study, no significant difference was found between the pre-treatment and post-treatment CCT, CTmin, and corneal volume values. Topical latanoprost itself may have played a role in reducing corneal densitometry values through its effect on the organization of collagen fibrils [23, 24]. However, Sen et al. [25] investigated the effect of topical latanoprost on corneal transparency and found that corneal densitometry values remained unchanged during the first month of treatment, with a subsequent decrease observed three months after treatment initiation. Therefore, it can be suggested that the lower corneal densitometry results observed in this study are associated with the effects of NO on the cornea.

A major strength of this study is its prospective design, which enables a systematic evaluation of the short-term effects of latanoprostene bunod on corneal and anterior segment parameters through objective and quantitative imaging techniques. The inclusion of a carefully selected study population—consisting solely of patients with POAG and no additional ocular conditions—enhances the validity of the findings by reducing potential confounding factors. Furthermore, the use of advanced imaging modalities, such as Scheimpflug tomography and specular microscopy, ensures precise and reproducible measurements of corneal transparency, thickness, and endothelial cell properties. However, certain limitations should be acknowledged. The relatively small sample size may restrict the generalizability of the results, and the brief follow-up period does not provide insights into the long-term effects of latanoprostene bunod on corneal health. Additionally, the participants in this study were only evaluated using the Snellen chart to assess BCVA, which may limit the evaluation of the clinical significance of the observed increase in corneal transparency. More comprehensive evaluations of visual function could capture the subtleties of visual quality or other factors that might be influenced by changes in corneal transparency, such as contrast sensitivity and glare. Lastly, adding the study group consisting of patients treated with only topical latanoprost may enhance the validity of our study's results. We acknowledge that the knowledge about nitric oxide's role in improving corneal transparency remains uncertain; hence, we have analyzed these findings with caution, emphasizing the need for future histopathological and molecular studies to further investigate this potential mechanism. Future studies with larger sample sizes and longer follow-up durations are essential to further clarify the long-term impact of this treatment on corneal transparency and thickness.

In conclusion, this study provides new insights into the short-term effects of latanoprostene bunod on corneal and anterior segment parameters in patients with POAG. The findings suggest that latanoprostene bunod may enhance corneal transparency, as evidenced by a decrease in corneal densitometry values, while no significant changes were observed in corneal thickness or endothelial cell characteristics. However, long-term studies are warranted to assess the lasting effects of latanoprostene bunod on corneal health and its potential clinical significance on corneal transparency.

Author contributions Conception and design: Gözde Hondur, Ali Mert Kocer, Pınar Kosekahya Analysis and interpretation of the data: Aysel Uzun, Ali Mert Kocer, Kubra Özdemir Yalçınsoy Drafting of the paper: Ali Mert Kocer Revision: Gözde Hondur, Mehmet Murat Uzel Final approval: Gözde Hondur, Mehmet Murat Uzel, Ali Mert Kocer.

Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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