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Comparative Efficacy of Bimatoprost 0.03% and Travoprost 0.004% in Reducing Intraocular Pressure in Patients with Primary Open-Angle Glaucoma

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Abstract

Background: Glaucoma is a progressive optic neuropathy in which increased intraocular pressure (IOP) is a primary risk factor, leading to vision loss. The present study was initiated to compare the intraocular pressure (IOP)-lowering efficacy of 0.03% bimatoprost and 0.004% travoprost in patients with unilateral primary open-angle glaucoma (POAG).

Methods and Results: A total of 100 patients with POAG were treated with a single hypotensive agent during a 6-month follow-up period. The patients were divided into two groups. Group 1 patients (n=50) were treated with 0.03% bimatoprost, and Group 2 patients (n=50) were treated with 0.004% travoprost. We assessed the IOP at baseline, 1 week, 4 weeks, 12 weeks, and 6 months after treatment.

There were no significant differences between the two treatments at the initial stage. At 1 week and 4 weeks after treatment, the mean IOP significantly decreased from baseline in both groups. There were no significant differences between the two treatments at these stages. After 12 weeks of treatment, IOP continued to decrease effectively in both groups; however, 0.03% bimatoprost provided lower IOP values, and there was a significant difference between groups. In the bimatoprost group, compared to baseline, the reduction was 30.9%, while in the travoprost group, the reduction was 28.3% (P=0.043). The difference was greater after 6 months: in the bimatoprost group, the reduction was 33.0% compared to baseline, while in the travoprost group, the reduction was 29.7% (P=0.033).

Conclusion: The consistently lower IOP values for 0.03% bimatoprost treatment suggest it may be more effective in lowering IOP than 0.004% travoprost.(**International Journal of Biomedicine. 2025;15(3):527-530.**)

Keywords: glaucoma • intraocular pressure • bimatoprost • travoprost

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Abbreviations

IOP, intraocular pressure; **PGA**, prostaglandin analogues; **POAG,** primary open-angle glaucoma.

Introduction

Glaucoma is a progressive optic neuropathy in which increased intraocular pressure (IOP) is a primary risk factor, leading to vision loss. The treatment of glaucoma aims to decrease the IOP, which is the only treatable risk factor. However, in glaucomatous optic neuropathy, the continuous progression of glaucoma despite a decrease in IOP of ≥ 30% suggests that vascular, genetic, and other factors play an important role in the pathogenesis of the disease.

Lowering IOP reduces the risk of visual field loss in patients with glaucoma and ocular hypertension. Based on the findings of the Early Manifest Glaucoma Trial, it is suggested that every millimeter of IOP lowering corresponds to a reduction in the risk of glaucomatous progression of

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approximately 10%.[1] The current treatment paradigm for patients with glaucoma or ocular hypertension focuses on reducing the IOP to a target level sufficiently low to preserve the visual field.[2]

A primary goal of medical therapy in glaucoma is to reduce IOP. Due to the significant impact on the reduction of the IOP, prostaglandin analogues, used once daily, have become the most commonly used first-line agents in glaucoma and ocular hypertension.[3-7]

Bimatoprost 0.03% is a potent and highly efficacious monotherapy that allows many patients to achieve low target pressures.[8] Furthermore, several clinical trials have shown that bimatoprost 0.03% monotherapy lowers the IOP more effectively than either latanoprost or timolol.[8,9] Bimatoprost is a prostamide, a synthetic, prostaglandin (PG)-related molecule, that reduces the IOP by increasing both the pressure-sensitive (presumed trabecular meshwork) and the pressure-insensitive (presumed uveoscleral) outflow.[10]

Travoprost 0.004%, a synthetic prostaglandin, F2a receptor agonist, lowers the IOP by increasing the uveoscleral outflow. Many clinical trials have shown that the IOP-lowering efficacy of travoprost monotherapy is superior to that of timolol and roughly equivalent to that of latanoprost.[10-12] Our study aimed to compare the IOP-lowering efficacy of 0.03% bimatoprost and 0.004% travoprost in patients with unilateral primary open-angle glaucoma (POAG).

Materials and Methods

This prospective cohort study was conducted in the Ophthalmology Department at the University Clinical Center of Kosovo (Prishtina, Kosovo) from January 2023 to February 2025. A total of 100 previously untreated patients with newly diagnosed POAG were initially enrolled in the study and randomly assigned to one of two groups in a double-masked fashion. Group 1 patients (n=50) were treated with 0.03% bimatoprost, and Group 2 patients (n=50) were treated with 0.004% travoprost.

Medication was administered at 24-hour intervals (between 20.00 hours and 22.00 hours) every day for 6 months. No other IOP-reducing therapy was permitted.

Primary open-angle glaucoma was diagnosed according to the European Glaucoma Society, based on the presence of typical glaucomatous optic disc damage (asymmetry between the vertical cup: disc ratios > 0.2, thinning of the neuroretinal rim, optic disc haemorrhages, parapapillary atrophy) with glaucomatous visual field loss, open angles, and IOP levels > 21 mmHg.

Data Collection

Eligible patients for the study were patients of both genders, aged ≥ 18 years, and diagnosed as having unilateral primary open-angle glaucomawith IOP of 22–36 mmHg during the pre-study period, who had not received any prior medical treatment to lower IOP or alter cardiovascular status (such as adrenergic agents, calcium channel blockers, carbonic anhydrase inhibitors, angiotensin-converting enzyme inhibitors and/or angiotensin receptor blockers). All subjects included in the study did not have any cardiovascular disease, systemic hypertension, or diabetes mellitus. The exclusion criteria included any corneal abnormalities or other diseases that could interfere with accurate IOP measurement with a Goldmann applanation tonometer.

During the treatment period, scheduled visits were performed at baseline and at the end of weeks 1, 4, 12, and after 6 months of treatment. IOP was measured with a Goldmann applanation tonometer.

Statistical analysis was performed using the statistical software package SPSS version 22.0 (SPSS Inc., Armonk, NY: IBM Corp). Baseline characteristics were summarized as frequencies and percentages for categorical variables and mean (M) ± standard deviation (SD) for continuous variables. The unpaired t-test was used to compare two groups. Group comparisons concerning categorical variables were performed using chi-square test. A probability value of $P<0.05$ was considered statistically significant.

Results

The demographic characteristics of patients included in the study are presented in Table 1. In terms of gender, there were more females in both groups, however, with no significant difference between the groups ($P>0.05$). At baseline, the IOP for both groups were very similar: 26.2±1.9 mmHg in Group 1 and 26.3±1.9 mmHg in Group 1 ($P=0.958$) (Table 1).

Table 1.

Comparison of clinical and demographic characteristics between study groups

	Group 1 (n=50) (Bimatoprost)	Group 2 (n=50) (Travoprost)	*P*-value
Gender n (%) Male Female	 22 (44.0) 28 (56.0)	 21 (42.0) 29 (58.0)	0.999
Age (year) Mean ± SD	64.6 ± 10.6	70.6 ± 10.5	0.068
Mean Baseline IOP (mmHg) Mean ± SD	26.2 ± 1.9	26.3 ± 1.9	0.958

At 1 week and 4 weeks after treatment, the mean IOP significantly decreased from baseline in both groups. There were no significant differences between the two treatments at these stages. After 12 weeks of treatment, IOP continued to decrease effectively in both groups; however, 0.03% bimatoprost provided lower IOP values, and there was a significant difference between groups. In the bimatoprost group, compared to baseline, the reduction was 30.9%, while in the travoprost group, the reduction was 28.3% ($P=0.043$). The difference was greater after 6 months: in the bimatoprost group, the reduction was 33.0% compared to baseline, while in the travoprost group, the reduction was 29.7% ($P=0.033$) (Table 2).

Table 2.

Changes in IOP during scheduled visits in the study groups.

	Group 1 (n=50) (Bimatoprost)	Group 2 (n=50) (Travoprost)	*P*-value
Mean (mmHg) and %IOP change from baseline Mean ± SD (%)			
Baseline	-	-	
1 weeks	5.9 ± 2.0 (22.4%)	5.6 ± 2.0 (21.0%)	0.316
4 weeks	7.7 ± 2.1 (29.1%)	7.1 ± 2.2 (26.6%)	0.071
12 weeks	8.2 ± 2.1 (30.9%)	7.5 ± 2.4 (28.3%)	0.043
6 months	8.8 ± 2.3 (33.0%)	7.9 ± 2.1 (29.7%)	0.038
IOP (mmHg) (Mean ± SD)			
Baseline	26.2 ± 1.9	26.3 ± 1.9	0.958
1 weeks	20.4 ± 1.8	20.7 ± 1.8	0.318
4 weeks	18.6 ± 1.8	19.2 ± 1.6	0.067
12 weeks	18.1 ± 1.7	18.8 ± 1.5	0.045
6 months	17.5 ± 1.2	18.4 ± 1.5	0.0006

Discussion

The American Academy of Ophthalmology preferred practice patterns suggest that reductions of at least 20% from untreated IOP levels should be targeted as a goal of treatment to prevent glaucomatous progression.[13] The findings of our study show that patients treated with bimatoprost 0.03% achieved higher reductions of this magnitude, compared with patients treated with travoprost 0.004%. Moreover, almost 40% had a decrease of at least 30% from baseline. Patients treated with bimatoprost 0.03% were also more likely to experience better clinical outcomes compared to those treated with travoprost 0.004%. Due to poor treatment outcomes and the lack of efficacy, patients in the travoprost group were more likely to discontinue the treatment.

The primary goal of glaucoma treatment is to reduce IOP to the target pressure using a minimal number of medications. The prospective randomized study by Gandolfi et al. and also other studies confirm previous reports,[14-16] demonstrating that additional IOP lowering may be achieved by switching patients who are inadequately controlled on latanoprost to another PGA and reinforces the concept that changing therapy within the PGA class should be considered before adding a second medication if further IOP lowering is required.[17]

In a study by Cantor et al.,[2] there was no significance between-group differences observed in IOP at baseline, at 09:00, 13:00 or 16:00 h (*P*=0.741). After 6 months, both drugs significantly reduced IOP at every time point (*P*<0.001). After 6 months, the mean IOP reduction at 09:00 h was 7.1 mmHg (27.9%) with bimatoprost (n=76) and 5.7 mmHg (23.3%) with travoprost (n=81) (*P*=0.014). At 13:00 h, the mean IOP reduction was 5.9 mmHg with bimatoprost (25.3%) and 5.2 mmHg (22.4%) with travoprost (*P*=0.213). At 16:00 h, the mean IOP reduction was 5.3 mmHg (22.5%) with bimatoprost and 4.5 mmHg (18.9%) (*P*=0.207) with travoprost, similar to our findings. Our results also show that bimatoprost had a higher IOP-lowering efficacy.

In a study of the Egyptian population where seventy-two patients were included in the study, both treatment agents provided statistically significant IOP reductions from baseline at all visits (*P*<0.001); however, bimatoprost provided greater mean IOP reductions from baseline compared to travoprost at each visit. Mean IOP reductions were 8.77 mmHg (33.39%) and 8.42 mmHg (31.54%) at 2 weeks (*P*=0.703), and 8.47 mmHg (31.61%) and 7.84 mmHg (29.50%) at 6 months (*P*=0.536) for bimatoprost and travoprost, respectively. IOP in the two groups at 2 weeks were ≤18 mmHg in 20(58.8%) versus 19(50%) eyes (*P*=0.603), and ≤16 mmHg in 12(35%) versus 12(32%) eyes (*P*=0.456); and at 6 months ≤18 mmHg in 22(65%) versus 14(37%) eyes (*P*=0.045), and ≤16 mmHg in 12(35%) versus 7(18%) eyes (*P*=0.037) for bimatoprost and travoprost, respectively.[18] The data from the literature aligns with the findings of our study, where the superiority of bimatoprost in IOP lowering is shown. In terms of safety and side effects, both agents were proven to be safe. In conclusion, the consistently lower IOP values for 0.03% bimatoprost treatment suggest it may be more effective in lowering IOP than 0.004% travoprost.

Ethical Considerations

The study was conducted in accordance with the ethical principles of the WMA Declaration of Helsinki (1964, ed. 2013) and approved by the National Research Ethics Committee (Nr.12084). Written informed consent was obtained from all participants.

Competing Interests

The authors declare that they have no competing interests.

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