LETTERS IN BIOMATHEMATICS
An International Journal

RESEARCH ARTICLE

OPEN ACCESS

Impact of Essential Hypertension on Ocular Perfusion Pressure in Primary Open-Angle Glaucoma-A Case – control study

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ABSTRACT

Background: Primary open-angle glaucoma (POAG) is a progressive optic neuropathy in which vascular factors, including systemic hypertension and ocular perfusion pressure (OPP), may contribute to glaucomatous damage. The relationship between essential hypertension, OPP, and glaucoma severity remains complex.

Objectives: To compare OPP between POAG patients with essential hypertension and normotensive POAG patients and to evaluate differences in structural and functional parameters of glaucoma severity between the groups.

Methods: This hospital-based case–control study included 84 patients with POAG attending the Department of Ophthalmology, R. L. Jalappa Hospital and Research Centre, Kolar, over one year. Participants were divided into two groups of 42 each based on hypertension status. Systemic blood pressure, intraocular pressure (IOP), and OPP were assessed. Glaucoma severity was evaluated using optic disc examination, Humphrey visual field analysis, and optical coherence tomography measurements of retinal nerve fibre layer (RNFL) and ganglion cell complex (GCC) thickness. Appropriate statistical tests were applied, with $p<0.05$ considered significant.

Results: Mean OPP was significantly higher in hypertensive patients than in normotensive patients (54.16 ± 5.15 versus 44.58 ± 4.04 mmHg; $p<0.001$), despite comparable IOP

($p=0.838$). Hypertensive patients demonstrated significantly greater cup-to-disc ratios ($p=0.036$), reduced superior RNFL thickness ($p=0.010$), and reduced inferior GCC thickness ($p=0.049$). Although visual field mean deviation, pattern standard deviation, and visual field index indicated numerically greater functional impairment among hypertensive patients, these differences were not statistically significant.

Conclusion: Essential hypertension was associated with higher calculated OPP and greater structural glaucomatous abnormalities despite comparable IOP. Higher daytime OPP may not necessarily indicate protection against optic nerve damage. Assessment of systemic vascular factors alongside conventional glaucoma parameters may support comprehensive POAG management.

ARTICLE HISTORY
Received September 27, 2026
Accepted October 31, 2026

KEYWORDS
Primary Open-Angle Glaucoma; Essential Hypertension; Ocular Perfusion Pressure; Intraocular Pressure; Retinal Nerve Fibre Layer; Ganglion Cell Complex.

1. Introduction

Glaucoma is a leading cause of irreversible blindness worldwide and represents a significant public health concern. Primary open-angle glaucoma (POAG) is a chronic, progressive optic neuropathy characterized by retinal ganglion cell loss, optic nerve head damage, and corresponding visual field defects. Although elevated intraocular pressure (IOP) is the principal modifiable risk factor, increasing evidence suggests that vascular abnormalities and impaired ocular blood flow contribute significantly to glaucomatous optic nerve damage.[1,2]

Ocular perfusion pressure (OPP), determined by the relationship between systemic blood pressure and IOP, is an important indicator of the pressure driving ocular circulation. Mean OPP is commonly estimated as two-thirds of mean arterial pressure minus IOP. Reduced OPP may compromise optic nerve head perfusion, resulting in ischemic injury and progressive glaucomatous damage.[1,2]

Essential hypertension has a complex relationship with POAG. Chronic elevation of systemic blood pressure may induce vascular endothelial dysfunction, microvascular alterations, and impaired autoregulation of ocular blood flow. Conversely, excessive blood pressure reduction, particularly nocturnal hypotension associated with antihypertensive treatment, may decrease OPP and increase

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susceptibility to optic nerve ischemia.[2,3] Previous studies have demonstrated associations between abnormal blood pressure patterns, reduced OPP, and glaucomatous visual field damage; however, findings regarding the independent influence of systemic hypertension on POAG severity remain inconsistent.[4,5]

Furthermore, evidence comparing OPP and glaucomatous damage between hypertensive and normotensive individuals, particularly in rural populations, remains limited. Understanding these associations may facilitate improved identification of vascular risk factors and individualized management of POAG.

Therefore, the present case–control study aims to compare ocular perfusion pressure between patients with POAG and essential hypertension and those with POAG without systemic hypertension, and to evaluate the association between OPP and glaucoma severity in both groups within a rural population.

2. Material and Methods:

This hospital-based, case–control study was conducted over a period of one year in the Department of Ophthalmology, R. L. Jalappa Hospital and Research Centre, Kolar, Karnataka, India. Institutional Ethics Committee approval details was obtained prior to the start of the study from SDUAHER ethics committee [IEC NO:SUDUAHER/R&D/CEC/SDUMC-F/NF/-2025-26]. The study included 84 patients aged 40–75 years attending the ophthalmology outpatient department. Participants were recruited using a convenience sampling technique and categorized into two groups: Group I comprised 42 patients with primary open-angle glaucoma (POAG) and essential hypertension, whereas Group II comprised 42 patients with POAG who were systemically normotensive.

Patients aged 40–75 years with confirmed POAG were eligible for inclusion. POAG was diagnosed based on characteristic glaucomatous optic disc cupping, corresponding visual field defects, thinning of the retinal nerve fibre layer (RNFL) and neuroretinal rim on optical coherence tomography (OCT), and open anterior chamber angles on gonioscopy, irrespective of intraocular pressure (IOP).

Patients in the hypertensive group had previously diagnosed essential hypertension or newly detected hypertension, defined as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg. The comparison group included patients with POAG who were normotensive.

Patients with secondary glaucoma, angle-closure glaucoma, a history of ocular trauma, diabetes mellitus, significant systemic vascular diseases, or ocular surgery within the preceding six months were excluded. Additional exclusion criteria included refractive errors exceeding ±6 dioptres, media opacities interfering with optic nerve or retinal evaluation, and noncompliance with prescribed antihypertensive treatment.

The sample size was calculated based on the difference in mean ocular perfusion pressure (MOPP) between hypertensive and normotensive individuals reported by Khandelwal et al.[6] The mean MOPP values were 59.22 ± 6.06 mmHg and 55.41 ± 2.50 mmHg, respectively. Sample size estimation was performed using the formula for comparing two independent means, with reference to the anticipated difference in mean MOPP between the groups.[7] 42 participants were included in each group, resulting in a total sample size of 84, after accounting for an anticipated 10% nonresponse rate.

Written informed consent was obtained from all participants before enrolment. Demographic characteristics, relevant medical and ocular history, duration of systemic hypertension, and details of antihypertensive medications were recorded using a structured data collection form. Systemic blood pressure was measured in the right upper arm with the participant in a seated position using a mercury sphygmomanometer and the standard auscultatory technique. Systolic and diastolic blood pressures were determined using the first and fifth Korotkoff sounds, respectively, in accordance with the American Heart Association recommendations.[8] All participants underwent a comprehensive ophthalmic evaluation. Best-corrected visual acuity was assessed using a Snellen chart at a distance of six metres. Anterior segment examination was performed using slit-lamp biomicroscopy to identify ocular abnormalities and exclude secondary causes of glaucoma.

POAG severity was classified using visual field mean deviation, such as the Hodapp–Parrish–Anderson criteria. Intraocular pressure was measured using Goldmann applanation tonometry, preferably between 9:00 AM and 11:00 AM, to minimize the influence of diurnal variations. Gonioscopy was performed using a Goldmann three-mirror lens to assess anterior chamber angle configuration and confirm open angles corresponding to Shaffer grades III or IV. Dilated fundus examination was performed using a +90 dioptre lens at the slit lamp to evaluate the optic nerve head, vertical cup-to-disc ratio, neuroretinal rim configuration, and other characteristic features of glaucomatous optic neuropathy. Visual field assessment was performed using the Humphrey Field Analyzer with the 24-2 Swedish Interactive Threshold Algorithm (SITA) Standard protocol to identify characteristic glaucomatous field defects and assess functional disease severity. Spectral-domain OCT was performed to evaluate structural glaucomatous changes, including peripapillary RNFL thickness and macular ganglion cell complex thickness, wherever applicable. Only OCT scans with signal strength greater than 6/10 were considered for analysis.

Calculation of Ocular Perfusion Pressure: Mean arterial pressure (MAP) was calculated from the measured systolic and diastolic blood pressures using the following formula:

MAP = DBP + ⅓ (SBP − DBP)

Mean ocular perfusion pressure (MOPP) was estimated using the standardized formula:

MOPP = ⅔ × MAP − IOP

where SBP represents systolic blood pressure, DBP represents diastolic blood pressure, and IOP represents intraocular pressure. All measurements were expressed in mmHg.

The calculated MOPP values were compared between hypertensive and normotensive patients with POAG. The association between MOPP and glaucoma severity was evaluated using the available clinical, structural, and visual field parameters.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Epi Info version 7.2.1, and Jamovi version 2.6.26.0. Categorical variables were expressed as frequencies and percentages, whereas continuous variables were summarized using means and standard deviations. The distribution of continuous variables was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. The independent-samples Student's t-test was used to compare normally distributed continuous variables between the two groups, whereas the Mann–Whitney U test was applied to variables that did not follow a normal distribution. Associations between categorical variables were evaluated using the chi-square test, subject to the expected cell-frequency assumptions. All statistical tests were two-sided, and a p-value of less than 0.05 was considered statistically significant.

3. Results

Table 1: Baseline demographic and clinical characteristics of the study groups

	Characteristic	POAG + Essential Hypertension (n = 42)	POAG + Normotension (n = 42)	Test statistic	p-value
Age, years,	mean ± SD	61.33 ± 8.20	56.21 ± 8.59	*t* = 2.793	**0.006***
Age group, n (%)	<50 years	3 (7.1)	10 (23.8)	χ^2 = 9.279	**0.026***
	51–60 years	17 (40.5)	18 (42.9)		
	61–70 years	14 (33.3)	13 (31.0)		
	>70 years	8 (19.0)	1 (2.4)		
Sex, n (%)	Female	23 (54.8)	20 (47.6)	χ^2 = 0.429	0.513
	Male	19 (45.2)	22 (52.4)		
Eye, n (%)	Left	23 (54.8)	21 (50.0)	χ^2 = 0.191	0.662
	Right	19 (45.2)	21 (50.0)		
Family history of glaucoma, n (%)	No	34 (81.0)	30 (71.4)	χ^2 = 1.050	0.306
	Yes	8 (19.0)	12 (28.6)		
Diabetes mellitus, n (%)	No	29 (69.0)	37 (88.1)	χ^2 = 4.525	**0.033***
	Yes	13 (31.0)	5 (11.9)		
Smoking status, n (%)	Current	5 (11.9)	4 (9.5)	χ^2 = 0.650	0.723
	Former	6 (14.3)	4 (9.5)		
	Never	31 (73.8)	34 (81.0)		

BMI, kg/m²,	mean ± SD	25.03 ± 3.34	24.54 ± 3.50	*t* = 0.657	0.513
Antihypertensive treatment, n (%)	ACEI/ARB	6 (14.3)	-	-	-
	ACEI/ARB + CCB	9 (21.4)	-	-	-
	Beta blocker	6 (14.3)	-	-	-
	CCB	12 (28.6)	-	-	-
	Other	9 (21.4)	-	-	-
	Hypertension duration, years, mean ± SD	8.67 ± 4.08	-	-	-

Independent t test, Chi-square test

Abbreviations: POAG, primary open-angle glaucoma; SD, standard deviation; BMI, body mass index; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker.

In the present study, the mean age of subjects in Group A was 61.33 ± 8.20 years compared with 56.21 ± 8.59 years in Group B, and the difference was statistically significant (p = 0.006). In Group A, the majority of subjects were aged 51–60 years (40.5%), followed by 61–70 years (33.3%), >70 years (19.0%), and <50 years (7.1%). Similarly, in Group B, the

majority were aged 51–60 years (42.9%), followed by 61–70 years (31.0%), <50 years (23.8%), and >70 years (2.4%). There was a statistically significant difference in age-group distribution between the two groups (p = 0.026). Females constituted 54.8% in Group A and 47.6% in Group B, with no significant difference in sex distribution (p = 0.513). Left-eye involvement was observed in 54.8% and 50.0% of subjects in Groups A and B, respectively, with no significant difference (p = 0.662). A family history of glaucoma was present in 19.0% of Group A and 28.6% of Group B, with no statistically significant difference (p = 0.306). Diabetes mellitus was significantly more common in Group A (31.0%) than in Group B (11.9%) (p = 0.033). Most subjects were never smokers in both Group A (73.8%) and Group B (81.0%), with no significant difference in smoking status (p = 0.723). The mean BMI was 25.03 ± 3.34 kg/m² in Group A and 24.54 ± 3.50 kg/m² in Group B, with no statistically significant difference (p = 0.513). Among Group A subjects, calcium channel blockers were the most commonly used antihypertensive medication (28.6%), and the mean duration of hypertension was 8.67 ± 4.08 years.

Table 2: Comparison of systemic blood pressure parameters between POAG patients with essential hypertension and normotension

	POAG + Essential Hypertension (n = 42),		**POAG + Normotension (n = 42),**		**p-value**
Parameter	**Mean ± SD**	**Median**	**Mean ± SD**	**Median**	
SBP (mmHg)	145.60 ± 9.04	148	119.69 ± 6.86	120	<0.001*
DBP (mmHg)	86.43 ± 7.78	85	77.60 ± 6.31	78	<0.001*
MAP (mmHg)	106.15 ± 6.24	106.7	91.63 ± 4.79	91.2	<0.001*

Abbreviations: POAG, primary open-angle glaucoma; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; SD, standard deviation; CI, confidence interval.

In the present study, the mean systolic blood pressure was significantly higher in Group A (145.60 ± 9.04 mmHg) compared with Group B (119.69 ± 6.86 mmHg) (p < 0.001). Similarly, the mean diastolic blood pressure was significantly higher in Group A (86.43 ± 7.78 mmHg) than in Group B (77.60 ± 6.31 mmHg) (p < 0.001). The mean arterial pressure was also significantly higher in Group A (106.15 ± 6.24 mmHg) compared with Group B (91.63 ± 4.79 mmHg) (p < 0.001). Thus, all systemic blood pressure parameters were significantly higher among POAG subjects with essential hypertension.

Table 3: Comparison of ocular parameters between POAG patients with essential hypertension and normotension

Parameter	POAG + Essential Hypertension (n = 42)		POAG + Normotension (n = 42)		p-value
	Mean ± SD	Median	Mean ± SD	Median	
Visual acuity (LogMAR)	0.20 ± 0.13	0.2	0.17 ± 0.12	0.1	0.232
IOP (mmHg)	16.61 ± 2.70	17	16.50 ± 2.03	16.5	0.838
OPP (mmHg)	54.16 ± 5.15	53.9	44.58 ± 4.04	45	**<0.001***

In the present study, the mean visual acuity was 0.20 ± 0.13 LogMAR in Group A and 0.17 ± 0.12 LogMAR in Group B, with no statistically significant difference between the groups (p = 0.232). The mean intraocular pressure was also comparable between Group A (16.61 ± 2.70 mmHg) and Group B (16.50 ± 2.03 mmHg), with no statistically significant difference (p = 0.838). However, the mean ocular perfusion pressure was significantly higher in Group A (54.16 ± 5.15 mmHg) compared with Group B (44.58 ± 4.04 mmHg) (p < 0.001). Thus, ocular perfusion pressure showed a significant difference between the two groups, whereas visual acuity and intraocular pressure were comparable.

Table 4: Comparison of glaucoma duration and antiglaucoma medication use between study groups

Parameter	POAG + Essential Hypertension (n = 42)		POAG + Normotension (n = 42)		p-value
	Mean ± SD	Median	Mean ± SD	Median	
Glaucoma duration (years)	5.74 ± 3.55	5	5.43 ± 3.22	5	0.676
Number of antiglaucoma medications	1.98 ± 0.95	2	1.83 ± 0.91	2	0.483

In the present study, the mean duration of glaucoma was 5.74 ± 3.55 years in Group A and 5.43 ± 3.22 years in Group B. There was no statistically significant difference in glaucoma duration between the two groups (p = 0.676). The mean number of antiglaucoma medications used was 1.98 ± 0.95 in Group A compared with 1.83 ± 0.91 in Group B, and this difference was also not statistically significant (p = 0.483). Thus, both groups were comparable with respect to glaucoma duration and the number of antiglaucoma medications used.

Table 5: Comparison of cup-to-disc ratio between POAG patients with essential hypertension and normotension

Parameter	POAG + Essential Hypertension (n = 42)		POAG + Normotension (n = 42)		p-value
	Mean ± SD	Median	Mean ± SD	Median	
Cup-to-disc ratio	0.78 ± 0.10	0.80	0.74 ± 0.10	0.73	**0.036***

Independent t test

In the present study, the mean cup-to-disc ratio was 0.78 ± 0.10 in Group A compared with 0.74 ± 0.10 in Group B. The cup-to-disc ratio was significantly higher among subjects with POAG and essential hypertension compared with those with POAG and normotension, and the difference was statistically significant (p = 0.036). This indicates greater optic disc cupping in Group A.

Table 6: Comparison of visual field parameters between POAG patients with essential hypertension and normotension

Parameter	POAG + Essential Hypertension (n = 42)		POAG + Normotension (n = 42)		p-value
	Mean ± SD	Median	Mean ± SD	Median	
HVF MD (dB)	−9.22 ± 5.73	−8.1	−7.81 ± 5.46	−7.1	0.252

HVF PSD (dB)	6.00 ± 2.87	6.7	5.64 ± 2.55	5.2	0.554
VFI (%)	76.10 ± 18.99	83	81.07 ± 16.60	88	0.205

In the present study, the mean Humphrey Visual Field mean deviation (HVF MD) was −9.22 ± 5.73 dB in Group A and −7.81 ± 5.46 dB in Group B, with no statistically significant difference (p = 0.252). The mean pattern standard deviation (HVF PSD) was 6.00 ± 2.87 dB in Group A and 5.64 ± 2.55 dB in Group B, which was also not statistically significant (p = 0.554). The mean Visual Field Index was 76.10 ± 18.99% in Group A compared with 81.07 ± 16.60% in Group B, with no statistically significant difference (p = 0.205). Thus, although Group A showed numerically greater visual field impairment, none of the visual field parameters differed significantly between the two groups.

Table 7: Comparison of OCT-derived RNFL and GCC thickness between POAG patients with essential hypertension and normotension

Parameter	POAG + Essential Hypertension (n = 42)		POAG + Normotension (n = 42)		p-value
	Mean ± SD	Median	Mean ± SD	Median	
Average RNFL	66.67 ± 13.44	65	71.43 ± 13.44	73	0.108
Superior RNFL	67.88 ± 12.74	67	75.40 ± 13.22	75	**0.01***
Inferior RNFL	65.07 ± 13.94	62	70.02 ± 16.22	74	0.137
Average GCC	64.98 ± 11.52	68	69.83 ± 11.25	72	0.054
Superior GCC	66.36 ± 12.02	68	69.76 ± 11.13	72	0.182
Inferior GCC	63.05 ± 11.78	65	68.24 ± 12.04	73	**0.049***

In the present study, the mean average RNFL thickness was lower in Group A (66.67 ± 13.44 µm) compared with Group B (71.43 ± 13.44 µm), although the difference was not statistically significant (p = 0.108). The mean superior RNFL thickness was significantly lower in Group A (67.88 ± 12.74 µm) than in Group B (75.40 ± 13.22 µm) (p = 0.010). The mean inferior RNFL thickness was 65.07 ± 13.94 µm in Group A and 70.02 ± 16.22 µm in Group B, with no statistically significant difference (p = 0.137). The mean average GCC thickness was lower in Group A (64.98 ± 11.52 µm) compared with Group B (69.83 ± 11.25 µm), but the difference did not reach statistical significance (p = 0.054). The mean superior GCC thickness was 66.36 ± 12.02 µm in Group A and 69.76 ± 11.13 µm in Group B, with no significant difference (p = 0.182). However, the mean inferior GCC thickness was significantly lower in Group A (63.05 ± 11.78 µm) compared with Group B (68.24 ± 12.04 µm) (p = 0.049). Thus, significant thinning of the superior RNFL and inferior GCC was observed among POAG patients with essential hypertension.

4. Discussion

Primary open-angle glaucoma (POAG) is a progressive optic neuropathy characterized by structural damage to the optic nerve head and corresponding visual field loss. Although elevated intraocular pressure (IOP) remains the major modifiable risk factor, vascular factors, particularly systemic blood pressure and ocular perfusion pressure (OPP), are increasingly recognized as important contributors to glaucomatous damage.[9,10] The present study evaluated the influence of essential hypertension on OPP and glaucomatous optic nerve changes among 84 patients with POAG attending a tertiary care hospital in rural Karnataka. The findings demonstrated significantly higher calculated OPP among hypertensive patients despite comparable IOP levels. However, these patients also exhibited greater optic disc cupping and significant thinning of selected retinal nerve fibre layer (RNFL) and ganglion cell complex (GCC) parameters.

The mean age of hypertensive POAG patients was significantly higher than that of normotensive patients (61.33 ± 8.20 versus 56.21 ± 8.59 years; p = 0.006). This observation is clinically relevant because increasing age is an established risk factor for glaucoma and is also associated with a higher prevalence of systemic hypertension. Age-related vascular changes, including arterial stiffness, endothelial dysfunction, and reduced autoregulatory capacity, may influence optic nerve head circulation.[10] Therefore, the greater age of hypertensive participants should be considered when interpreting differences in structural glaucomatous damage. The two groups were comparable regarding sex distribution, family history of glaucoma, smoking status, and body mass index. However, diabetes mellitus was significantly more common in the hypertensive group (31.0% versus 11.9%; p = 0.033), representing another potential vascular confounding factor.

As expected, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were significantly higher among

hypertensive POAG patients. The mean systolic blood pressure was 145.60 ± 9.04 mmHg in the hypertensive group compared with 119.69 ± 6.86 mmHg in the normotensive group ($p < 0.001$). Similarly, mean arterial pressure was significantly higher among hypertensive patients (106.15 ± 6.24 versus 91.63 ± 4.79 mmHg; $p < 0.001$). These findings reflect the systemic haemodynamic differences between the study groups and provide a physiological basis for the observed variation in calculated OPP.

The principal finding of this study was the significantly higher mean OPP in hypertensive POAG patients (54.16 ± 5.15 mmHg) compared with normotensive POAG patients (44.58 ± 4.04 mmHg; $p < 0.001$). In contrast, mean IOP was comparable between the groups (16.61 ± 2.70 versus 16.50 ± 2.03 mmHg; $p = 0.838$). Since OPP is calculated using mean arterial pressure and IOP, the higher OPP observed in hypertensive patients was primarily attributable to their elevated systemic blood pressure rather than differences in IOP.

These findings are consistent with the observations of Khandelwal et al., who investigated the relationship between systemic hypertension, OPP, and open-angle glaucoma.[6] Their study demonstrated higher overall OPP among hypertensive individuals compared with normotensive participants. However, variations in OPP were observed among hypertensive patients with glaucoma, particularly in relation to antihypertensive treatment. These findings suggest that the relationship between systemic hypertension and ocular perfusion is complex and may depend on blood pressure control, treatment status, and underlying vascular changes. The present study similarly demonstrates that elevated systemic blood pressure increases calculated daytime OPP, although this does not necessarily indicate adequate optic nerve head perfusion.

Ocular perfusion pressure represents the pressure gradient responsible for maintaining ocular blood flow. However, calculated OPP is an indirect estimate and does not measure actual blood flow through the optic nerve head. Under physiological conditions, ocular vascular autoregulation maintains relatively stable blood flow despite fluctuations in systemic blood pressure and IOP. Chronic hypertension may impair this mechanism through vascular remodelling, endothelial dysfunction, and increased vascular resistance. Consequently, patients with longstanding hypertension may experience inadequate tissue perfusion despite having relatively high calculated OPP.[10]

Caprioli and Coleman et al emphasized the importance of blood pressure and perfusion pressure in glaucomatous optic neuropathy, particularly when vascular autoregulation is impaired.[10] Their observations support the possibility that systemic hypertension may influence glaucoma through mechanisms beyond its immediate effect on calculated OPP. Therefore, the higher OPP observed in the hypertensive group should not be interpreted as evidence of better ocular circulation or protection against glaucomatous damage.

Another important consideration is the effect of antihypertensive therapy on ocular perfusion. In the present study, the mean duration of hypertension was 8.67 ± 4.08 years, and calcium channel blockers were the most commonly prescribed antihypertensive medications. Although systemic blood pressure was higher in the hypertensive group during examination, these measurements may not reflect blood pressure variations throughout the day and night.

Melgarejo et al. reported that abnormalities in 24-hour blood pressure regulation and reduced ocular perfusion were associated with progression of open-angle glaucoma.[11] Excessive reductions in blood pressure, particularly during the night, may compromise optic nerve head perfusion in susceptible individuals. This may be relevant in hypertensive patients receiving antihypertensive treatment, although the present study did not include ambulatory blood pressure monitoring. Therefore, nocturnal hypotension and treatment-related fluctuations in OPP remain possible explanations rather than demonstrated mechanisms for the observed structural differences.

The present study also demonstrated significantly greater optic disc cupping among hypertensive POAG patients. The mean cup-to-disc ratio was 0.78 ± 0.10 in the hypertensive group compared with 0.74 ± 0.10 in the normotensive group ($p = 0.036$). Increased cupping reflects structural changes associated with glaucomatous optic nerve damage. The finding of greater cupping despite comparable IOP suggests that factors other than measured IOP may contribute to differences in optic nerve morphology between the groups. Chronic vascular abnormalities associated with hypertension could be one such factor, although the observational design does not establish a causal relationship.

Structural assessment using optical coherence tomography further demonstrated differences between the groups. The mean superior RNFL thickness was significantly lower among hypertensive patients (67.88 ± 12.74 µm) than among normotensive patients (75.40 ± 13.22 µm; $p = 0.010$). Similarly, inferior GCC thickness was significantly reduced in the hypertensive group (63.05 ± 11.78 versus 68.24 ± 12.04 µm; $p = 0.049$). Although average RNFL and average GCC thicknesses were also lower among hypertensive patients, these differences did not reach statistical significance.

These findings indicate greater thinning in selected structural parameters among patients with coexisting hypertension and POAG. Reduced RNFL thickness reflects loss of retinal ganglion cell axons, while GCC thinning indicates damage involving retinal ganglion cell layers. Such structural alterations are important indicators of glaucomatous neurodegeneration. The association between hypertension and greater structural damage may be related to chronic microvascular dysfunction, impaired autoregulation, or other systemic vascular factors.[9,10] However, the observed differences may also be influenced by age and associated systemic conditions.

Interestingly, the structural differences were not accompanied by statistically significant differences in visual field parameters.

The mean Humphrey visual field mean deviation was −9.22 ± 5.73 dB in hypertensive patients compared with −7.81 ± 5.46 dB in normotensive patients (p = 0.252). Pattern standard deviation and visual field index were also comparable between the groups. Although hypertensive patients showed numerically greater functional impairment, these differences were not statistically significant.

This discrepancy between structural and functional findings may reflect differences in the sensitivity of OCT and automated perimetry to glaucomatous damage. Structural abnormalities can sometimes be detected before corresponding functional changes become evident on conventional visual field testing. However, the cross-sectional nature of the present study prevents conclusions regarding the sequence or progression of structural and functional damage. Longitudinal assessment would be required to determine whether the observed OCT differences predict subsequent visual field deterioration.

The relationship between OPP and glaucoma has also been examined in a systematic review and meta-analysis by Kim et al., which highlighted the association between ocular perfusion parameters and open-angle glaucoma risk.[9] However, OPP is influenced by both blood pressure and IOP, and its interpretation requires consideration of these individual components. The present findings add to this evidence by demonstrating that higher calculated OPP may coexist with greater structural glaucomatous abnormalities in patients with essential hypertension.

Importantly, the mean duration of glaucoma and the number of antiglaucoma medications were comparable between the two groups. Mean glaucoma duration was 5.74 ± 3.55 years in hypertensive patients and 5.43 ± 3.22 years in normotensive patients (p = 0.676). Similarly, the number of antiglaucoma medications did not differ significantly. These findings suggest broadly comparable recorded glaucoma duration and treatment burden, although differences in long-term IOP control and treatment adherence cannot be excluded.

The clinical implications of this study are particularly relevant to glaucoma management in patients with systemic hypertension. Assessment of IOP alone may not adequately represent the vascular factors contributing to optic nerve damage. Blood pressure status, antihypertensive treatment, and structural optic nerve parameters should also be considered during clinical evaluation. In patients showing progressive glaucomatous damage despite apparently controlled IOP, further assessment of systemic blood pressure patterns may be useful.[10,11] Nevertheless, antihypertensive treatment should not be altered solely on the basis of a single calculated OPP measurement.

The present study has several limitations. First, the hospital-based design, convenience sampling, and relatively small sample size may limit the generalizability of the findings.

Second, the hypertensive group was significantly older and had a higher prevalence of diabetes mellitus, both of which may influence ocular vascular health and glaucoma severity. Third, blood pressure and IOP were assessed at a single examination, preventing evaluation of diurnal fluctuations and nocturnal hypotension. Fourth, calculated OPP does not directly measure optic nerve head blood flow or vascular autoregulatory function. Finally, the study design does not establish whether hypertension preceded or contributed to the observed structural damage. Larger prospective studies incorporating ambulatory blood pressure monitoring, serial OCT measurements, and visual field assessments are required to clarify these relationships.

5. Conclusion

The present study demonstrated a significant association between essential hypertension and ocular perfusion pressure (OPP) among patients with primary open-angle glaucoma (POAG). Hypertensive patients exhibited significantly higher OPP compared with normotensive patients, despite comparable intraocular pressure levels. However, increased OPP was not associated with reduced glaucomatous optic nerve damage. Patients with coexisting essential hypertension showed significantly greater optic disc cupping, superior retinal nerve fibre layer thinning, and inferior ganglion cell complex thinning, indicating greater structural glaucomatous damage. Although visual field parameters suggested relatively greater functional impairment in hypertensive patients, the differences were not statistically significant. These findings highlight the complex relationship between systemic hypertension, ocular perfusion, and glaucomatous optic neuropathy. They also suggest that elevated calculated OPP does not necessarily reflect adequate optic nerve head perfusion or protection against glaucomatous damage.

The study emphasizes the importance of considering systemic vascular factors alongside intraocular pressure in the comprehensive evaluation and management of POAG. Chronic hypertension-related vascular alterations and impaired ocular blood flow regulation may contribute to structural optic nerve damage despite apparently adequate ocular perfusion pressure. Regular monitoring of systemic blood pressure, intraocular pressure, optic nerve head morphology, and optical coherence tomography parameters may facilitate early identification of glaucomatous changes in hypertensive patients. However, the observed associations do not establish an independent causal effect of hypertension on glaucoma severity. Further large-scale prospective studies incorporating ambulatory blood pressure monitoring and longitudinal structural and functional assessments are warranted to clarify the influence of systemic hypertension and ocular perfusion pressure on glaucomatous progression and to guide individualized management strategies.

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