Original Article

# Prevalence of pre-perimetric primary open angle glaucoma in hypertensives of North India

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## Abstract

**Background:** Systemic hypertension is alleged to increase the risk of glaucoma. As clinically Primary Open angle Glaucoma (POAG) is diagnosed only after approximately 40% of ganglion cell loss has occurred, therefore this study was commenced with an aim to determine the prevalence of pre-perimetric glaucomatous damage and its association with systemic hypertension using optical coherence tomography (OCT). **Materials and Methods:** A total of 680 study participants were enrolled in this cross-sectional study. Among them 340 patients were of systemic hypertension (Group 1) and 340 patients without hypertension (Group 2). All patients underwent detailed history, ocular and systemic examination including slit lamp examination, fundus examination by +90 D lens, Humphrey field analyser for field charting and OCT for nerve fiber analysis. For glaucomatous nerve damage. **Results:** Group 1 and Group 2 had Male: Female ratio of 1:8 and 1:9, respectively ($P = 0.809$). Maximum participants 48.8% and 54.4% in Group 1 and Group 2, respectively, were in age group 50–59 years. Statistically significant difference was seen in the percentage of pre-perimetric glaucomatous patients between the two groups ($P < 0.001$). On OCT analysis between pre-perimetric glaucomatous eyes and healthy eyes significant difference in thickness was seen in temporal inner macula, inferior outer macula, temporal outer macula, superior outer macula and nasal outer macula. Significant difference in volume was seen for inferior temporal and nasal outer macula ($P < 0.001$). **Conclusion:** In hypertensives, glaucomatous optic nerve damage starts much earlier before the obvious clinical signs of POAG appear, as compared to normotensive individuals.

**Keywords:** Glaucoma, hypertensives, OCT, retinal nerve fiber layer

## Background

Glaucoma is defined as an optic neuropathy characterized by slow, progressive degeneration of retinal ganglion cells (RGCs) and their axons, resulting in a distinct appearance of the optic disk and a concomitant pattern of visual loss.[1] The World Health Organization (WHO) has identified glaucoma to be the first leading cause of blindness in the world, followed by cataracts.[2] Because of its asymptomatic nature up to a very advanced stage, it is often termed as a 'silent killer' as it leads to an irrecoverable visual loss.[3,4] A recent report estimated that there are more than 70 million people worldwide with glaucoma[5] and glaucoma per-se is accountable for 8% of all global blindness.[3] Among the various cause listed by the National Blindness Survey for bilateral blindness glaucoma stands at fourth with a prevalence of 3.2%.[4] Glaucoma is divided into primary and secondary glaucoma based on the cause. In primary glaucoma the eye does not have any pre-existing disease. Primary Glaucoma is again classified into primary open-angle glaucoma (POAG) and primary closed-angle glaucoma (PCAG). The most commonest form of glaucoma which accounts for 90% of all cases is the Open angle Glaucoma.[6] POAG is described as an optic neuropathy with 'a characteristic acquired atrophy of the optic nerve and

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loss of retinal ganglion cells and their axons' developing in the presence of open anterior chamber angles, and manifesting characteristic visual field loss abnormalities and no obvious causative ocular or systemic conditions.[7] POAG inheritance is multifactorial and polygenic, occurs commonly among the elderly, rarely seen earlier than 40 years of age.[8] Intraocular pressure, age, family history, race, and potential vascular disease are some of the known risk factors of glaucoma.[9] Among the vascular aspects of POAG, systemic hypertension which is a major health condition that affects above 25% of adult population across the globe and tends to contribute an increase in intraocular pressure via overproduction or impaired outflow of aqueous humor.[10] Systemic hypertension, however, affects other systems also and ocular system is one among them with the most prominent ocular outcomes being glaucoma. It was suggested by the on-going research studies that for both glaucoma as well as systemic hypertension altered epithelial sodium transport in the distal nephron and ciliated epithelium is responsible.[11] Hypertension increases the risk of progression and worsening of glaucoma and many postulates have been determined such as microvascular damage that directly caused by hypertension which subsequently compromises the blood flow to the optic nerve,[12,13] and besides impairment of autoregulation of the posterior ciliary circulation.[14] In addition to these mechanisms, the antihypertensive therapy can induce hypotensive episodes during night times, that could further damage the optic nerve.[12] Glaucomatous optic neuropathy is depicted as loss of retinal ganglion cells, which on histopathological view shows a decrease in the retinal nerve fiber layer (RNFL) thickness. Though, glaucoma patients suffer a loss of more than 40% of retinal ganglion cell axons before an evident circumpapillary visual field defect.[15] As systemic hypertension is positively correlated with Glaucoma, early identification of glaucoma becomes vital in order to enable proper monitoring and treatment against further disease progression and to curtail the risk of irreversible visual field loss. The present study is an attempt to screen glaucoma in patients with hypertensives at primary care level. Therefore, this study was undertaken to find out the prevalence of Pre-perimetric Primary Open Angle Glaucoma in patients with systemic hypertension by measurement of retinal nerve fibre layer thickness and macular thickness.

## Materials and Methods

This was a prospective observational-cross-sectional study at a tertiary care centre of North India. Total of 680 study participants, attending ophthalmology OPD primarily or referred, during October 2018 to Dec 2019, were enrolled for the study. The study was initiated after obtaining approval from institutional ethics committee. (IHEC/ERA/2018/0012) After getting Informed written consent from the participants, the study participants were screened on the basis of their blood pressure criteria as per JNC 8 guideline for classification of B.P in adults.[16] Hypertension as per the Joint National Committee (JNCC) was defined as a systolic blood pressure ≥140 mmHg and/or a diastolic blood pressure ≥90 mmHg. Enrolled patients were divided into two groups, Group 1 and Group 2 of 340 patients each according to the presence or absence of systemic hypertension, respectively. Patients with media opacity precluding good-quality OCT scans such as corneal opacity, cataracts, retinal diseases affecting the retinal nerve fibre layer thickness and the visual field; certain conditions preventing reliable applanation tonometry; on medications known to affect visual field sensitivity; with the findings of secondary glaucoma; pre-diagnosed Glaucoma; history of intraocular diseases were excluded from the study. If both the eyes of any participant met all criteria, the thinner eye cpRNFLT (circum-papillary nerve fibre layer thickness) was enrolled in this study.

### Data collection

A detailed and thorough ocular and medical history was elicited and all the participants underwent a comprehensive ophthalmological assessment that included best corrected visual acuity as well as slit lamp and funduscopic examination by + 90 D lens, gonioscopy, IOP measurement, systemic blood pressure measurement, visual field examination, and OCT examination. IOP was determined with Goldmann applanation tonometry under local anesthesia and the median value of three measurements was taken. Color stereoscopic optic disc photographs and red-free nerve fiber layer photographs were taken on the Zeiss Fundus camera FF 450 with Visupac System 451 (Carl Zeiss Ophthalmic Systems, Jena, GmBH, Germany). Diagnosis of pre-perimetric Glaucoma was made as follows open angle method on gonioscopy with grade 3 or 4 as per Shaffer classification. The other criteria were refractive error range between +3.00 and -8.00 diopters and best-corrected visual acuity more than 20/40. In addition, intraocular pressure of 21 mmHg or less in minimum three separate examinations, abnormal circum-papillary RNFL thickness in minimum one clockwise OCT scan sector between 6, 7, 8, 10, 11, and 12 o'clock (6, 5, 4, 2, 1 and 0 o'clock in left eye), confirmed in three minimum examinations and a normal visual field. Optic disc suspicious for glaucoma defined as having features suggestive of glaucomatous optic neuropathy such as cup-disc ratio >0.6, any diffuse or focal neuroretinal rim thinning, any disc hemorrhage, and/or any RNFL defects on the red-free photograph. Systemic blood pressure was measured by sphygmomanometer, according to the standard technique, in the brachial artery at the height of the heart with the participant in sitting position. The average of three consecutive measurements of systolic and diastolic B.P was used in the analysis.

### Visual field evaluation and OCT examination

Visual field was measured by static automated perimetry using the Swedish Interactive Threshold Algorithm (Standard 24–2) of a Humphrey Field Analyzer (Carl Zeiss Meditec Inc., Dublin, California). Visual field measurements with fixation losses of more than 20%, or with over 33% false positives or false negatives were excluded from the analysis. Optical coherence tomography (OCT; Humphrey systems Inc., Dublin, CA) is a noninvasive, noncontact method that allows cross-sectional,

*in vivo* imaging of intraretinal layers.[3] Anatomic layers within the retina can be imaged and quantitative assessment of RNFL thickness and macular thickness can be performed based on the different reflectivity properties of different layers. Spectral-domain (SD-) OCT, with its improved resolution, scan speed, and reproducibility compared with its predecessor, time-domain OCT, potentially leads to earlier and more accurate detection of glaucoma progression. Spectral-domain OCT has been used to evaluate the patterns of progressive RNFL defect and to estimate the number of retinal ganglion cells. Images with signal strength less than 6 were considered of poor quality and excluded from the data analysis. The circum-papillary RNFL thickness (cpRNFLT), the RNFLT in 4 quadrants, and 12-o'clock positions were used in the evaluation. RNFL defects were assessed clockwise in the right eyes and counter clockwise in the left eyes. In the 12 sector analysis at ONH, 7, 8, 10, 11, and 12 o'clock sectors in right eye is in correspondence to 5, 4, 2, 1, and 0 o'clock sectors in left eye, respectively. The OCT software automatically classified all RFNLT as within normal limits or abnormal (out of 95 percentile from age-matched healthy eye).

## Statistical analysis

The data were entered and analysed with SPSS IBM version 21.0. Means and proportions were calculated. An independent t-test was used to compare the differences in the macular thickness, macular volume, peripapillary RNFL thickness, and image quality scores between early glaucomatous eyes and normal eyes. Comparisons to find differences in image quality scores between macular and peripapillary RNFL measurements were also made. *P* value of <0.05 is considered to be significant.

## Results

In the present study, a total of 340 individuals known to have systemic hypertension were enrolled as cases (Group 1) and total of 340 subjects not having systemic hypertension as controls (Group 2). Amongst the hypertensives majority cases (85%) had systolic blood pressure within range of 140–159 mm Hg. The male and female ratio was 1:8 in Group 1 and 1:9 in Group 2. There was no statistically significant difference in the distribution of gender in the two groups. ($\chi 2 = 0.059$; *P* value = 0.809) [Table 1].

In present study, maximum participants 48.8% and 54.4% of Groups 1 and 2, respectively, were in age group 50–59 years. Pre-perimetric Glaucoma was observed in 66 (9.4%) participants of Group 1 and 19 (5.5%) participants of Group 2 and this difference was statistically significant (P < 0.001). With respect to age distribution of pre-perimetric Glaucoma in hypertensives and controls, majority were in age category of 50–59 years, about 39.3% in Group 1 and 42.1% in Group 2 [Table 2].

Risk of having pre-perimetric Glaucoma in hypertensives as compared to non hypertensives was OR (95% CI) = 3.47 (2.56–4.13).

**Table 1: Age distribution of study participants (*n*=680)**

| | Group 1 (*n*=340) | | Group 2 (*n*=340) | | Total (*n*=680) | |
|---|---|---|---|---|---|---|
| | *n* | % | *n* | % | *n* | % |
| Age (years) | | | | | | |
| 40-49 | 124 | 36.5 | 105 | 30.8 | 229 | 33.6 |
| 50-59 | 166 | 48.8 | 185 | 54.4 | 351 | 51.6 |
| 60-69 | 32 | 9.4 | 33 | 9.7 | 65 | 9.5 |
| >70 | 18 | 5.2 | 17 | 5 | 35 | 5.1 |

$\chi^2$=2.65; *P*=0.449

**Table 2: Pre-perimetric glaucoma among the study participants (*n*=680)**

| | Pre-perimetric glaucoma in Group 1 (*n*=66) | | Pre-perimetric glaucoma in Group 2 (*n*=19) | |
|---|---|---|---|---|
| | *n* | % | *n* | % |
| Age (years) | | | | |
| 40-49 | 19 | 28.7 | 6 | 31.5 |
| 50-59 | 26 | 39.3 | 8 | 42.1 |
| 60-69 | 12 | 18.1 | 3 | 15.7 |
| >70 | 9 | 13.6 | 2 | 10.5 |

Results of SD-OCT macular parameters (thickness and volume) in pre-perimetric glaucoma and healthy eyes are shown in Table 3. Significant differences in thickness were found for temporal inner macula (mm), inferior outer macula (mm), temporal outer macula (mm), superior outer macula (mm), nasal outer macula (mm), while there was statistically significant differences in volume for Inferior outer macula, temporal outer macula, and nasal outer macula ($mm^3$) (p value (<0.001)).

On comparing the results of SD-OCT Peripapillary Retinal Nerve Fibre Layer (RNFL) Thickness parameters in pre-perimetric and healthy eyes we found statistically significant differences at the Inferior quadrant (mm), and at 6 o'clock (mm), 7o'clock (mm), 8 o'clock (mm), and 9 o'clock segments (mm) (p < 0.001) [Table 4].

## Discussion

Hypertension (HTN) is one of the commonest systemic disease and is a recognized risk factor for cardiovascular disease while glaucoma remains to be one of the commonest causes of blindness. In the pathogenesis of primary open-angle glaucoma (POAG), it is generally agreed that, in addition to ocular hypertension (OH), other risk factors may also play an important and, in certain cases, even a predominant role.[17] Particularly worthy of note are risk factors of a vascular nature like systemic hypertension, atherosclerosis, vasospasm, and other vascular diseases.[18] The pathways through which HTN predisposes to POAG is a complex one and still remains to be fully understood. High blood pressure may increase intraocular pressure by increased production of aqueous humor (through elevated ciliary blood flow and capillary pressure), and decreased aqueous humour drainage as a result of increased episcleral venous pressure.[19] Primary open angle glaucoma is an important health concern across the world as it is usually

**Table 3: Comparison of SD-OCT macular thickness and volume in pre-perimetric and healthy eyes**

| Parameters | Pre-Perimetric Glaucoma (mean±SD) | Healthy eyes (mean±SD) | *t*-test | *P* |
|---|---|---|---|---|
| Fovea thickness (mm) | 225.3±17.4 | 229.6±18.2 | 1.329 | 0.186 |
| Inferior inner macula (mm) | 270.8±48.2 | 295.8±14.1 | 3.153 | 0.002 |
| Temporal inner macula (mm) | 178.9±9.1 | 191.8±9.3 | 7.663 | <0.001 |
| Superior inner macula (mm) | 294.0±12.0 | 293.3±11.7 | 0.319 | 0.750 |
| Nasal inner macula (mm) | 296.8±15.8 | 303.2±13.4 | 2.272 | 0.024 |
| Inferior outer macula (mm) | 224.3±17.4 | 240.1±13.2 | 5.181 | <0.001 |
| Temporal outer macula (mm) | 230.0±11.3 | 246.5±10.3 | 8.084 | <0.001 |
| Superior outer macula (mm) | 239.6±12.1 | 257.0±11.1 | 7.951 | <0.001 |
| Nasal outer macula (mm) | 258.9±16.4 | 270.3±13.2 | 3.931 | <0.001 |
| Fovea volume ($mm^3$) | 0.16±0.1 | 0.15±0.1 | 0.543 | 0.588 |
| Inferior inner macula ($mm^3$) | 0.41±0.3 | 0.43±0.2 | 0.387 | 0.700 |
| Temporal inner macula ($mm^3$) | 0.42±0.2 | 0.49±0.1 | 2.081 | 0.039 |
| Superior inner macula ($mm^3$) | 0.46±0.2 | 0.43±0.2 | 0.815 | 0.416 |
| Nasal inner macula ($mm^3$) | 0.48±0.2 | 0.43±0.2 | 1.358 | 0.176 |
| Inferior outer macula ($mm^3$) | 1.19±0.1 | 1.29±0.1 | 5.432 | <0.001 |
| Temporal outer macula ($mm^3$) | 1.21±0.1 | 1.31±0.1 | 5.432 | <0.001 |
| Superior outer macula ($mm^3$) | 1.27±0.1 | 1.33±0.1 | 3.259 | 0.001 |
| Nasal outer macula ($mm^3$) | 1.41±0.1 | 1.41±0.1 | 1.000 | <0.001 |
| Total volume ($mm^3$) | 7.13±0.2 | 7.2±0.3 | 1.684 | 0.094 |

**Table 4: Comparison of SD-OCT Peripapillary Retinal Nerve Fiber Layer (RNFL) Thickness in pre-perimetric and healthy eyes**

| Parameters | Pre-Perimetric Glaucoma (mean±SD) | Healthy-eyes (mean±SD) | '*t*' | *P* |
|---|---|---|---|---|
| Superior quadrant (mm) | 115.7±15.4 | 124.9±11.3 | 3.424 | 0.001 |
| Nasal quadrant (mm) | 91.0±14.2 | 98.0±12.5 | 2.747 | 0.007 |
| Inferior quadrant (mm) | 110.1±19.2 | 137.8±15.4 | 8.164 | <0.001 |
| Temporal quadrant (mm) | 81.3±13.3 | 89.1±12.9 | 3.207 | 0.002 |
| 12 o'clock (mm) | 111.2±21.6 | 124.1±18.3 | 3.350 | 0.001 |
| 1 o'clock (mm) | 115.1±17.2 | 123.8±18.6 | 2.698 | 0.008 |
| 2 o'clock (mm) | 98.4±15.7 | 105.3±17.4 | 2.329 | 0.021 |
| 3 o'clock (mm) | 82.9±13.4 | 87.0±12.7 | 1.681 | 0.095 |
| 4 o'clock (mm) | 87.9±18.0 | 91.9±13.3 | 1.272 | 0.205 |
| 5 o'clock (mm) | 113.4±16.5 | 117.0±17.2 | 1.174 | 0.242 |
| 6 o'clock (mm) | 1179±26.8 | 143.6±23.9 | 5.331 | <0.001 |
| 7 o'clock (mm) | 102.6±36.2 | 146.0±20.4 | 7.065 | <0.001 |
| 8 o'clock (mm) | 79.2±21.1 | 91.1±14.4 | 3.261 | 0.001 |
| 9 o'clock (mm) | 70.8±11.3 | 79.9±12.2 | 4.297 | <0.001 |
| 10 o'clock (mm) | 91.7±18.2 | 98.6±17.2 | 2.084 | 0.039 |
| 11 o'clock (mm) | 121.9±27.1 | 136.7±18.7 | 3.154 | 0.002 |
| Average NFL (mm) | 105.5±11.4 | 117.4±9.2 | 3.424 | 0.001 |

silent and progressive nature. This is also leading cause of blindness. Systemic hypertension alone seems to be one of the key player in determining the prevalence and severity of POAG. As people with systemic hypertension are at high risk of developing Glaucoma, it is essential that this blinding disease be diagnosed at its preliminary stage so that timely intervention can be done. Since RNFL abnormalities are known to precede visual functional damage, it is necessary to measure RNFL thickness in an attempt to identify pre-perimetric glaucoma. This was assessed by spectral domain OCT as it detects Glaucomatous damage earlier than conventional automated perimetry. The participants included in this observational cross-sectional study design were labelled as cases (Group 1) and controls (Group 2) based on their systolic blood pressure level in accordance with the definition and guidelines given by JNC-8.[16] In the present study, maximum number of participants were in age group 50–59 years and did not show any significant difference between the two groups similar to the findings of He *et al*.[20] who also found maximum number of their patients in 50–59 years of age. This is because both hypertension and POAG occurs, in elderly, rarely seen earlier than 40 years of age. In the Barbodos Eye Study,[21] the prevalence of POAG was especially high at older age and in men. Among participants ≥50 years old, one in 11 had POAG, and prevalence increased to one in six at age 70 years or older. In our study, with respect to gender proportion, males 65.4% out-numbered females 34.5% but

with no significant difference between the groups. ($\chi 2 = 0.059$; *P* value = 0.809)

Our study using SD-OCT showed comparable performance of macular parameters for the diagnosis of early glaucoma to RNFL parameters, suggesting an improved diagnostic ability of macular parameters by SD-OCT. These individuals were labelled as having pre-perimetric Glaucoma. On comparing the results of SD-OCT Peripapillary Retinal Nerve Fibre Layer (RNFL) Thickness parameters in pre-perimetric and healthy eyes significant differences were seen at the Inferior quadrant (mm), and at 6 o'clock (mm), 7 o'clock (mm), 8 o'clock (mm), and 9 o'clock segments (mm) ($p < 0.001$) while the results of SD-OCT macular parameters (thickness and volume) in pre-perimetric glaucoma and healthy eyes showed significant differences in thickness for temporal inner macula (mm), Inferior outer macula (mm), temporal outer macula (mm), superior outer macula (mm), and nasal outer macula (mm), and statistically significant differences in volume for inferior outer macula, Temporal outer macula and nasal outer macula ($mm^3$) ($p < 0.001$)). An explanation for this observation may be seen in the results of a post-mortem histological study which demonstrated that up to 40% of nerve fibres may be lost from the optic disc before the development of visual field defects in patients with ocular hypertension.[15]

Mok KH *et al.*[22] demonstrated that patients with ocular hypertension (OHT) have thinner RNFL thickness measured by OCT compared to normal eyes, suggesting that a subgroup of OHT patients may in fact have early glaucomatous structural damage not detected by standard tests.

Kanamori *et al.*[23] have reported a strong correlation between RNFL thickness and visual field disturbance while Greenfield *et al.*[24] showed significant correlation between macular thickness and visual field disturbance in moderately advanced glaucoma. There are studies which proves that the risk of conversion of defective visual fields in glaucoma suspect (GS) eyes increased with the increase in RNFL damage. Therefore, it is possible that the thinner RNFL in GS is an early form of glaucoma threat which precedes detectable optic nerve or visual field defects.

In our study we found a positive correlation between hypertension and Glaucomatous RNFL changes. Similar to the results of present study, Kaman *et al.*[25] in their study also showed a nominal increase in glaucoma prevalence among hypertensives (3.75%) as compared to normal population (2.56%) and showed that pre-perimetric glaucoma conditions like IOP rise was significantly correlated with blood pressure levels. All these studies prove that pre-perimetric glaucoma is much more pronounced in hypertensives as compared to normotensives and the observations of present study are akin with all these observations. This suggest that, in hypertensives, glaucomatous optic nerve damage starts much earlier before the obvious clinical signs of POAG appear, as compared to in normotensives individuals. There are several possible pathophysiological mechanisms involved in hypertensive damage to the optic nerve head. Systemic hypertension could initially increase blood flow to the anterior optic nerve, but with sustained hypertension, microvascular damage and impaired blood flow could ensue, with resultant ischemic damage to anterior optic nerve structures.[12,13] Hypertension could also interfere with the auto-regulation of posterior ciliary perfusion to the optic nerve, already found to be impaired in OAG cases.[14] Anti- hypertensive treatment could also induce hypotensive episodes, particularly at night which could reduce optic nerve head blood flow below a critical level, resulting in further reduction of mean perfusion pressure against OH.[12]

As the patients with hypertension screened for glaucoma help in early diagnosis of the disease in pre-pathogenesis phase and the present study was able to screen and find glaucoma among the study population.

## Conclusion

The patients of systemic hypertension are at high risk of glaucoma and hypertension is contributing to both physiological as well as pathological damage in the eye. POAG is associated with pre-existing HTN, therefore, it is recommended that ocular examination should be taken as a key ingredient of glaucoma control strategy among hypertensives. As controlling the intraocular pressure (IOP) at an early stage in glaucoma has been shown to slow down or stop progression of the disease, therefore, it is crucial to screen all the patients with systematic hypertension for primary open angle glaucoma by regular monitoring of intraocular pressure.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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### Conflicts of interest

There are no conflicts of interest.

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