"COMPARISION OF CENTRAL CORNEAL THICKNESS IN PRIMARY OPEN ANGLE GLAUCOMA, PSEUDO EXFOLIATIVE GLAUCOMA, OCULAR HYPERTENSION AND NORMAL POPULATION."

By

DR. POOJITHA M, M.B.B.S

Dissertation submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, CENTRE, TAMAKA, KOLAR

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY

IN OPHTHALMOLOGY

Under the guidance of

DR. RASHMI G

M.B.B.S., M.S.

DEPARTMENT OF OPHTHALMOLOGY

SRI DEVARAJ URS MEDICAL COLLEGE

TAMAKA, KOLAR.

JUNE/JULY 2023

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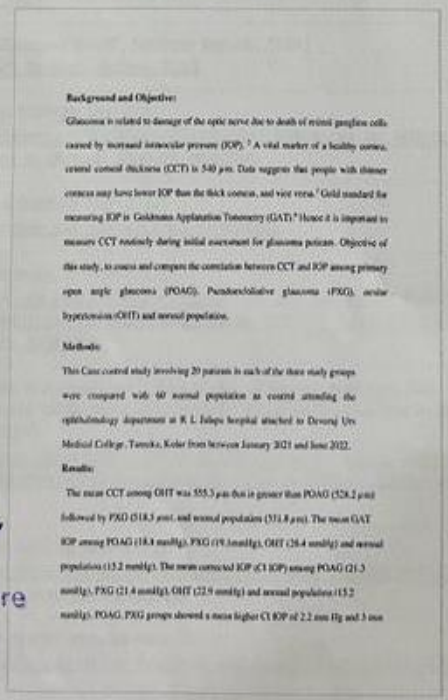

Background and Objective:

Glaucoma is related to damage of the optic nerve due to death of retinal ganglion cells caused by increased intraocular pressure (IOP). [1] A vital marker of a healthy cornea, central corneal thickness (CCT) is 540 μm. Data suggests that people with thinner corneas may have lower IOP than the thick corneas, and vice versa. [2] Gold standard for measuring IOP is Goldmann Applanation Tonometry (GAT). [3] Hence it is important to measure CCT routinely during initial assessment for glaucoma patients. Objective of this study, to assess and compare the correlation between CCT and IOP among primary open angle glaucoma (POAG), Pseudoexfoliative glaucoma (PXG), ocular hypertension (OHT) and normal population.

Methods:

This Case control study involving 20 patients in each of the three study groups were compared with 60 normal population as control attending the ophthalmology department at R. L. Jalappa hospital attached to Devaraj Urs Medical College, Tamaka, Kolar from between January 2021 and June 2022.

Results:

The mean CCT among OHT was 555.3 μm thus is greater than POAG (526.2 μm) followed by PXG (518.3 μm), and normal population (571.8 μm). The mean GAT IOP among POAG (18.8 mmHg), PXG (19.1mmHg), OHT (26.4 mmHg) and normal population (15.2 mmHg). The mean corrected IOP (CI IOP) among POAG (21.3 mmHg), PXG (21.4 mmHg), OHT (22.9 mmHg) and normal population (15.2 mmHg). POAG, PXG groups showed a mean higher CI IOP of 2.2 mm Hg and 3 mm

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LIST OF ABBREVATIONS

IOP	Intraocular pressure
RGC	Retinal ganglion cell
CCT	Central corneal thickness
GAT	Goldmann tonometry
POAG	Primary open angle glaucoma
PXG	Pseudoexfoliative glaucoma
OHT	Ocular hypertension
ACA	Anterior chamber angle
TM	Trabecular meshwork
SC	Schlemm's canal
AS-OCT	Anterior segment optical coherence tomog-raphy
AOD	Angle opening distance
RNFL	Retinal nerve fiber layer
ACV	Anterior chamber volume
ACW	Anterior chamber width
VHG	Van Herick's grading
HTN	Hypertension
SWAP	short-wavelength automated perimetry
FDT	Frequency doubling technology
MDP	Motion displacement perimetry

SS	Scleral spur
SL	Schwalbe's line
CB	Ciliary body band
CDR	Cup-to-disk ratio
NRR	Neuroretinal rim
HFA	Humphrey field analyser
OHTS	Ocular hypertension treatment study
UP	Ultrasonic pachymetry
OPT	Optical pachymetry
OCT	Optical coherence tomography
PPA	Peripapillary alterations
ONH	Optic nerve head
GCIPL	Ganglion cell inner plexiform layer
PEX	Pseudoexfoliation syndrome
SLT	Selective laser trabeculoplasty
DM	Diabetes mellitus
TGF-β1	Transforming growth factor-beta 1
PE	Pseudoexfoliative eyes
OPA	Ocular pulse amplitudes
DCT	Pascal dynamic contour tonometry
CIOP	Corrected IOP
NTG	Normal tension glaucoma
CC	Corneal curvature

ABSTRACT

Background and Objective:

Glaucoma is related to damage of the optic nerve due to death of retinal ganglion cells caused by increased intraocular pressure (IOP). [2] A vital marker of a healthy cornea, central corneal thickness (CCT) is 540 µm. Data suggests that people with thinner corneas may have lower IOP than the thick corneas, and vice versa.[7] Gold standard for measuring IOP is Goldmann Applanation Tonometry (GAT).[9] Hence it is important to measure CCT routinely during initial assessment for glaucoma patients. Objective of this study, to assess and compare the correlation between CCT and IOP among primary open angle glaucoma (POAG), Pseudoexfoliative glaucoma (PXG), ocular hypertension (OHT) and normal population.

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Conclusion and interpretation

Overall, positive correlation was noted between IOP and CCT among POAG, PXG and the OHT patients. CCT was highest among OHT, while POAG and PXG had CCT thinner than normal population. Within the morbid groups, CCT was lower among PXG. Also, among POAG and PXG who had thinner CCT had underestimation of GAT IOP but the CI IOP was little higher, while in OHT group who had thicker CCT showed overestimation of GAT IOP but the CI IOP was little lower. Further, multi-centric, prospective studies must be conducted between the groups of patients to establish the impact of CCT on these types of glaucoma.

Keywords: central corneal thickness, Intra ocular pressure, Primary open angle glaucoma, Pseudoexfoliative glaucoma, Ocular hypertension.

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INTRODUCTION

1. INTRODUCTION

A set of progressive optic neuropathies called glaucoma are defined as changes in optic nerve head (ONH) due to degradation of retinal nerve fibre layers (RNFL) and retinal ganglion cells (RGC).[1] Glaucoma is related to damage to the optic nerve caused by raised IOP, that can lead to death of RGC.[2] The primary cause of permanent blindness globally is glaucoma, linked to a lower quality of life.[3]

A vital marker of a healthy cornea, central corneal thickness (CCT) aids in diagnosis of corneal disorders. The corneal stroma, which is thought to be 450µ in diameter in the middle, makes up the majority of the average conventional CCT, which is roughly 540 µ. This stroma provides crucial structural integrity and is crucial for maintaining corneal transparency.[4] As eyes with corneal thickness of </= 555µm showed larger risk in developing glaucoma than with a corneal thickness of >/= 588 µm, central corneal thickness of ocular hypertension patients is also thought to be a potent predictor of glaucoma development.[5]

Any new patient who is suspected of developing glaucoma must have a central cornea thickness (CCT) examination.[6] The CCT which affects IOP readings, might have an impact on diagnosing, screening, and treatment of glaucoma. Data suggests that people with thinner corneas may have lower IOP than those with thick corneas, and vice versa.[7]

As it helps in interpretation of IOP data, risk stratification, setting of a target IOP, studies in the past have advocated that measuring of CCT routinely has to be included in assessment of all glaucoma patients initially. However, rather than relying solely on

the first CCT, following clinical therapy should take into account glaucoma investigation progress.[8]

There are several instruments for measuring IOP, and the majority of them are susceptible to changes in CCT. GAT which is a gold standard for measuring IOP, was created using a 500 μm thick cornea.[9] Though, it was shown that during cannulation trials CCT of about 520 μm was most reliable with GAT. The relationship between IOP, CCT in GAT is favourable.[10–12]

Additionally, CCT has been found to be a significant indicator of the likelihood that ocular hypertension-related primary open angle glaucoma (POAG) may develop.[5]

Thus, the role of CCT in diagnostic as well as prognostic attributes of glaucoma has been shown in previous studies. However, there is lacunae of studies on assessing the CCT between various types of glaucoma along with relation between CCT, IOP in our current study settings. Hence, we conducted the following study.

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AIMS & OBJECTIVES

2. AIMS & OBJECTIVES

AIM

- To assess and compare correlation between Central corneal thickness and IOP among POAG, PXG, OHT and normal population.

OBJECTIVES

- To assess central corneal thickness in POAG, PXG, OHT and normal eye.
- To compare and correlate between CCT and IOP in POAG, PXG, OHT and normal eye.

REVIEW OF LITERATURE

3. REVIEW OF LITERATURE

Glaucoma:

Glaucoma is one of the primary causes of lifelong blindness that occurs among older persons.[13] Adult glaucoma falls into two groups: primary open and closed angle glaucoma, and secondary open and closed angle glaucoma. A recognizable ONH changes and gradual vision loss are the effects seen in optic neuropathy, which is due to acquired loss of RGC and axons of optic nerve.[14]

History of glaucoma

Glaukos was a term used by the ancient Greeks to designate healthy, glaucous irides rather than sick eyes (light blue, gray, or green).[15] Glaucoma has a long history and has a hazy reputation as a disease. In 400 BC, the Ancient Greeks were the first to describe the condition that we now refer to as "glaucoma." The term "Glaykoseis" was initially used by Hippocrates to describe a sickness that mostly affects the elderly and causes blindness. The basic steps in comprehending the eye condition "glaucoma" will be discussed in this session. Since its invention during the time of Hippocrates, the concept of glaucoma has undergone a significant modification. The word "glaucoma" was used in Hippocratic texts to describe blindness that seemed to grow with increasing age and was accompanied by a fixed look of the pupil. Richard Banister was the person to identify precise connection between increased IOP and glaucoma in 1622.[16]

The discovery of physostigmine in the calabar bean in 1862 marks the beginning of glaucoma pharmacology. Some 40 years later, the ability of epinephrine to reduce intraocular pressure was discovered. With the emergence of beta blockers, prostaglandin analogues, and carbonic

anhydrase inhibitors throughout the 20th century, drug research and development significantly increased.[17]

- ***Prevalence***

Around the world, glaucoma-related visual neuropathy affects 60 million people. The open-angle form is more prevalent in the African population. Those of African ancestry are up to 15 times more likely than other ethnic groups to go blind from open-angle glaucoma.[14] Closed angle glaucoma is most common in the Inuit population and afflict women more frequently than males and those with Asian ancestry at a greater rate, despite the fact that these people tend to have shallower anterior chambers.[18] Japanese people are most likely to have normal-tension glaucoma.

Age is significant risk factor for the loss of RGC across all kinds. Other risk factors of glaucoma are primary relative (mother, father, brother, sister, or child) with the disease, as well as those who have high blood sugars, hypertension (HTN), cardiac diseases, trauma to eye, thinner corneas, previous retinal detachment, eye tumours or inflammation, long-term steroid usage. In India, there are thought to be 1.12 crore people with glaucoma who are 40 years of age or older. 0.65 crore people are impacted with POAG. There are 0.25 crore population with POAG. There may be 2.76 crore people with primary angle-closure illness in some form. Hence the majority of those with the disease go undetected, detecting and treating those with disease presents significant challenges. [19]

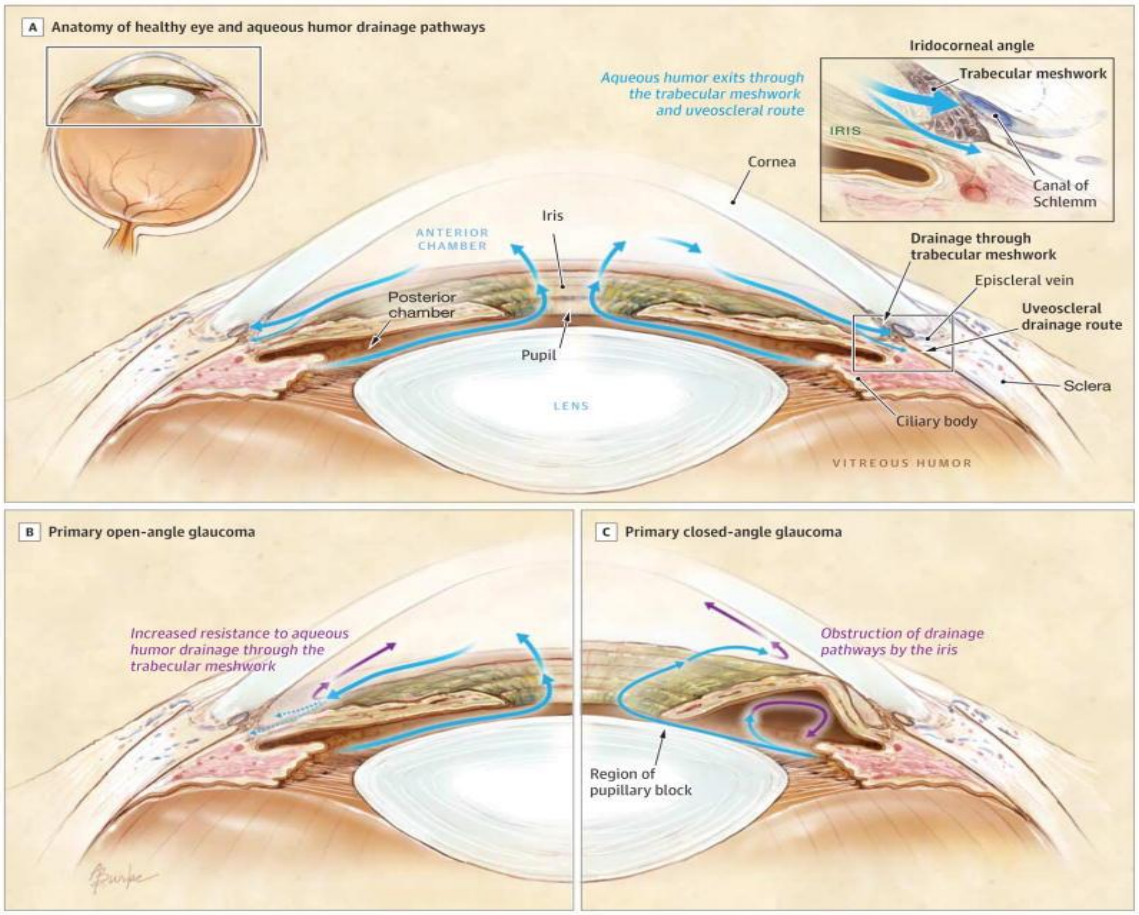
- ***Pathophysiology***

Anatomical considerations

The balance between aqueous humor secretion in the posterior chamber from ciliary body, drainage of aqueous from anterior chamber angle, either through the trabecular

outflow or the uveoscleral outflow controls IOP (normal range 10 - 21 mm Hg). An increased production of aqueous humor outflow or reduced outflow facility causes increased IOP.[14]

Figure 1: Glaucomatous and healthy eye's aqueous humour drainage pathways



Although the pathogenesis of glaucoma is unknown, RGC depletion is related with intraocular pressure. In open angle glaucoma patients the aqueous outflow via the trabecular meshwork is more restricted. In contrary, angle-closure glaucoma, the iris often blocks access to the drainage routes.[2]

"As a result of IOP, posterior components of the eye, particularly the lamina cribrosa and adjacent tissues undergo mechanical stress.[20] RGC axons exit the eye via a perforation in the sclera near the lamina. Stress and strain caused by intraocular pressure can cause the lamina cribrosa to compress, deform, and remodel. As a result, mechanical axonal injury and axonal transport may be interrupted, preventing the retrograde release of vital trophic substances from their brainstem origin to retinal ganglion cells.[21,22] Studies with experimentally induced Studies with experimentally induced OHT in cats and monkeys have shown that both or-

thograde and retrograde axonal transmission are blocked at the lamina cribrosa level.[23] Early in pathogenesis of glaucoma in experimental systems, disrupted axonal transport occurs, resulting in vesicle collections and disruption of microtubules and neurofilaments in prelaminar and postlaminar areas. Postmortem human eyes with glaucoma also showed similar ultrastructural alterations in the optic nerve fibers.[20] The IOP induced metabolic stress demands high levels of energy that can be challenging to satisfy since retinal ganglion cells and astrocytes may also have mitochondrial malfunction.[24]"

- **Types of Glaucoma**

Glaucoma is classified based on the aetiology as follows:[25]

Primary glaucoma's[25]

- Open-angle glaucoma
- Normotensive glaucoma
- Closed angle glaucoma
- Congenital glaucoma

Secondary glaucoma's[25]

- Pseudo exfoliation glaucoma
- Neovascular glaucoma
- Uveitic glaucoma
- Pigmentary glaucoma

- ***Diagnosis***

  - ✓ **Anterior segment examination**

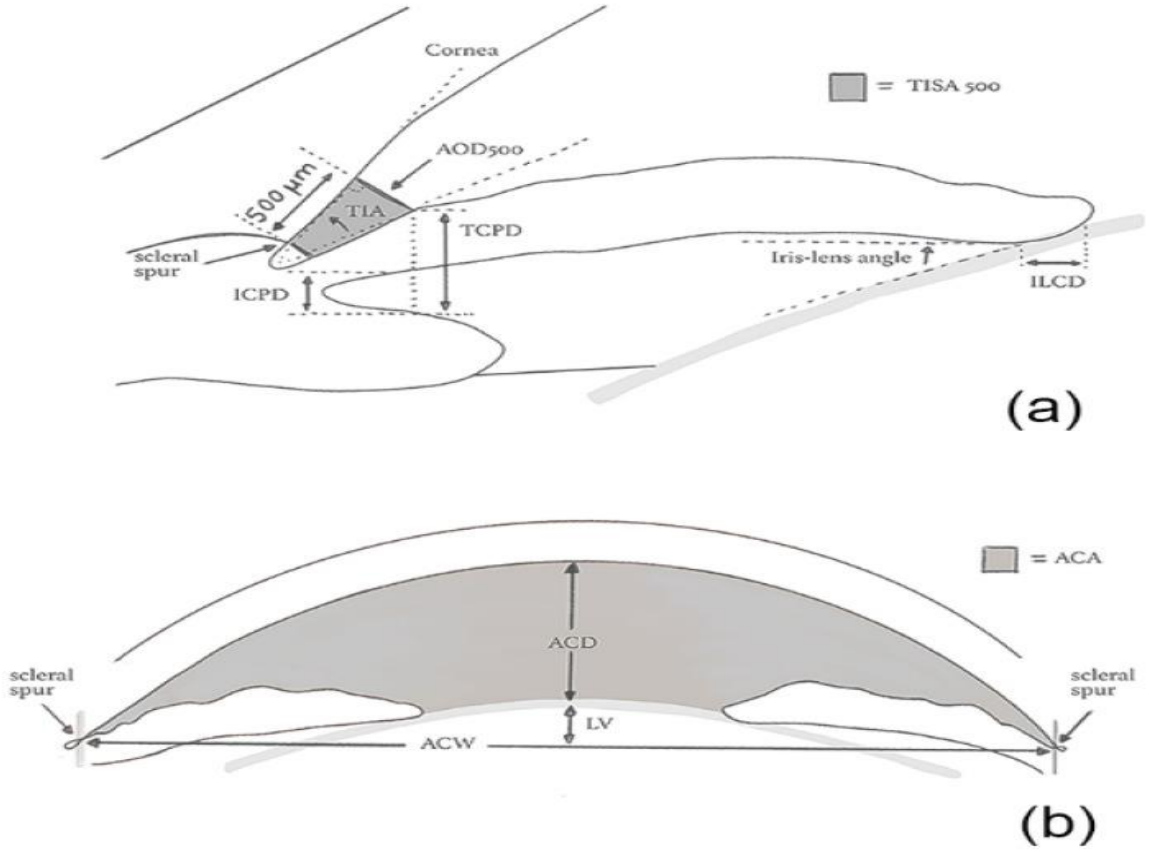
A vital component of examination is assessment of the angle of anterior chamber (ACA).[26] A thorough evaluation of the ACA should always be carried out on patients who have glaucoma or glaucoma suspicions. This will provide direct visibility of the primary structures responsible for the drainage of the aqueous humour, which will directly alter intraocular pres-

sure.[27,28]A number of findings, including abnormal iris insertion, abnormal blood vessels in the angle, blood in the Schlemm's canal (SC), abnormal trabecular meshwork (TM) pigmentation, the presence of synechiae, angle recession, dysgenesis of anterior segment and other abnormalities may be related to impaired aqueous humor outflow.[28]

The objective assessment is provided by Anterior segment optical coherence tomography (AS-OCT), few technologies are developed recently for making of ACA evaluation more practical, quantitative.[29]

Few quantitative parameters that have been anticipated to describe the anatomical features of the anterior segment are angle opening distance (AOD), anterior chamber width (ACW), Van Herick's grading (VHG) and anterior chamber volume (ACV).[29]

Figure 2: Parameters of Anterior segment[29]



- **Gonioscopy**

"Gonioscopy is essential for ACA evaluation of glaucomatous eyes because it serves as the clinical reference standard. The iris root, ciliary body band (CB), scleral spur (SS), the non-pigmented and pigmented TM and the Schwalbe's line (SL) are the angle structures visible with gonioscopy from posterior to anterior.[30]"

Different grading systems of angles[30]

To offer an uniform explanation of ACA anatomy , gonioscopy grading systems were developed. Today, a variety of grading systems are in use.[30]

The visualization of the angle structures is the foundation of the Scheie grading system. Following is the detailed explanation of the Scheie system:

Table 1: Scheie classification system[30]

Grade	Visibility	Interpretation
Wide	Wide	Open, all structures visible
I	Slightly Narrowed	Ciliary body visible, but recess obscured by the last roll of the iris
II	Apex not visible	Ciliary body not visible
III	Posterior half of trabecular meshwork not visible	Ciliary body, scleral spur, and posterior half of the trabecular meshwork not visible
IV	None of the angle structures visible	Cliary body, scleral spur, and trabecular meshwok not visible

Angularity is the basis of the Shaffer system. It employs an number system, which is the opposite to Scheie system.

Table 2: Shaffer's grading system[30]

Angular Grade	Width (in degrees)	Grade	Clinical Interpretation
Wide Open Angle	45-35	4	Angle closure impossible in both Grades 3 and 4
	35-20	3	
Narrow Angle	20	2	Angle closure possible
Narrow Angle, extreme	10 or less	1	Angle closure probable, eventually
Narrow Angle, slit	Critically narrowed angle, quite possibly against the trabecular meshwork beyond Schwalbe's line	-	-
Narrow angle, partial or complete closure	0	0	Angle closed in part or all of circumference

Spaeth[30]

The Spaeth method is far more intricate and specifies each specific anatomical aspect in great detail. The posterior trabecular meshwork's pigmentation, iris insertion, angularity, configuration are all described using this approach. Iris Insertion is usually designated by letter A-E, indicating the depth of the insertion. Iris angularity which is the angle between 1st line that lies parallel to trabecular meshwork while the 2nd line is a tangent to the surface of the anterior iris, in general is in the range from 10 degree to 40 degree. Iris configuration denotes the iris' shape between its course from the margin of papilla to the point where it is inserted. S Steep or convex configuration, B anteriorly Bowing, P Plateau, R or F Regular or flat, Q Queer – deeply concave Pigmentations takes a grading between 0 and 4, according to the intensity of pigmentation present in the posterior TM. Additionally, dynamic or compressive

gonioscopy is taken into consideration by the Spaeth system.

Other screening procedures that can be done to evaluate the patients with glaucoma are measurement of IOP, fundoscopic examination, visual field tests, OCT.

- **Intra ocular pressure (IOP) and its measurement**

"IOP measures the amount of force the aqueous humour exerts on the interior surface area of the anterior eye because the pressure is the measure of force per area. IOP = (F/C) + P, where F denotes aqueous flow rate, C denotes aqueous outflow, and P is the episcleral venous pressure, can potentially be used to calculate IOP." The IOP will unavoidably change if any of these factors change or fluctuate. IOP is an intricate balance between aqueous humour generation and drainage.[31] Abrupt IOP changes can result in mechanical stress and ischemic effect on RNFL. Conversely, rapid changes in the IOP can result in the formation of micro emboli from dissolved gases in the microvasculature that will increase in size and can lead to blockage in the microvessels and leadng to ischemia.[32]

Since the 19th century, a variety of devices known as tonometers have been suggested to measure IOP.[33,34] These devices may be divided into two primary groups according to their functioning principle: indentation Tonometers and applanation Tonometers.

GAT is a commonly used method for measuring IOP since it estimates the pressure within the anterior eye based on how resistant a small portion of the cornea is flattened.

The usual range for pressures is 11 to 21 mmHg, and a diurnal variation in IOP is anticipated, with greater pressures commonly reported in the morning.[31]

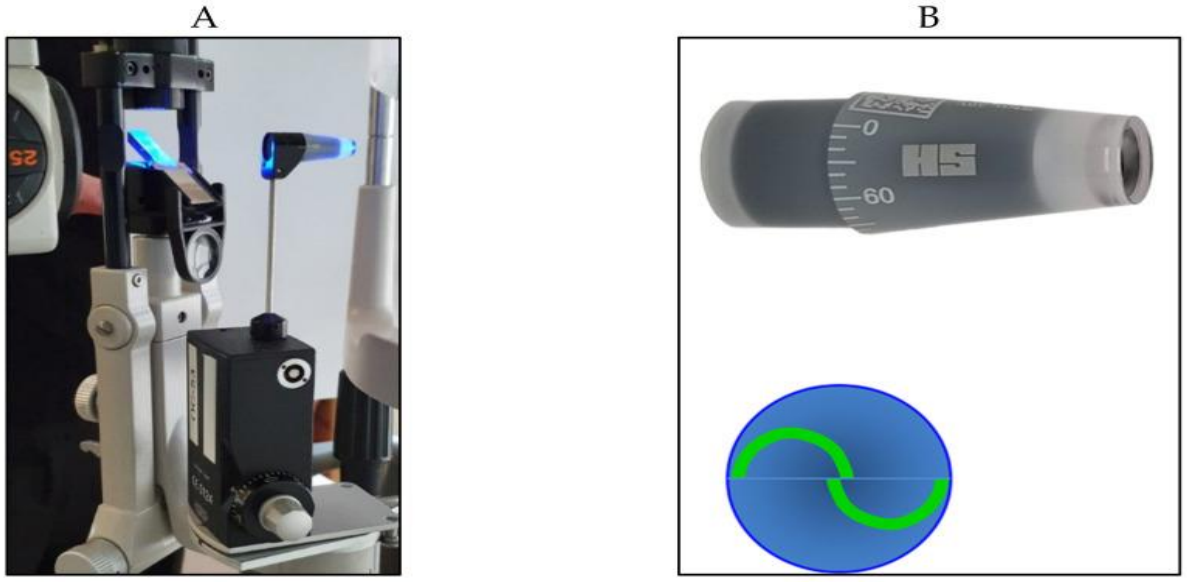
Although GAT is still primary method for determining IOP, rebound tonometry utilising portable tonometers has become a useful method for determining IOP in an emergency situation.[31]

✓ ***Goldmann Applanation Tonometry (GAT)***

Hans Goldmann initially developed the Goldmann applanation tonometer (GAT) in 1948, and still regarded as the gold standard today. Tonometer is necessarily mounted onto slit lamp.[9]

A truncated cone with blue light-illumination that is of 7.35 mm^2 surface area and 3.06 mm in diameter is touched against the centre of anaesthetized cornea after staining with 1% sodium fluorescein dye. The doubling prism that is inserted in the cone divides the circular meniscus on the flattened cornea into two arcs, which must be positioned to provide an accurate and uniform applanation.[35]

Figure 3: Goldmann Applanation Tonometer positioned to the slit lamp[35]



GAT depends on "Imbert-Fick principle, which assumes that pressure inside a sphere is equal to the force required to flatten its surface divided by the area flattened."[36] The corneal wall's innate stiffness or biomechanical characteristics are not taken into consideration by this approach. It only functions based on the tear meniscus's capillary attraction force that resists corneal stiffness at 3.06 mm in diameter.

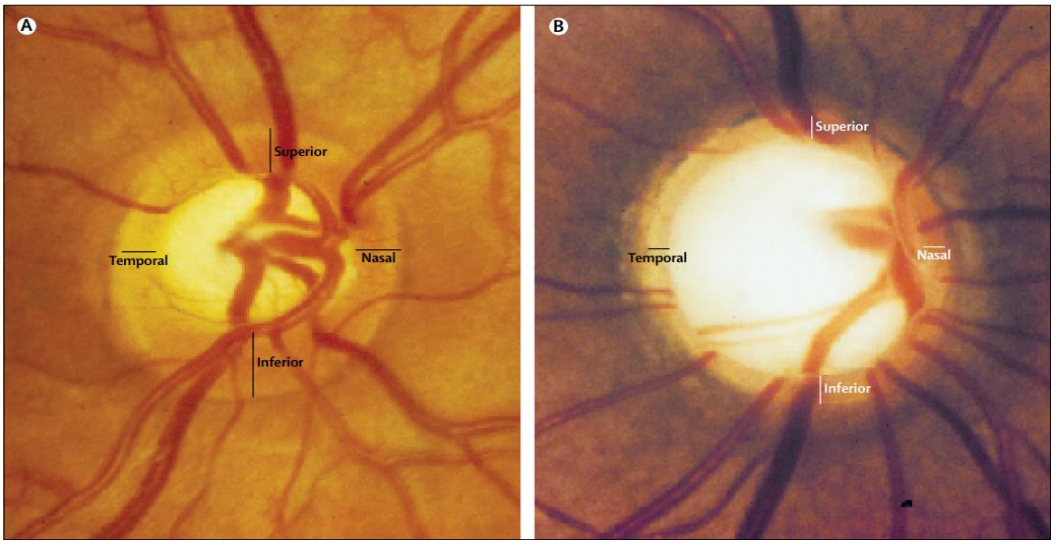
However, as recently noted by Gazzard et al., there are a number of disadvantages that should be taken into consideration.[37] GAT is impacted by corneal characteristics, such as curvature

of cornea, CCT when it deviates significantly from the normal (540 microns)[38,39] Additionally, GAT readings might vary as they are subjective. The visual acuity testing, pachymetry for measuring corneal thickness, tracking of developing alterations in the RNFL and visual field abnormalities are other important examinations for diagnosing glaucoma. Currently, the American Academy of Ophthalmology advises individuals with glaucoma risk factors to get complete eye exams on a regular basis, with the frequency depending on age, race, and family history.[40]

- **Fundus examination[41]**

Fundus examination will be helpful to differentiate the glaucomatous from non- glaucomatous eyes. The different characteristics to look for during fundus examination are disc size and shape, optic cup depth and shape, cup-to-disk ratio (CDR), the neuroretinal rim (NRR), presence of disc haemorrhage and its location, RNFL defects, parapapillary chorioretinal atrophy.

Figure 4: Ophthalmoscopic images of glaucomatous and healthy optic discs[42] (Jonas)

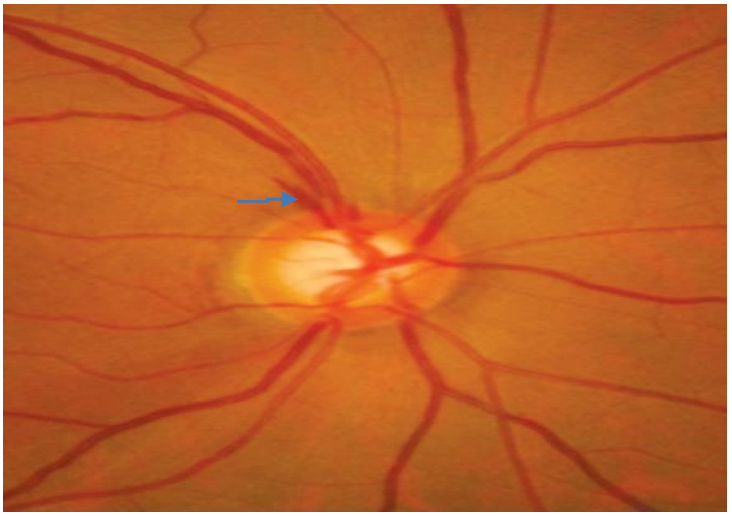


A) “The NRR of healthy disc has its typical form, with the inferior area being the broadest, followed by superior region, then nasal region and lastly the temporal region (ISNT rule).”

B) Glaucomatous optic disc has thinner NRR than that of the healthy optic disc, leading to optic cup to become wider, deep.

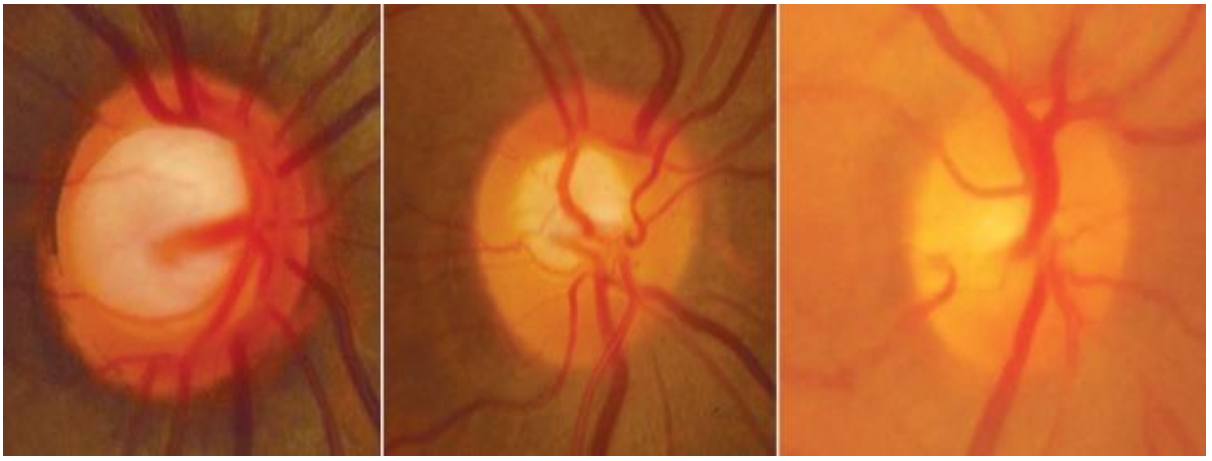
A disc haemorrhage is a splinter or flame-shaped that is perpendicular to the disc margin and orientated radially. These are situated in the neighboring superficial retinal nerve fibre and the prelaminar region of the optic disc. In the general population, disc hemorrhage occurs between 0.6 and 1.4 percent of the time, but in glaucoma patients, it occurs between 1.9 and 16.9 percent of the time.[42] According to reports, overall incidence of optic disc hemorrhage’s is 0.5 percent among ocular hypertensives, 2.5 percent in the eyes with POAG.[44] In 9 years of follow-up, 5.4 percent of incidence is noted in primary angle closure glaucoma (PACG).[45] Prevalence of optic disc haemorrhages is more in eyes with normotensive glaucoma (5-9 percent) than POAG (4. 3 percent) or secondary open angle glaucoma (1.2 percent). According to reports, bilateral haemorrhages occur between 5.6 and 16.7 percent of the time while repeated haemorrhages occur between 45.6 and 53.6 percent of the time.

Figure 5: Splinter haemorrhage, a classic glaucoma complication



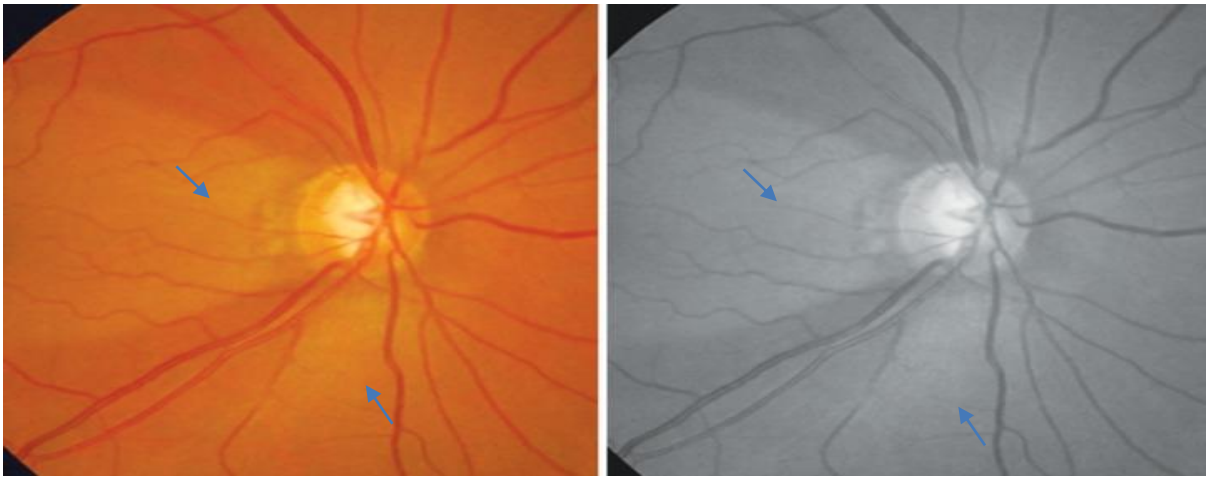
The vertical CDR and vertical diameter are positively correlated. A huge disc will typically have huge cup, a medium sized disc will have a medium-sized cup and a small disc often none at all. It is important to remember that we may overlook early or even moderately advanced glaucoma by only assessing glaucoma based on the C:D ratio.[41]

Figure 6: A large disc and a large cup, an average-sized disc and an average-sized cup, and a small disc with no cup.



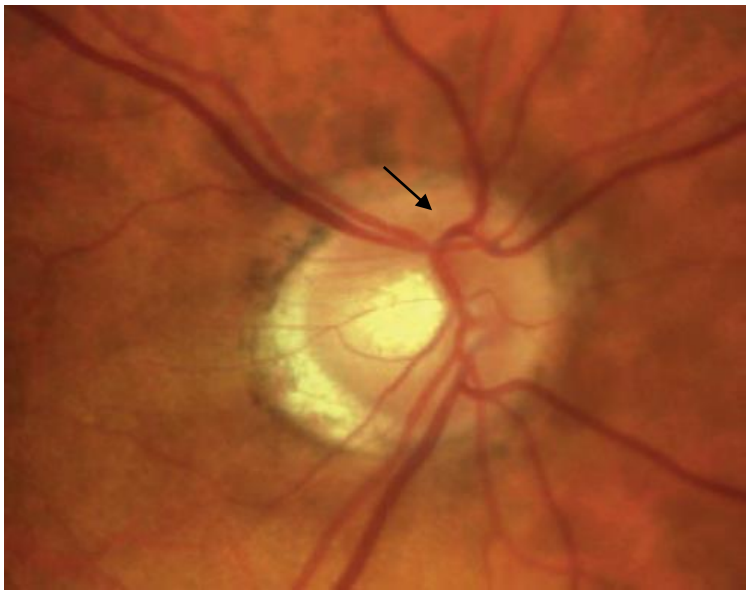
Examining RNFL is crucial for detecting early glaucomatous alterations even before perimetry. Examining the RNFL may also help to distinguish between glaucoma and nonglaucoma.[42] RNFL defects can be localised wedge shaped or diffuse defects. Red free light or OCT or confocal scanning laser tomography can be used to see the defects better.

Figure 7: Superior and inferior RNFL defects with bipolar notching.



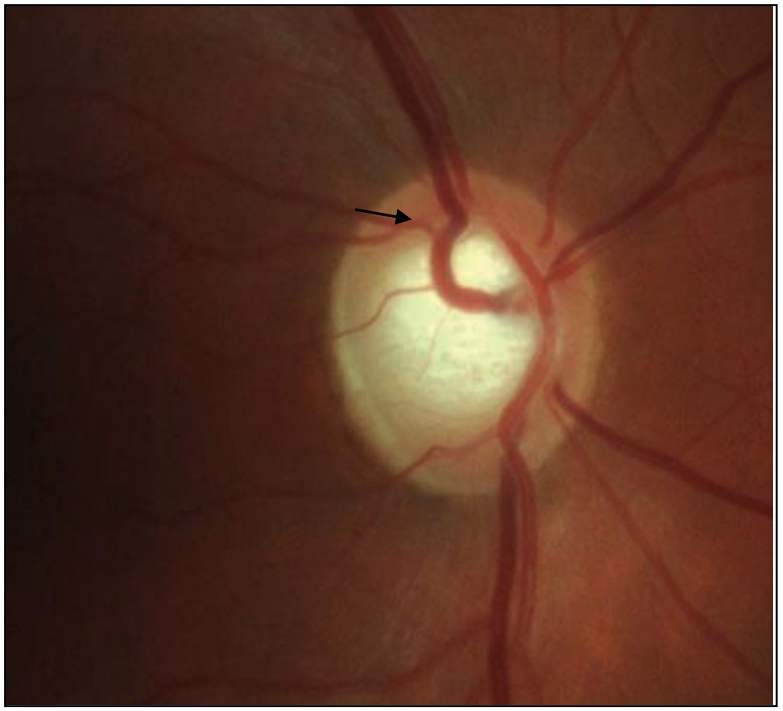
Baring of circumlinear vessels refers to the space between a superficial blood vessel travelling from the superior/inferior portion of the disc towards the macula and the disc margin.

Figure 8: Baring of circumlinear vessels



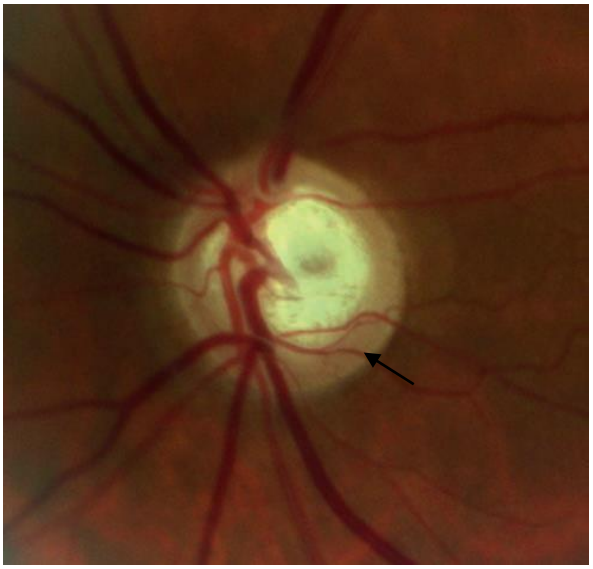
Bayonetting of vessels denotes to kinking/bending of the vessel over the edge of the cup.

Figure 9: Bayonetting of vessels



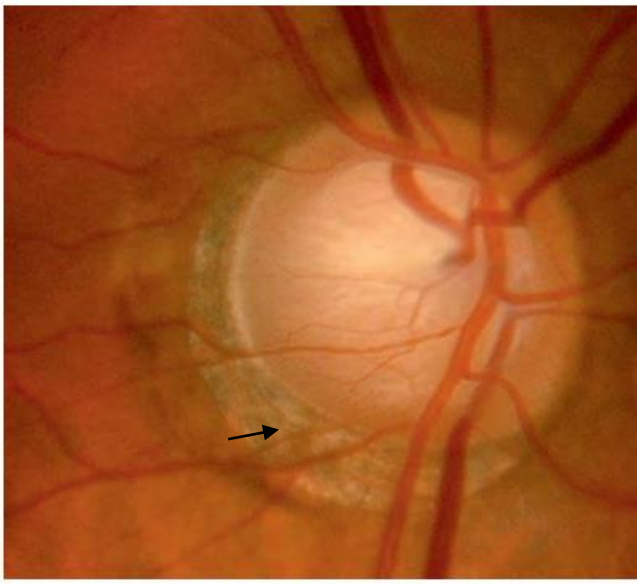
The laminar dot sign appears as glaucoma progresses. As the NRR retreats, the lamina cribrosa produces fenestrations that resemble grey dots. It can also be present in healthy eyes.

Figure 10: Laminar dot sign



Peripapillary alterations (PPA) include alpha/outer zone atrophy from superficial changes in the retinal pigment epithelium. The beta/inner zone is because of chorioretinal atrophy. In glaucoma, these 2 zones are wider and more prevalent.

Figure 11: Peripapillary changes



- **Perimetry:**[46]

Perimetry is majorly of two types. Depending on the moving stimulus it is classified into static or kinetic. In static type the stimulus presented is stationary. In Kinetic type the moving stimulus is of specific shape, brightness, size is used to detect the field defect. Static type is superior and more reliable than kinetic perimetry for recognition of field defects due to glaucoma. The Humphreys visual field analyser (HFA) is an example of static type of perimeter.[46]

Humphreys visual field analyser (HFA):[47]

Humphrey field analyser (HFA) is used to detect monocular visual field, but it may also be used for screening, monitoring, and helping with the diagnosis and monitoring of specific diseases like glaucoma and even brain lesions. Visual field anomalies indicates the damage in visual system that can be anywhere from retina to brain's visual cortex.

Because non-congruous defects such as superior defect in one eye, inferior defect in the other eye might go missing when both eyes are tested together, this can be due to overlapping of normal field region in one eye to defects in the other eye and can seem as normal binocular vision. visual field defects in early glaucoma are subtle, can be missed easily. Even the automated and sensitive visual field analyser cannot detect the field loss in glaucoma until there is loss of at least 30% of RGC axons. To detect field loss in glaucoma the defects in upper, lower hemi-fields that is below, above the horizontal raphe are tested. However, for detection of neurological field loss defects on either side of the vertical meridian are seen. "The newer visual field testing equipment that are easier, reliable and affordable than standard perimetry such as frequency doubling technology (FDT), short-wavelength automated perimetry (SWAP) and motion displacement perimetry (MDP) are available and can help in detecting glaucoma.[47]"

Figure 12: Humphreys visual field analyser[47]

Figure 13: Normal visual field of left eye[47]

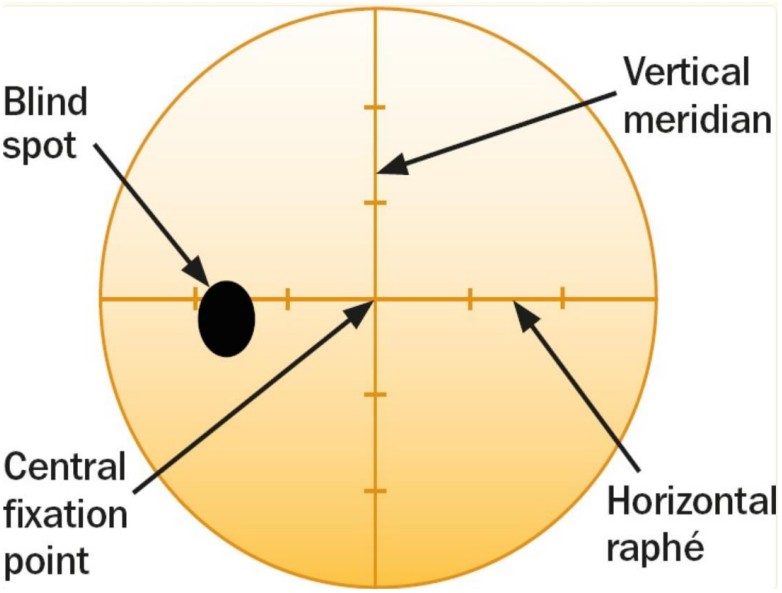
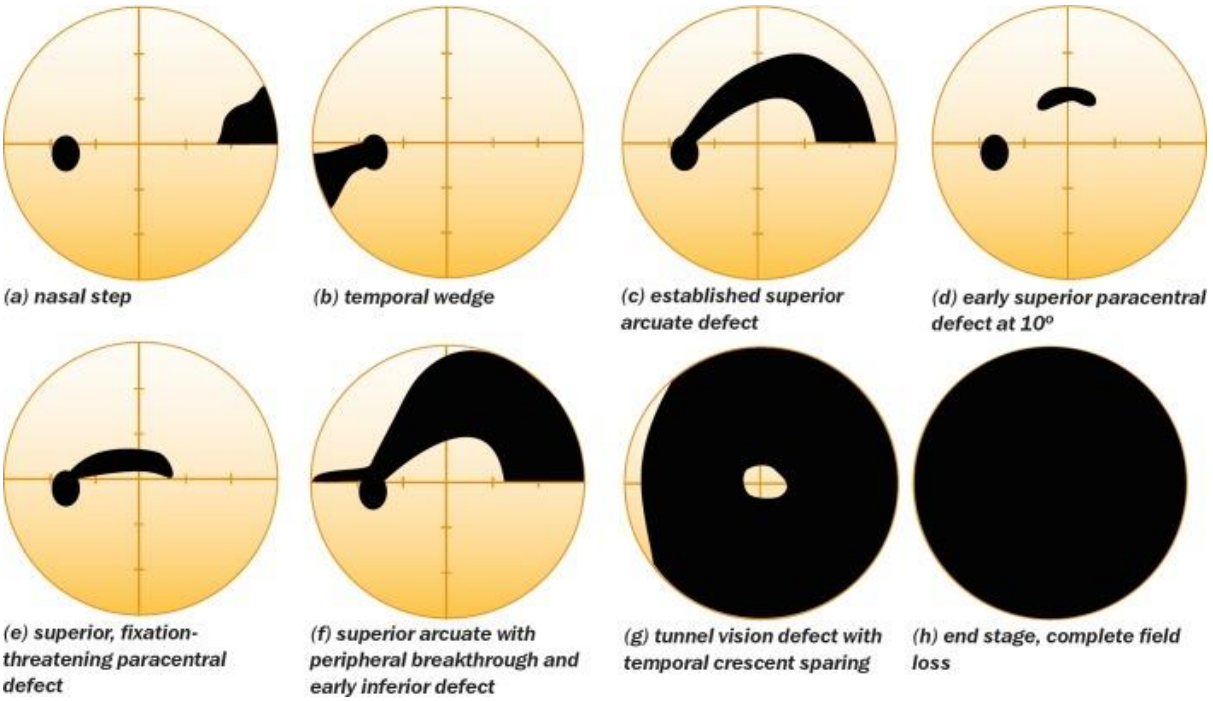


Table 3: Anderson criteria

Criteria for early defect
Mean deviation no worse than –6 dB
On pattern deviation plot, <25% of points depressed below the 5% level and <15% of points depressed below the 1% level
No point within central 5° with sensitivity <15 dB
Criteria for moderate defect
Mean deviation worse than –6 dB but no worse than –12 dB
On pattern deviation plot, <50% of points depressed below the 5% level and <25% of points depressed below the 1% level
No point within central 5° with sensitivity ≤0 dB
Only 1 hemifield containing a point with sensitivity <15 dB within 5° of fixation
Criteria for severe defect
Mean deviation worse than –12 dB
On pattern deviation plot, >50% of points depressed below the 5% level or >25% of points depressed below the 1% level
Any point within central 5° with sensitivity ≤0 dB
Both hemifields containing point(s) with sensitivity <15 dB within 5° of fixation

The diffuse visual field defects are seen with cataract's or corneal opacities etc. Visual field defects in glaucoma are non-specific, even though typical loss is related to arrangement of RGC axons within the RNFL.[47]

Figure 14: Types of Glaucomatous field defects[47]



- **Measuring the CCT**

Any new patient who is suspected of developing glaucoma must have a central cornea thickness (CCT) examination.[6] The CCT was said to be a important risk factor for ocular hypertension progression to primary open angle glaucoma(POAG) according to Ocular Hypertension Treatment Study (OHTS). [48]

The most popular technique for measuring CCT is ultrasonic pachymetry (UP), which necessitates the use of topical anaesthesia and a compliant patient because it needs contact with the eye and is generally simple to use, repeatable, and inexpensive. Other non-contact imaging methods include OCT, rotating Scheimpflug cameras, optical low-coherence reflectometry, specular microscopy and slit-scanning optical pachymetry. [6]

Ultrasonic Pachymetry:

"The gold standard method for measuring of CCT. Works on a principle that the measurement of time difference between echoes of ultrasonic signal pulses from the transducer of probe and the reflected signals from anterior and posterior corneal surface to the transducer" Obtaining the corneal thickness at a single location on the cornea is easily done using ultrasonic pachymetry. If more than one spot on the corneal surface is required, the measurement can be repeated. The morphological analysis is carried out by a specialised computer and determines the corneal thickness.[49] The benefit of ultrasonic pachymetry is its simple to use and can handle the equipment efficiently with little training. An ultrasonic pachymeter is portable and practical due to its tiny size. Since it is an applanation device that needs anaesthesia before use, this test should be carried out after the slit-lamp examination.[50]

- ***Management:***

Main goal of treatment is to achieve target IOP that is "the range of IOP below which further damage to the optic nerve head is unlikely. The target pressure is set as an individual basis,

based on the baseline IOP, level of pre-existing damage, rapidity with which the damage has occurred, systemic associations. Target IOP = INITIAL PRESSURE (1- INITIAL PRESSURE/100) Z+2, where Z is a constant. The target pressure for early glaucoma are < 21 mm Hg, < 18 mm Hg for moderate glaucoma, < 15 mm Hg for advanced glaucoma. Revised."

Medical Management[51,52]

Table 4: Medical management of glaucoma

Treatment category	Treatment type		Mechanism of action
Drugs	Local application	Prostaglandin analogs	Improved uveoscleral and trabecular outflow
		Beta-blockers	Reduced aqueous humor production
		Alpha $_2$ -adrenergic agonists	Reduced aqueous humor production, increased uveoscleral outflow
		Carbonic anhydrase inhibitors	Reduced aqueous humor production
		Miotic agents	Widening of the chamber angle
	Systemic intake	Carbonic anhydrase inhibitors	Reduced aqueous humor production
		Osmotically active substances (mannitol IV)	Osmotic removal of water from the globe

There are several different chemical types that may be used topically to lower intraocular pressure.[51] "Prostaglandin analogues (bimatoprost reduces the intraocular pressure by 5.61 mm Hg, latanoprost reduces the intraocular pressure by 4.85 mm Hg, travoprost reduces the intraocular pressure by 4.83 mm Hg, tafluprost reduces the intraocular pressure by 4.37 mm Hg), followed by beta-blockers (levobunolol reduces the intraocular pressure by 4.51 mm Hg, timolol reduces the intraocular pressure by 3.70 mm Hg, carteolol reduces the intraocular pressure by 3.44 mm Hg, levobetaxolol reduces the intraocular pressure by 2.56 mm Hg,

betaxolol reduces the intraocular pressure by 2.24 mm Hg), alpha$_2$-adrenergic agonists (brimonidine reduces the intraocular pressure by 3.59 mm Hg, apraclonidine reduces the intraocular pressure by 2.52 mm Hg), and carbonic anhydrase inhibitors (dorzolamide reduces the intraocular pressure by 2.49 mm Hg, brinzolamide reduces the intraocular pressure by 2.42 mm Hg) are the suggested common agents.[52]"

Operative techniques[51]

Table 5: Surgical management of glaucoma

Operative interventions	Laser therapy	Laser trabeculoplasty	Increased outflow of aqueous humor via the canal of Schlemm
		Cyclophotocoagulation	Reduced aqueous humor production
	Surgery	Cyclocryocoagulation	Reduced aqueous humor production
		Minimally invasive procedure	For example, implantation of a stent in the canal of Schlemm to lessen the outflow resistance of the trabecular meshwork
		Non-filtering procedure	For example, deep sclerotomy: widening of the outflow pathways without incising the eye
		Filtering procedure	For example, trabeculectomy: creation of an accessory pathway for the aqueous humor to flow out of the eye under the conjunctiva

Laser[51]

Laser therapy is considered if medical treatment is not adequately lowering the IOP or obtaining target pressure is failed. Laser can moderately lower IOP by increasing aqueous outflow

following laser trabeculoplasty or decreasing aqueous generation after cyclophotocoagulation. About 20% of IOP can be reduced by cyclophotocoagulation. Major complication of cyclophotocoagulation can be insufficient or extreme pressure reduction, inflammation.[51]

Surgical management[51]

A failed maximum tolerable medical therapy, likelihood that laser therapy may be insufficient or unsuitable. Early surgery may result in a better long-term prognosis for the therapy of choice in advanced diseases, but risks are calculated carefully on individual basis.

There are filtering and non-filtering methods of minimally invasive glaucoma surgery. Minimally invasive technique, involves inserting a stent into the Schlemm canal to reduce resistance of outflow via TM. Unless the glaucoma is very mild, this procedure, which may be combined with cataract surgery, does not typically reduce IOP sufficiently.

Although a filtering method seems to have fewer adverse effects than minimally invasive glaucoma surgery, it also decreases intraocular pressure less significantly. To decrease the generation of aqueous humour, cyclocryocoagulation is used. To enlarge the outflow routes without incising the eye, deep sclerotomy is performed. The purpose of trabeculectomy is to provide a secondary conduit for the aqueous fluid to exit the eye via the conjunctiva.

In a filtering surgery, an alternate aqueous flow pathway is created. Trabeculectomy is a standard type of filterating procedure. In advanced glaucoma patients trabeculectomy is preferred than laser trabeculoplasty. Deep sclerectomy and canaloplasty are other surgical methods with low complication risk.[51]

- **PRIMARY OPEN ANGLE GLAUCOMA (POAG)**

"Primary Open-angle glaucoma is a chronic, progressive, and irreversible multifactorial optic neuropathy that is characterized by an open angle of the anterior chamber, optic nerve head changes, progressive loss of peripheral vision followed by central visual field loss. Intraocular pressure is an important and modifiable risk factor, it can also occur due to death of RGC.[53,54]"

- **Prevalence**

The prevalence of POAG is more worldwide, escpecially in African and Western population.[55,56]

- **Risk factors**

Elevated intraocular presssure, elderly age group, black race, myopia, thinner CCT, family history of POAG are significant risk factors.

Ocular perfusion pressure, ocular blood flow, myopia, and optic disc haemorrhages are ocular risk factors that have been suggested.

Systemic factors include smoking, lipid dysregulation, African ancestry, atherosclerosis, genetic factors, obesity, low blood pressure (especially at night), type 2 diabetes mellitus (DM), vasospasm, migraine, stress, and primary vascular dysregulation.[57,58,59]

- **Pathophysiology**

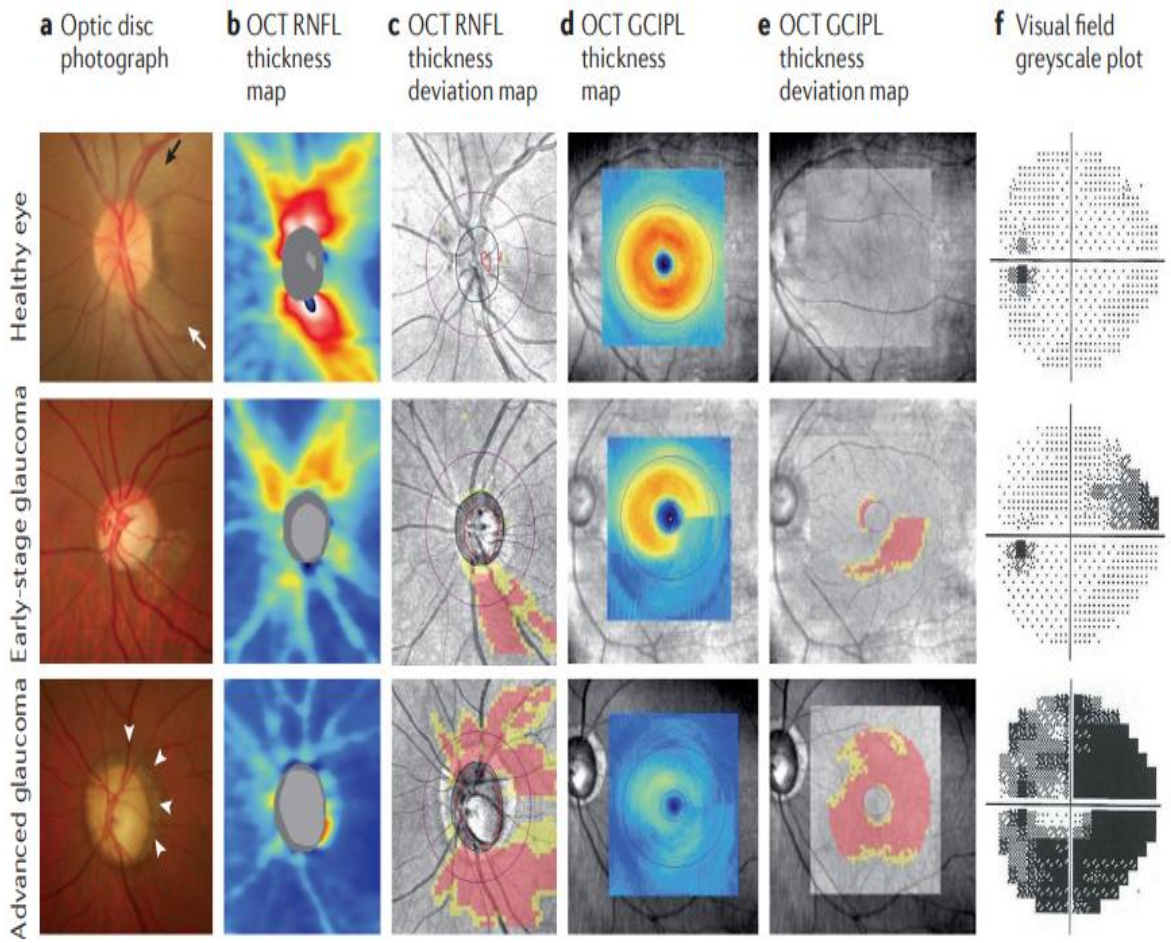
Persistently elevated IOP has been related to the pathophysiology of POAG and leads to gradual loss of vision. "It is thought that genetic anomalies set off a chain of actions that causes glaucomatous optic nerve damage and remodelling. The myocilin, optineurin and cytochrome CYP1B1 genes have been implicated with development of glaucoma.[60,61]" Furthermore, single-nucleotide polymorphisms and sequence variations have been related to POAG in genome studies.[62–65] Impact of several systemic factors on glaucoma development is still up for debate, notwithstanding the influence of genetic mutations and variations.

- **Diagnosis**

Several diagnostic methods can be used to clinically examine open-angle glaucoma, however the following triad has traditionally served as the basis for diagnosis:[66]

1. Changes in Optic disc
2. Changes in Visual field
3. Raised IOP

Figure 15: Optic disc changes & field defects in POAG



When compared to the healthy eye, optic disc pictures (part a) of early glaucoma (middle panel) and advanced glaucoma (bottom panel) both reveal a narrower NRR with an elevated CDR (upper panel). Alterations in the supero-temporal quadrent (black arrow) infero-temporal quadrent (white arrow) of ONH are common locations for RNFL and ONH changes. In advanced glaucoma patients, beta-zone PPA is seen (arrowheads). RNFL loss in both early-stage and severe glaucoma, according to OCT. Maps showing thickness of RNFL (part b) and OCT- RNFL thickness deviation maps (part c). On the maps showing thickness of RNFL, warmer colours indicate thick RNFLs and cooler colours indicate thin RNFLs. The deviation maps are built using information gathered from healthy people. The 99th percentile and 95th percentile ranges are shown by the red and yellow areas, respectively. During early and late stage glaucoma both ganglion cell layer and inner plexiform layer are lost, as shown by OCT ganglion cell inner plexiform layer (GCIPL) thickness maps (part d) and OCT GCIPL thickness deviation maps (part e). According to part b and part c's descriptions, maps are colour coded. The diffuse loss of fields in advanced glaucoma (part f) and early superonasal abnormalities in early glaucoma are shown in the visual field greyscale plots.[67]

Management

Preventing the advancement of alterations to the ONH & reducing risk of visual field degradation are the objectives of POAG therapy.

Target IOP was a notion that was created to help with this objective.

Initially, medications are needed (for example, prostaglandin analogues like latanoprost or tafluprost, beta-blockers like timolol), and occasionally surgery like laser trabeculoplasty or filtration operation.[68]

- **PSEUDO EXFOLIATIVE GLAUCOMA (PXG)**

Pseudo exfoliation syndrome (PEX) is a systemic condition typically manifests as whitish-gray protein deposition on the corneal endothelium, iris, lens, ciliary epithelium, and trabecular meshwork in the eyes. The periocular tissues, conjunctiva, epithelium of lens, TM, iris, ciliary processes are the source of material, according to pathologic analysis, despite attempts to identify it being unsuccessful. This material that is insoluble, floats in the aqueous humour, and then deposited in the TM. The endothelial cells of TM continue to produce the proteinaceous substance that clogs trabecular gaps and focally collapses Schlemm's canal. IOP rises and aqueous humour outflow declines as a result.[69]

PEX may be bilaterally or unilaterally present. The most common cause of secondary open-angle glaucoma. PEX is closely related to increased IOP in about 44 percent of patients and the subsequent onset of PXG.[70]

- **Prevalence**

Prevalence of PXG is between 0.3 percent to 30 percent in persons who are 60 or older. Between 60 and 70 million people worldwide are affected by PEX.[71,72] Pseudoexfoliation syndrome, however, considerably increases the risk of developing elevated IOP in people who are afflicted, and it causes glaucoma in 15 to 30 percent of cases.[69]

- **Risk factors**

The following have been identified as PEX risk factors:[73]

- Elderly age
- Nordic and Mediterranean racial groups

- **Pathophysiology**

Although the precise cause of PEX is unknown, the disease's link with the human leukocyte antigen suggests a hereditary factor to it.[74]

Uncertainty exists about the precise pathophysiological mechanisms underlying PEX. But PEX is now widely acknowledged to be a fibrillopathy. PEX material develops due to an aberrant buildup of elastic microfibrils, which are made up of proteins including vitronectin, fibrillin-1, clusterin, fibrillin-2, lysyl oxidase among others. Microfibrils are created by the accumulation of fibrillin molecules, and once they are crosslinked, PEX fibrils result. One important facilitator of the aberrant buildup of PEX material is thought to be transforming growth factor-beta 1 (TGF-β1). Synthesis higher β quantities have been discovered in aqueous humour of PEX eyes. Ascorbic acid levels have reduced and oxidative stress indicators have risen in aqueous humour, providing evidence that oxidative stress contributes to the development of PEX. PEX is also linked to anterior chamber hypoxia and hypoperfusion of the iris.[74]

- **Diagnosis**

Every patient who is having PEX should have a gonioscopy and slit-lamp examination. The risk of developing OHT and PXG is more so, baseline IOP should be evaluated during diagnosis and then in any case once a year after that. Given that PEX is a clinical diagnosis, genetic testing of the eyes is not frequently done.[74]

IOP calculation: Pseudoexfoliation caused by glaucoma has a pronounced diurnal IOP change and is related with elevated intraocular pressure. Therefore, it is vital to check IOP during the course of the day.

Optic disc changes

A higher CDR than 0.5, an imbalance in CDR between the two eyes and disc haemorrhages

are classic symptoms of glaucomatous damage.[75]

Figure 16: A patient with Pseudoexfoliative glaucoma's left eye[73]

Type of field defects

As a basic assessment in glaucoma suspects and for tracking the disease progression in confirmed glaucoma cases, static automated perimetry is helpful. It is common to observe diffuse visual fields, superonasal field defects are often more severe.[75]

Optical coherence tomography (OCT): Glaucomatous damage is related to RNFL (retina nerve fibre layer) thinning in the peripapillary area.[76]

- **Management**

There is no medication to stop the accumulation of PEX material in the eyes. Regular examination for early identification and treatment is the main focus. Medical care is the primary line of defence for PXG. Prostaglandin analogues, beta-blockers, carbonic anhydrase inhibitors, or a combination of them are among the topical drugs that can decrease IOP in PXG. In general, PXG is less responsive to medical treatment than POAG. In order to prevent more intrusive surgery, selective laser trabeculoplasty (SLT) may be utilised as the first line of therapy.[74]

- **OCULAR HYPERTENSION (OHT)**

"An intraocular pressure (IOP) more than 21 mm Hg, a healthy optic nerve, and a normal visual fields are the criteria for defining ocular hypertension.[77] It has been described as the contrast of normal-pressure glaucoma, in which progressive glaucomatous optic neuropathy occurs despite a normal IOP.[78]"

- **Prevalence**

Ocular hypertension affects between 2.7 and 3.8 percent of the general population. OHT rises with age, from 1.7 to 2.7 percent in the 40 to 49 year age range, 2.7 to 4.6 percent in the 50 to 59 year range, to 4.1 to 7.5 percent in those over the age of 80.[79-81]

- **Risk factors:[82]**

- Increased aqueous production.
- Poor aqueous drainage.
- Steroids.
- Trauma to eye
- Race, age, and family history are risk factors.

- **Diagnosis:**

Disc changes

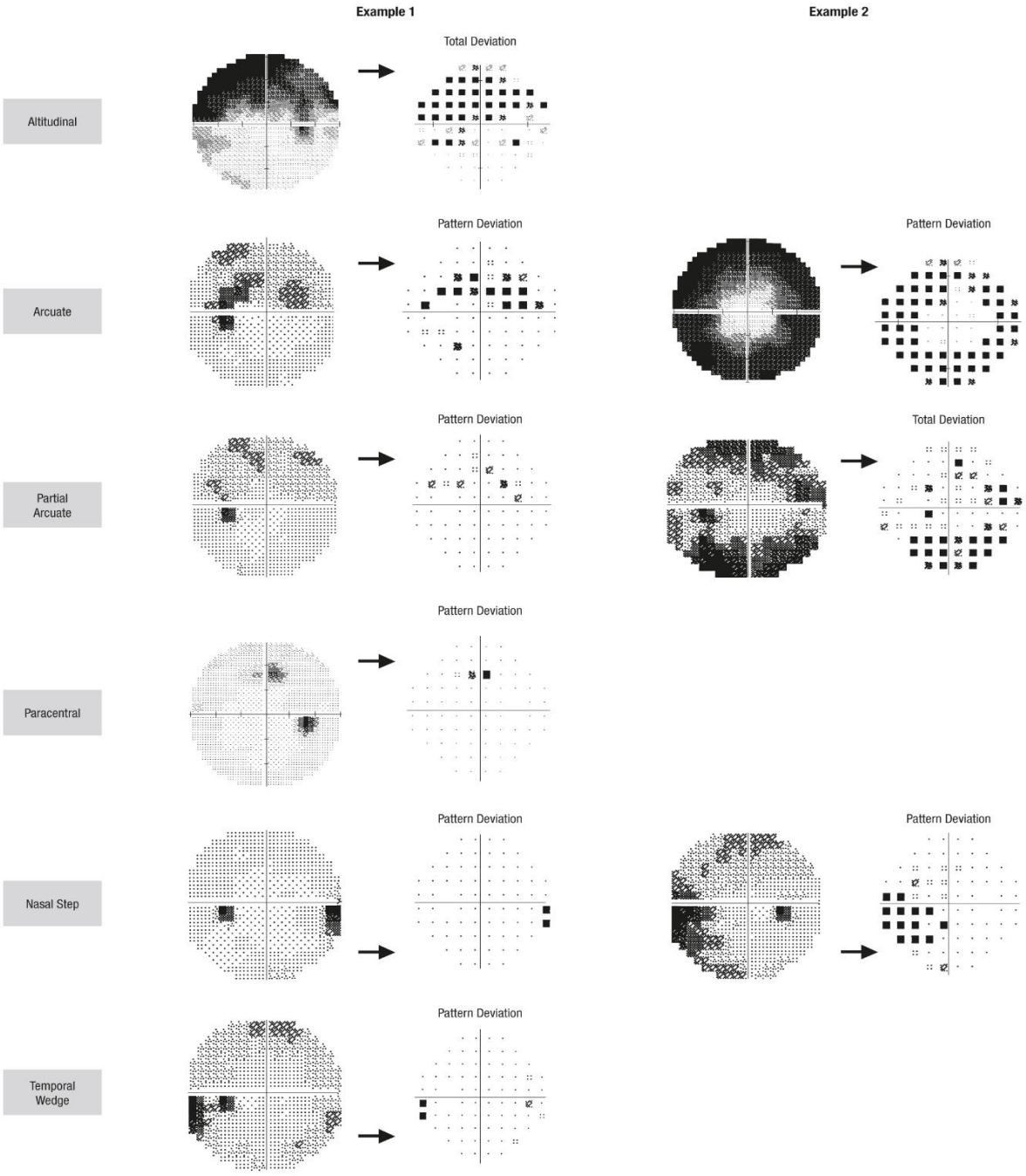
The primary characteristics of the disc alterations in ocular hypertension eyes are

Increased cupping with pallor. Pallor is preceded by cupping. Cupping is contour of the optic disc, whereas pallor absence of blood vessels.[83]

Type of field defects[84]

The visual field abnormalities seen with OHT group is paracentral scotoma being the most common followed by diffuse widespread loss and central visual field loss.[84]

Figure 17: Total or pattern deviation plots are used to categorise aberrant nerve fibre bundles[84]



- **Management**

In those with increased IOP, topical ocular hypotensive therapy proved successful in delaying or avoiding the onset of POAG. Incidence of glaucoma is reduced by 50 percent to 60 percent

when ocular hypertension is treated with topical medications, according to the OHTS.[77] Considering the risk variables such as CCT, family history, and life expectancy they should be treated with prostaglandin analogue, selective laser trabeculoplasty (SLT), is the surgical management.[85]

- ***Relation between CCT and IOP/Glaucoma***

Studies by Ehlers and Hansen and Whitacre et al. said that the significant variable in assessment of IOP is measuring CCT among the patients undergoing refractive and corneal transplant surgery, also in contact lens wearers. In individuals with ocular hypertension, CCT was a reliable indicator of the onset of POAG. This study specifically showed that participants with lower CCT readings had a higher probability of developing POAG (1.71 for 40 μm reduction in central corneal thickness). Furthermore, glaucoma risk was three times higher in those with CCTs of 555 μ or less than it was in people with CCTs of 588 μ or above.[5]

Furthermore, individuals with various forms of glaucoma have shown variances in mean CCT. The findings together imply that CCT levels have an impact on IOP readings. In reality, because individuals with NTG often have thin corneas and glaucoma suspicious patients have thick corneas, misdiagnosis frequently happens when CCT is not taken into account.[36,86–88]

Studies comparing the CCT in POAG and/or PXG and/or OHT and/or normal eye.

Gorezis et al. investigated and assessed the CCT in various glaucoma forms. In the following patient groups, an observational cross-sectional investigation evaluated CCT under a specular microscope: 60 eyes with POAG, 50 with PXG, 50 with OHT, and 60 had neither (control group). Pseudoexfoliative glaucoma patient's CCT was much thinner ($P < 0.05$) than those with ocular hypertension, and vice versa. These findings suggests the idea that CCT should

be taken into account when assessing instances of PXG and OHT since it differs in various kinds of glaucoma.[89]

Yagci et al. had set out to ascertain the association between CCT and IOP assessed by GAT in glaucomatous, OHT, and normal eyes. The study involved 125 individuals in total 50 were normal healthy individuals, and 26 with POAG, 25 with pseudo exfoliative glaucoma (PXG), 24 with OHT. CCT values were determined using an ultrasonic pachymeter, whilst IOP values were determined using a Goldmann applanation tonometer. CCT values in the OHT group (595.75±22.52 micrometer) were higher than those in the normal group (526.28±31.73 micrometer), the POAG group (539.92±21.50 micrometer), and the PXG group (526.28±21.50 micrometer). (533.96±29.25 micrometer) ($p<0.05$). The authors of this study found that one-third of the IOP readings for the OHT group when reinterpreted using the CCT formulas were normal.[90]

Using optical low coherence reflectometry, **Ventura et al**. measured the central corneal thickness in patients with normal tension glaucoma, primary open angle glaucoma, pseudoexfoliation glaucoma, or ocular hypertension, as well as that of healthy individuals. Using Goldmann applanation tonometry, IOP was assessed. Ocular hypertension patients' central corneal thickness was significantly greater (p or <0.05) than that of healthy people, patients with normal tension glaucoma, primary open angle glaucoma and pseudoexfoliation glaucoma; there were no significant differences among the latter four groups. Additionally, patients with ocular hypertension were considerably younger than any of the three glaucoma groups ($p <0.05$). This study demonstrates that a considerable proportion of ocular hypertension patients have normal IOPs after making the necessary corrections for outliers in their central corneal thickness.[91]

According to **Shetgar et al.** CCT of POAG, OHT with that of Normal Tension Glaucoma (NTG). CCT's were measured using UP and IOP was measured with GAT for all the sub-

jects. They observed statistically significant ($p<0.05$) difference in mean CCT between the groups. The CCT among NTG patients was significantly lower than the controls and POAG patients, whereas central corneal thickness was significantly higher in OHT patients than controls and POAG. The CCT of POAG patients did not differ significantly from the controls.[92]

Kumar et al. in their study examined the impact of CCT on clinical care in patients with NTG, POAG, OHT, and normal population. GAT was employed to measure the intraocular pressure. CCT was determined using UP. The mean CCT was 503.91±11.31µm, 504.36 ±11.07 µm in the right and left eye respectively among NTG group. The mean CCT of POAG patients was 525.25± 23.59 µm and 526.38± 21.98 µm in the right and left eye, respectively. 572.25± 22.71 µm and 572.67± 22.20 µm was the mean CCT among OHT group in right and left eye respectively. This study confirms that CCT was significantly higher among OHT group than controls and patients with POAG. In contrast, CCT is significantly lower in NTG group when compared to controls and POAG. There is no discernible difference between POAG and control groups.[93]

In a south Indian population, **Natarajan et al.** compared the central corneal thickness of individuals with primary open angle glaucoma with healthy controls. There were 50 primary open angle glaucoma patients and 50 controls in total for the study. The mean central corneal thickness in the control group was 536 µ (462-608 µ), but it was 531 µ (476-609 µ) in the group with primary open angle glaucoma. Between patients with primary open angle glaucoma and the healthy controls, there was no appreciable variation in the central corneal thickness.[94]

Yazgan et al. compared the biomechanical characteristics of the corneas of healthy individuals with those who have pseudoexfoliation syndrome (PEX) and pseudo exfoliative glaucoma. (PXG). CCT was measured using ultrasound pachymetry (UP). Healthy subjects, PEX and PXG groups had mean CCTs of 546.3± 28, 525.5± 35, and 509± 36, respectively. Addi-

tionally, there were significant variations in CCT across the three groups ($p<0.05$). They came to the conclusion that CCT had reduced, which was more pronounced in PXG patients than in PEX patients.[95]

CCT and corneal curvature (CC) of pseudo exfoliative (PE) eyes with glaucoma, without glaucoma are measured by **Ozcura et al**. All PE and non-glaucomatous PE eyes had substantially lower mean CCT than control eyes with P value <0.05 and P value <0.05, respectively). Among PXG and control eyes, CCT did not vary. ($P>0.05$). CCT was noticeably narrower in PEX-treated eyes compared to control eyes, but there was no discernible difference between PXG-treated and control eyes.[96]

Pseudoexfoliative glaucoma (PXG) corneas' biomechanical characteristics were evaluated by **Ozkok et al**., and they were compared to those of individuals with primary open-angle glaucoma (POAG). The mean CCT did not significantly differ across the groups.[97]

In their cross-sectional study, **Vieira et al.** found that the central corneal thickness (CCT) was significantly thinner in POAG (526± 40.0 µm) and PXG (520± 38.2 µm) than in the control group. The study included 146 eyes from 146 subjects, including 37 healthy eyes, 23 eyes with OHT, and 86 glaucoma eyes (70 with POAG and 16 with PXG). (553 ± 28.8 µm).[98]

To compare healthy eyes and POAG eyes**, Juan-marcos et al**. measured the CCT and corneal endothelial morphometry in PE eyes with POAG and without POAG. The mean CCT did not differ significantly among the four groups.[99]

Past Studies comparing CCT and IOP:

This prospective research study was done to compare IOP and ocular pulse amplitudes (OPA) in patients with POAG and PXG by **Moghimi et al**. IOP was assessed using the Pascal dynamic contour tonometry (DCT) and the Goldmann applanation tonometry (GAT) and also measured CCT. GAT IOP and CCT in the POAG and PXG groups were associated ($r = 0.40$,

P<0.05) although DCT IOP and CCT were not associated (r = 0.35, P-0.05). DCT IOP and CCT did not correspond.[100]

In order to determine whether there is any fluctuation in the central corneal thickness and intraocular pressure in PE, as well as whether there is any connection between them, **Syed et al** conducted research. 85 individuals with PEX who did not have glaucoma had 141 eyes each subjected to a prospective observational study. At four different intervals throughout business hours, CCT and IOP were assessed using a portable ultrasonic pachymeter (DGH Technology INC. Pachette 2, USA) and a Goldmann applanation tonometer, respectively. Over the course of the 4 sessions, there was a statistically significant (P<0.05) decrease of about 10 μ in mean CCT and 1.4 mmHg in mean IOP. In PEX eyes, IOP and CCT are consistently correlated in a major way throughout the day. (*P* <0.05). The CCT measurements reveal considerable daytime thinning, and a corresponding decline in IOP was also seen. Our research demonstrates that the diurnal fluctuation of CCT and IOP are significantly correlated.[101]

Kniestedt et al. evaluated the connection between central corneal thickness (CCT), Goldmann applanation tonometry (GAT), PASCAL dynamic contour tonometry (DCT), and glaucoma stage in their prospective, cross-sectional tricenter observation research. Following ultrasonography pachymetry, intraocular pressure was assessed using Goldmann applanation tonometry and PASCAL. While PASCAL is not substantially related to central corneal thickness, intraocular pressure as measured by Goldmann applanation tonometry exhibits a strong link with it. Patients with advanced stages of glaucoma are more likely to have thin central corneal thickness. One potential contributing cause is the underestimation of intraocular pressure by Goldmann applanation tonometry.[102]

Iyamu et al. conducted a research to see if CCT is a more accurate predictor than intraocular pressure (IOP) in identifying those who are more likely to develop glaucoma early on. Based on clinical traits of ocular risk factors, 65 participants were divided into normals, ocular hy-

pertensives, and glaucoma subjects. A Goldmann applanation tonometer placed on a slit lamp was used to measure the IOP. The central corneal thickness (CCT) of both eyes was measured using the Sonomed PacScan 300AP Biometric/Pachymeter prior to applanation tonometry. Unpaired t-test; $p<0.05$; the difference in mean IOP between normal and glaucoma participants was statistically significant. The mean CCT between normal and glaucoma participants also differed significantly ($p<0.05$). In glaucoma patients, the connection between CCT and Age was marginally significant, but it was not significant in normal people. According to a linear model, CCT will decline by 7 im per 10 years. For ocular hypertensives, a substantial correlation between CCT and IOP was discovered, with a prediction of an increase of 0.70 mmHg for every 10 µ in corneal thickness. For glaucoma patients, there was a modest correlation between CCT and IOP, with an indication of an increase in intraocular pressure of 0.35 mmHg for every 10 µ in corneal thinning. When paired with other ocular risk factors, the central corneal thickness is a stronger indicator of people who are more likely to develop primary open-angle glaucoma than intraocular pressure.[103]

Han et al. examined children's intraocular pressure (IOP) and risk factors for glaucoma. In a multivariable study, the following IOP-related parameters were evaluated: thicker CCT [standardized correlation coefficient (SRC) = 0.201, $P<0.05$) Greater refraction (SRC = 0.090, P 0.001), lower mean corneal curvature (SRC = 0.123, P 0.001), shorter axial length (SRC = 0.086, P = 0.036), and a deeper anterior chamber depth (SRC = 0.059, P = 0.009) were all observed.[104]

According to the level of intraocular pressure (IOP) and the stage of congenital glaucoma, **Khamroeva et al**. calculate the values of central corneal thickness (CCT) in children. An automated non-contact tonometer/pachymeter made by NIDEK was

used to calculate CCT. They came to the conclusion that edema brought on by increased IOP is related with thicker CCT.[105]

Wei et al. used the NT-530P to map out the distributions and connections between the central corneal thickness (CCT) and intraocular pressure (IOP) in Chinese young people. CCT and IOP showed a significant connection in a linear regression study (r = 0.44, P 0.05). IOP will rise by 0.32 mm Hg for every 10 μ rise in CCT, according to the linear regression equation IOP = 2.35± 0.032 CCT. Corrected IOP (CIOP) had a mean of 15.32± 2.38 mmHg and no correlation to CCT. For every 10 μ increase in CCT, the IOP rises by 0.32 mmHg. Correctly assessing the real IOP depends on knowing the CCT value.[106]

MATERIAL & METHOD

4. MATERIALS AND METHODS

SOURCE OF DATA:.

A total 120 patients. 20 patients in each of the three study groups that is POAG, PXG, OHT were compared with 60 normal population as control in this case control study, visiting the outpatient department of Ophthalmology at R.L.J. HOSPITAL AND RESEARCH CENTRE, attached to SRI DEVARAJ URS MEDICAL COLLEGE.

STUDY DESIGN: Case control study

STUDY PERIOD: January 2021 – June 2022

INCLUSION CRITERA:

1. POAG: IOP prior to treatment >21mm of Hg, current IOP on treatment </=21mmhg, open angle on gonioscopy with optic disc changes and glaucomatous field defects.
2. PXG: IOP prior to treatment >21 mm hg, current IOP on treatment </=21 mm of Hg, pseudo exfoliative material in the anterior segment with optic disc changes and glaucomatous field defects.
3. Ocular hypertensive: IOP>21mmhg on at least 2occassions, healthy optic disc with no glaucomatous field defects.
4. Normals: IOP</=21mmhg, normal optic discs, open angle on gonioscopy, no suspicion of glaucoma.

EXCLUSION CRITERIA:

1. Evidence of other anterior segment pathology including corneal opacities, corneal oedema and keratoconus.

2. Previous intraocular or corneal surgery

3. Diabetes mellitus

4. Use of contact lens

5. Any other optic nerve pathology or intracranial disease

6. Anterior segment infections and inflammation

Ethical clearance

Prior to the commencement, the study was approved by the Ethics and Research Committee, Sri Devraj Urs medical college, Kolar.

Informed Consent

All the patients fulfilling selection criteria were explained about the nature of the study. A written informed consent was obtained from all the participants before enrolment (Annexure II and III).

METHOD OF COLLECTION OF DATA

1. Visual Acuity was done for both distant and near vision using Snellen's chart and jaeger's chart respectively.

2. Slit lamp bio microscopy to asses anterior chamber depth, any anterior segment abnormalities like corneal opacities, corneal edema and keratoconus, accumulation of PEX material.

3. Ultrasonic pachymetry: Patient is made to sit comfortably on a chair, anaesthetic eyedrops of 0.5% procaine are instilled into the eye before the acquisition of corneal thickness. The probe of pachymetry is placed on the corneal surface centrally in order to obtain the CCT.

4. Fundus examination by direct and indirect ophthalmoscopy, including optic disc evaluation to see for any glaucomatous disc changes.

5. Assessment of Intraocular Pressure using Goldmann Applanation Tonometer: “Patient is made to sit comfortably near the slit lamp, anaesthetic eye drops 0.5% procaine drops are instilled into eye and stained with 1% sodium fluorescein dye strip. Tonometer is kept perpendicular to the cornea and observed monocularly through the biprism at lower magnification. Tonometer is advanced forward to the patient until the tip gently touches the cornea and the semicircular mires are seen. Adjust until the two semicircles are of equal size, optimum thickness and seen in the center of the field of view. Tension knob is rotated until inner borders of the fluorescein rings meet each other at

6. the midpoint of their pulsations. The reading measured in grams is multiplied by 10 to get the IOP in millimeters of mercury.”

7. Gonioscopy – Shaffer’s grading system used

8. Humphery visual field analysis

The findings were documented and patients found to have POAG, PXG, OHT are identified and compared with normal population.

SAMPLE SIZE ESTIMATION

Was estimated by using the difference in Mean IOP between control and PXG from the study Yagci et. al. as 15.58 ± 2.40mmhg and 17.55 ± 4.87 mm of Hg. Using these values at 95% Confidence limit and 80% power sample size of 59 was obtained in each group by using the below mentioned formula and Med calc sample size software. With 10% nonresponse sample size of 59+ 5.9 ≈ 65 cases will be included in each group.

$$\text{Sample size} = \frac{2SD^2(Z_{\alpha/2} + Z_{\beta})^2}{d^2}$$

SD – Standard deviation = From previous studies or pilot study

$Z_{\alpha/2} = Z_{0.05/2} = Z_{0.025} = 1.96$ (From Z table) at type 1 error of 5%

$Z_{\beta} = Z_{0.20} = 0.842$ (From Z table) at 80% power

d = effect size = difference between mean values

So now formula will be

$$\text{Sample size} = \frac{2SD^2(1.96 + 0.84)^2}{d^2}$$

STATISTICAL METHODS USED FOR THIS STUDY

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 26.0 version software. Categorical data was represented in the form of Frequencies and proportions. Normality of Continuous variables were tested by Kolmogorov Smirnov test and found to be not normally distributed. Mann-Whitney test was applied to test the association between various groups and continuous variables Spearman correlation was used to test the association between continuous variables. Appropriate graphs were made for the results. A p value of <0.05 is considered statistically significant.

RESULTS

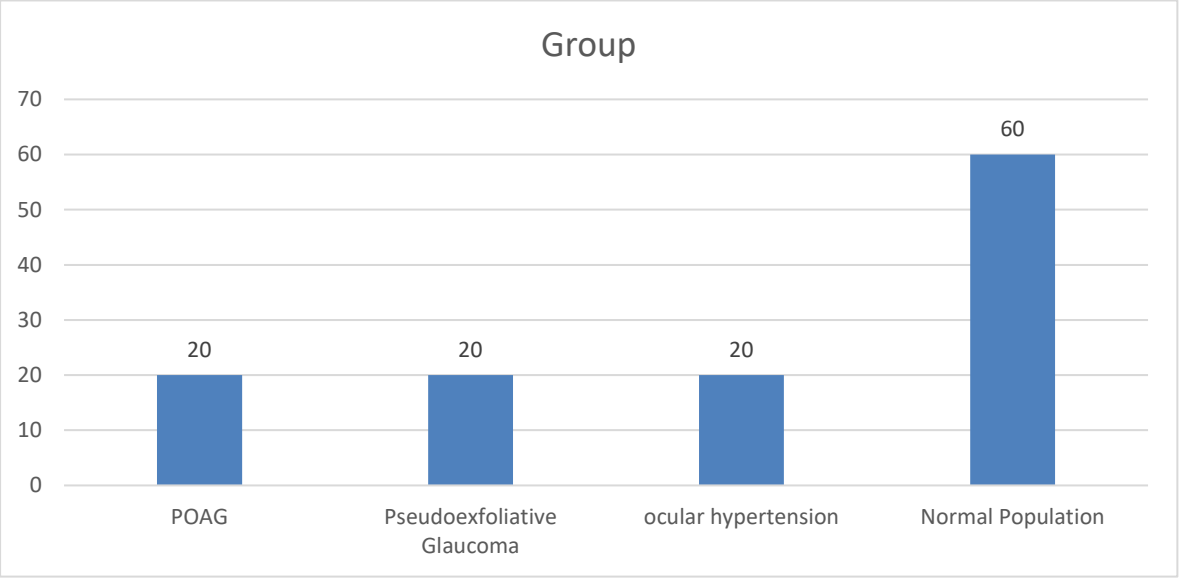
5. RESULTS

20 patients in each of the three study group were there, with 60 normal population as control.

Table :6 Distribution of study group

Group	Frequency	Percent
POAG	20	16.7
Pseudoexfoliative Glaucoma	20	16.7
ocular hypertension	20	16.7
Normal Population	60	50.0
Total	120	100.0

Graph 1: Distribution of study group

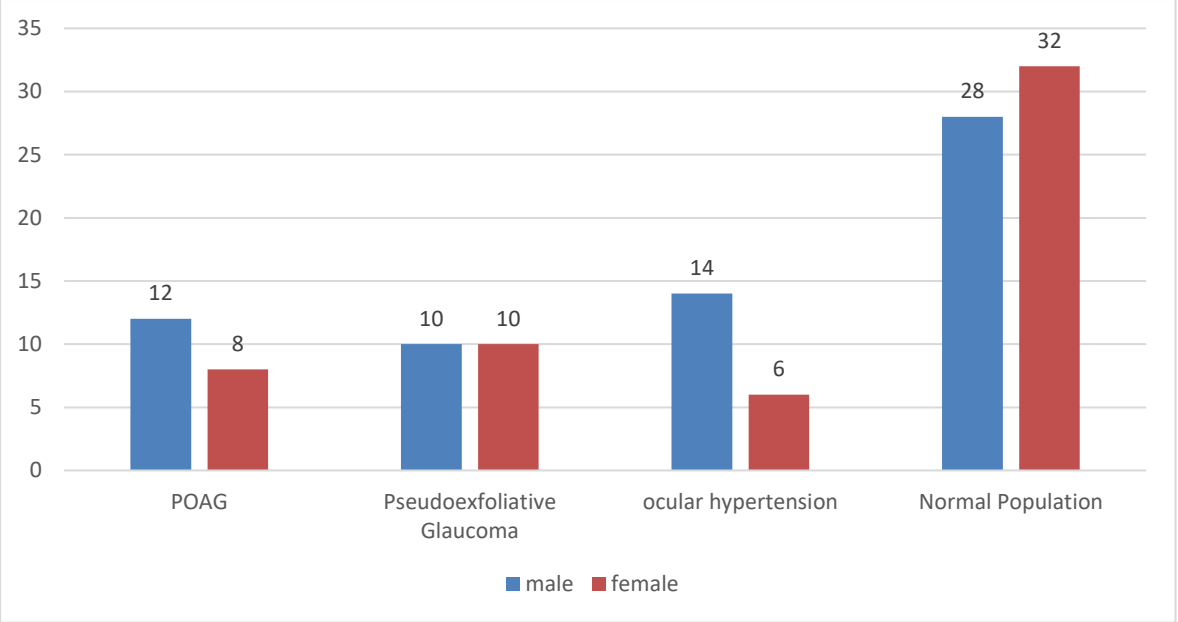


Males were the majority in POAG (60%) and ocular hypertension group (70%). Males and females were equal in PXG group. Females were the majority in normal population (53.3%)

Gender distribution

Group		**Sex**		
		Male	Female	Total
POAG	Frequency	12	8	20
	Percentage	60.0%	40.0%	100.0%
Pseudoexfoliative glaucoma	Frequency	10	10	20
	Percentage	50.0%	50.0%	100.0%
Ocular hypertension	Frequency	14	6	20
	Percentage	70.0%	30.0%	100.0%
Normal population	Frequency	28	32	60
	Percentage	46.7%	53.3%	100.0%
Total	Frequency	64	56	120
	Percentage	53.3%	46.7%	100.0%

Table 7: Gender wise distribution of study groups



Graph 2: Gender wise distribution of study groups

POAG:

The mean age among POAG group is 62.3 years, mean CCT is 528.2 µm, mean of GAT IOP is 19.1 mmHg, mean of CI IOP 21.3 mmHg and the difference in IOP is 2.2 mmHg.

Parameter	**Mean**	**SD**	**Median**	**IQR**
Age (years)	62.3	5.8	63	58,66
CCT (µm)	528.2	12.7	520	518,539.5
GAT IOP (mm Hg)	19.1	4.6	19	16,24
CI IOP (mm Hg)	21.3	2.8	22	20,23.5
IOP difference (mm Hg)	2.2	3	4	0,4

Table 8: Mean of age, GAT IOP, CI IOP and IOP difference among POAG group

Pseudoexfoliative Glaucoma (PXG):

The mean of age, CCT, GAT IOP, CI IOP 62.7 year, 518.5 µm, 18.4 mmHg, 21.4 mmHg respectively and the difference in IOP among the PXG group is 3 mmHg.

Parameter	Mean	SD	Median	IQR
Age (years)	62.7	8.4	60.5	56.5,68.3
CCT (µm)	518.5	10.6	516	512,528
GAT IOP (mm Hg)	18.4	5.3	18	14,24
CI IOP (mm Hg)	21.4	3.5	22	18,24
IOP difference (mm Hg)	3	2.1	4	0,4

Table 9: Mean of age, GAT IOP, CI IOP and IOP difference among PXG group

Ocular hypertension:

The mean of age, CCT, GAT IOP, CI IOP and the difference in IOP among the ocular hypertension patients were 56.7 year, 555.3 µm, 26.4 mmHg, 22.9 mmHg and -3.5 mmHg, respectively.

Parameter	Mean	SD	Median	IQR
Age (years)	56.7	6.6	57	51,62
CCT (µm)	555.3	12.8	559	540.5,566
GAT IOP (mm Hg)	26.4	7.4	28	18.5,32
CI IOP (mm Hg)	22.9	5.4	24	17,26
IOP difference (mm Hg)	-3.5	2.9	-6	-6,0

Table 10: Mean of age, GAT IOP, CI IOP and IOP difference among OHT group

Normal population

The mean of age, CCT, GAT IOP, CI IOP were 57.3 year, 531.8 µm, 15.2 mmHg, 15.2 mmHg respectively and there was no difference in IOP noted among the normal population.

Parameter	Mean	SD	Median	IQR
Age (years)	57.3	7.3	56	52,63
CCT (µm)	531.8	7.1	533	524,540
GAT IOP (mm Hg)	15.2	2.6	16	12,18
CI IOP (mm Hg)	15.2	2.6	16	12,18
IOP difference (mm Hg)	0	0	0	0,0

Table 11: Mean of age, GAT IOP, CI IOP and IOP difference among Normal population.

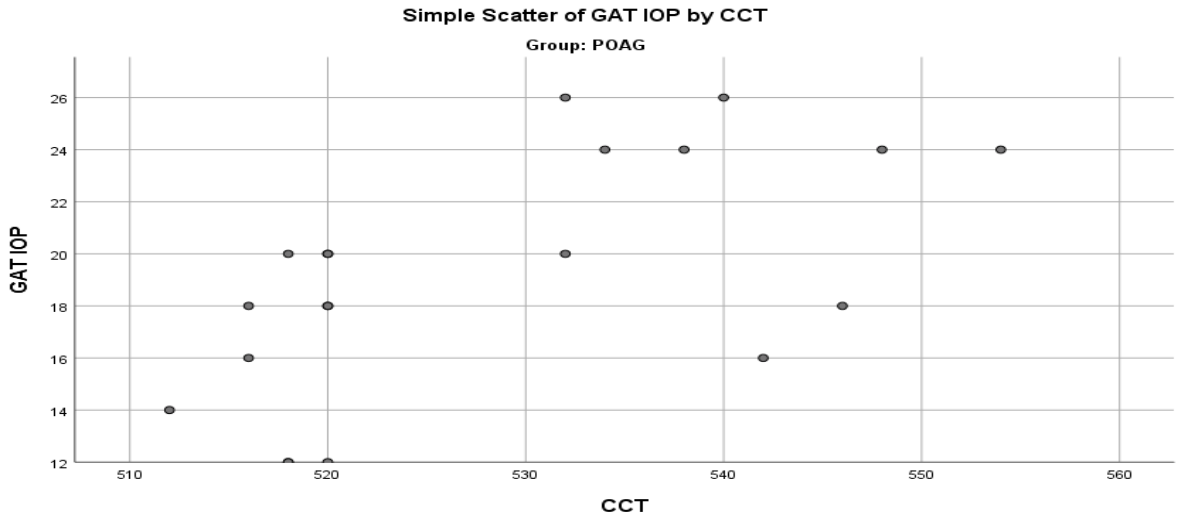
p value <0.05 indicates statistically significant association.

Correlation between the CCT and various IOPs among POAG

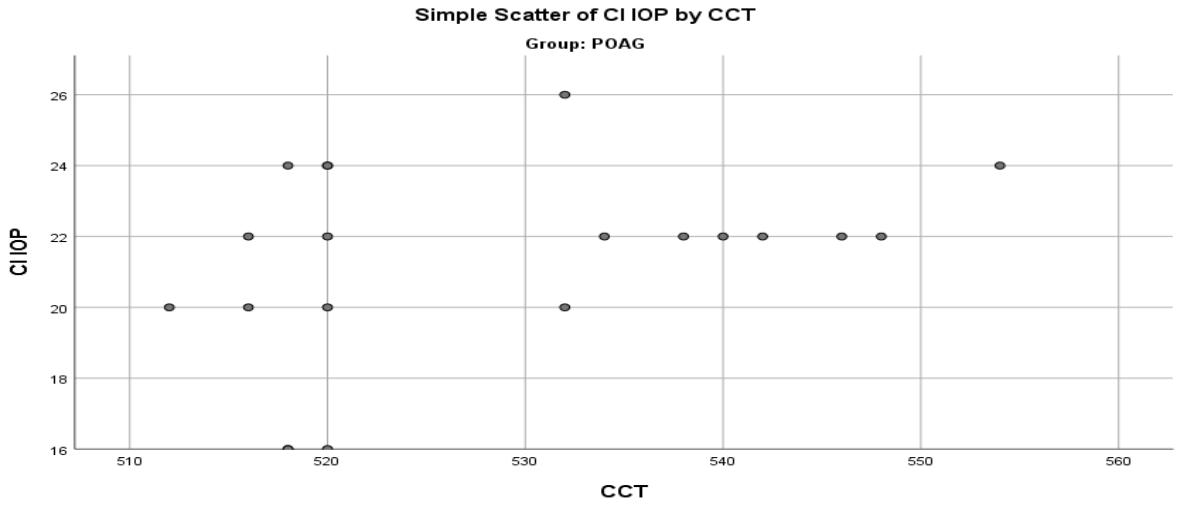
Significant positive correlation was noted between IOP based on GAT and the CCT among POAG patients.

IOP's (mm Hg)	Spearman correlation coefficient	p value
GAT	0.605	0.005
CI	0.386	0.093
Difference IOP	-0.575	0.008

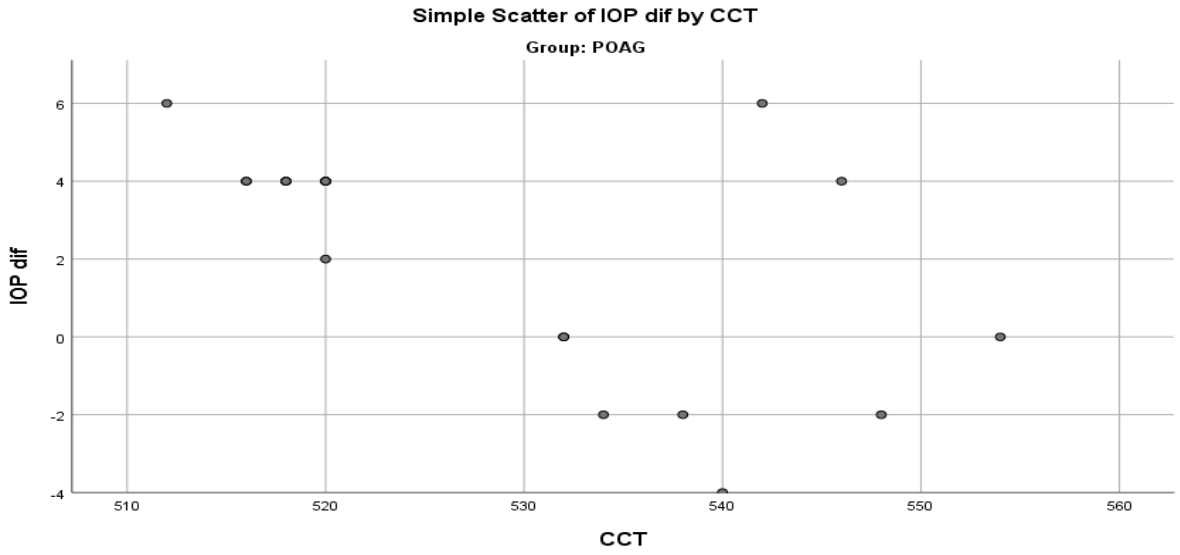
Table 12: Correlation between the CCT and various IOP's among POAG group



Graph 3: Scatter diagram of GAT IOP and CCT among POAG group



Graph 4: Scatter diagram of CI IOP and CCT among POAG group



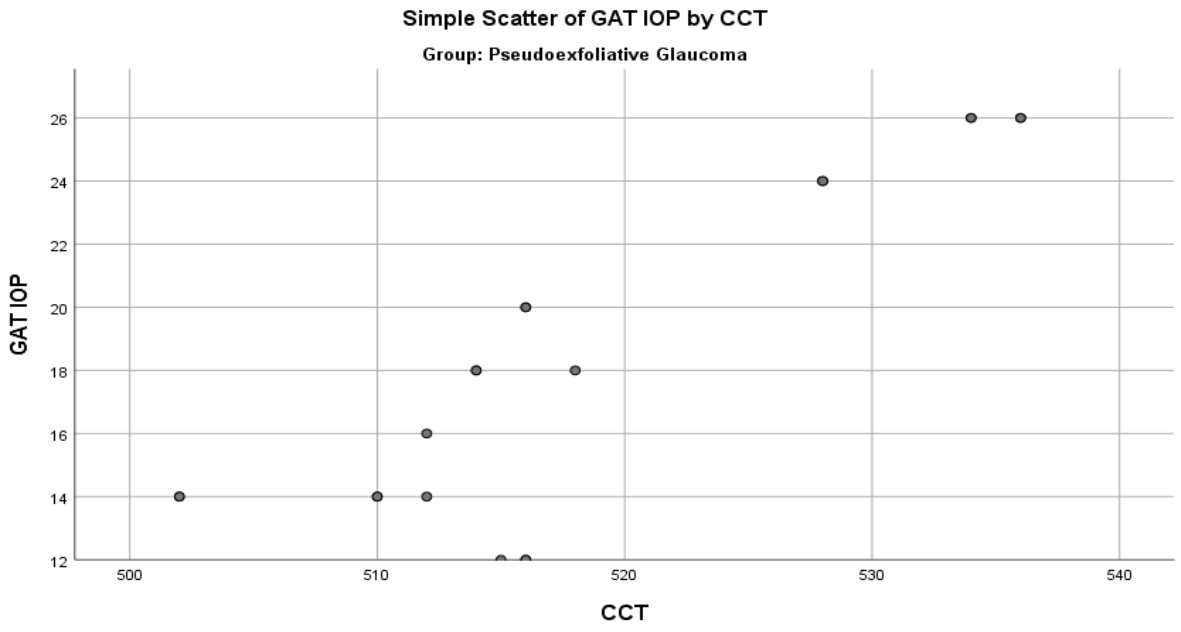
Graph 5: Scatter diagram of difference in IOP and CCT among POAG group

Correlation between the CCT and various IOP's among Pseudoexfoliative Glaucoma

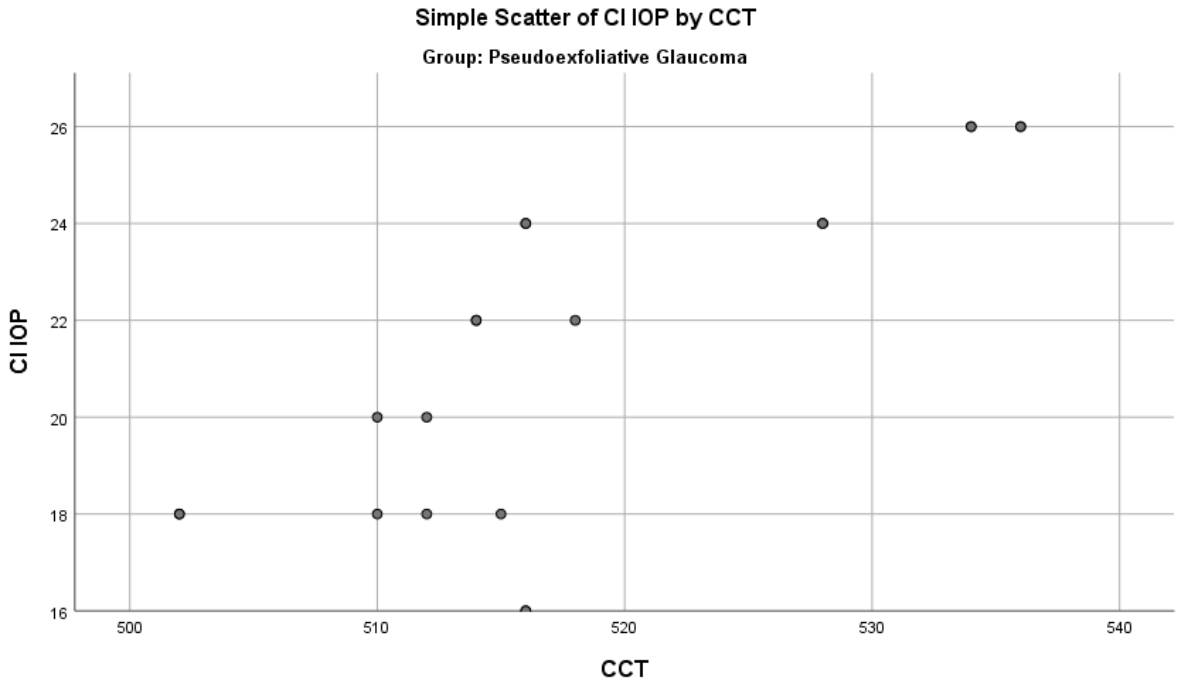
Significant positive correlation was noted between IOP and the CCT among Pseudoexfoliative Glaucoma patients.

Table 13: Correlation between the CCT and various IOP's among PXG group

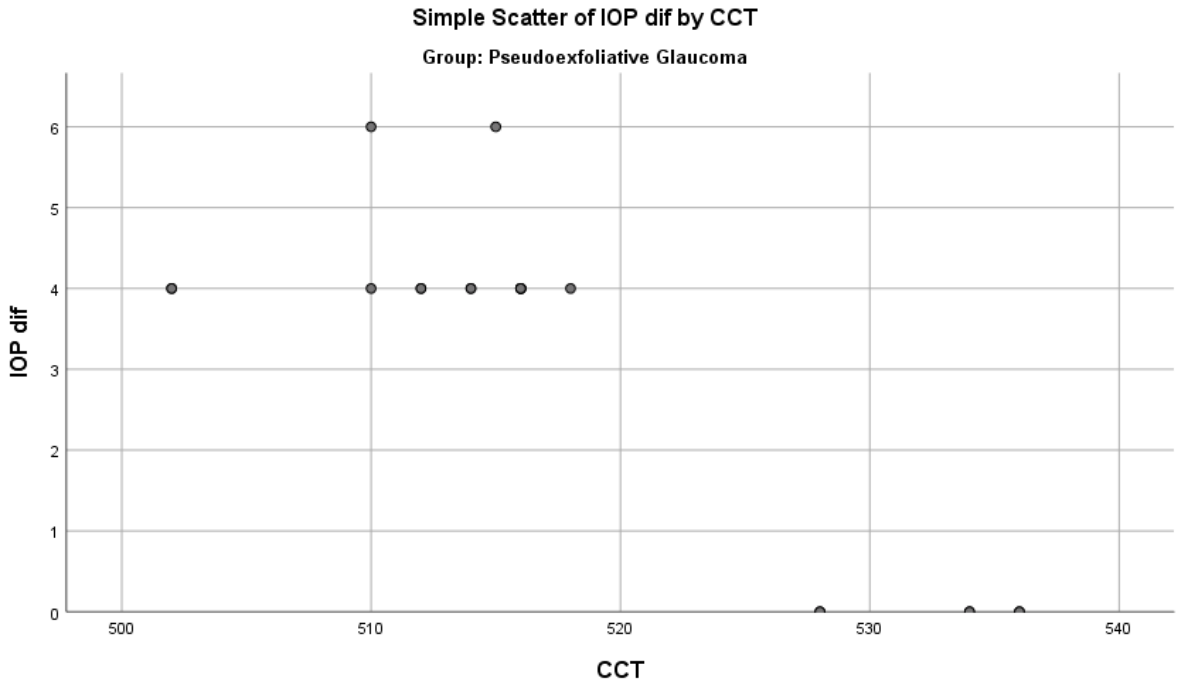
IOP's (mm Hg)	Spearman correlation coefficient	p value
GAT	0.738	<0.001
CI	0.742	<0.001
Difference IOP	-0.761	<0.001



Graph 6: Scatter diagram of GAT IOP and CCT among PXG group



Graph 7: Scatter diagram of CI IOP and CCT among PXG group



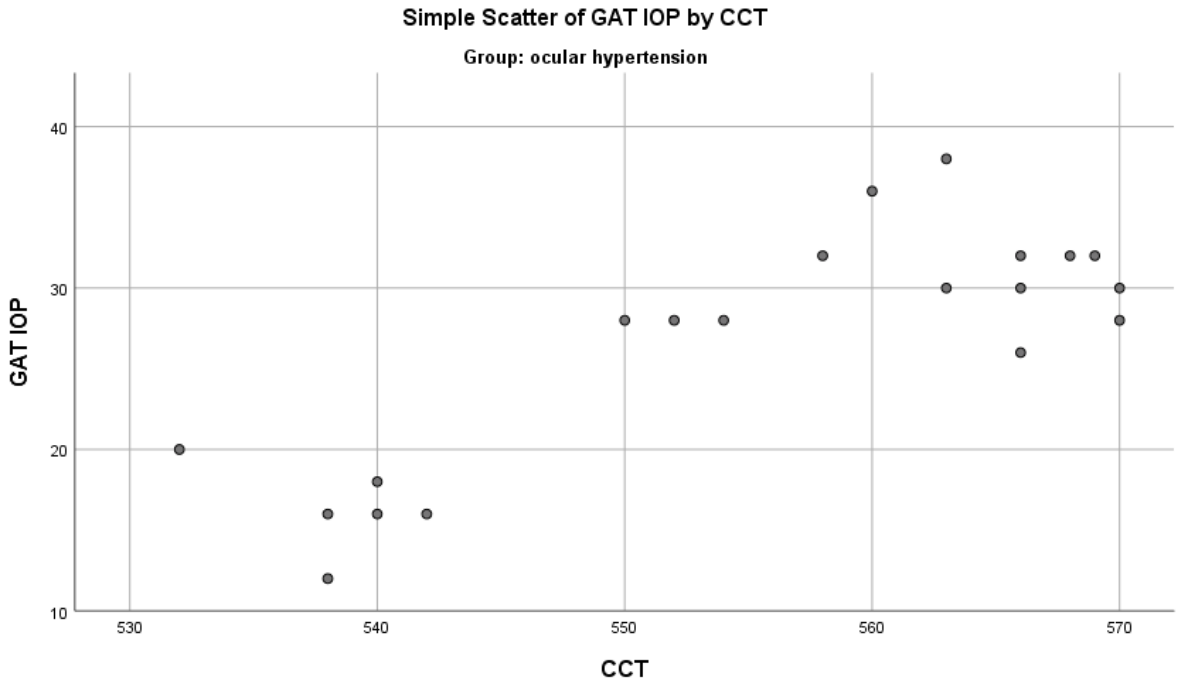
Graph 8: Scatter diagram of difference in IOP and CCT among PXG group

Correlation between the CCT and various IOPs among ocular hypertension

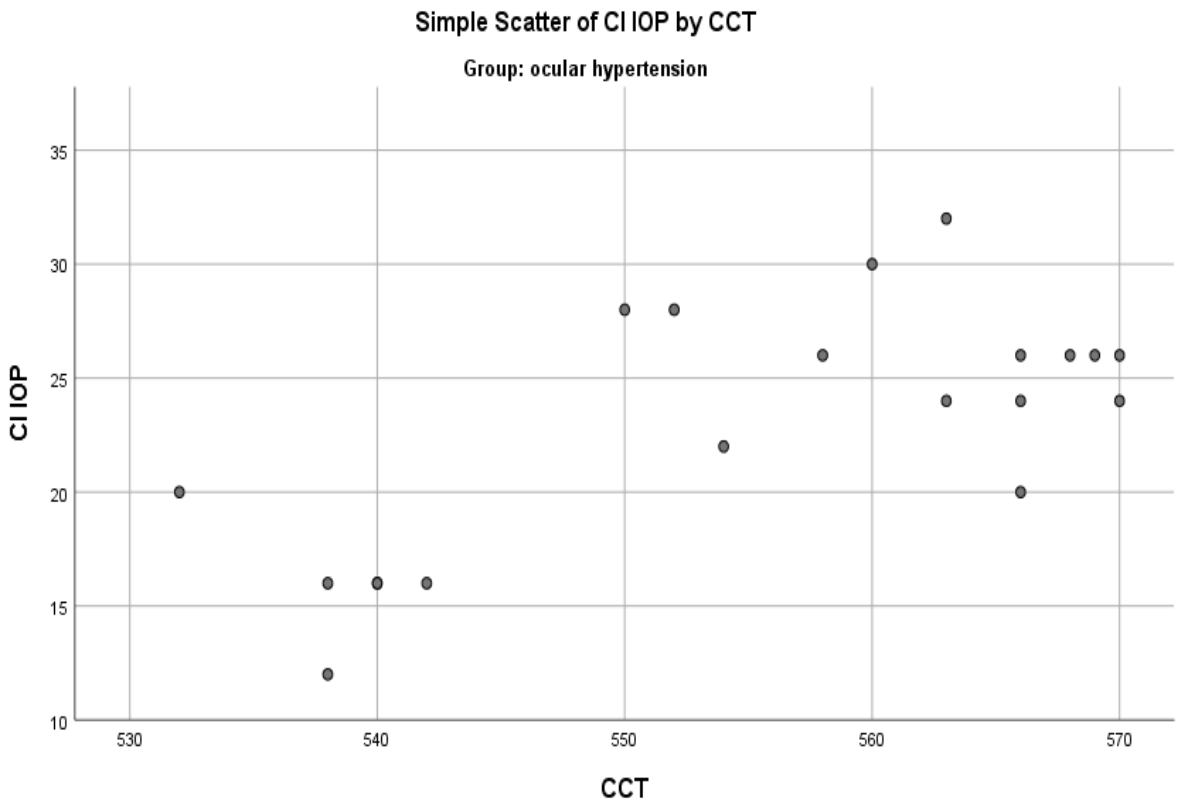
Significantly positive correlation was noted between IOP and the CCT among ocular hypertension patients.

IOP's (mm Hg)	Spearman correlation coefficient	p value
GAT	0.676	0.001
CI	0.519	0.019
Difference IOP	-0.750	<0.001

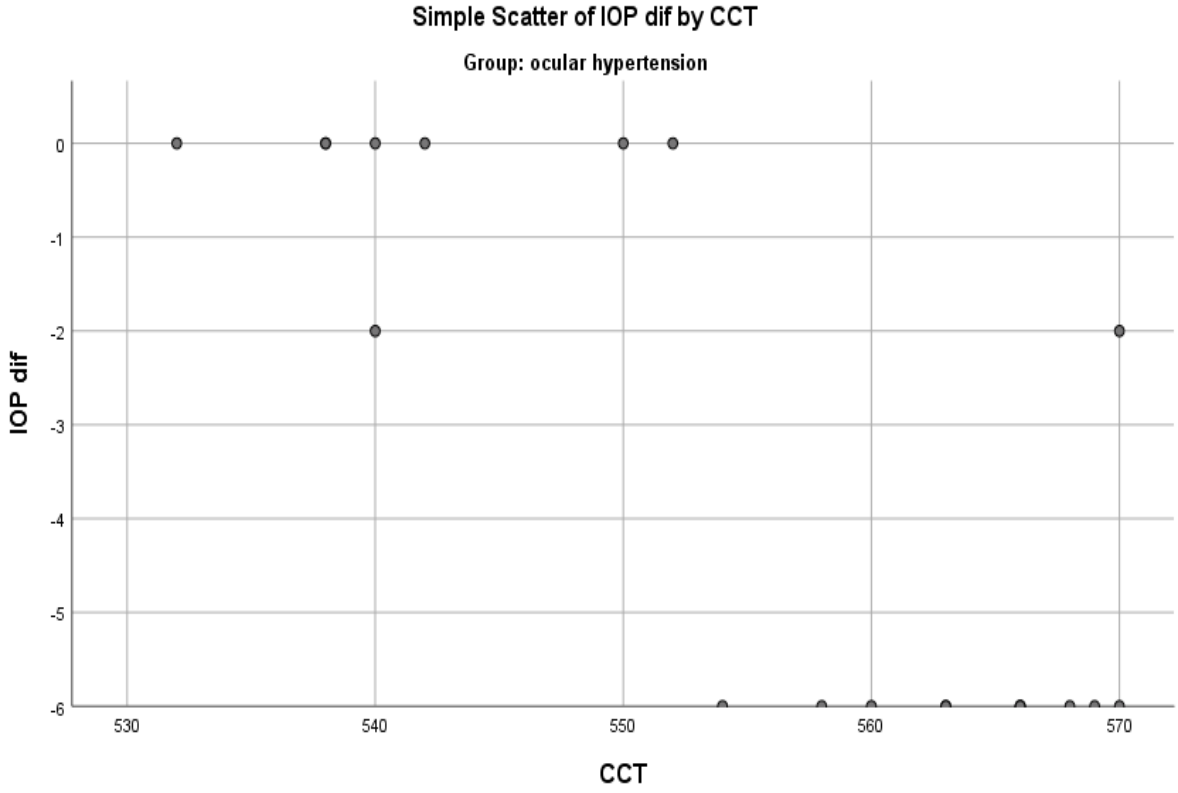
Table 14: Correlation between the CCT and various IOP's among OHT group



Graph 9: Scatter diagram of GAT IOP and CCT among OHT group



Graph 10: Scatter diagram of CI IOP and CCT among OHT group



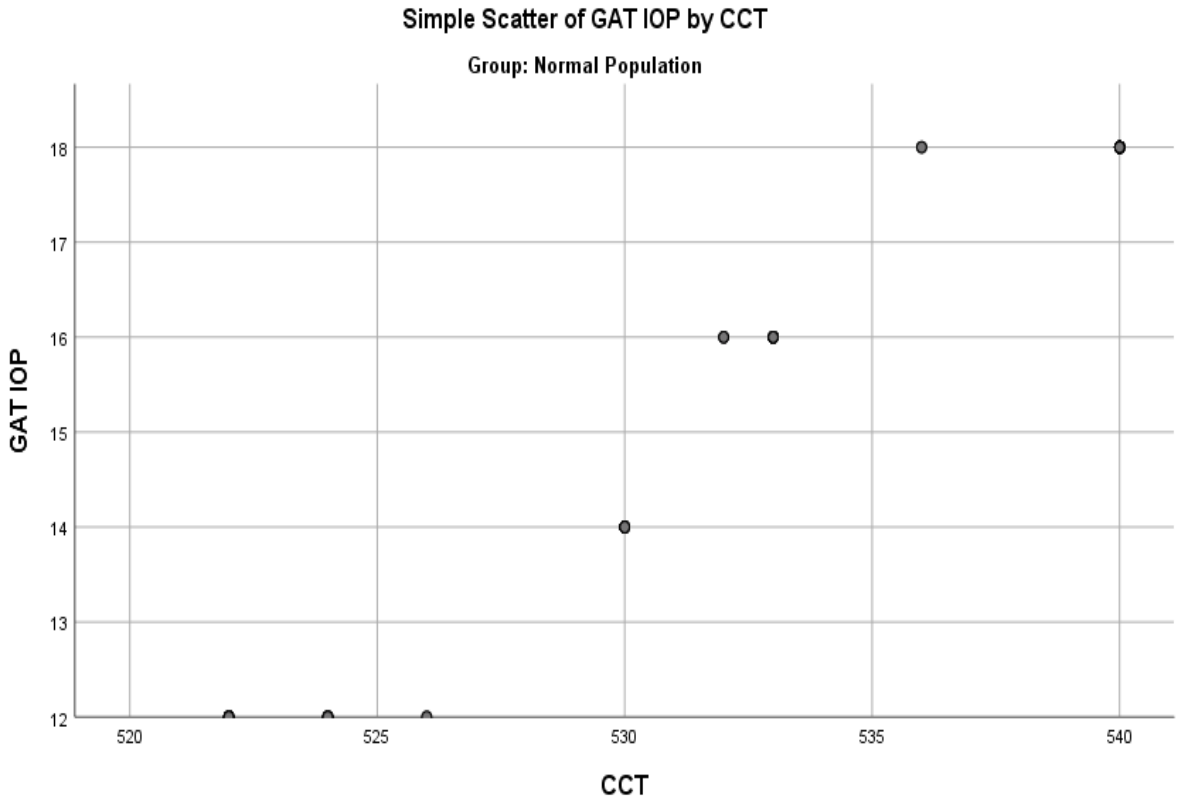
Graph 11: Scatter diagram of difference in IOP and CCT among OHT group

Correlation between the CCT and various IOPs among normal population

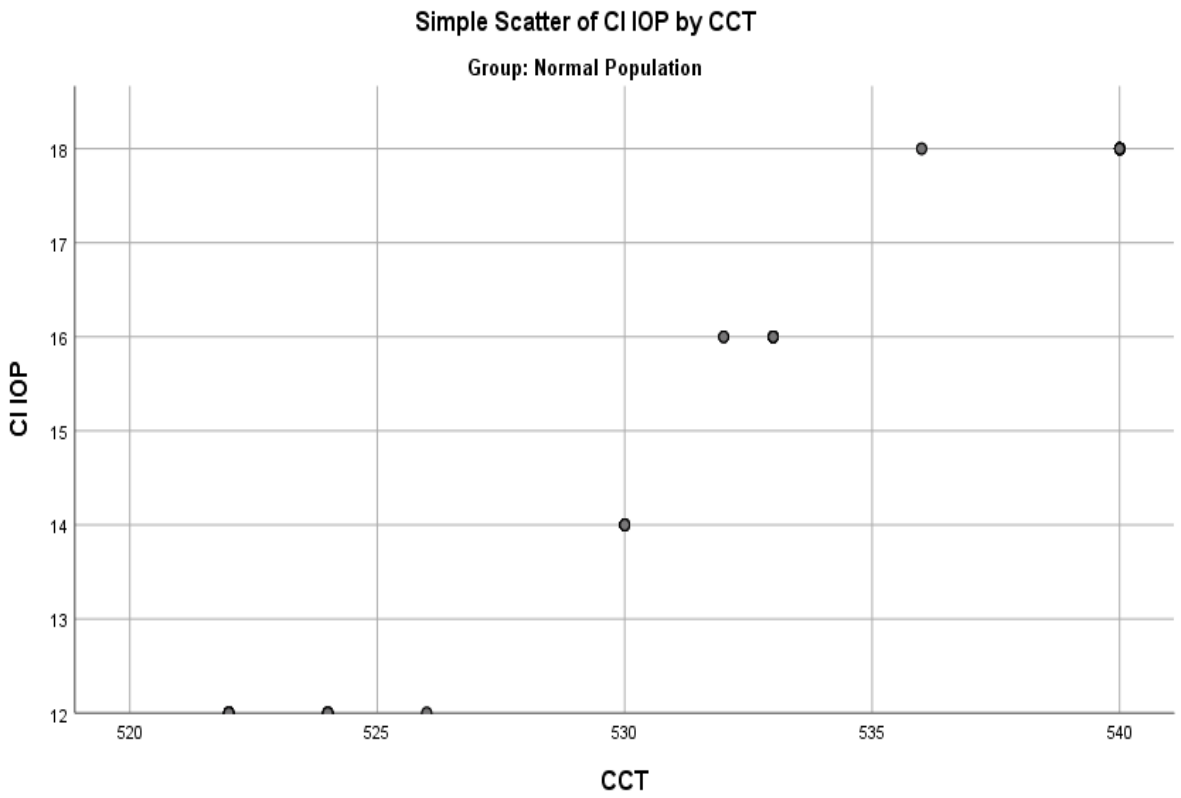
Significantly positive correlation was seen between IOP and the CCT among normal population.

IOP's (mm Hg)	Spearman correlation coefficient	p value
GAT	0.976	<0.001
CI	0.976	<0.001
Difference IOP	-	-

Table 15: Correlation between the CCT and various IOP's among Normal population group



Graph 12: Scatter diagram of GAT IOP and CCT among Normal population group



Graph 13: Scatter diagram of CI IOP and CCT among normal population group

Comparison of CCT & IOP between study groups and normal population

CCT was significantly lower among the POAG and Pseudoexfoliative Glaucoma patients than the normal population, while it was higher among the ocular hypertension patients than normal population.

Significantly low CCT was noted among PXG group than POAG patients and ocular hypertension patients.

Significantly low CCT was noted among the POAG patients than the ocular hypertension patients.

IOP between groups:

IOP was significantly higher among the POAG group, Pseudoexfoliative Glaucoma and ocular hypertension patients than the normal population.

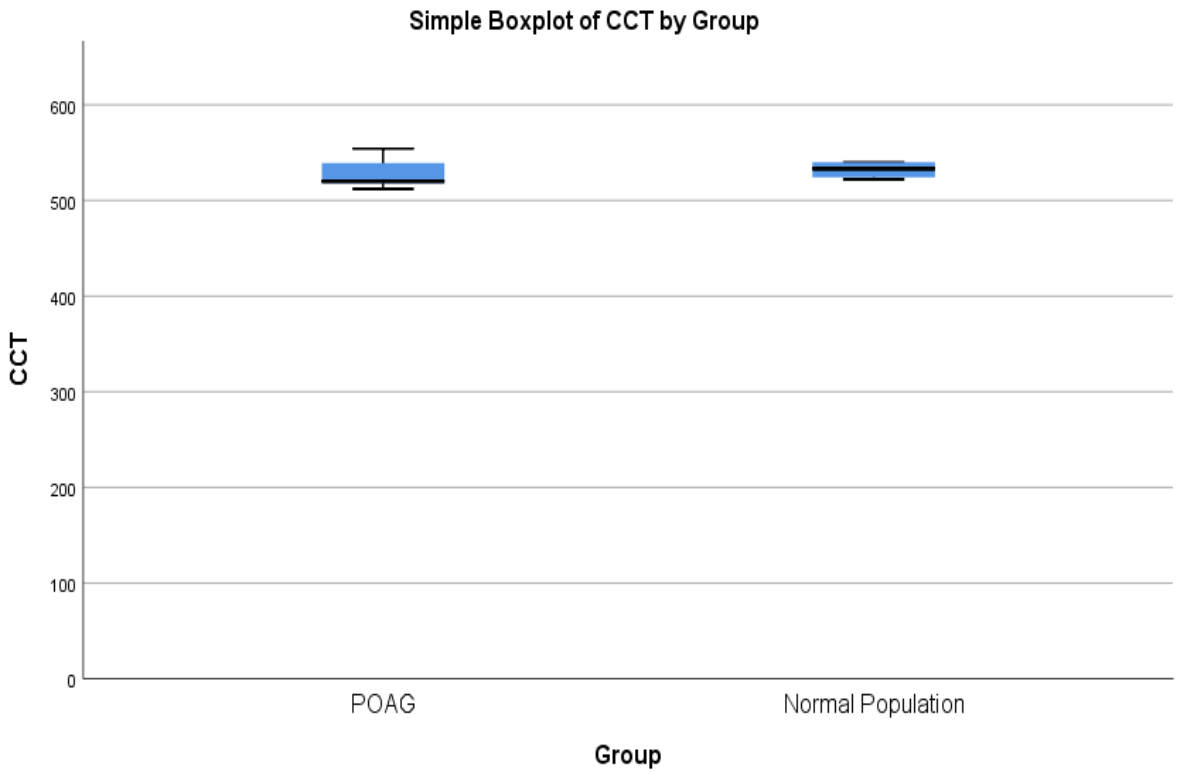
Significantly higher IOP was reported among ocular hypertension patients than POAG and

Pseudoexfoliative Glaucoma

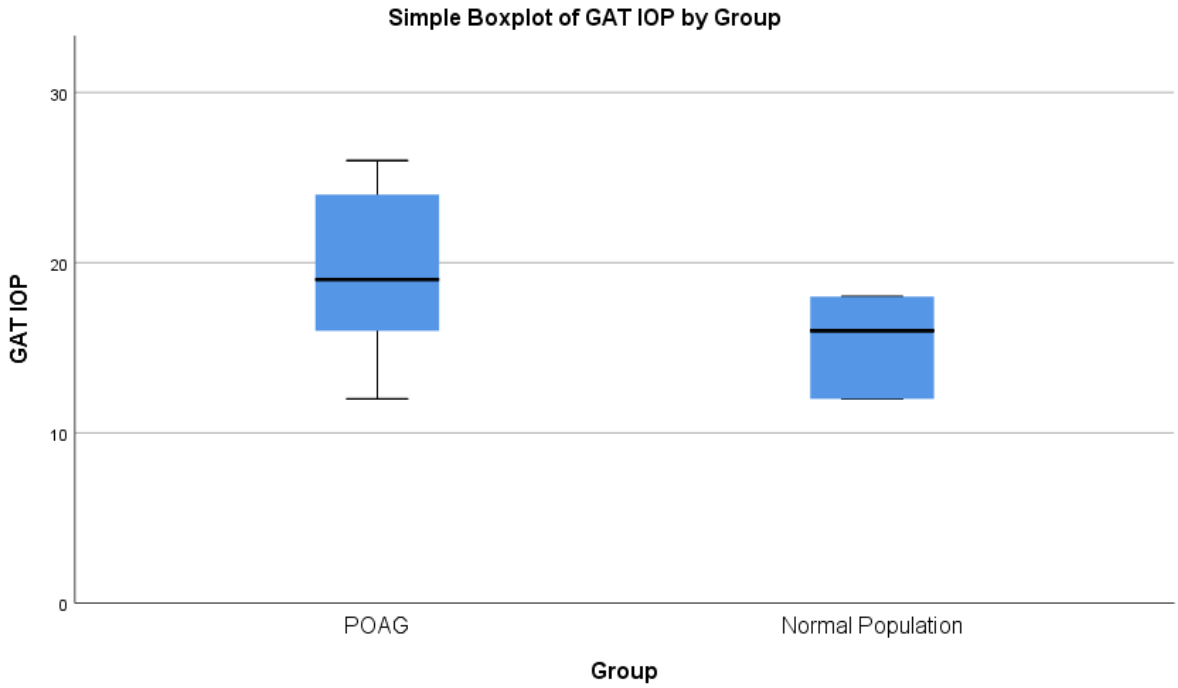
No significant difference in IOP was noted among POAG and Pseudoexfoliative Glaucoma.

Parameter	**Group**	**N**	**Mean Rank**	**p value**
CCT (µm)	POAG	20	31.65	0.047
	Normal Population	60	43.45	
	Total	80		
GAT IOP (mm Hg)	POAG	20	56.15	<0.001
	Normal Population	60	31.90	
	Total	80		
CI IOP (mm Hg)	POAG	20	66.30	<0.001
	Normal Population	60	31.90	
	Total	80		
Difference IOP (mm Hg)	POAG	20	54.00	<0.001
	Normal Population	60	36.00	
	Total	80		

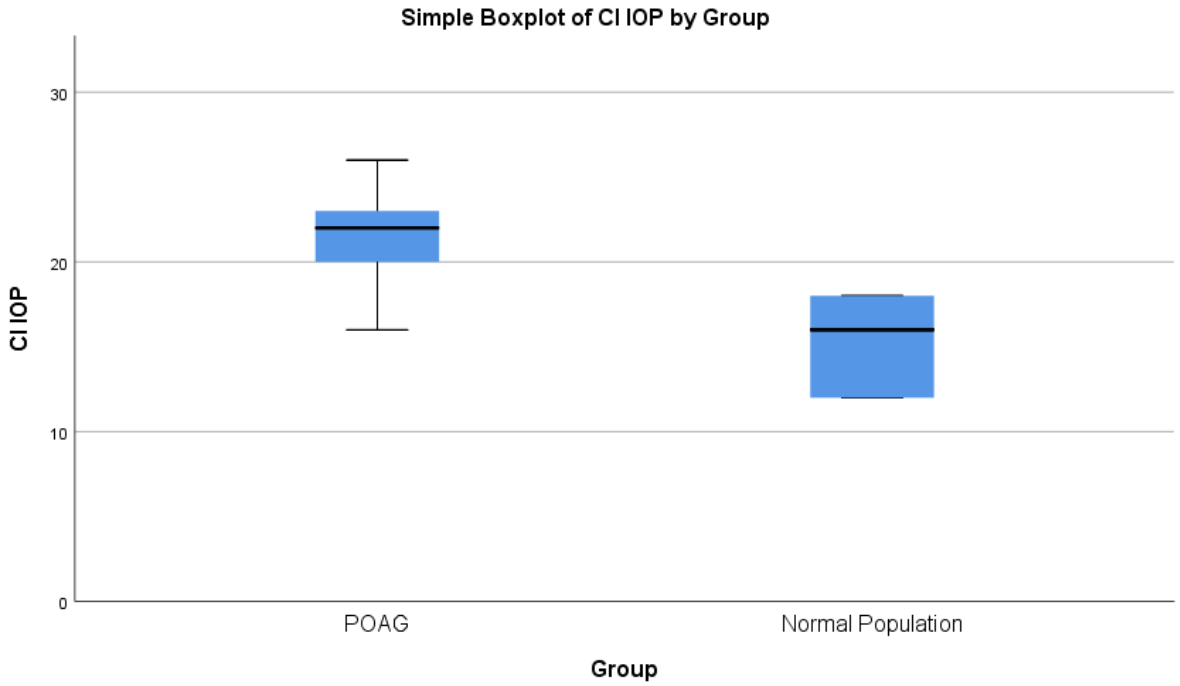
Table 16: Comparison of CCT, IOP among POAG group and normal population



Graph 14: Boxplot showing comparison of CCT among POAG group and Normal population



Graph 15: Boxplot showing comparison of GAT IOP among POAG group and Normal population

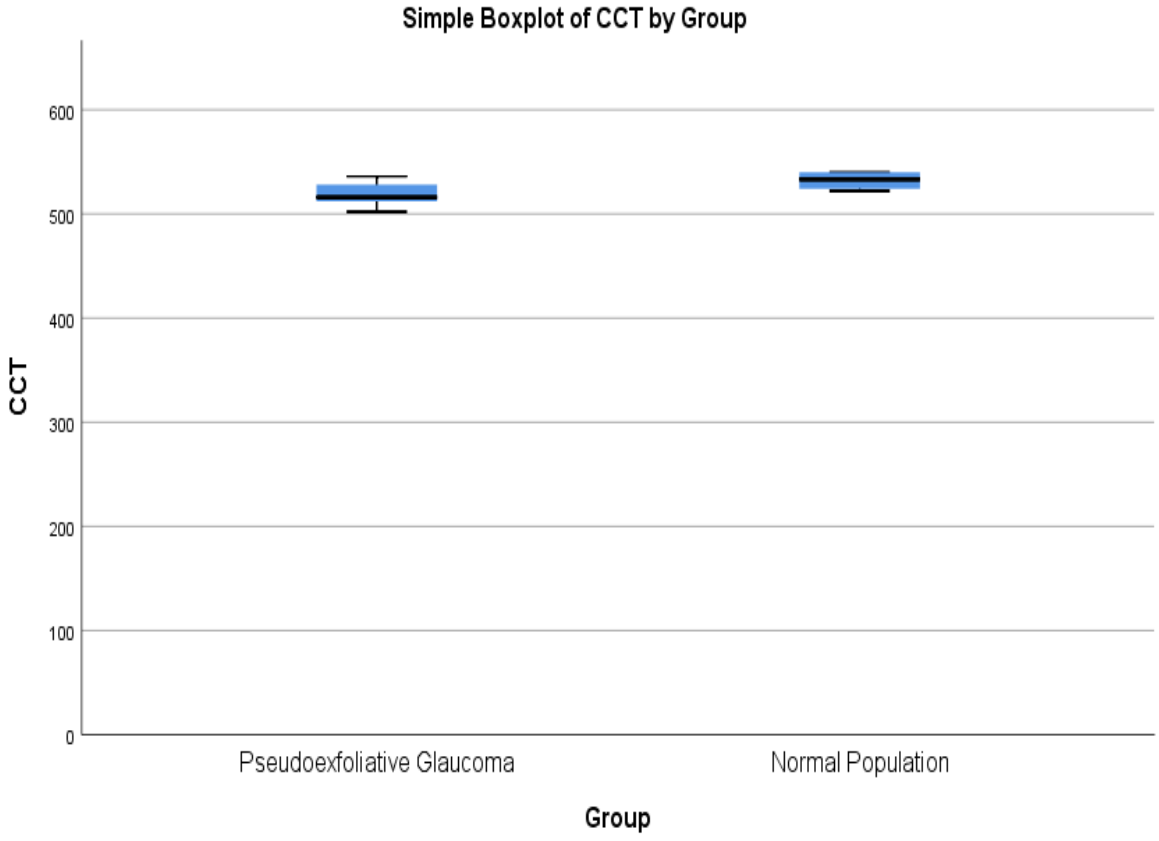


Graph 16: Boxplot showing comparison of CI IOP among POAG group and Normal population

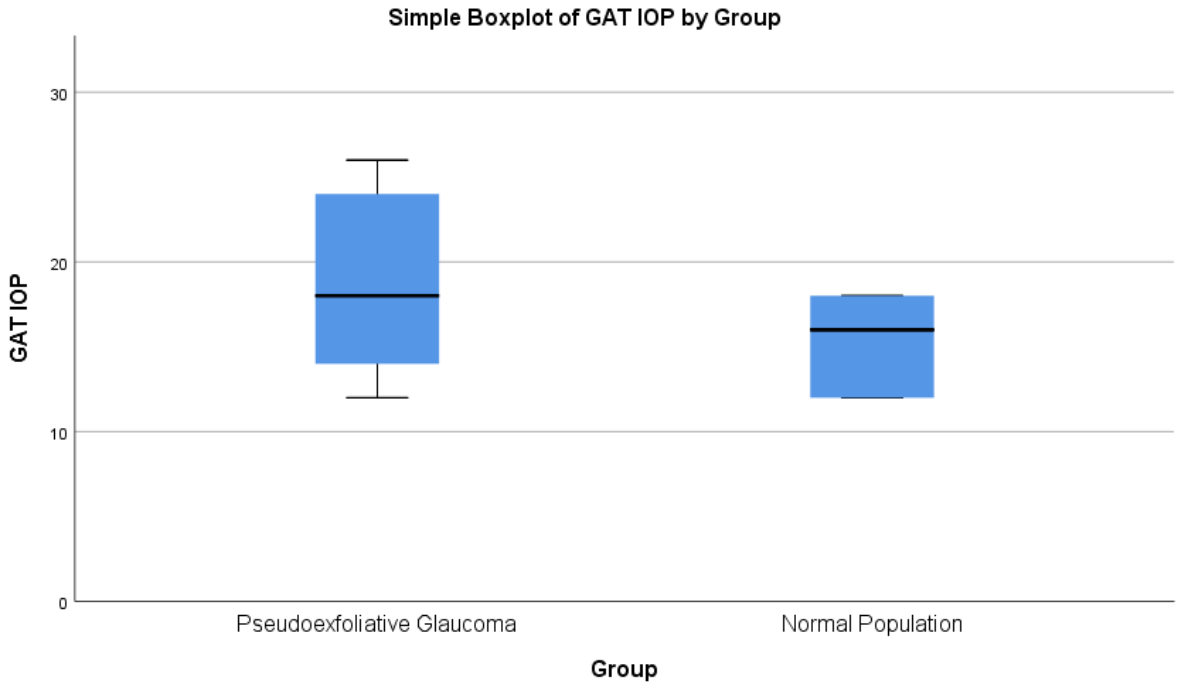
Parameter	**Group**	**N**	**Mean Rank**	**p value**
CCT (µm)	Pseudoexfoliative Glaucoma	20	20.20	<0.001
	Normal Population	60	47.27	
	Total	80		
GAT IOP (mm Hg)	Pseudoexfoliative Glaucoma	20	50.70	0.019
	Normal Population	60	37.10	
	Total	80		
CI IOP (mm Hg)	Pseudoexfoliative Glaucoma	20	64.95	<0.001
	Normal Population	60	32.35	

	Total	80		
Difference IOP (mm Hg)	Pseudoexfoliative Glaucoma	20	61.50	<0.001
	Normal Population	60	33.50	
	Total	80		

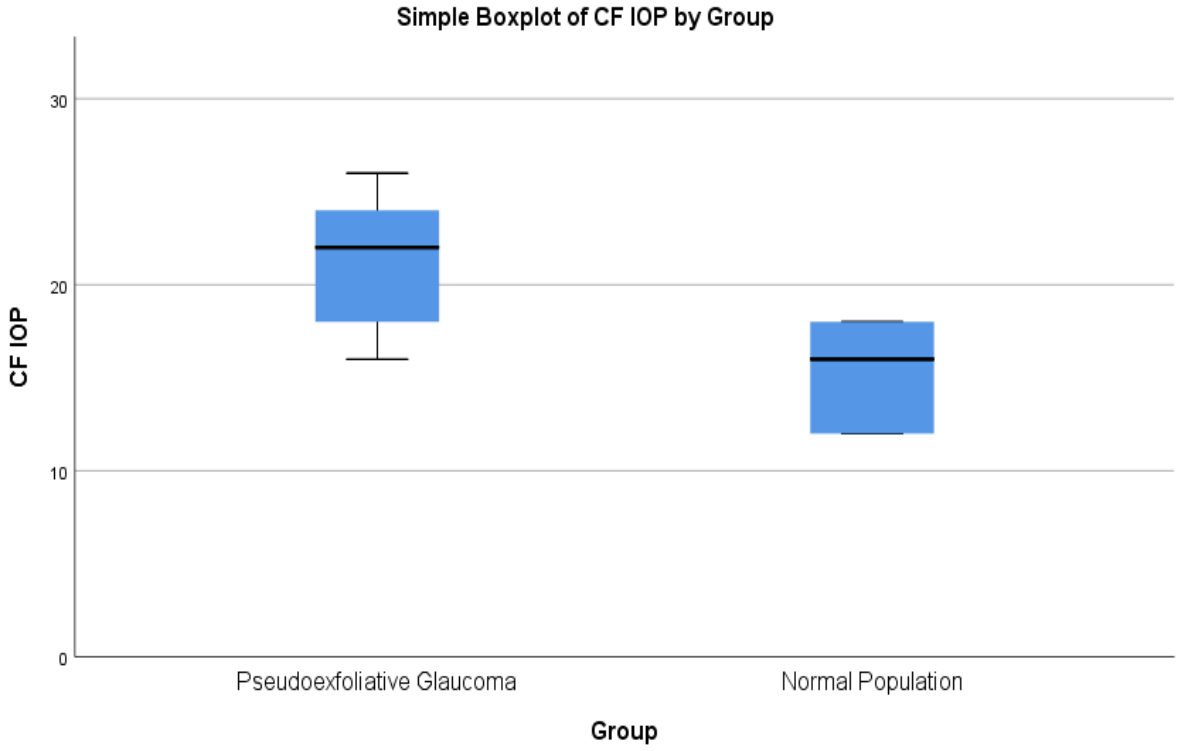
Table 17: Comparison of CCT & IOP between Pseudoexfoliative Glaucoma and normal population



Graph 17: Boxplot showing comparison of CCT among PXG group and Normal population



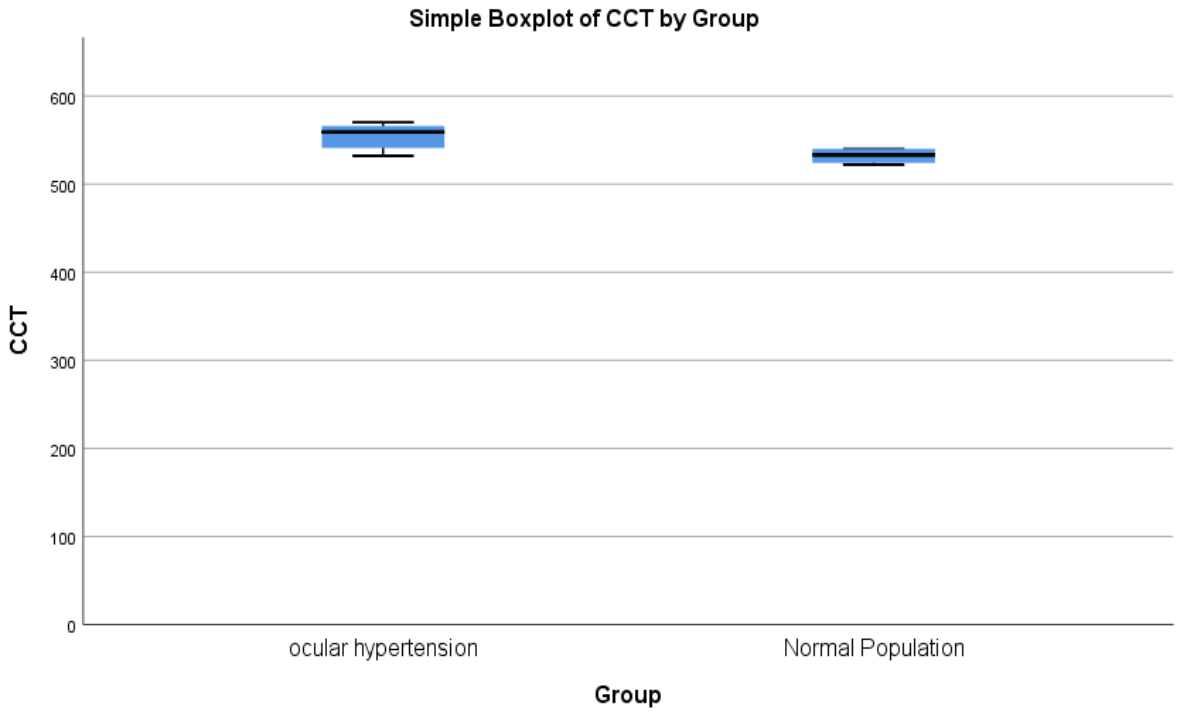
Graph 18: Boxplot showing comparison of GAT IOP among PXG group and Normal population



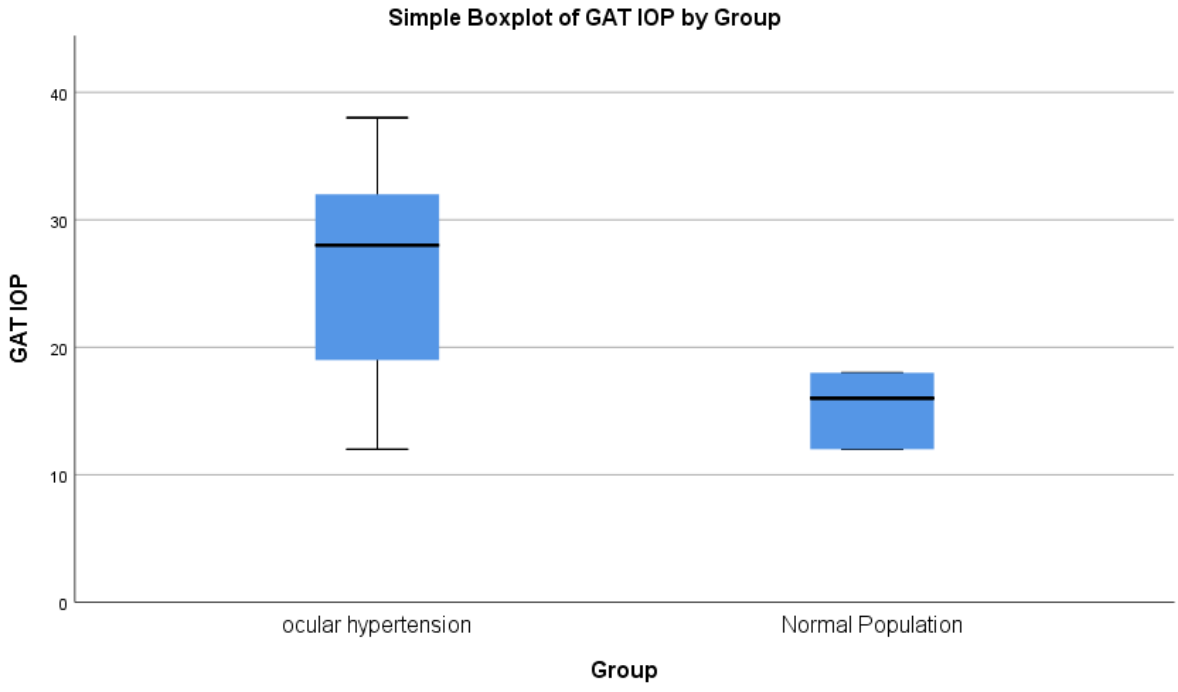
Graph 19: Boxplot showing comparison of CI IOP among PXG group and Normal population

Parameter	Group	N	Mean Rank	p value
CCT (µm)	ocular hypertension	20	65.88	<0.001
	Normal Population	60	32.04	
	Total	80		
GAT IOP (mm Hg)	ocular hypertension	20	63.25	<0.001
	Normal Population	60	32.92	
	Total	80		
CI IOP (mm Hg)	ocular hypertension	20	62.40	<0.001
	Normal Population	60	33.20	
	Total	80		
Difference IOP (mm Hg)	ocular hypertension	20	21.00	<0.001
	Normal Population	60	47.00	
	Total	80		

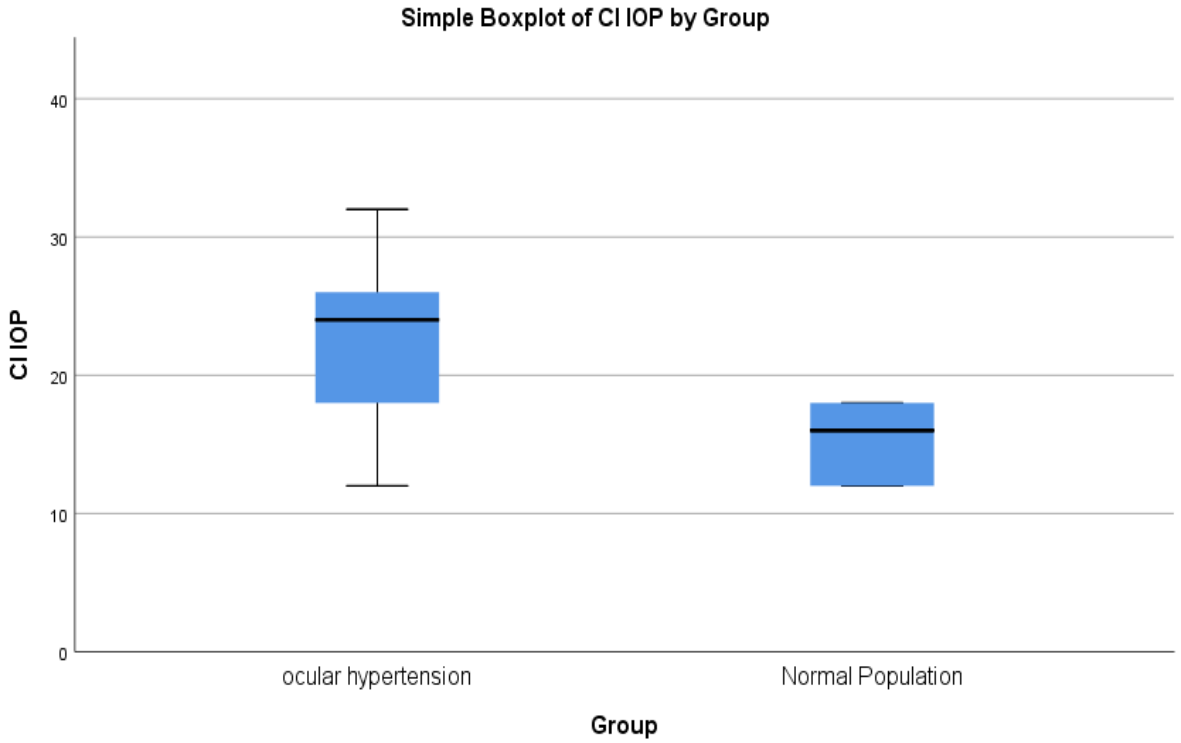
Table 18: Comparison of CCT & IOP between ocular hypertension and normal population



Graph 20: Boxplot showing comparison of CCT among OHT group and Normal population



Graph 21: Boxplot showing comparison of GAT IOP among OHT group and Normal population

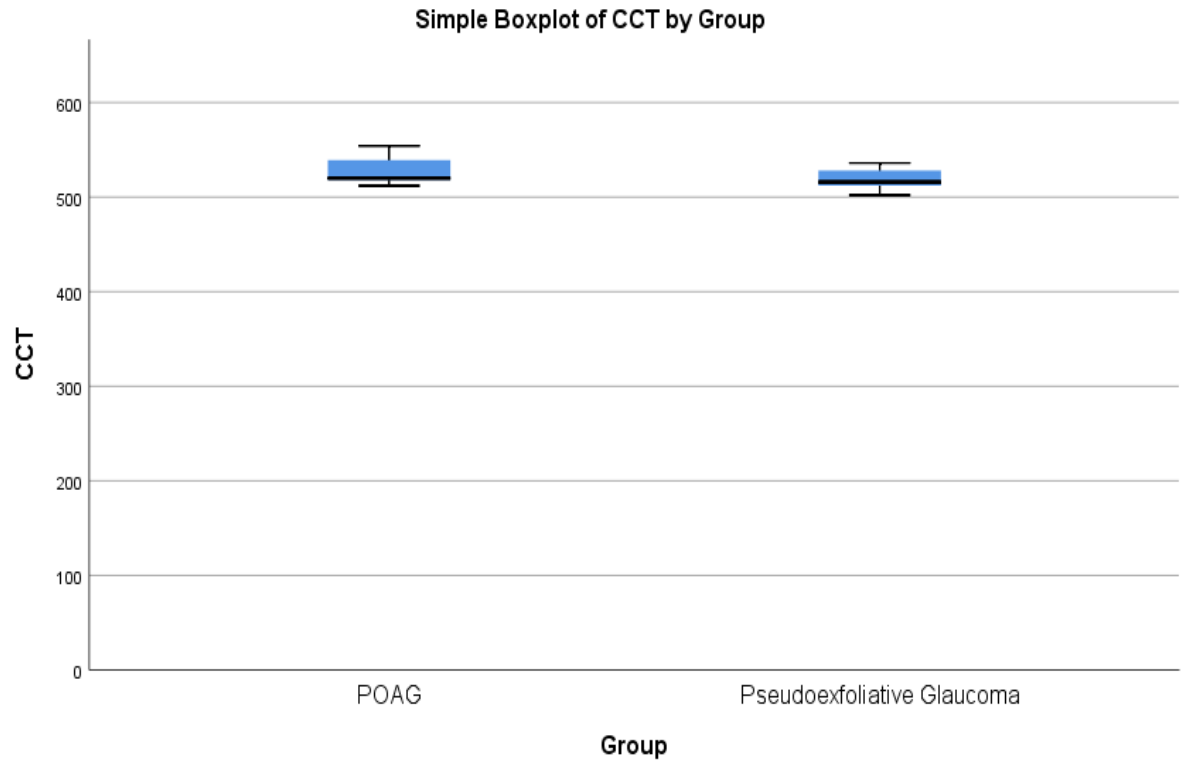


Graph 22: Boxplot showing comparison of CI IOP among OHT group and Normal population

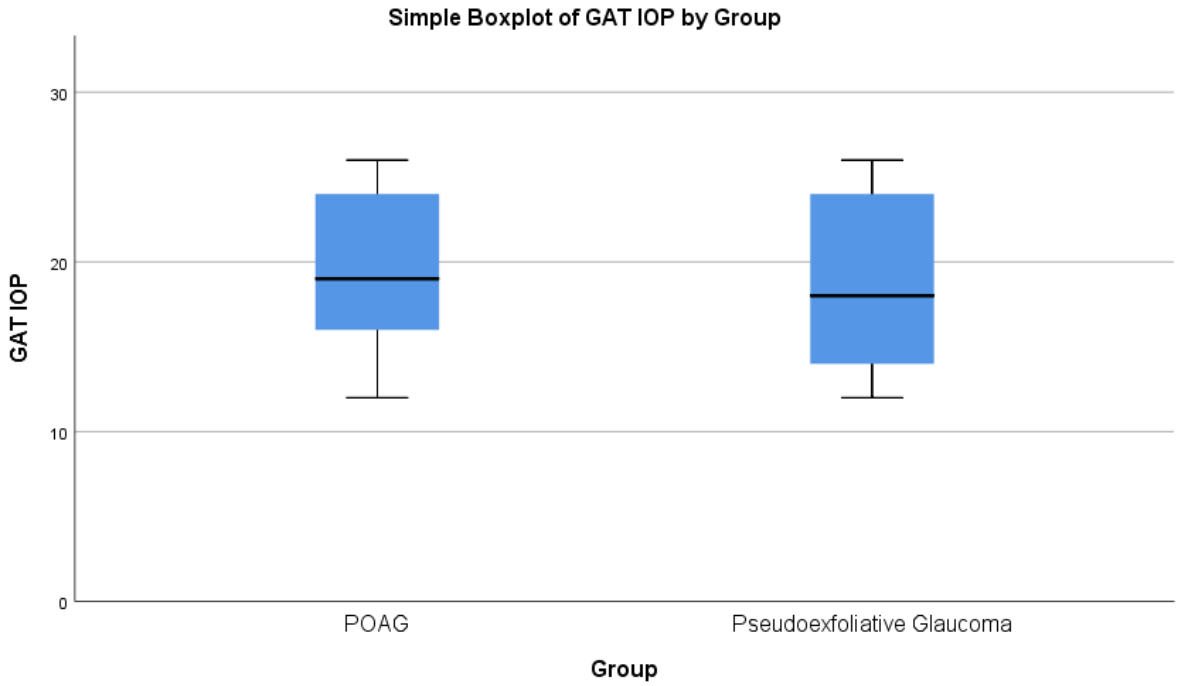
Parameter	**Group**	**N**	**Mean Rank**	**p value**
CCT (µm)	POAG	20	25.83	0.003
	Pseudoexfoliative Glaucoma	20	15.18	
	Total	40		
GAT IOP (mm Hg)	POAG	20	21.35	0.659
	Pseudoexfoliative Glaucoma	20	19.65	
	Total	40		

CI IOP (mm Hg)	POAG	20	20.15	0.862
	Pseudoexfoliative Glaucoma	20	20.85	
	Total	40		
Difference IOP (mm Hg)	POAG	20	19.15	0.478
	Pseudoexfoliative Glaucoma	20	21.85	
	Total	40		

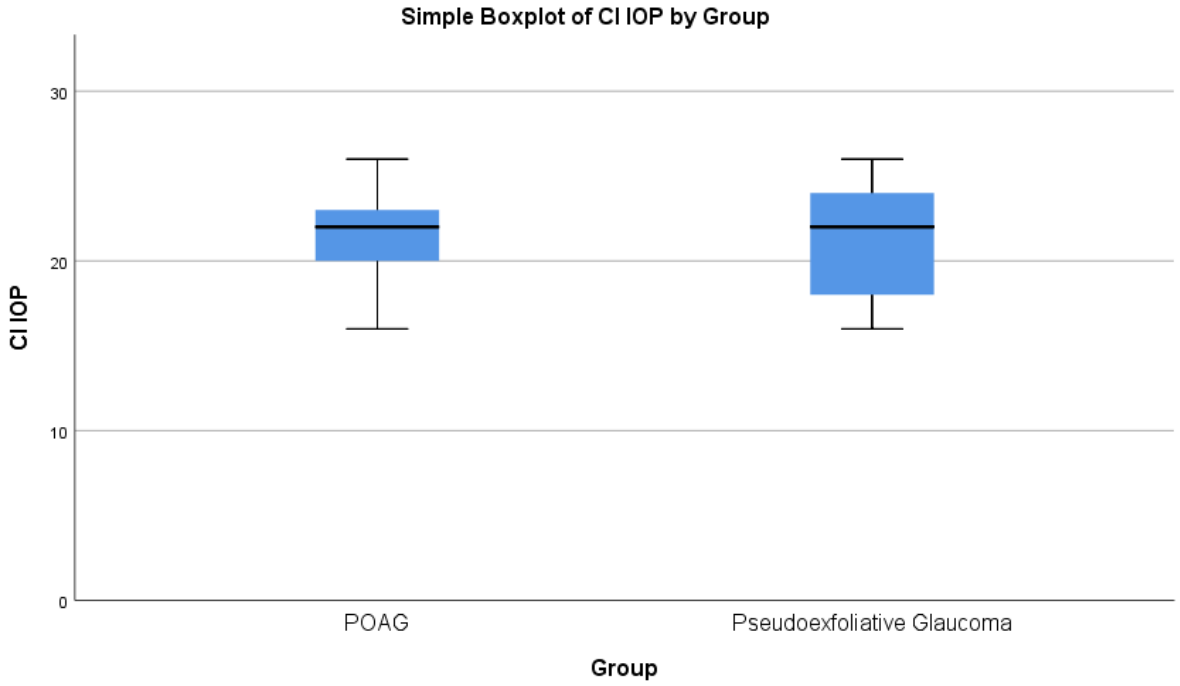
Table 19: Comparison of CCT & IOP between POAG and Pseudoexfoliative Glaucoma



Graph 23: Boxplot showing comparison of CCT among POAG group and PXG



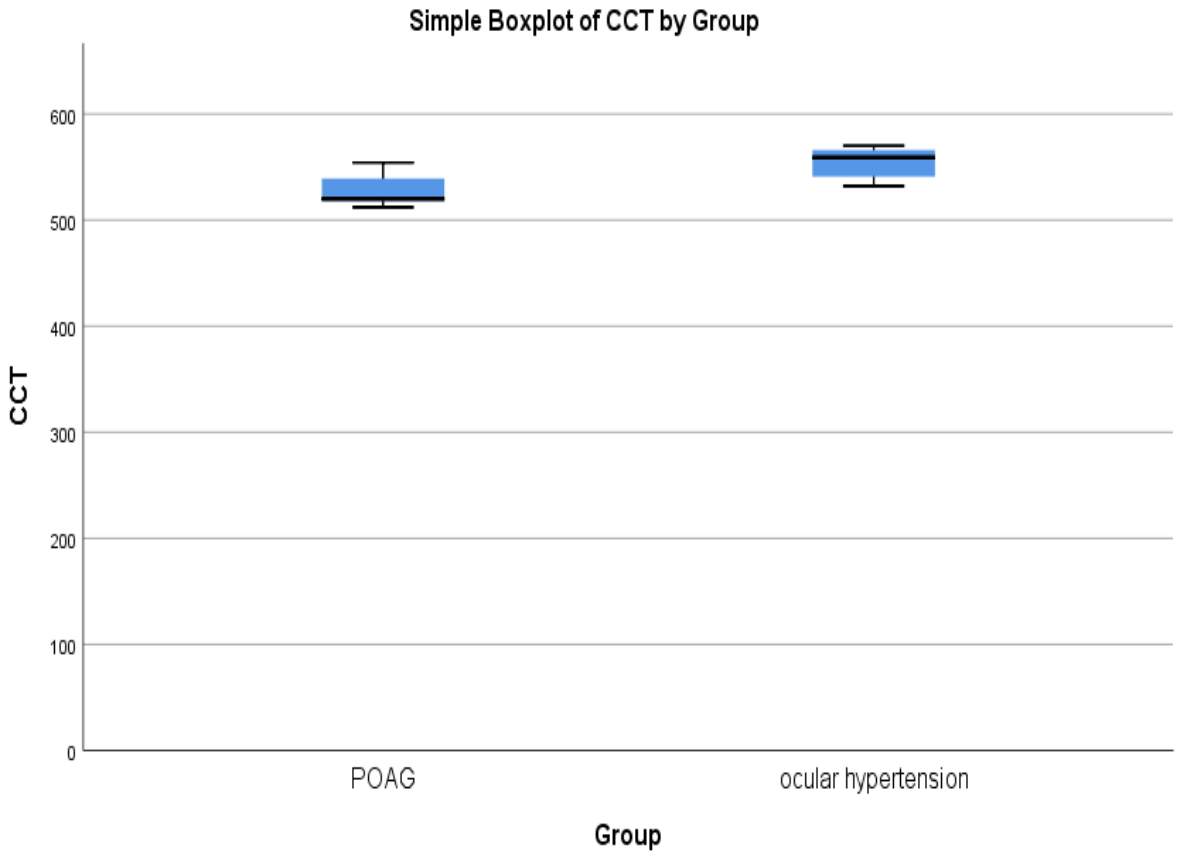
Graph 24: Boxplot showing comparison of GAT IOP among POAG group and PXG



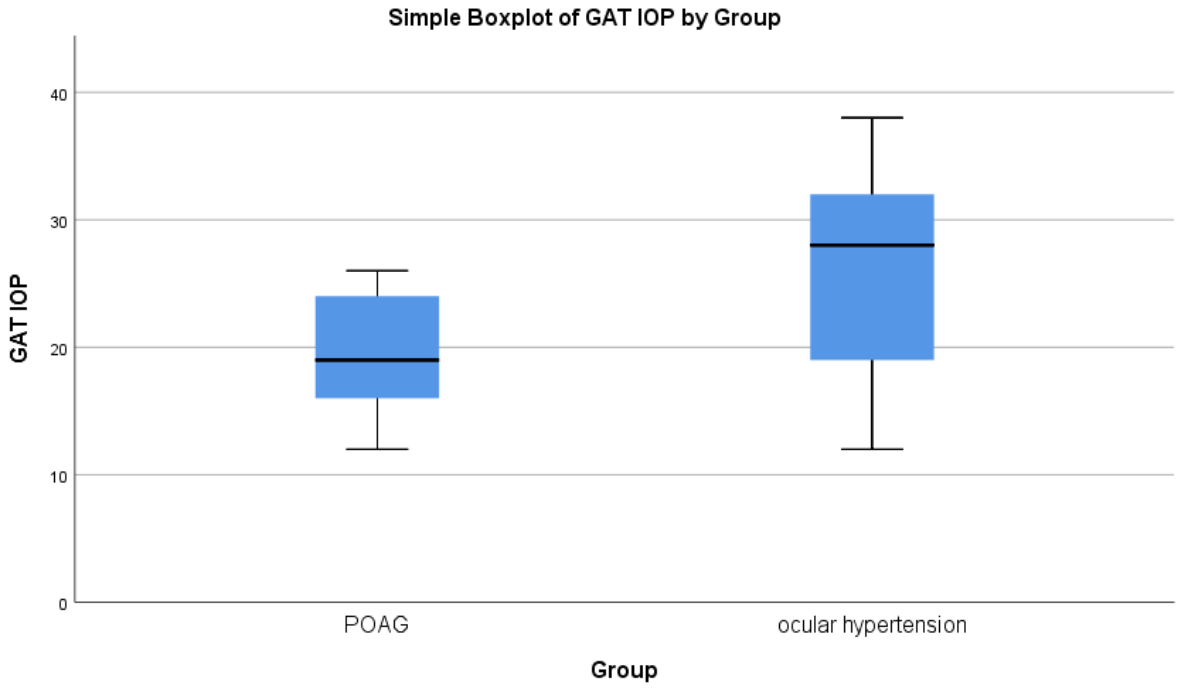
Graph 25: Boxplot showing comparison of CI IOP among POAG group and PXG

Parameter	Group	N	Mean Rank	p value
CCT (µm)	POAG	20	12.20	<0.001
	ocular hypertension	20	28.80	
	Total	40		
GAT IOP (mm Hg)	POAG	20	14.73	0.001
	ocular hypertension	20	26.28	
	Total	40		
CI IOP (mm Hg)	POAG	20	17.73	0.134
	ocular hypertension	20	23.28	
	Total	40		
Difference IOP (mm Hg)	POAG	20	28.33	<0.001
	ocular hypertension	20	12.68	
	Total	40		

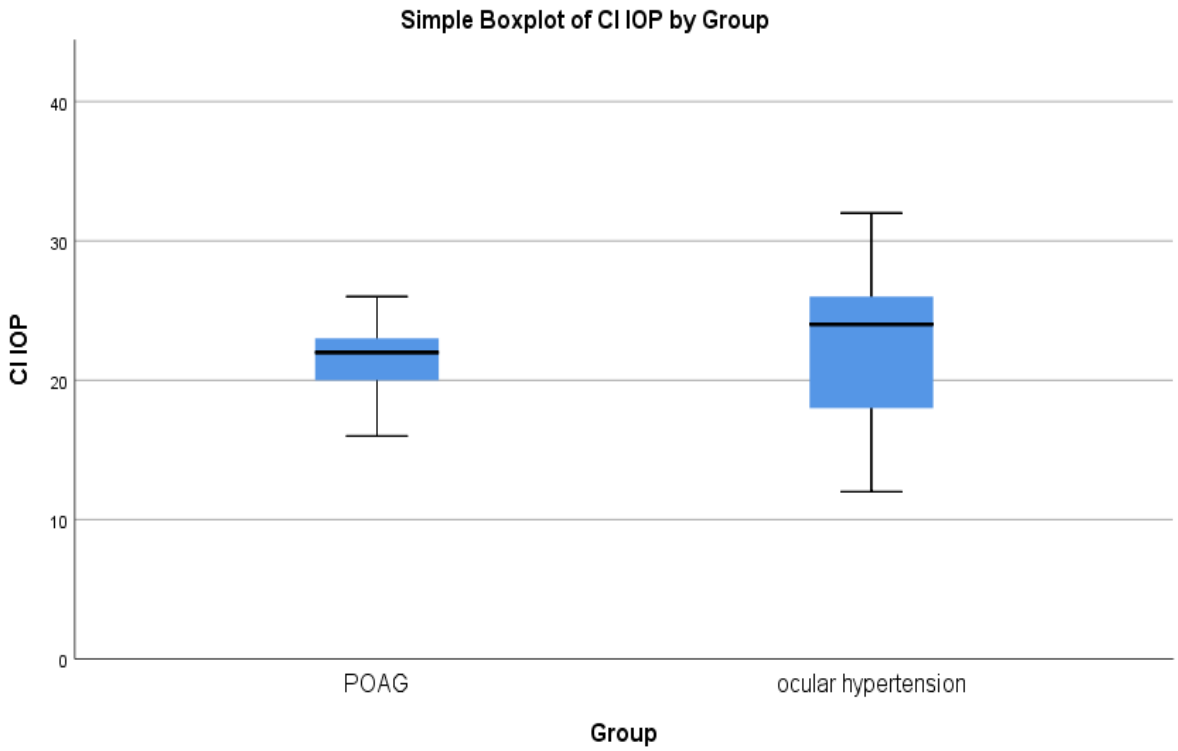
Table 20: Comparison of CCT & IOP between POAG and ocular hypertension



Graph 26: Boxplot showing comparison of GAT IOP among POAG group and OHT



Graph 27: Boxplot showing comparison of CCT among POAG group and OHT

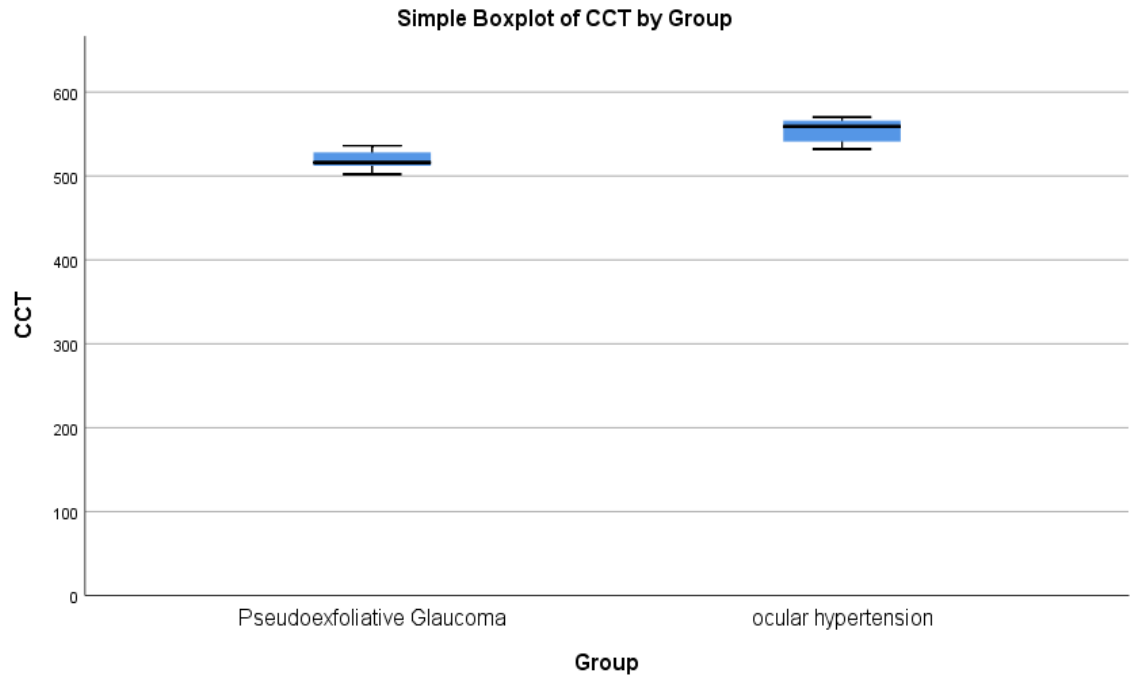


Graph 28: Boxplot showing comparison of CI IOP among POAG group and OHT

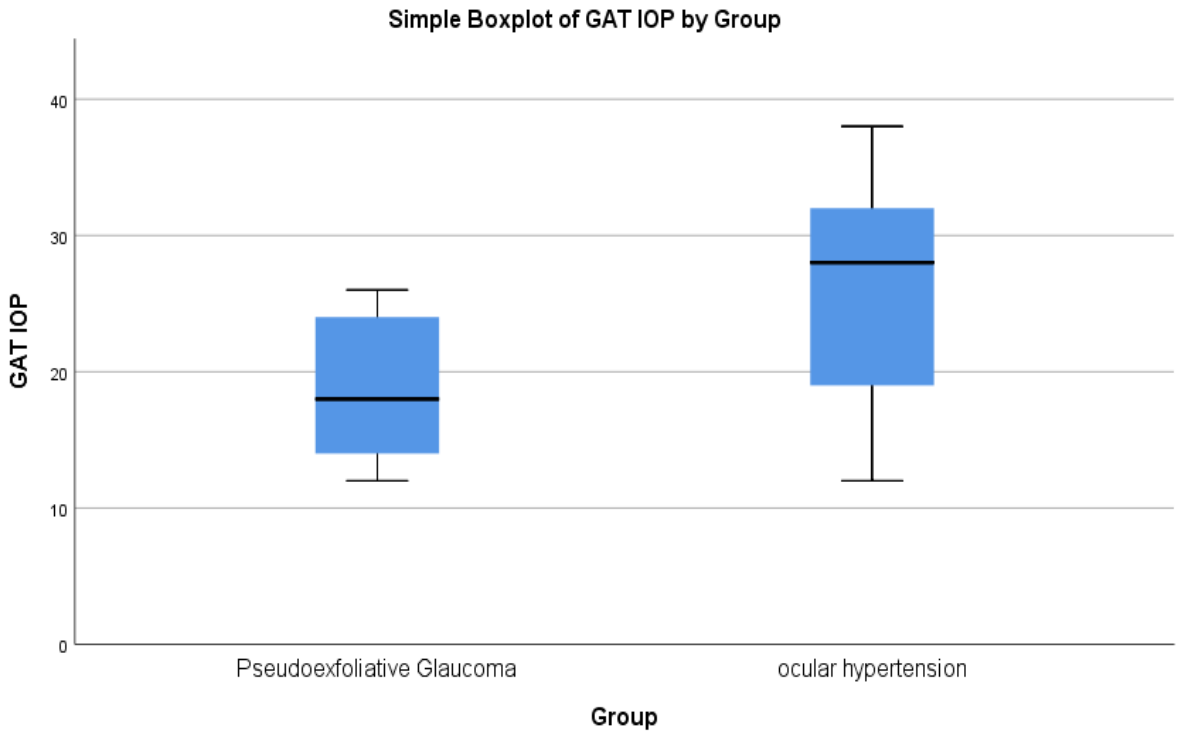
Parameter	**Group**	**N**	**Mean Rank**	**p value**
CCT (µm)	Pseudoexfoliative Glaucoma	20	10.70	<0.001
	ocular hypertension	20	30.30	
	Total	40		
GAT IOP (mm Hg)	Pseudoexfoliative Glaucoma	20	14.08	<0.001
	ocular hypertension	20	26.93	
	Total	40		
CI IOP (mm Hg)	Pseudoexfoliative Glaucoma	20	18.38	0.253

	ocular hypertension	20	22.63	
	Total	40		
Difference IOP (mm Hg)	Pseudoexfoliative Glaucoma	20	29.45	<0.001
	ocular hypertension	20	11.55	
	Total	40		

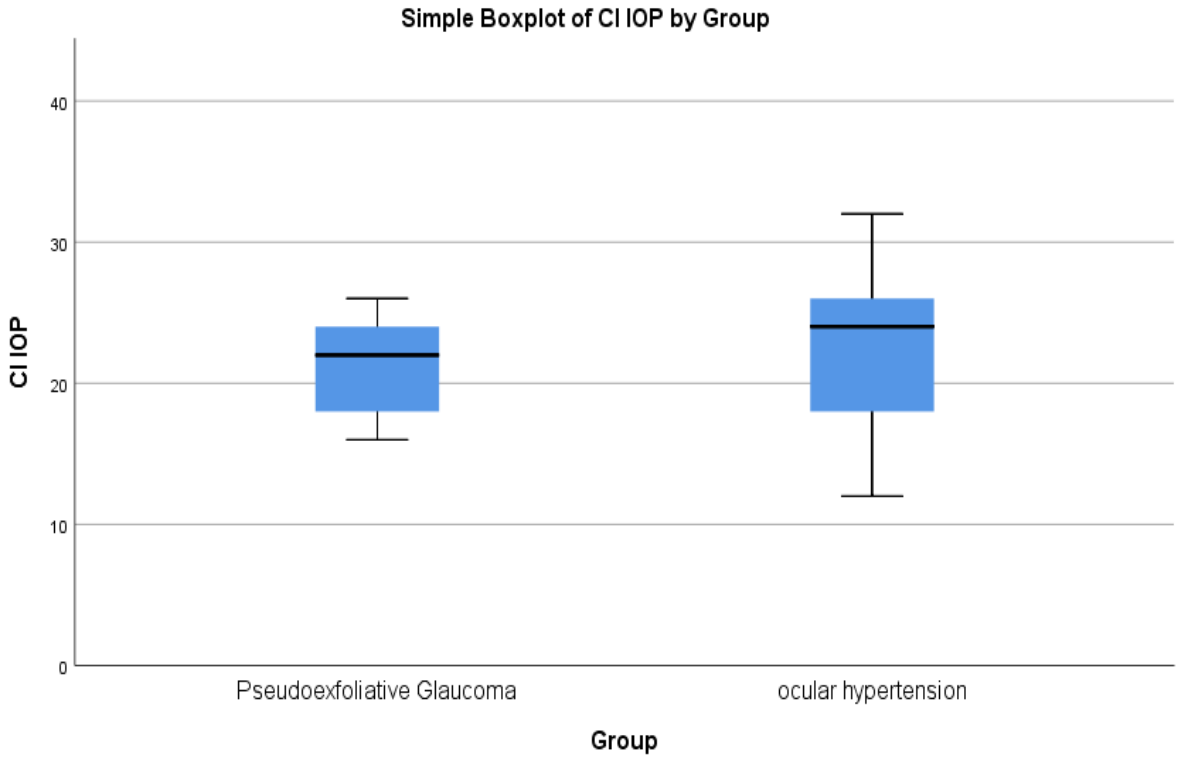
Table 21: Comparison of CCT & IOP between Pseudoexfoliative Glaucoma and ocular hypertension



Graph 29: Boxplot showing comparison of CCT among PXG group and OHT



Graph 30: Boxplot showing comparison of GAT IOP among PXG group and OHT

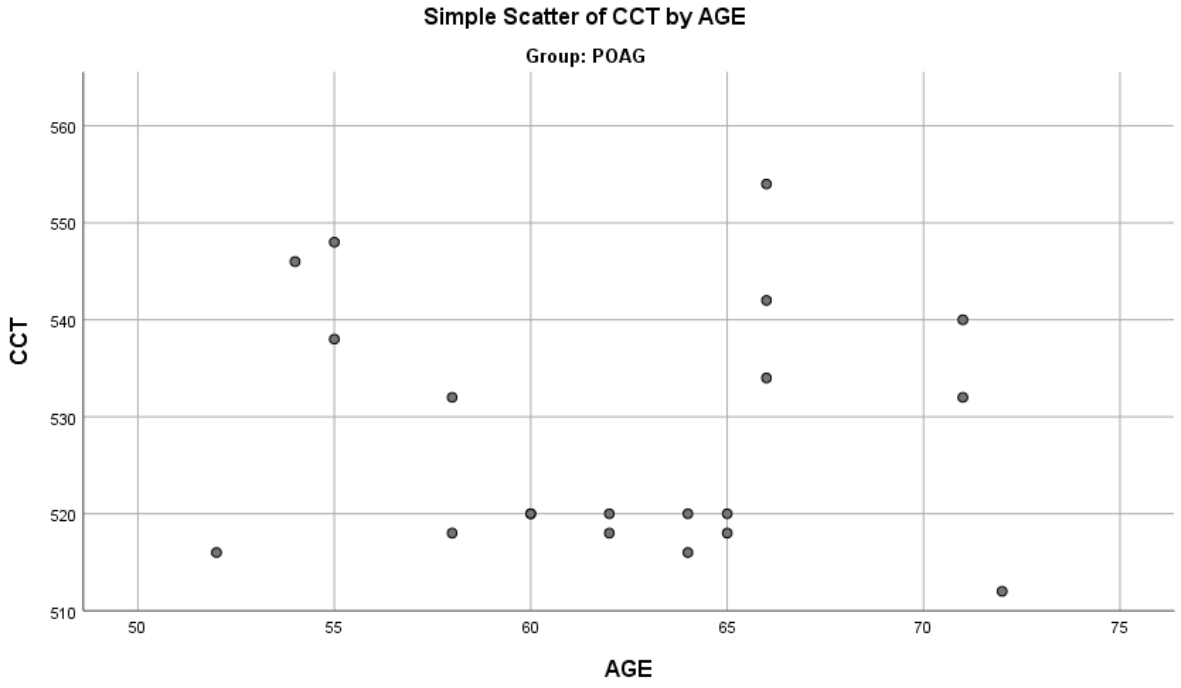


Graph 31: Boxplot showing comparison of CI IOP among PXG group and OHT

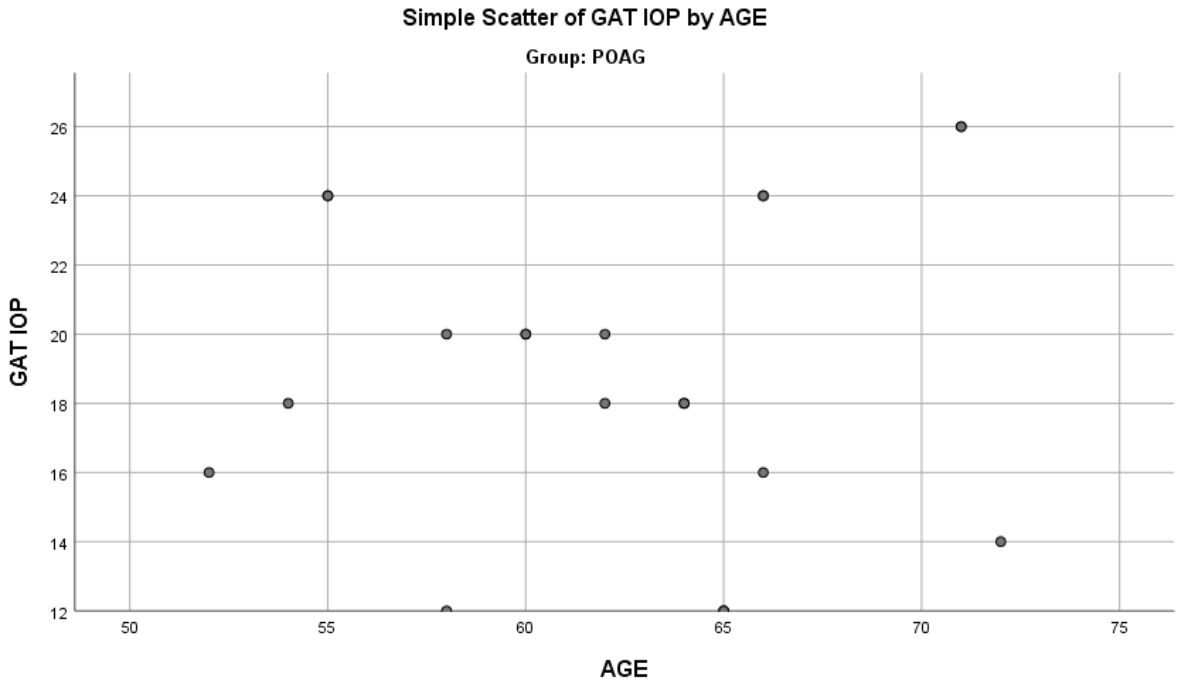
No correlation was noted between age and CCT/IOP values of POAG, patients.

IOP's (mm Hg)	Spearman correlation coefficient	p value
CCT (µm)	0.002	0.994
GAT	0.102	0.668
CI	0.135	0.571
Difference IOP	0.034	0.887

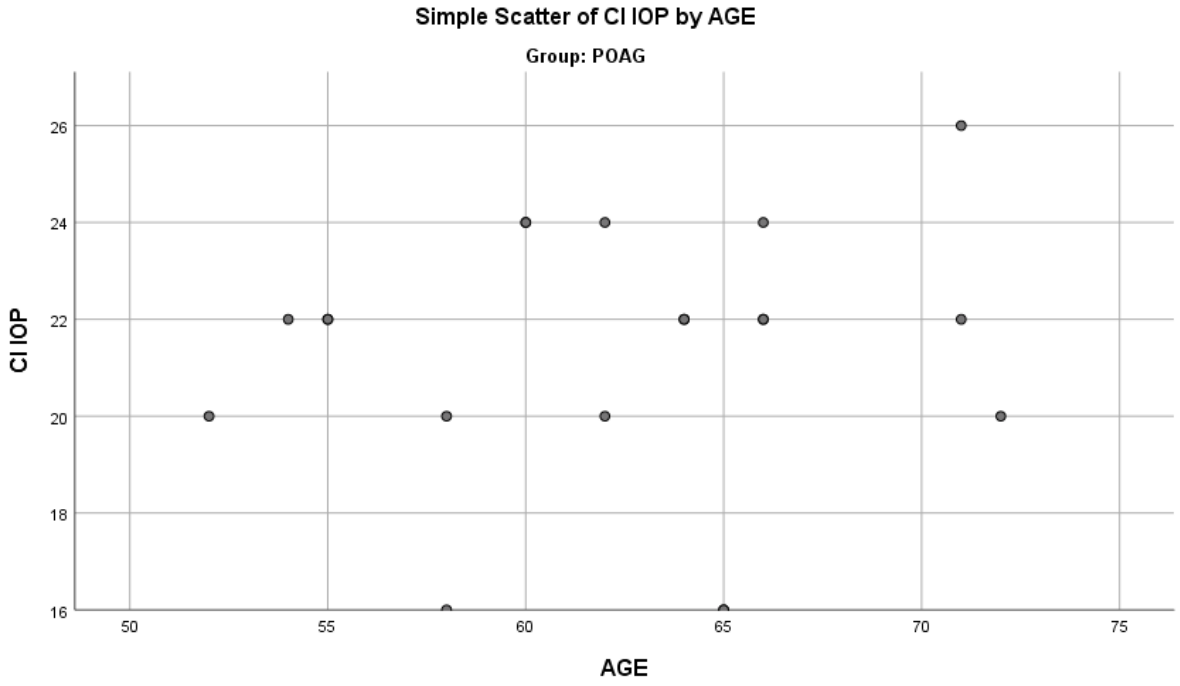
Table 22: Correlation between the Age, CCT and various IOPs among POAG



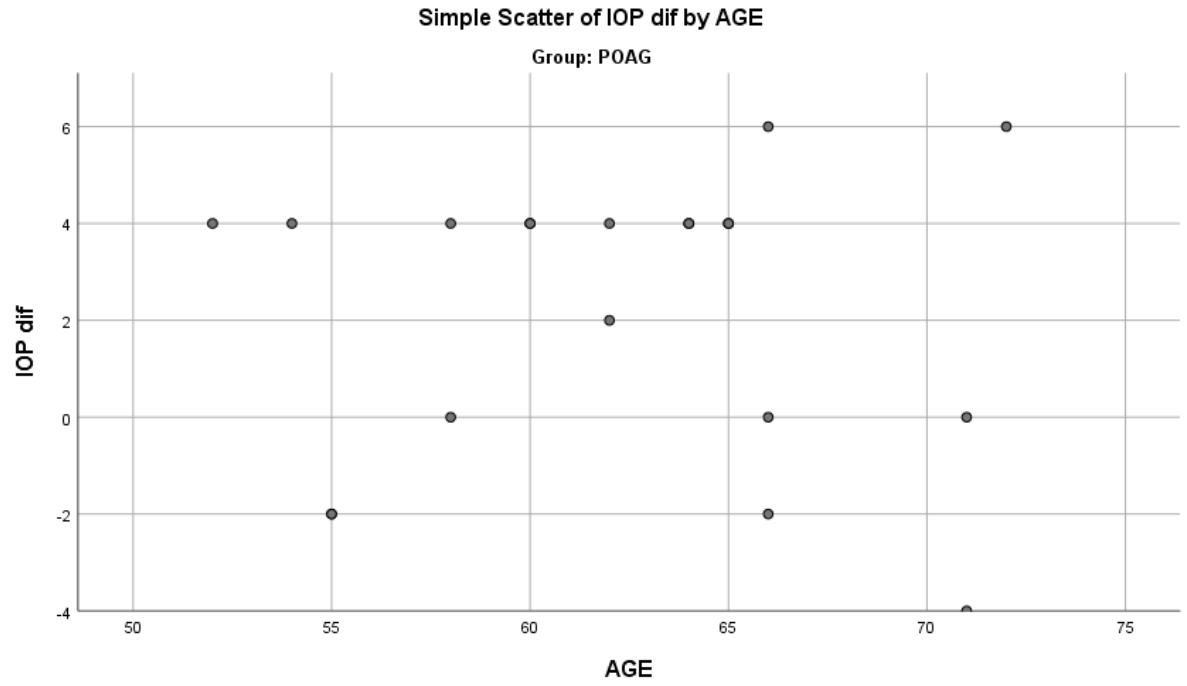
Graph 32: Scatter diagram showing the relationship between CCT and age among POAG group



Graph 33: Scatter diagram showing the relationship between GAT IOP, age among POAG group



Graph 34: Scatter diagram showing the relationship between CI IOP, age among POAG group

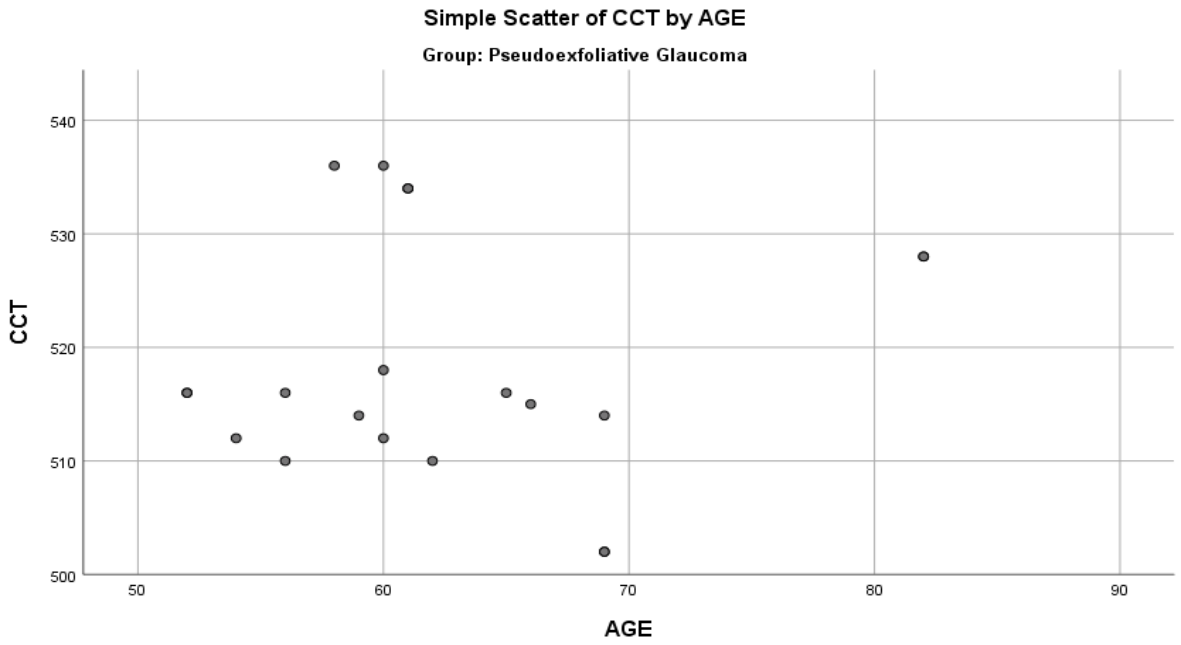


Graph 35: Scatter diagram showing the relationship between difference in IOP, age among POAG group

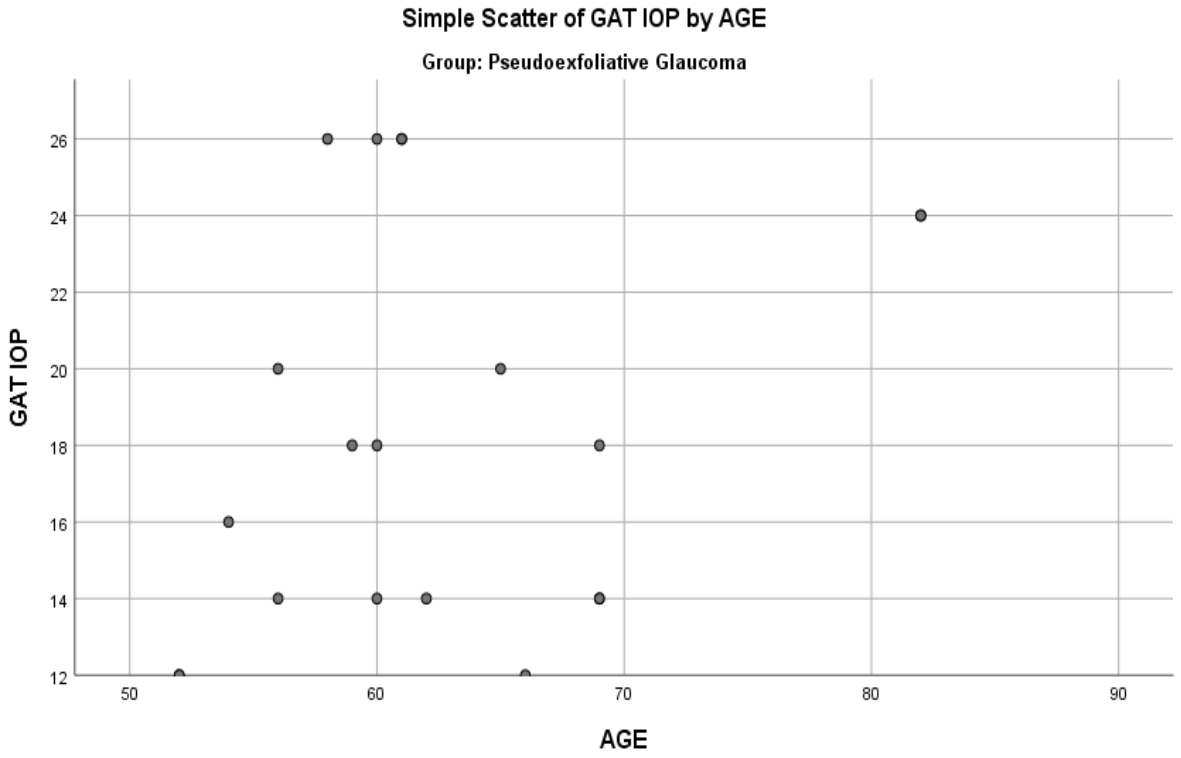
No correlation was noted between age and the CCT/IOP values of Pseudoexfoliative Glaucoma patients.

IOP's (mm Hg)	Spearman correlation coefficient	p value
CCT (µm)	-0.059	0.804
GAT	0.217	0.359
CI	0.181	0.444
Difference IOP	-0.237	0.315

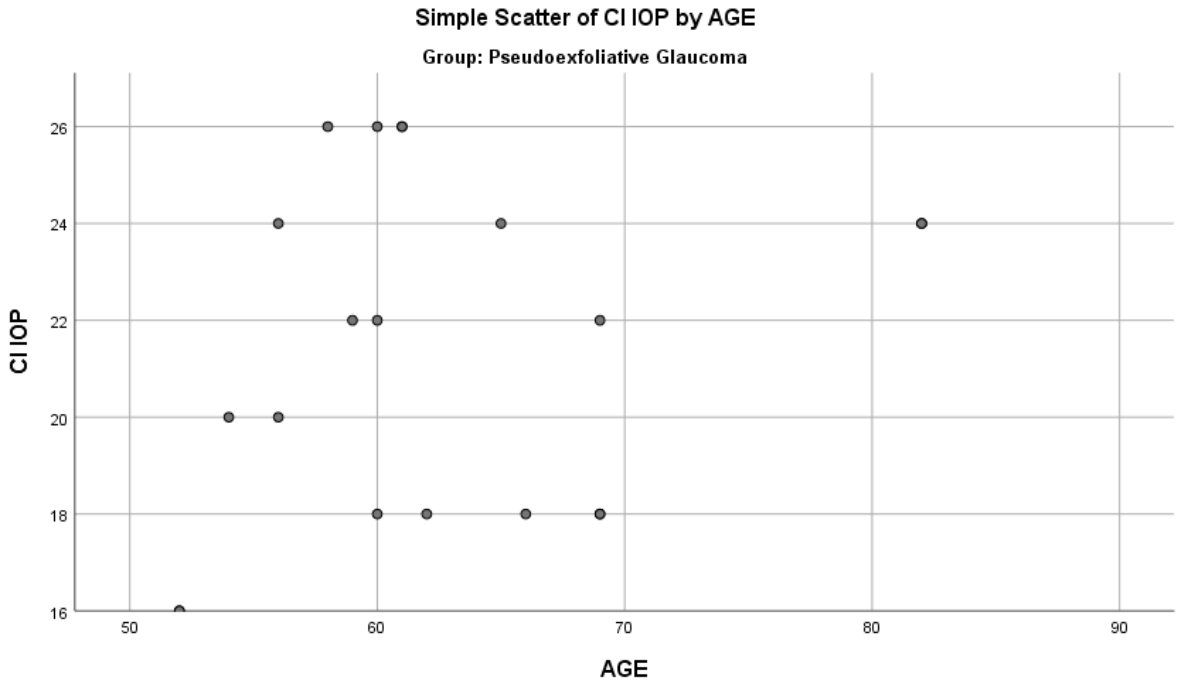
Table 23: Correlation between the Age, CCT and various IOPs among Pseudoexfoliative Glaucoma



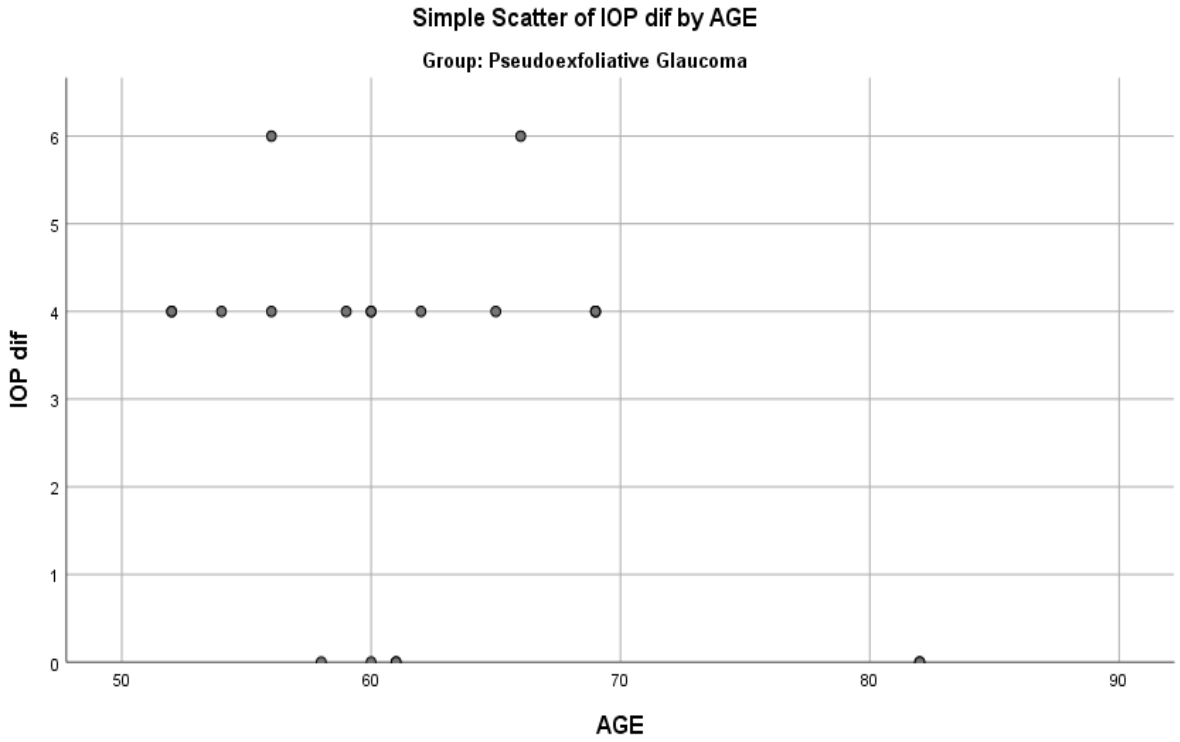
Graph 36: Scatter diagram showing the relationship between CCT, age among PXG group



Graph 37: Scatter diagram showing the relationship between GAT IOP, age among PXG group



Graph 38: Scatter diagram showing the relationship between CI IOP, age among PXG group

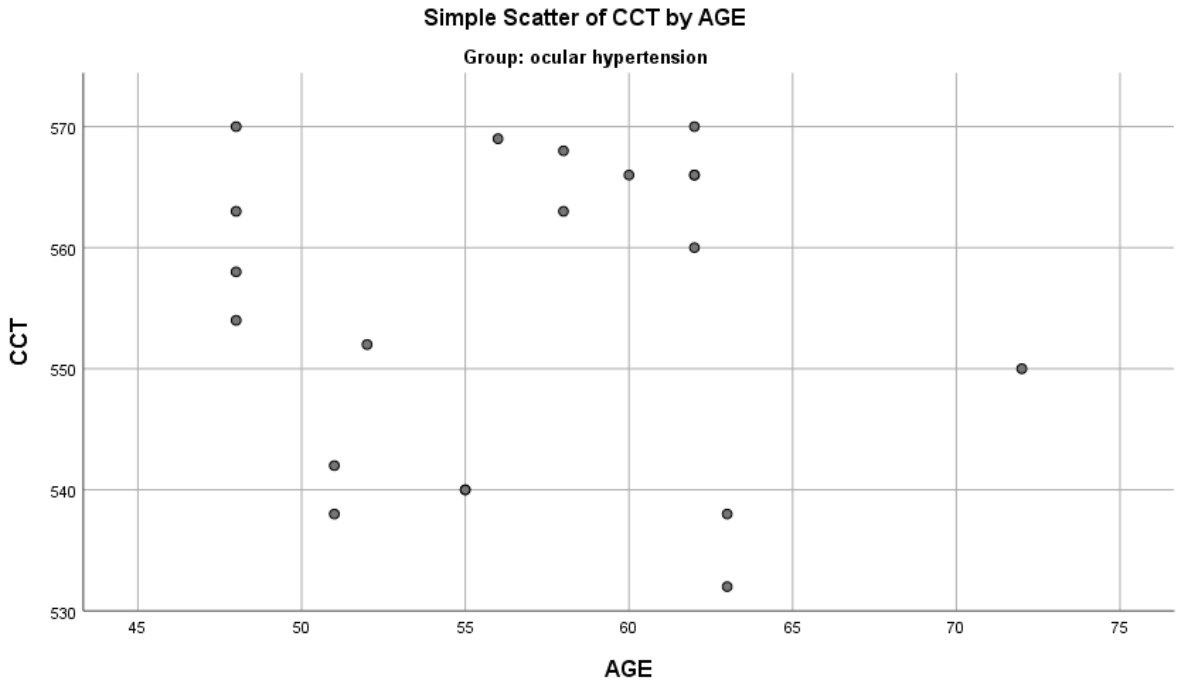


Graph 39: Scatter diagram showing the relationship between difference in IOP, age among PXG group

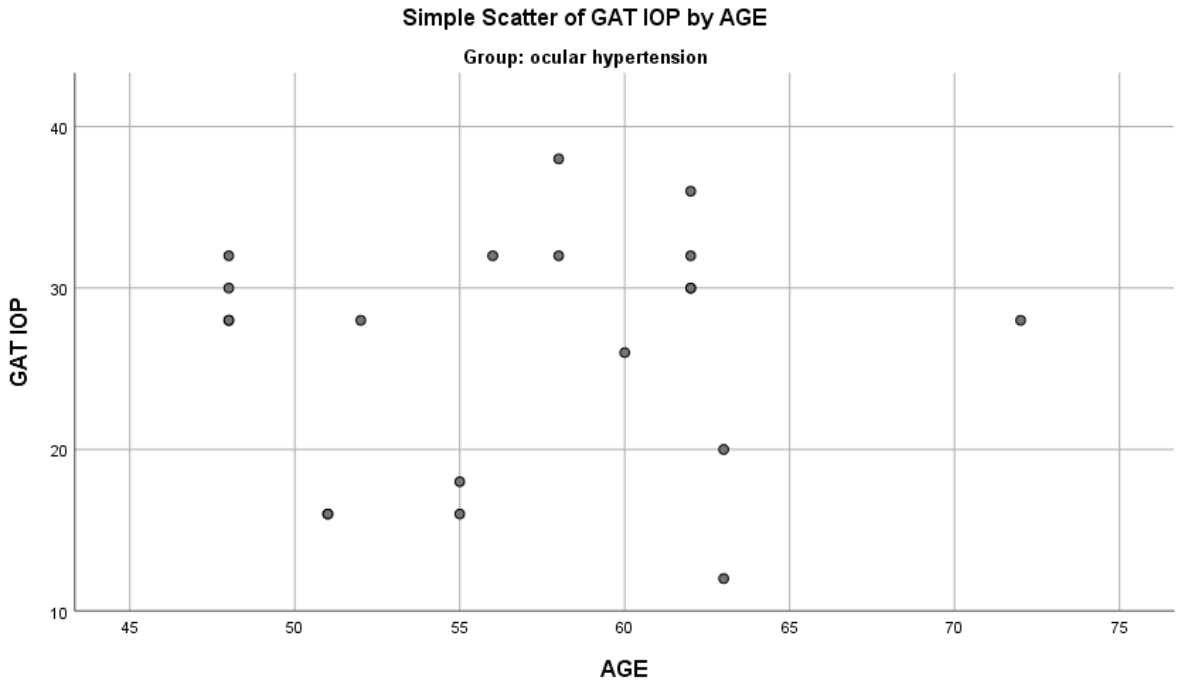
There was no correlation between age and the CCT/IOP values of Ocular hypertension patients.

IOP's (mm Hg)	Spearman correlation coefficient	p value
CCT (µm)	-0.063	0.791
GAT	0.049	0.836
CI	0.087	0.717
Difference IOP	0.077	0.748

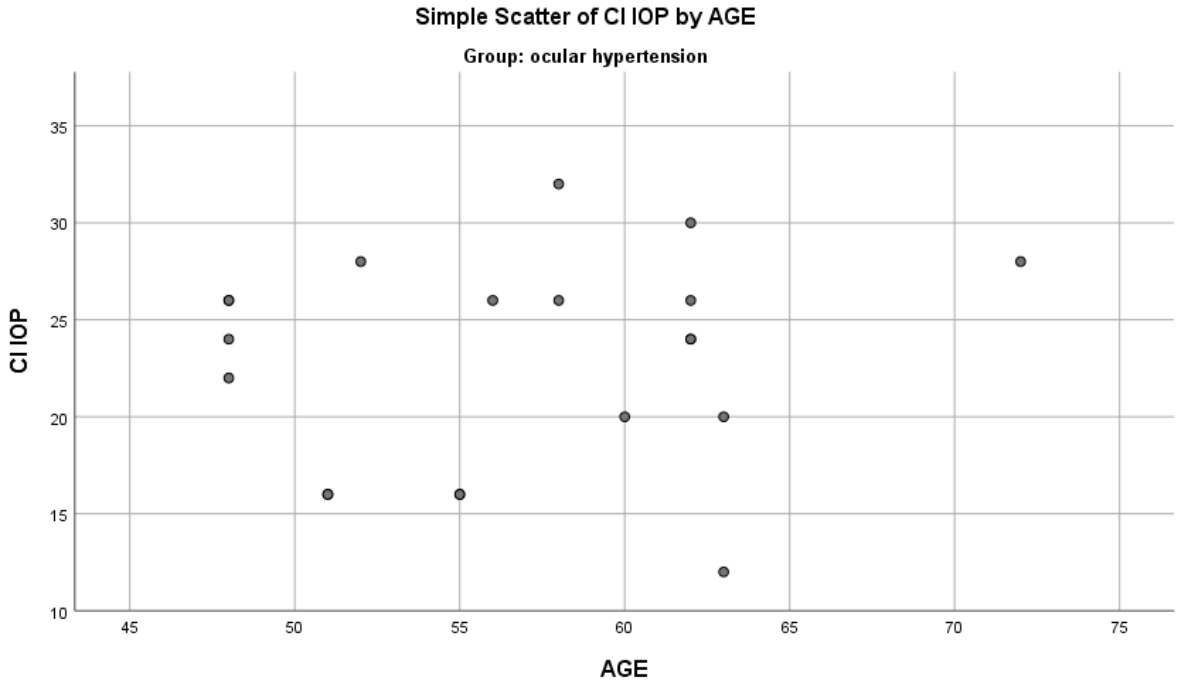
Table 24: Correlation between the Age, CCT and various IOPs among Ocular hypertension



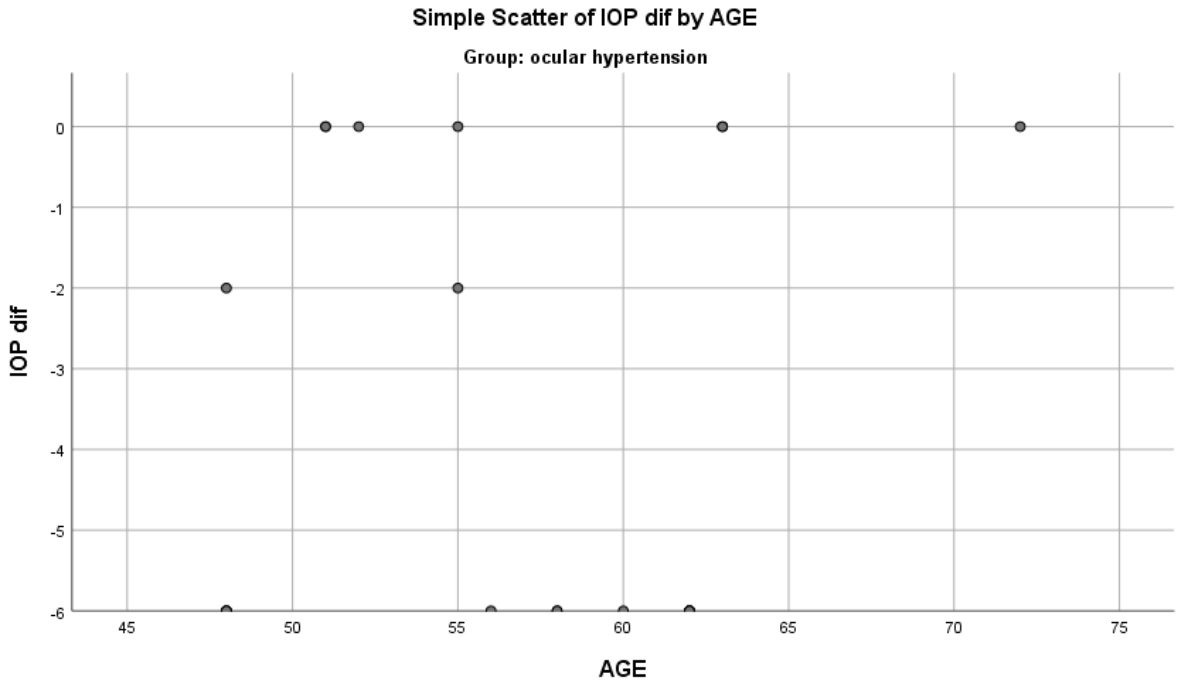
Graph 40: Scatter diagram showing relation between CCT and age among OHT group



Graph 41: Scatter diagram showing relation between GAT IOP and age among OHT group



Graph 42: Scatter diagram showing relation between CI IOP and age among OHT group

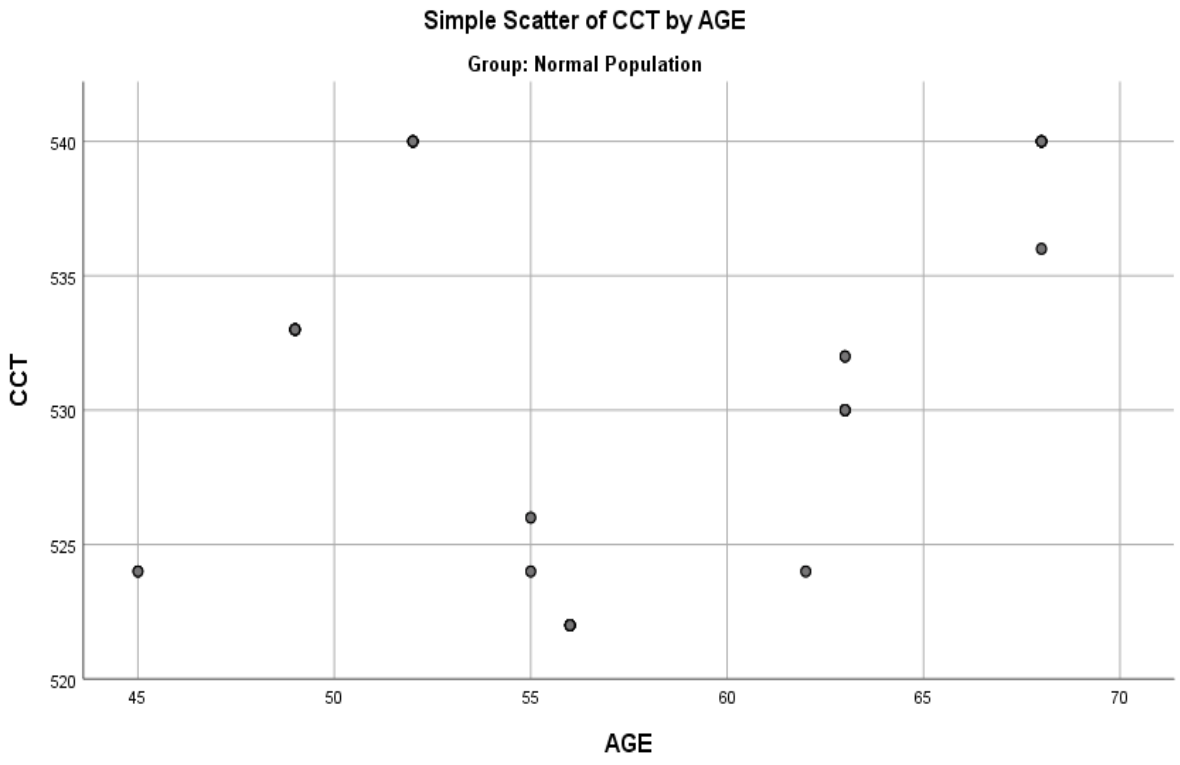


Graph 43: Scatter diagram showing relation between difference in IOP and age among OHT group

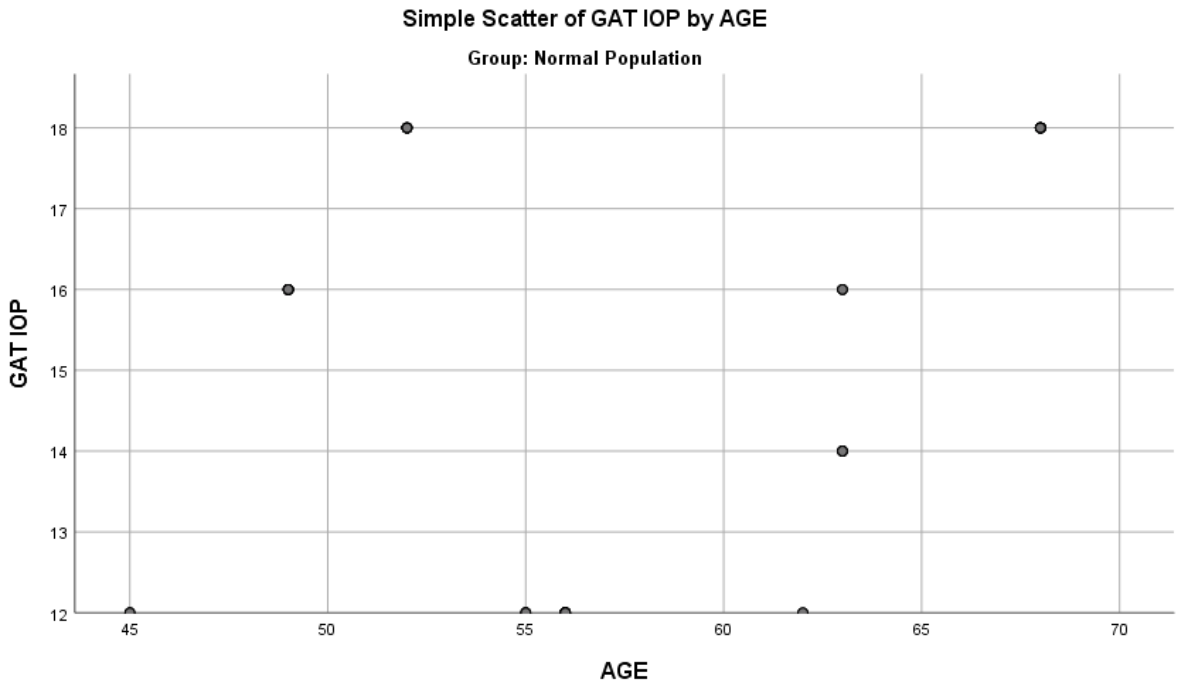
There was no correlation between age and the CCT/IOP values of normal population.

IOP's (mm Hg)	**Spearman correlation coefficient**	**p value**
CCT (µm)	0.130	0.323
GAT	0.216	0.097
CI	0.216	0.097
Difference IOP	-	-

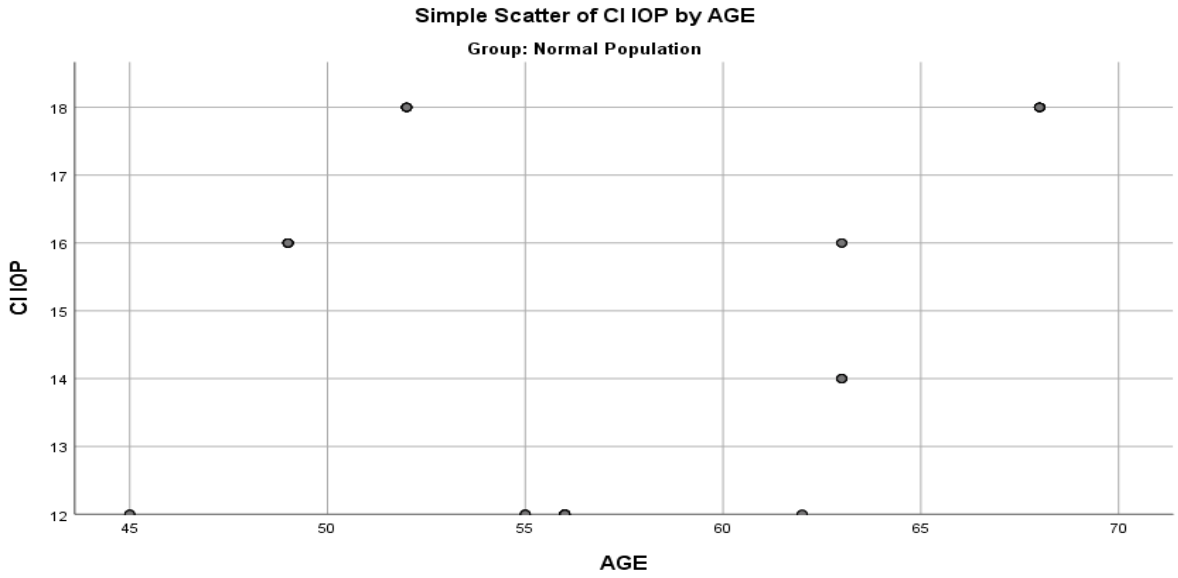
Table 25: Correlation between the Age, CCT and various IOPs among normal population



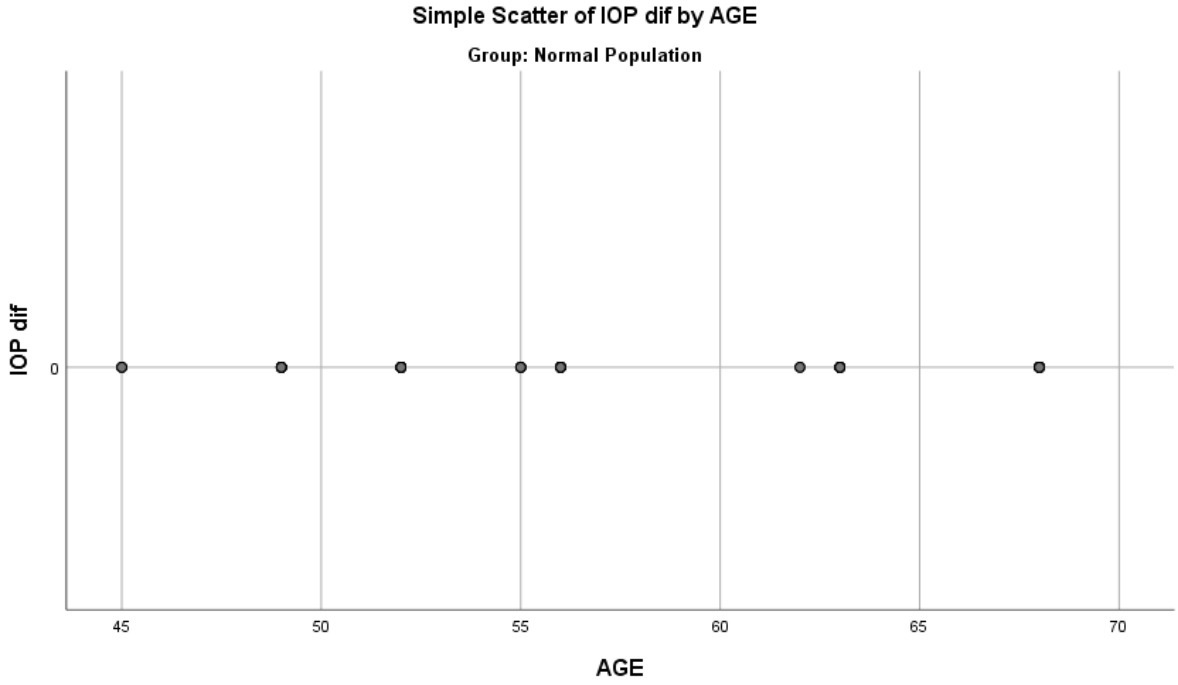
Graph 44: Scatter diagram showing relation between CCT and age among Normal population group



Graph 45: Scatter diagram showing relation between GAT IOP and age among Normal population group



Graph 46: Scatter diagram showing relation between CI IOP and age among Normal population group

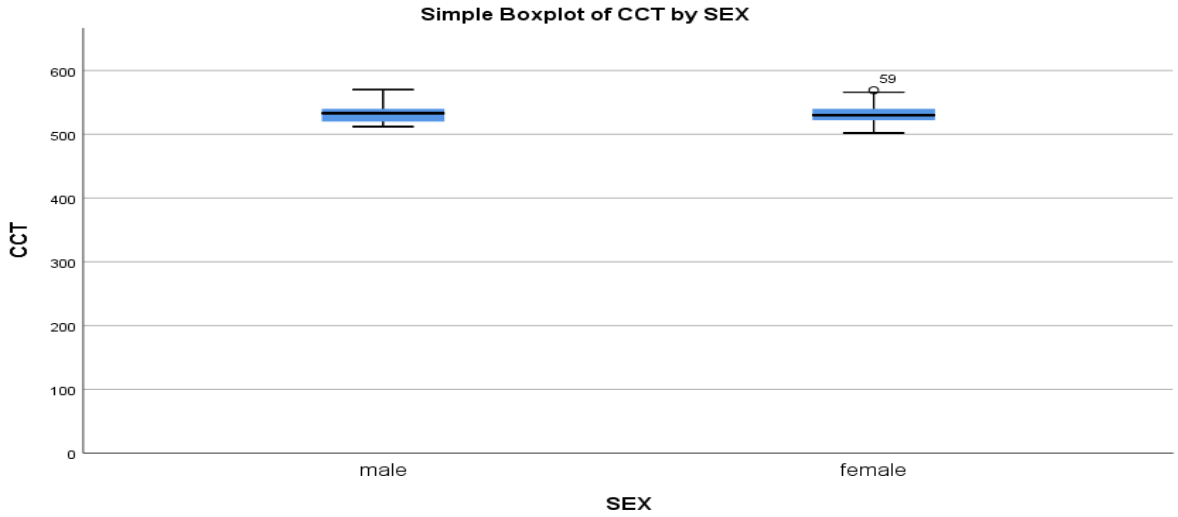


Graph 47: Scatter diagram showing relation between difference in IOP and age among Normal population group

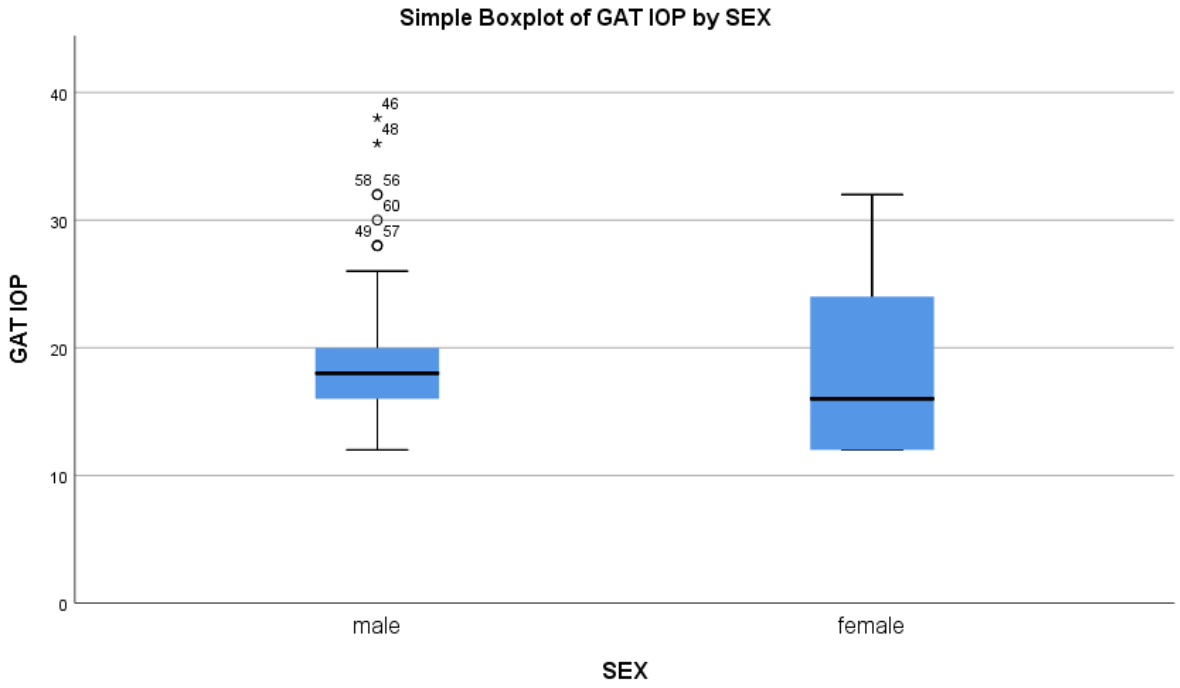
No significant difference was noted in the CCT and IOPs of males and females of patients in all groups.

Parameter	SEX	N	Mean Rank	P value
CCT (µm)	Male	64	62.05	0.599
	Female	56	58.72	
	Total	120		
GAT IOP (mm Hg)	Male	64	63.28	0.341
	Female	56	57.32	
	Total	120		
CI IOP (mm Hg)	Male	64	63.81	0.259
	Female	56	56.71	
	Total	120		
Difference IOP (mm Hg)	Male	64	64.66	0.103
	Female	56	55.74	
	Total	120		

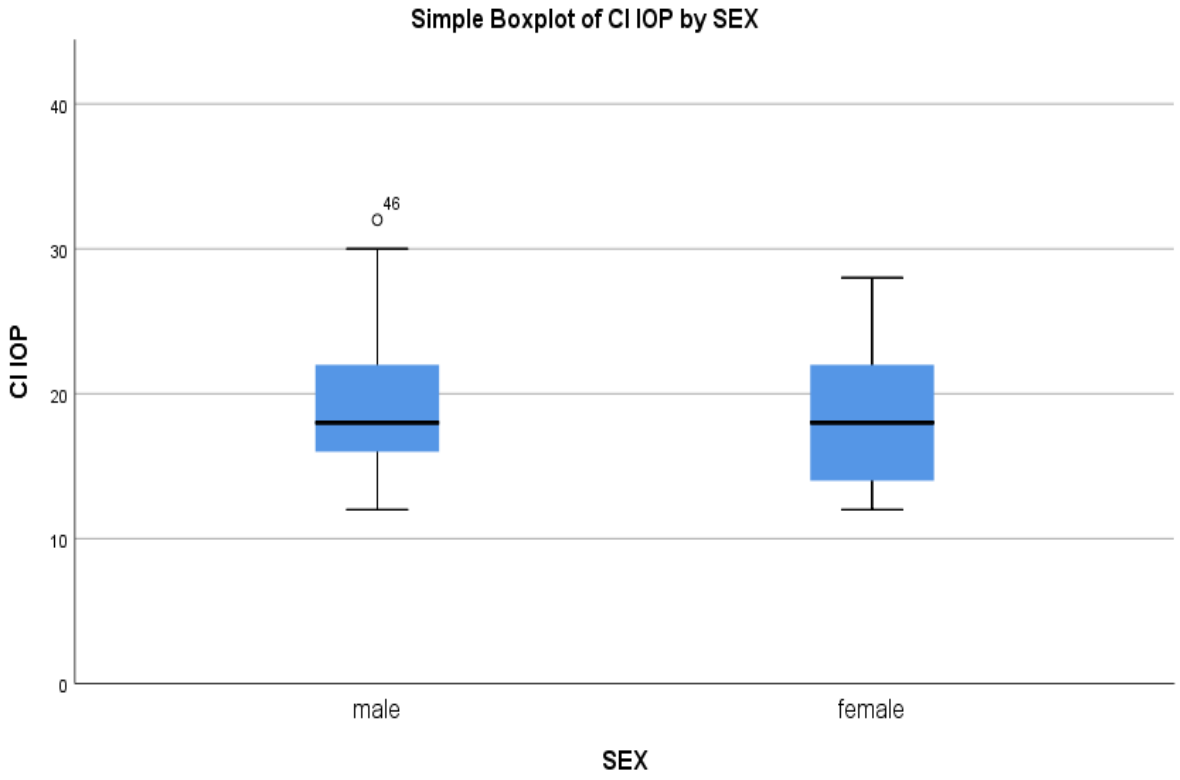
Table 26: Comparison of CCT & IOP between males and females (overall)



Graph 48: Boxplot comparing CCT Gender wise



Graph 49: Boxplot comparing GAT IOP Gender wise

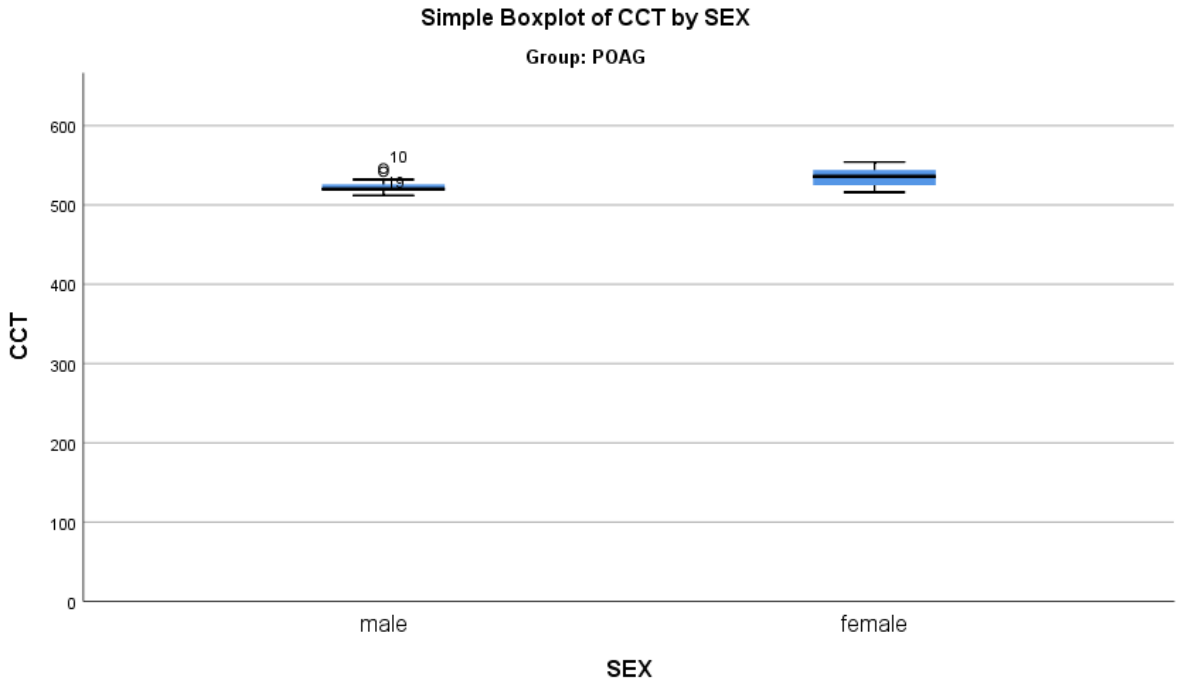


Graph 50: Boxplot comparing CI IOP Gender wise

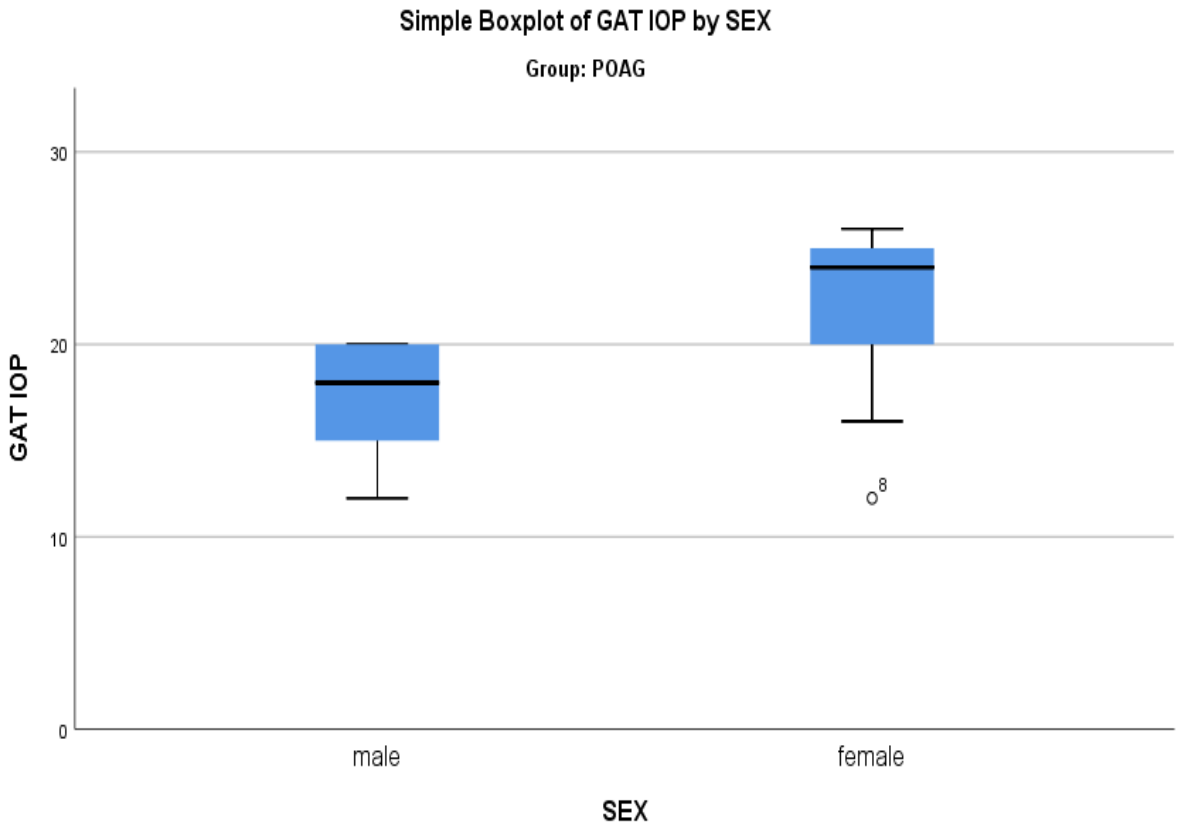
No significant difference was seen in the CCT and IOPs of males and females of patients in POAG group.

Group	Parameter	SEX	N	Mean Rank	P value
POAG	CCT (µm)	Male	12	8.83	0.135
		Female	8	13.00	
		Total	20		
	GAT IOP (mm Hg)	Male	12	8.13	0.025
		Female	8	14.06	
		Total	20		
	CI IOP (mm Hg)	Male	12	9.92	0.624
		Female	8	11.38	
		Total	20		
	Difference IOP (mm Hg)	Male	12	13.42	0.005
		Female	8	6.13	
		Total	20		

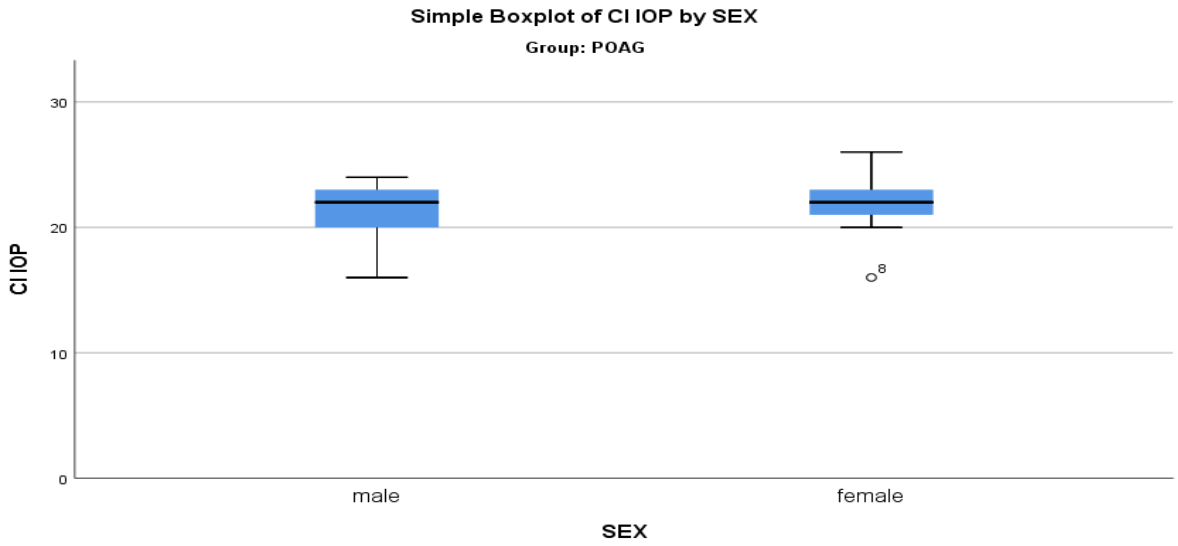
Table 27: Comparison of CCT & IOP between males and females among POAG



Graph 51: Boxplot comparing CCT Gender wise among POAG group



Graph 52: Boxplot comparing GAT IOP Gender wise among POAG group



Graph 53: Boxplot comparing CI IOP Gender wise among POAG group

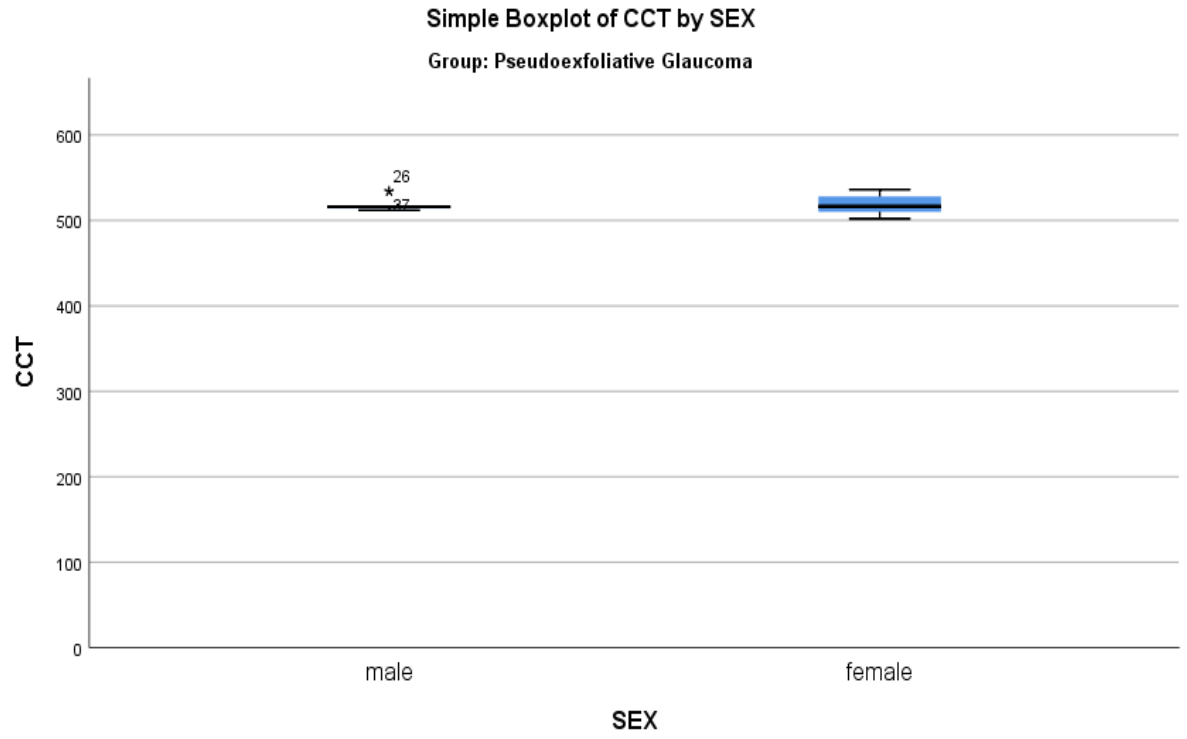
Pseudoexfoliative Glaucoma

No significant difference was noted in the CCT and IOPs of males and females of patients in PEX group.

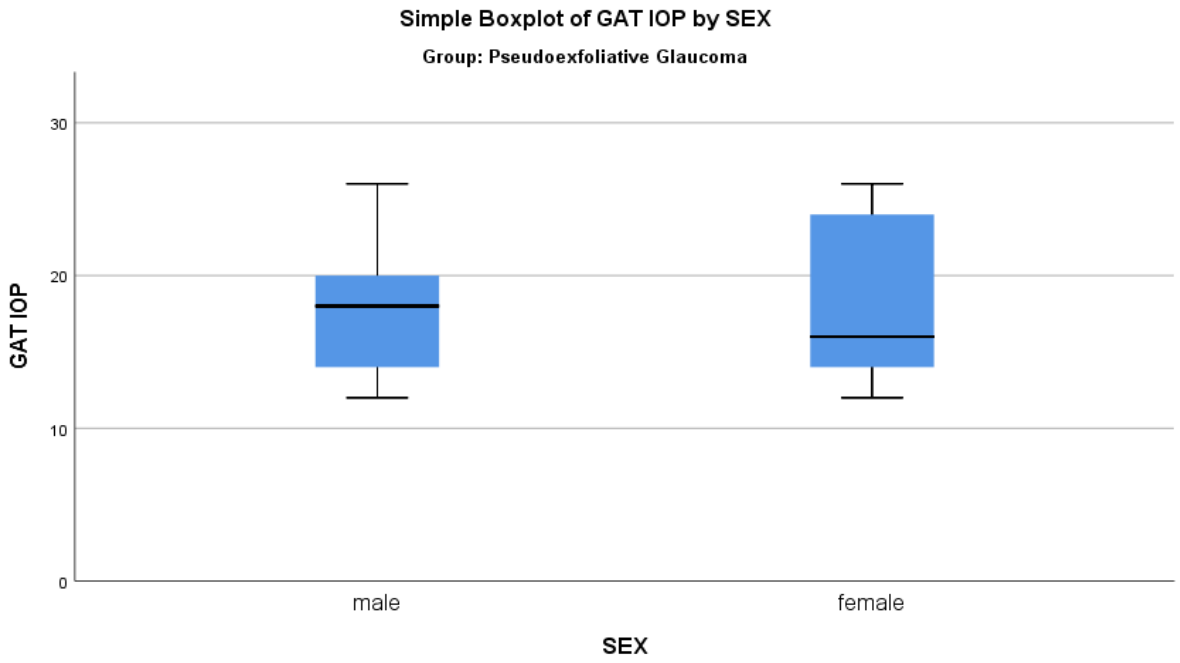
Group	Parameter	SEX	N	Mean Rank	P value
PXG	CCT (µm)	Male	10	10.70	0.912
		Female	10	10.30	
		Total	20		
	GAT IOP (mm Hg)	Male	10	10.50	1.000
		Female	10	10.50	
		Total	20		
	CI IOP (mm Hg)	Male	10	10.45	0.971
		Female	10	10.55	

		Total	20		
	Difference IOP (mm Hg)	Male	10	10.70	0.912
		Female	10	10.30	
		Total	20		

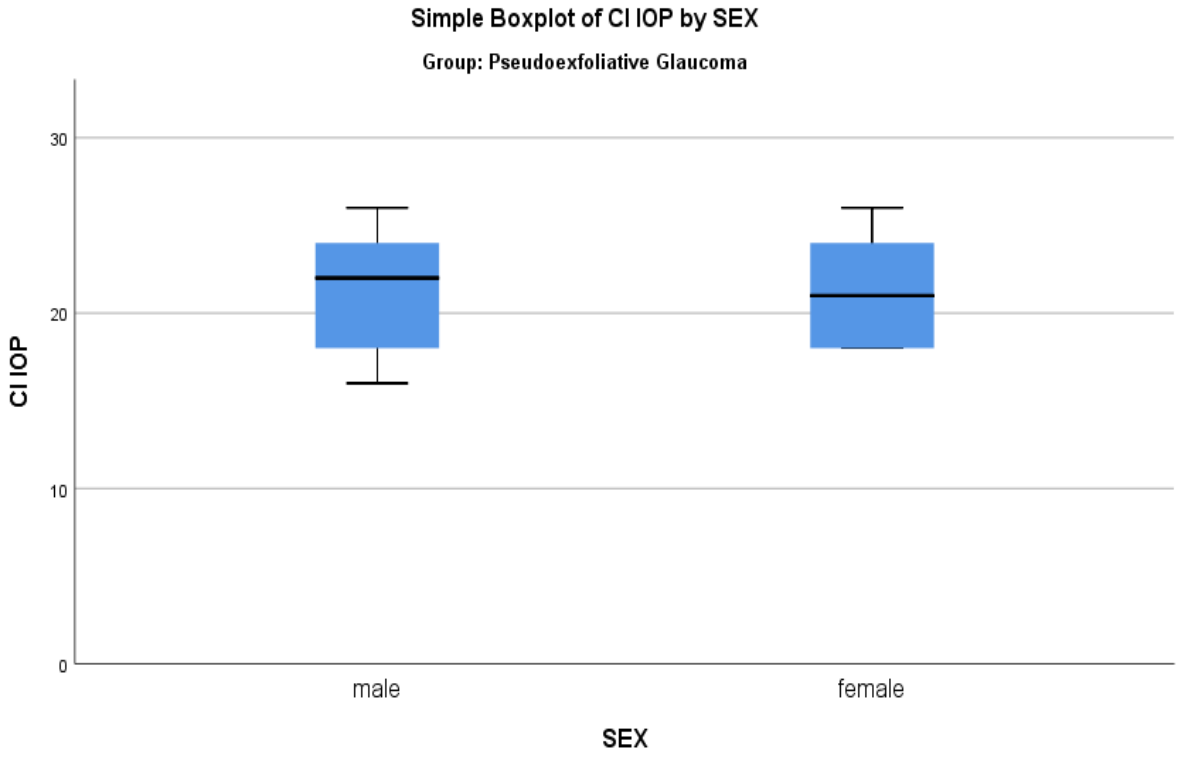
Table 28: Comparison of CCT & IOP between males and females among PXG



Graph 54: Boxplot comparing CCT Gender wise among PXG group



Graph 55: Boxplot comparing GAT IOP Gender wise among PXG group



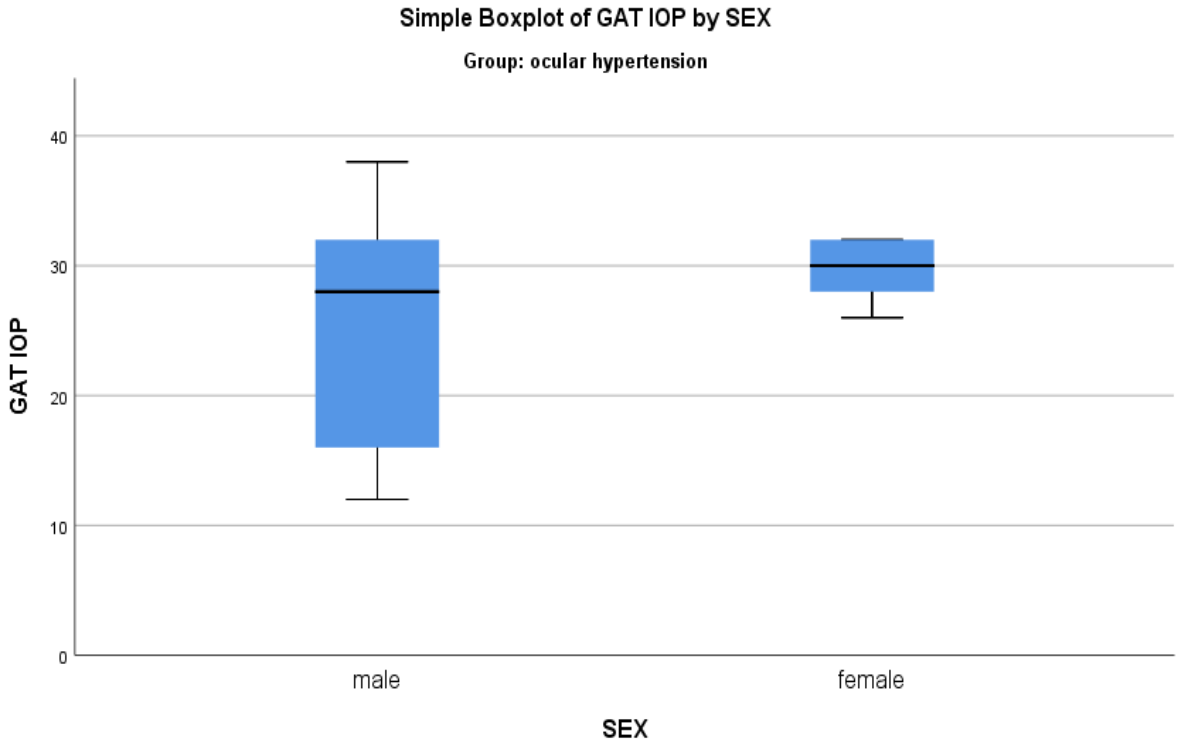
Graph 56: Boxplot comparing CI IOP Gender wise among PXG group

No significant difference was noted in the CCT and IOPs of males and females of patients in ocular hypertension group.

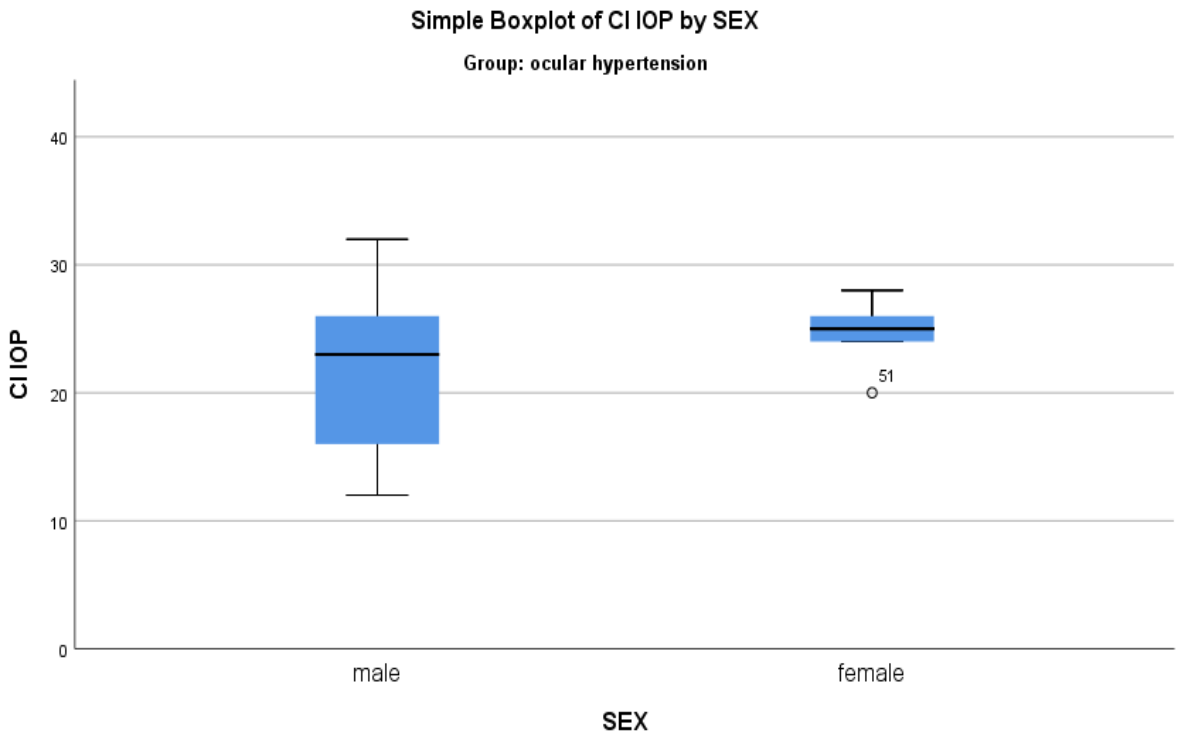
Group	Parameter	SEX	N	Mean Rank	P value
OHT	CCT (µm)	Male	14	9.39	0.207
		Female	6	13.08	
		Total	20		
	GAT IOP (mm Hg)	Male	14	9.61	0.312
		Female	6	12.58	
		Total	20		
	CI IOP (mm Hg)	Male	14	9.86	0.494
		Female	6	12.00	
		Total	20		
	Difference IOP (mm Hg)	Male	14	11.64	0.207
		Female	6	7.83	
		Total	20		

Table 29: Comparison of CCT & IOP between males and females among ocular hyper-tension

Graph 57: Boxplot comparing CCT Gender wise among OHT group



Graph 58: Boxplot comparing GAT IOP Gender wise among OHT group



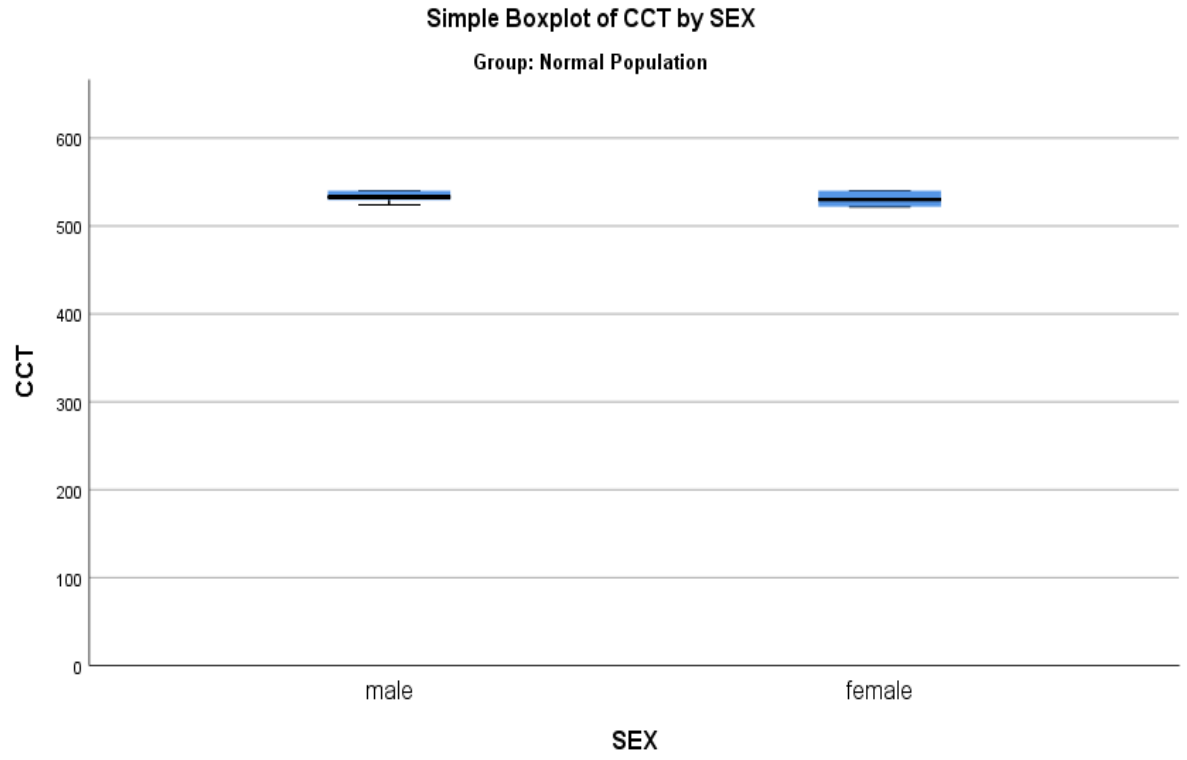
Graph 59: Boxplot comparing CI IOP Gender wise among OHT group

No significant difference was noted in the CCT and IOPs of males and females of patients in normal population.

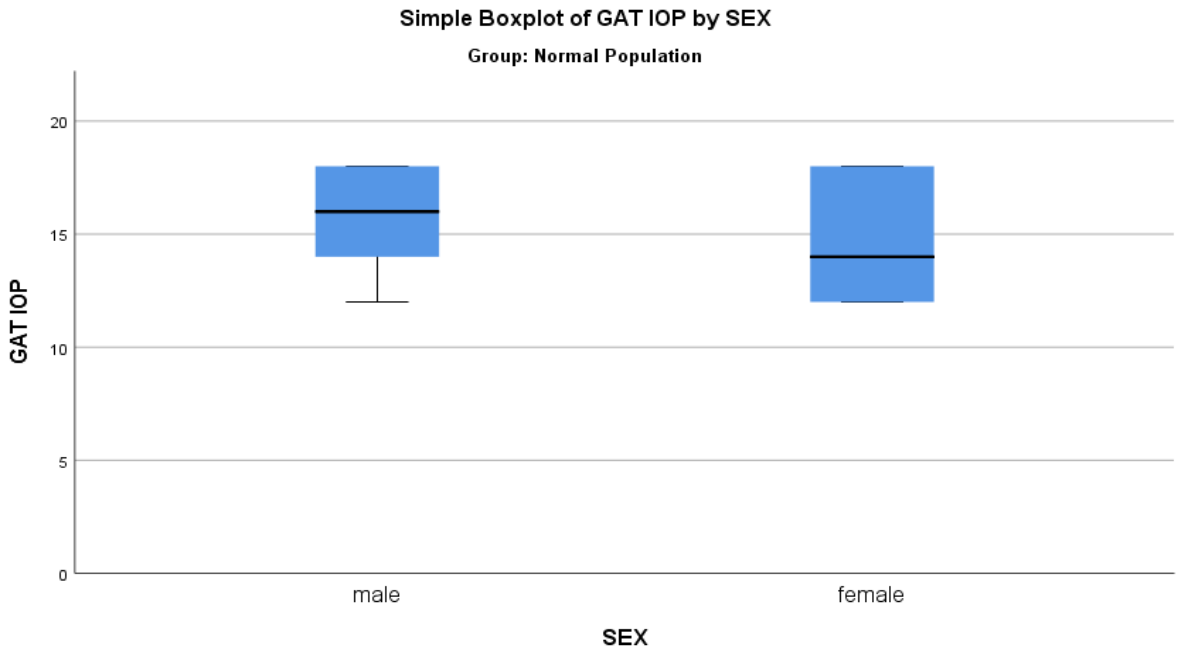
Group	Parameter	SEX	N	Mean Rank	P value
Normal population	CCT (µm)	Male	28	35.86	0.023
		Female	32	25.81	
		Total	60		
	GAT IOP (mm Hg)	Male	28	34.29	0.099
		Female	32	27.19	
		Total	60		
	CI IOP (mm Hg)	Male	28	34.29	0.099

		Female	32	27.19	
		Total	60		
	Difference IOP (mm Hg)	Male	28	30.50	1.000
		Female	32	30.50	
		Total	60		

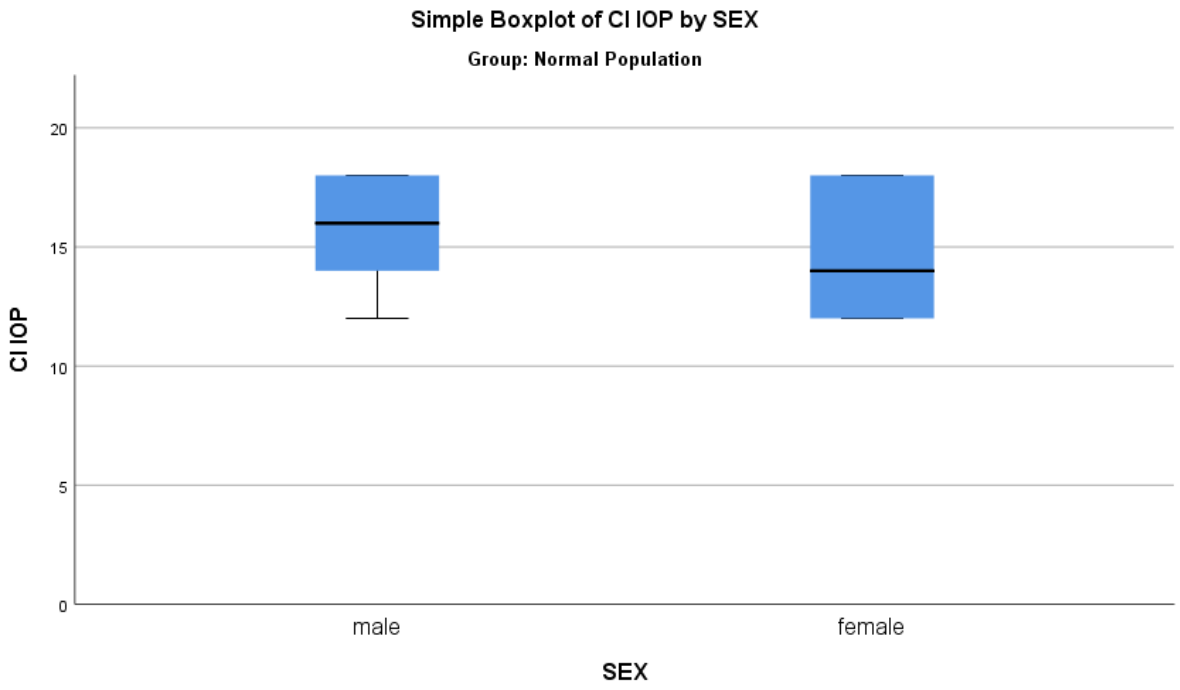
Table 30: Comparison of CCT & IOP between males and females among Normal Population



Graph 60: Boxplot comparing CCT Gender wise among Normal population group



Graph 61: Boxplot comparing GAT IOP Gender wise among Normal population group



Graph 62: Boxplot comparing CI IOP Gender wise among Normal population group

DISCUSSION

6. DISCUSSION

The current case control study was carried out in Ophthalmology department, R.L.Jalappa. Hospital and Research Centre, Sri Devaraj Urs Medical College, Tamaka, Kolar between January 2021 and June 2022 with the aim to assess and compare the correlation between CCT and IOP among the patients with POAG, PXG, OHT and normal population. 20 patients in each of the three study groups were compared with 60 normal population as controls. Intra-ocular pressure being the modifiable risk factor of glaucoma and can be the main parameter that can help in diagnosis. IOP reduction of about 1 mm Hg had shown to lower incidence of glaucomatous field loss by 10% in glaucoma patients.[100] Hence accurate measurement of IOP has a vital role in glaucoma. But IOP measurement with GAT can be influenced by corneal factors like central corneal thickness and curvature and can lead to either underestimation or overestimation of IOP.[101] The risk of glaucoma is higher among the patients with CCT of >/= 555µm than in the patients with CCT of >/=588 µm.[5] So CCT measurement became a stand-ard procedure in diagnosing and glaucoma monitoring. Thus, it is important to assess CCT in the glaucoma patients and the relationship between the CCT and the IOP might also varies between different types of glaucoma.

Moghimi et al determined the relation between the IOP and CCT in their study between POAG and pseudoexfoliative syndrome patients from Tehran, Iran.[101] **Ozkok et al** evaluated the IOP and CCT, among the PEX and POAG patients from Turkey.[97] **Kniestedt et al** as-sessed the correlation of CCT with IOP among glaucoma patients in USA.[102] **Shetgar et al** in their study from Karnataka, evaluated the CCT between the patients with NTG, POAG and ocular hypertension.[92] Similar to **Shetgar et al, Kumar et al** also compared the CCT and IOP between similar patient groups from Puducherry.[92.93] **Copt et al** evaluated the CCT in glaucoma and OHT patients from Switzerland. **Natarajan et al** compared the CCT between

POAG and normal patients from their study in South India.[94] **Yazgan et al** evaluated the IOPs and CCT between the PEX patients with and without glaucoma from Turkey.[95] **Yagci et al** assessed the CCT & IOP values among the various types of glaucoma, similar to our study, among patients of Ankara, Turkey.[90]

The present study had males in majority among POAG (60%) and ocular hypertension group (70%). Males and females were equal in PXG group. **Shetgar et al, Yazgan et al**, **Copt et al, Ozkok et al and Kumar et al** also had males in majority in their study.[92,93,95,97,107] In contrast, **Moghimi et al, Yagci et al and Wei et al** had majority of females.[90,100,106]

In the current study, sex had no significant impact on the CCT, while majority of studies reported males having increased CCT than females.[6] No significant difference was noted in CCT and IOP's between males and females among all the groups. **Kumar et al** showed no significant change in the CCT with respect to gender.[93]

The mean age of the POAG patients, PXG, ocular hypertension patients and normal population were 62.3 years, 62.7 years, 56.7 years and 57.3 years, respectively. **Shetgar et al, Kumar et al** reported a similar pattern of mean age with 56.2 years and 55.6 years, respectively.[92,93] **Moghimi et al** had much older patients in POAG (68.9 years) as well as in pseudoexfoliative syndrome (71.8 years) in their study.[100] **Ozkok et al** also had older patients in POAG (67.6 years) and PEX group (70.3 years).[97] **Yagci et al** included younger patients among the OHT (50.4 years), while PXG patients were older (69.1 years) and POAG patients were of similar age (60.4 years) to our study.[90] **Copt et al** had older patients in POAG (68.8 years) than the OHT group (60.3 years) in their study.[107] **Ventura et al** study had older patients in POAG (71.7 years) and PXG (76 years) and younger patients in ocular hypertension group (51.7 years).[91]

This study found no correlation between age, CCT/IOP values of POAG, Pseudoexfoliative Glaucoma and Ocular hypertension patients, which is in contrast to majority of previous

studies.[6] Past studies have reported an inverse relation in age and corneal thickness that is older the patient thinner was the CCT.[6,93]

The mean CCT of POAG , PXG, ocular hypertension patients and normal population were 528.2, 518.5, 555.3 and 531.8 µm respectively. The mean CCT among the POAG (525.3-526.4 µm) was similar in **Shetgar et al,** while they reported higher CCT (572.6 µm) among the ocular hypertension patients in comparison to our study.[92] **Kumar et al** reported similar mean CCT among POAG (525.3 µm) and normal population (527.7 µm) similar to our study, while CCT of OHT patients were higher (572.3 µm).[93] The POAG patients in **Moghimi et al** had a thicker CCT (537.8 µm) while PXG group had thinner CCT (506.5 µm).[100] In contrast, **Ozkok et al** reported thicker CCT in both POAG (549.1 µm) and PXG patients (546.1 µm) than our study.[97] **Copt et al** also reported thicker CCT among POAG (543 µm) and OHT (583 µm) than our study.[107] **Yagci et al** observed a thicker CCT among POAG (539.9), PXG (526.3 µm) and OHT patients (595.8 µm) than the patients in our study.[90] **Yazgan et al** reported CCT thickness of 509 & 525.5 µm among PXG patient with and without glaucoma.[95] **Natarajan et al** observed a mean CCT thickness of 531 to 552 µm, increasing according to the age.[94] **Ventura et al** reported thinner CCT among POAG (515 µm) and PXG (507 µm) and thicker CCT among OHT patients (563 µm) than our study.[91]

The present study showed, significantly lower CCT among the POAG and Pseudoexfoliative Glaucoma patients than the normal population, while it was higher among the ocular hypertension patients than normal population. **Shetgar et al and Kumar et al** also reported similar findings between ocular hypertension and normal population and no difference was seen between POAG and control group.[92,93] No difference in the CCT was noted by **Natarajan et al** between the POAG and normal population.[94] **Vieira et al** also reported thinner CCT among the POAG than normal people.[98]

We found CCT to be significantly lower among Pseudoexfoliative Glaucoma patients than

the POAG and ocular hypertension patients. No such difference was reported by **Ozkok et al** between POAG and PXG patients.[97] **Yazgan et al** reported significantly thinner CCT among the PXG patients.[95]

Significantly lower CCT was noted among POAG patients than the ocular hypertension patients, which is same as the observation by **Shetgar et al and Kumar et al.**[92,93] **Gorezis et al** reported a similar finding of significantly thinner CCT among the PXG patients and thicker CCT among the OHT group.[89] **Ventura et al** reported similar significantly higher thickness among OHT patients than all other types of glaucoma and the normal population.[108]

Thinner CCT among the Pseudoexfoliative Glaucoma and POAG indicates much severe optic nerve damage among these patients.[72]

The mean GAT IOP of the POAG patients, PXG, ocular hypertension patients and normal population were 19.1, 18.4, 26.4 and 15.2 mmHg respectively. **Ozkok et al, Yagci et al** reported a lower IOP by GAT among the POAG (16.5 & 15.9 mmHg) as well as PXG group (14.7 & 17.6 mmHg).[90,97] OHT group in **Yagci et al** also found to have lower IOP (23 mmHg) compared to our study.[90] In contrast, POAG as well as OHT reported higher IOP among the patients of **Copt et al**.[107] This might be due to varied severity of the glaucoma patients included in their studies and our study.

The mean CI IOP of the POAG patients, PXG, ocular hypertension patients and normal population were 21.3, 21.4, 22.9 and 15.2 mmHg respectively. **Shetgar et al and Kumar et al** reported higher corrected IOP among POAG than ours (29.6 & 29.6 mmHg), while it was lower in the Ocular hypertension group (24.5 & 24.5 mmHg).[92,93] **Ozkok et al** also had lower IOP after correcting for the CCT in both POAG (16.9 mmHg) and PXG (16.7 mmHg) than ours.[97]

The IOP difference in the POAG patients and PXG were 2.2 mmHg and 3 mmHg respectively which was underestimated when calculated with GAT. Ocular hypertension patients had

overestimation of IOP of about 3.5 mmHg when calculated with GAT. **Copt et al** recorded a mean overestimation of 2.3 mm Hg among the ocular hypertension group, which was lower than our findings.[107] Corrected IOP and GAT IOP difference was significantly higher in POAG and PXG patients, while it was lower in OHT than normal population in our study. In contrary, no such difference was noted between POAG and PXG patients by **Ozkok et al** but found a higher difference among PXG than POAG group.[97]

IOP was significantly higher among the POAG group, Pseudoexfoliative Glaucoma and ocular hypertension patients than the normal population. **Shetgar et al** showed similar findings in their comparison of POAG and ocular hypertension patients with normal population.[92] **Iyamu et al** also found a similar difference in IOP between normal and glaucoma patients.[103]

Significantly higher IOP was reported among ocular hypertension patients than POAG and Pseudoexfoliative Glaucoma, in our study. In contrast, **Shetgar et al** found a higher IOP among POAG patients than OHT.[92]

A positive correlation was noted between IOP based on GAT and CCT among POAG, Pseudoexfoliative Glaucoma and ocular hypertension patients. This is similar to the findings of **Moghimi et al** who reported significant positive correlation in CCT and GAT IOP among PXG and POAG.[100] In contrast, **Yagci et al** showed such significant association in the ocular hypertension group only, while other groups had no such correlation.[90] **Kniestedt et al** also reported a significant correlation between the GAT based IOP and the CCT of the glaucoma patients.[102] **Iyamu et al** in their study also noted positive correlation between CCT and IOP, which is same as our study.[103] For ocular hypertensives, a substantial association has been discovered between CCT and IOP, with a prediction of 0.70 mmHg rise in IOP for every 10 µm increase in corneal thickness. The same correlation pattern was observed even among Children with glaucoma[105]

CONCLUSION

7. CONCLUSION

Overall, a positive correlation was noted between the intraocular pressure and the central corneal thickness among POAG, Pseudoexfoliative Glaucoma and the ocular hypertension patients. CCT was highest among the ocular hypertensives, while POAG and Pseudoexfoliative Glaucoma patients had CCT thinner than normal population. Within the morbid groups, CCT was lower among Pseudoexfoliative Glaucoma patients than the POAG patients and ocular hypertension patients. CCT was also lower among the POAG patients than the ocular hypertension patients. Also, among the patients with POAG and PXG who had thinner CCT showed underestimation of IOP when measured with GAT but the corrected IOP was little higher, while in OHT group who had thicker CCT showed overestimation of IOP when measured with GAT but the corrected IOP was little lower.

Further, multi-centric, prospective studies must be conducted between the groups of patients to establish the impact of central corneal thickness on these types of glaucoma.

SUMMARY

8. SUMMARY

- CCT may have an impact on the diagnosis, screening, treating patients with glaucoma and ocular hypertension.
- This case- control study was conducted to assess CCT among POAG, PXG, OHT and compared with normal population.
- The study also compared and evaluated the correlation between CCT and IOP among POAG, PXG, OHT and normal eye.
- The study included 60 patients with POAG, PXG, OHT and 60 normal population
- It was conducted among the patients visiting the outpatient of Ophthalmology department at R.L.J. Hospital And Research Centre, Sri Devaraj Urs Medical College between January 2021 to June 2022
- The mean age of POAG, Pseudoexfoliative Glaucoma, Ocular hypertension patients and normal population was 62.3 years, 62.7 years,56.7 years & 57.3 years, respectively.
- The mean CCT among POAG, Pseudoexfoliative Glaucoma, Ocular hypertension patients and normal population was 528.2, 518.5, 555.3 and 531.8, respectively.
- The mean IOP (GAT) among POAG, Pseudoexfoliative Glaucoma, Ocular hypertension patients and normal population was 19.1, 18.4, 26.4 & 15.2 respectively.
- The mean IOP (CI) among POAG, Pseudoexfoliative Glaucoma, Ocular hypertension patients and normal population was 21.3, 21.4, 22.9 & 15.2, respectively.
- Significant positive correlation has been noted between IOP, CCT among POAG, Pseudoexfoliative Glaucoma, OHT patients and normal population.

- Significantly lower CCT was noted among the POAG and PXG patients than the normal population, while it was higher among the ocular hypertension patients than normal population.
- Significantly lower CCT was noted among PXG patients than the POAG patients and ocular hypertension patients.
- Significantly lower CCT was noted among the POAG patients than the ocular hypertension patients.
- IOP was significantly higher among the POAG group, Pseudoexfoliative Glaucoma and ocular hypertension patients than the normal population.
- Significantly higher IOP was reported among ocular hypertension patients than POAG and Pseudoexfoliative Glaucoma
- No significant difference was noted in IOP between the POAG and Pseudoexfoliative Glaucoma.
- No correlation was noted between age and the CCT/IOP values of POAG, Pseudoexfoliative Glaucoma and Ocular hypertension patients.
- No significant difference was noted in CCT and IOPs between males and females among POAG, PXG and OHT.
- POAG and PXG groups showed a mean higher IOP of 2.2, 3 mmHg, respectively, in the corrected IOP than the calculated IOP by GAT, which shows that thinner CCT groups (POAG, PXG) have falsely low IOP but when corrected, IOP is seen little higher.
- Patients with OHT showed mean lower IOP of 3.5 mmHg in the corrected IOP than the calculated IOP, which shows that thicker cornea groups (OHT group) have falsely high IOP when calculated but when corrected IOP will be little less

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9. BIBLIOGRAPHY

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ANNEXURE

10.ANNEXURE-1

CASE PROFORMA

Patient Name: - UHID: -

Age: - Gender: -

Address: -

BREIF HISTORY:- **PAST HISTORY:-**	
PERSONAL HISTORY: -	Appetite/ diet Bowel and bladder Sleep Habits
GENERAL PHYSICAL EXAMINATION **VITALS: -**	Pallor / icterus/ cyanosis/ clubbing/ edema PR: - BP: - RR: - TEMP:
SYSTEMIC EXAMINATION	CVS: - RS: - P/A: - CNS:

OCULAR EXAMINATION

TESTS	RE	LE
1. HEAD POSTURE **2. OCULAR POSTURE** 3. **FACIAL SYMMETRY**		
4. EXTRAOCULAR MOVEMENTS a) Ductions b) Versions c) Vergence		
5. VISUAL ACUITY: a) Distant b) Near		
6. ANTERIOR SEGMENT a. Lids and Adnexa b. Conjunctiva c. Cornea d. Anterior chamber e. Iris f. Pupillary reaction- • Direct • Indirect • Consensual g. Lens h. Anterior Vitreous a.		
7. RETINOSCOPY		
8. CENTRAL CORNEAL THICK-NESS-		
9. FUNDUS a. Distant direct ophthalmosco-py b. Direct ophthalmoscopy		

c. Indirect ophthalmoscopy		
10. Intraocular pressure by Goldmann applanation tonometer		
11. Gonioscopy a. shaffer's grading		
12. Humphrey visual field analysis		

ANNEXURE-II

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Case no:

IP no:

TITLE:

COMPARISION OF CENTRAL CORNEAL THICKNESS IN PRIMARY OPEN ANGLE GLAUCOMA, PSEUDO EXFOLIATIVE GLAUCOMA, OCULAR HYPERTENSION AND NORMAL POPULATION

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of personal information as outlined in this consent form.

I understand the purpose of this study, the risks and benefits of the technique and the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

I have had the opportunity to ask questions regarding the various aspects of this study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw the participation from this study at any time and this will not change the future care.

Participation in this study does not involve any extra cost to me.

Name	Signature	Date	Time
Patient:			
Witness:			
Primary Investigator/ Doctor:			

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR - 563101.

ತಿಳಿವಳಿಕೆಯ ಸಮ್ಮತಿ ನಮೂನೆ

ಕೇಸ್ ಸಂಖ್ಯೆ:

ಐಪಿ ಸಂಖ್ಯೆ:

ಶೀರ್ಷಿಕೆ:

"ಪ್ರೈಮರಿ ಒಪೆನ್ ಅಂಗಲ್ ಗ್ಲೋಕಾಮ, ಸೂಡೋ, ಎಕ್ಸ್ ಫೊಲಿಯೇಟಿವ್ ಗ್ಲೋಕಾಮ, ಓಕುಲರ್ ಹೈಪರ್ ಟೆನ್ಷಿಎನ್ ಉಳ್ಳ ರೋಗಿಗಳಲ್ಲಿ ಮತ್ತು ಸಾಮಾನ್ಯ ಜನರಲ್ಲಿ ಮಧ್ಯ ಕಾರ್ನಿಯಾದ ದಪ್ಪವನ್ನು ಹೋಲಿಕೆಯ ಅಧ್ಯಯನ"

ನಾನು, ಅಂಗೀಕರಿಸಿದ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಮತ್ತು ಈ ಸಮ್ಮತಿಯ ರೂಪದಲ್ಲಿ ವಿವರಿಸಿರುವಂತೆ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನು ಅನುಮೋದಿಸಲು ಒಪ್ಪುತ್ತೇನೆ.

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ತಂತ್ರದ ಅಪಾಯಗಳು ಮತ್ತು ಪ್ರಯೋಜನಗಳನ್ನು ಮತ್ತು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಲ್ಪಟ್ಟಿರುವ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವ ಮಾಹಿತಿಯ ಗೌಪ್ಯತೆಗೆ ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳನ್ನು ಕುರಿತು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ ಮತ್ತು ನನ್ನ ತೃಪ್ತಿಗೆ ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರ ನೀಡಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಿಂದ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಾನು ಹಿಂಪಡೆಯಲು ಮುಕ್ತವಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ಭವಿಷ್ಯದ ಕಾಳಜಿಯನ್ನು ಬದಲಿಸುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆ ನನಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚ ಒಳಗೊಳ್ಳುವುದಿಲ್ಲ.

ಹೆಸರು	ಸಹಿ/ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿಯ ಹೆಸರು			
ಸಾಕ್ಷಿಗಳ ಹೆಸರು			
ಪ್ರಾಥಮಿಕ ಸಂಶೋಧಕರು/ ವೈದ್ಯರು			

ANNEXURE-III

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

PATIENT INFORMATION SHEET

This information is to help you understand the purpose of the study "COMPARISION OF CENTRAL CORNEAL THICKNESS IN PRIMARY OPEN ANGLE GLAUCOMA, PSEUDO EXFOLIATIVE SYBDROME, OCULAR HYPERTENSION AND NORMAL POPULATION". You are invited to take part voluntarily in this research study, it is important that you read and understand the purpose, procedure, benefits and discomforts of the study.

1. What is the purpose of this study?

   Is to study and compare the correlation between CCT and IOP among the patients with POAG, PXG, OHT and NORMAL POPULATION.

2. What are the various investigations being used? Are there any associated risks?

   Absolutely no risks are associated with various investigations involved in this study such as

   i) Ultrasonic pachymetry

3. What is the benefit for me as a participant?

   Participation in this research study may not change the final outcome of your eye condition. However, patients in the future may benefit as a result of knowledge gained from this study. You will not be charged extra for any of the procedures performed during the research study. Your taking part in this study is entirely voluntary. You may refuse to take part in the study or you may stop your participation in the study at any time, without a penalty or loss of any benefits to which you were otherwise entitled before taking part in this study.

CONFIDENTIALITY

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. Your original records may be reviewed by your doctor or ethics review board.

For further information/ clarification please contact Dr.RASHMI.G, SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.Contact no: 9886998871 or 8494906087 to Dr.POOJITHA M

ರೋಗಿಯ ಮಾಹಿತಿ ಪತ್ರ

“ಪ್ರೈಮರಿ ಒಪೆನ್ ಅಂಗಲ್ ಗ್ಲೋಕಾಮ, ಸೂಡೋ ಎಕ್ಸ್ ಫೊಲಿಯೇಟಿವ್ ಗ್ಲೋಕಾಮ, ಓಕುಲರ್ ಹೈಪರ್ ಟೆನ್ಷಂನ್ ಉಳ್ಳ ರೋಗಿಗಳಲ್ಲಿ ಮತ್ತು ಸಾಮಾನ್ಯ ಜನರಲ್ಲಿ ಮಧ್ಯ ಕಾರ್ನಿಯಾದ ದಪ್ಪವನ್ನು ಹೋಲಿಕೆಯ ಅಧ್ಯಯನ”. ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳಲು ಸಹಾಯ ಮಾಡುವುದು. ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಿಮ್ಮನ್ನು ಆಹ್ವಾನಿಸಲಾಗಿದೆ, ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಕಾರ್ಯವಿಧಾನ, ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಅಸ್ವಸ್ಥತೆಗಳನ್ನು ನೀವು ಓದುವುದು ಮತ್ತು ಅರ್ಥಮಾಡಿಕೊಳ್ಳುವುದು ಮುಖ್ಯವಾಗಿದೆ.

1. ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶವೇನು?

ಪ್ರೈಮರಿ ಓಪನ್ ಆಂಗಲ್ ಗ್ಲುಕೋಮಾ, ಸ್ಯೂಡೋ ಎಕ್ಸ್‌ಫೋಲಿಯೇಟಿವ್ ಗ್ಲಾಕೋಮಾ, ಆಕ್ಯುಲರ್ ಹೈಪರ್ಟೆನ್ಷನ್ ಮತ್ತು ಸಾಮಾನ್ಯ ಜನಸಂಖ್ಯೆಯಲ್ಲಿ ಸೆಂಟ್ರಲ್ ಕಾರ್ನಿಯಲ್ ಥಿಕ್ನೆಸ ರೋಗಿಗಳಲ್ಲಿ ಸಿಸಿಟಿ ಮತ್ತು ಐಒಪಿ ನಡುವಿನ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ಅಧ್ಯಯನ ಮಾಡುವುದು ಮತ್ತು ಹೋಲಿಸುವುದು.

2. ಯಾವ ತನಿಖೆಗಳನ್ನು ಬಳಸಲಾಗುತ್ತದೆ? ಯಾವುದೇ ಸಂಬಂಧಿತ ಅಪಾಯಗಳಿವೆಯೇ?

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಒಳಗೊಂಡಿರುವ ವಿವಿಧ ತನಿಖೆಗಳೊಂದಿಗೆ ಯಾವುದೇ ಅಪಾಯಗಳಿಲ್ಲ

I. ಅಲ್ಟ್ರಸೋನಿಕ್ಪ್ಯಾಕಿಮೆಟ್ರಿ

3. ಭಾಗವಹಿಸುವವನಾಗಿ ನನಗೆ ಏನು ಪ್ರಯೋಜನ?

ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆಯು ನಿಮ್ಮ ಕಣ್ಣಿನ ಸ್ಥಿತಿಯ ಅಂತಿಮ ಫಲಿತಾಂಶವನ್ನು ಬದಲಿಸಬಾರದು. ಆದಾಗ್ಯೂ, ಭವಿಷ್ಯದಲ್ಲಿ ರೋಗಿಗಳು ಈ ಅಧ್ಯಯನದಿಂದ ಪಡೆದ ಜ್ಞಾನದ ಫಲಿತಾಂಶವಾಗಿ ಪ್ರಯೋಜನ ಪಡೆಯಬಹುದು. ಸಂಶೋಧನಾ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಡೆಸಿದ ಯಾವುದೇ ಪ್ರಕ್ರಿಯೆಗಳಿಗೆ ನಿಮಗೆ ಹೆಚ್ಚುವರಿ ಶುಲ್ಕ ವಿಧಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನೀವು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದಕ್ಕೆ ಮುಂಚಿತವಾಗಿ ನೀವು ಯಾವುದೇ ಅರ್ಹತೆಯಿಂದ ಯಾವುದೇ ದಂಡ ಅಥವಾ ನಷ್ಟವಿಲ್ಲದೆಯೇ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನೀವು ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಗೌಪ್ಯತೆ

ನಿಮ್ಮ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯನ್ನು ಅಧ್ಯಯನದ ವೈದ್ಯರು ಮತ್ತು ಸಿಬ್ಬಂದಿ ಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದು ಮತ್ತು ಸಾರ್ವಜನಿಕವಾಗಿ ಲಭ್ಯವಿರುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮೂಲ ದಾಖಲೆಗಳನ್ನು ನಿಮ್ಮ ವೈದ್ಯರು ಅಥವಾ ನೈತಿಕ ವಿಮರ್ಶೆ ಮಂಡಳಿ ಪರಿಶೀಲಿಸಬಹುದು. ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ

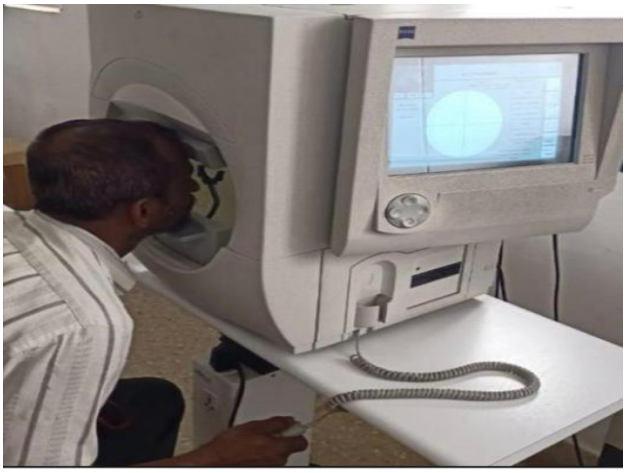
ಡಾ. ರಶ್ಮಿ.ಜಿ,

ಡಾ. ಪೂಜಿತ. ಎಂ

ಎಸ್ ಡಿ ಯು ಎಮ್ ಸಿ.

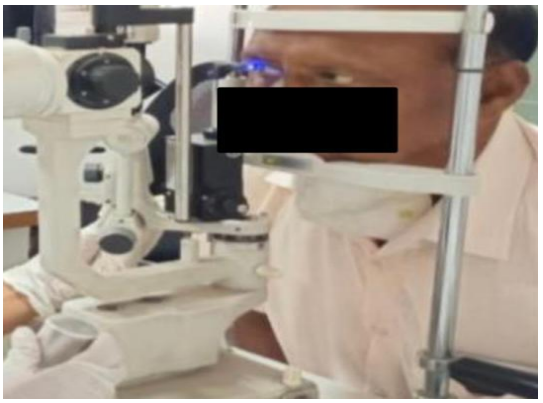
ಟಮಕ, ಕೋಲಾರ ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 9886998871 ಅಥವಾ 8494906087.

ANNEXURE-IV



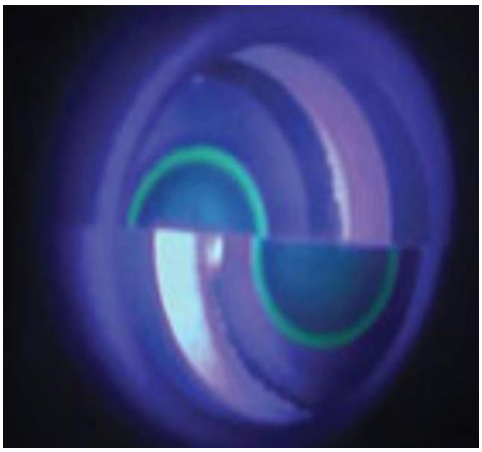
PHOTOGRAPH 1 : HUMPHREYS VISUAL FIELD ANALYSER

PHOTOGRAPH 2: GONIOSCOPY



PHOTOGRAPH 3: GOLDMANN APPLANATION TONOMETRY

PHOTOGRAPH 4: LE- CCT measurement by Ultrasonic Pachymetry



PHOTOGRAPH 5: Slit lamp ocular view of fluorescein semicircles indicating IOP during applanation tonometry.

ANNEXURE-V

KEY TO MASTER CHART

1. **M-** Male
2. **F-** Female
3. **NP–** Normal population
4. **POAG-** Primary open angle glaucoma
5. **PXG-** Pseudo exfoliative glaucoma
6. **OHTN-** Ocular hypertension
7. **CCT-** Central corneal thickness
8. **GAT IOP-** IOP measured by Goldmann Applanation Tonometry
9. **CI IOP-** Corrected IOP

MASTER CHART

UHID	AGE	SEX	GROUP	CCT (µm)	GAT IOP (mmHg)	CI IOP (mmHg)
901842	62	M	NP	524	12	12
887081	49	M	NP	533	16	16
881892	52	F	NP	540	18	18
902795	63	F	NP	530	14	14
897906	56	F	NP	522	12	12
899202	68	M	NP	540	18	18
896895	55	M	NP	526	12	12
879001	49	M	NP	533	16	16
896869	52	F	NP	540	18	18
896291	63	F	NP	532	16	16
896519	56	F	NP	522	12	12
668558	68	M	NP	540	18	18
893986	45	F	NP	524	12	12
864271	49	M	NP	533	16	16
894847	52	F	NP	540	18	18
894818	63	F	NP	530	14	14
890073	56	F	NP	522	12	12
881807	68	M	NP	540	18	18
700605	56	F	NP	522	12	12
937161	68	M	NP	540	18	18
929291	55	M	NP	524	12	12
927711	49	M	NP	533	16	16
911471	52	F	NP	540	18	18
942599	63	F	NP	530	14	14
941214	56	F	NP	522	12	12
939906	68	M	NP	536	18	18

UHID	AGE	SEX	GROUP	CCT (µm)	GAT IOP (mmHg)	CI IOP (mmHg)
936102	68	M	NP	540	18	18
938771	55	M	NP	526	12	12
934211	49	M	NP	533	16	16
933906	52	F	NP	540	18	18
941968	63	F	NP	532	16	16
921729	56	F	NP	522	12	12
948745	68	M	NP	540	18	18
933386	45	M	NP	524	12	12
980112	49	M	NP	533	16	16
945009	52	F	NP	540	18	18
941267	63	F	NP	530	14	14
977143	56	F	NP	522	12	12
943750	68	M	NP	540	18	18
943766	56	F	NP	522	12	12
943754	68	M	NP	540	18	18
943771	55	M	NP	524	12	12
930391	49	M	NP	533	16	16
814048	52	F	NP	540	18	18
813983	63	F	NP	530	14	14
817830	56	F	NP	522	12	12
816148	68	M	NP	536	18	18
711982	62	M	NP	524	12	12
812123	49	M	NP	533	16	16
813893	52	F	NP	540	18	18
802189	52	F	NP	540	18	18
803590	63	F	NP	532	16	16

UHID	AGE	SEX	GROUP	CCT (µm)	GAT IOP (mmHg)	CI IOP (mmHg)
802702	56	F	NP	522	12	12
577924	68	M	NP	540	18	18
793638	45	F	NP	524	12	12
603421	49	M	NP	533	16	16
793523	52	F	NP	540	18	18
797761	63	F	NP	530	14	14
790423	56	F	NP	522	12	12
792665	68	M	NP	540	18	18
828038	62	M	POAG	520	18	20
828671	71	F	POAG	532	26	26
850762	66	F	POAG	554	24	24
822836	60	M	POAG	520	20	24
830629	65	M	POAG	520	12	16
698307	64	M	POAG	516	18	22
824865	55	F	POAG	548	24	22
824165	58	F	POAG	518	12	16
831461	72	M	POAG	512	14	20
831463	66	M	POAG	542	16	22
849987	52	F	POAG	516	16	20
836412	71	F	POAG	540	26	22
833806	66	F	POAG	534	24	22
506490	60	M	POAG	520	20	24
830629	65	M	POAG	518	12	16
830130	64	M	POAG	520	18	22
830147	55	F	POAG	538	24	22
827681	62	M	POAG	518	20	24

UHID	AGE	SEX	GROUP	CCT (µm)	GAT IOP (mmHg)	CI IOP (mmHg)
845977	54	M	POAG	546	18	22
848737	58	M	POAG	532	20	20
895200	62	F	OHTN	566	30	24
895204	51	M	OHTN	542	16	16
898328	63	M	OHTN	538	12	12
898339	48	F	OHTN	558	32	26
856896	55	M	OHTN	540	16	16
893850	58	M	OHTN	563	38	32
873110	48	M	OHTN	554	28	22
901794	62	M	OHTN	560	36	30
886198	72	M	OHTN	550	28	28
901797	52	F	OHTN	552	28	28
901800	60	F	OHTN	566	26	20
858831	51	M	OHTN	538	16	16
820819	63	M	OHTN	532	20	20
859859	48	F	OHTN	563	30	24
860119	55	M	OHTN	540	18	16
860470	58	M	OHTN	568	32	26
860522	48	M	OHTN	570	28	26
861422	62	M	OHTN	566	32	26
862784	56	F	OHTN	569	32	26
865218	62	M	OHTN	570	30	24
874926	59	M	PXG	514	18	22
874537	65	M	PXG	516	20	24
881676	69	F	PXG	502	14	18
874040	60	F	PXG	536	26	26

UHID	AGE	SEX	GROUP	CCT (µm)	GAT IOP (mmHg)	CI IOP (mmHg)
877190	82	F	PXG	528	24	24
876484	61	M	PXG	534	26	26
876859	52	M	PXG	516	12	16
877031	60	M	PXG	512	14	18
879001	56	F	PXG	510	14	20
886193	66	F	PXG	515	12	18
891572	62	F	PXG	510	14	18
896452	69	M	PXG	514	18	22
881936	56	M	PXG	516	20	24
902859	69	F	PXG	502	14	18
873110	58	F	PXG	536	26	26
911010	82	F	PXG	528	24	24
908309	61	M	PXG	534	26	26
911471	52	M	PXG	516	12	16
908487	54	M	PXG	512	16	20
907260	60	F	PXG	518	18	2