A Dissertation on

A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS (PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER)

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the Regulationsfor the Award of the Degree of

M.S. (BRANCH - III) OPHTHALMOLOGY

REG NO. 221913055

GOVT. STANLEY MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 001

MAY 2022

CERTIFICATE

This is to certify that the study entitled **"A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS (PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER)"** is the result of original work carried out by **Dr. P. NANTHINI** , under my supervision and guidance at **GOVT. STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of **M.S Degree in Ophthalmology** , a course from **2019 to 2022** at Govt. Stanley Medical College, Chennai.

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DECLARATION

I hereby declare that this dissertation entitled "**A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS (PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER)**" is a bonafide and genuine research work carried out by me under the guidance of **Prof. Dr. K. VINAYAGAMURTHY M.S.,** Professor, Department of Ophthalmology, Government Stanley Medical College and Hospital, Chennai - 600 001.

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ACKNOWLEDGEMENT

I express my immense gratitude to the Dean, **Prof. Dr. P.BALAJI M.S., FRCS,PhD.** Government Stanley Medical College**,** Chennai for giving me the opportunity to work on the study.

With overwhelming respect and gratitude, I wish to express my heartfelt thanks to my teacher and guide **Prof. Dr.K.VINAYAGAMURTHY M.S.,** Professor, Department of Ophthalmology Government Stanley Medical College **,** Chennai-01**.** for his valuable advice, and guidance, in throughout the completion of the study.

I would like to thank **Prof. Dr.THANGERANI RAAJASEHARAN M.S.,D.O.,** Head of Department for her valuable guidance and suggestions for the study.

I am very grateful to **Prof. Dr.ARUNA ARUMUGAM M.S.,D.O.** for her timely suggestions and guidance.

I specially thank my co-guides , Prof. **Dr.R.SARAVANAN M.S**., and Asst. Prof. **Dr.K.VINODHINI M.S.,D.O** for their valuable suggestions, constant motivation and supervision throughout my thesis work.

My heartfelt thanks to my teachers and Assistant Professors **Dr. K. SIVAKUMAR M.S,DR. S.VENKATESH M.S.,** and **Dr.A.KAVITHA D.O** for rendering their for supervision and assistance throughout the progress of work.

I am thankful to all my colleagues and friends for their support.

I am deeply indebted to all my patients for their sincere cooperation for completion of this study.

Finally, my heartfelt thanks to my family for their encouragement, motivation and timely help during the course of this study.

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Document Information

Analyzed document	thesis final.docx (D123711704)
Submitted	2021-12-27T03:36:00.0000000
Submitted by	P. NANTHINI
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LIST OF ABBREVIATION

SPOAG	Primary Open Angle Glaucoma
SAP	Standard Automated Perimetry
IOP	Intraocular Pressure
TM	Trabecular Meshwork
CCT	Central Corneal Thickness
OHTS	Ocular Hypertension Treatment Study
EGPS	European Glaucoma Prevention Study
SWAP	Short-Wavelength Automated Perimetry
OCT	Optical Coherence Tomography
AS OCT	Anterior Segment - Optical Coherence Tomography
GAT	Goldmann Applanation Tonometer
NCT	Non- contact tonometer
RNFL	Retinal Nerve Fibre Layer
ONH	Optic Nerve Head
HFA	Humphrey Field Analyzers
dB	Decible
SITA	Swedish Interactive Thresholding Algorithm
MD	Mean Deviation
PSD	Pattern Standard Deviation
CPSD	Corrected Pattern Standard Deviation
SF	Short - term Fluctuation
GHT	Glaucoma Hemifield Test
VFI	Visual Field Index
NTG	Normal Tension Glaucoma
FDT	Frequency – doubling technology
ALT	Argon Laser Trabeculoplasty
OHT	Ocular Hypertension
OR	Odds Ratio
BP	Blood Pressure
SHTN	Systemic Hypertension
DM	Diabetes Mellitus
CD ratio	Cup Disc ratio
V1	Visit 1
V2	Visit 2
V3	Visit 3

INTRODUCTION

POAG (primary open-angle glaucoma) is one of the most common causes of blindness around the globe. Until substantial anatomic and functional damage occurs, patients are frequently asymptomatic. Therefore, visual field evaluation is crucial in the treatment and follow-up of glaucoma patients, particularly in mild to severe cases[1,2].

According to glaucoma experts, early diagnosis and treatment of glaucoma are critical for managing the condition and preventing visual loss. The diagnosis of glaucoma requires a thorough examination of the patient's medical history and clinical findings, including visual function tests and imaging procedures[2]. However, the glaucoma community is divided on what constitutes the earliest indications of glaucoma, and individuals with contradicting functional and structural outcomes may be difficult to manage. For these reasons, selective perimetry, formerly thought to be a supplementary test, became standard practise[3,4]. Some of the risk factors are associated with the development of POAG like, increase in IOP, age , familiarity , female sex, hypertension[5] .

Standard automated perimetry (SAP) is the current clinical standard for visual function testing and is essential for glaucoma diagnosis and management.

In SAP, Patients' perceptions in the damage region demand a brighter stimulus of the same size or a bigger stimulus of the same brightness as their visual field deficiencies progress[8]. The retina's spatial summation qualities make more significant, darker stimuli are more reproducible with defect depth than more minor, brighter stimuli.

The present study was carried out to assess the prevalence of primary open-angle glaucoma among glaucoma suspect patients with or without risk factors(predisposing to glaucoma) with the help of automated perimetry (Humphrey field analyzer) to detect the early field changes , to know the progression of visual field defects and also to know the effective time to treat the patients and to know the associated risk factors which correlates to POAG development.

EPIDEMIOLOGY

Prevalence

Primary open-angle glaucoma (POAG) is more common in certain ethnic groups and ethnicities than others. When compared to blacks, whites have a lesser prevalence (1.3 percent) (4.7 percent). Singapore has a Chinese population of 2.4 percent, a Japanese population of 2.6 percent, and an Indian population of 1.7 percent, although Alaskan Inuits (0.1 percent) and Mongols (0.5 percent) have lower prevalence rates. In Ghana, the frequency is 8% among those over the age of 40. POAG is influenced more by age than by race or ethnicity. POAG is rare in those under the age of 40. The prevalence increased from 0.6 percent in those aged 40 to 49 to 7.33 percent in those aged 80 and more[7,8].

Incidence

The incidence during a four-year period was determined to be 2.2 percent 2. It was shown to be age dependent, ranging from 1.2 percent in those aged 40 to 49 to 4.2 percent in those aged 70 and more. POAG has been shown to have a comparable age-dependent incidence rate in studies from Framington, Rotterdam, Australia, and Minnesota[8].

CLINICAL RISK FACTORS

Early intervention and illness prevention may be possible if risk factors are identified.

GENERAL

1. Age: The prevalence rates double every decade beyond 40 years (relative risk of 2), and are nearly ten times higher in the over 80 age group than in the 40 to 49 age group[9]. The percentage of people with optic nerve injury and vision loss rises from 1% in those under the age of 40 to 3 to 8 times greater in people over the age of 70.

2. Race: Blacks have the greatest prevalence, followed by Whites, Hispanics, and South Asians, while North Asians have the lowest incidence[10].

3. Family history: The chance of having POAG increases with first degree relatives. Around 10%–20% of glaucoma patients have a family history of the disease[11].

4. Diabetes: Diabetes affects the vascular tissues in eye and thereby, the retinal neurons and glial components are prone for damage, resulting in retinal ganglion cell death. Neurons and glia that are already vulnerable owing to diabetes are particularly vulnerable to additional stress, such as high intraocular pressure induced by POAG. Diabetes has a greater frequency of POAG and ocular hypertension than the general population[12]. Patients with POAG and steroid responders have been demonstrated to have a greater prevalence of diabetes or a positive glucose tolerance test. Diabetes appears to influence the nature of visual field loss in POAG patients, with a prevalence of inferior field loss of 64.4 percent in diabetics versus 36.4 percent in non-diabetics, respectively, and a 32 percent prevalence of diabetes among POAG patients with primarily inferior loss, compared to 13 percent in those without such a defect[13].

5. Systemic hypertension: When compared to individuals with lower systolic blood pressure, those with a systolic blood pressure exceeding 130 mmHg had a greater prevalence of open angle glaucoma. Normal tension glaucoma is more likely to have nocturnal arterial

hypotension than primary open angle glaucoma with high intraocular pressure (IOP). Blood pressure rise leads to compromise in optic disc circulation resulting to decreased perfusion of the optic disc which contributes to the development of glaucoma[14].

6. Migraine and Vasospasm: Migraine has been identified as a risk factor for open angle glaucoma, since it has been linked to temporary abnormalities in ocular blood flow and peripheral vasospasm. This is more often seen in those who have normotensive glaucoma[15].

OCULAR

1. Intraocular pressure: Elevation of IOP causes apoptosis of ganglion cell neurons through blockage of retrograde axonal transport .The incidence of POAG was reduced by 60% when IOP was reduced by 23% on average. Lowering the pressure causes less progression and more stable visual fields[16,17].

2. Features of the optic nerve head and peripapillary Progressive visual field loss is a concern associated with disc haemorrhages. It's linked to glaucoma with normal tension[18]. Peripapillary atrophy is associated with glaucoma but not exclusive to it. As glaucoma progresses, peripapillary atrophy may develop. With POAG, zone beta atrophy is more prevalent.

3. Myopia: Myopia is a risk factor for glaucoma, with a greater frequency among myopes with a Dioptre difference of more than 6 dioptres[19].

4. Others: Thin corneal thickness , pseudoexfoliation syndrome and pigment dispersion syndrome are more associated with increased risk for progression[20].

ANGLE OF ANTERIOR CHAMBER:

Schwalbe's line, trabecular meshwork, canal of Schlemn and the sclera spur are the main structures forming the anterior chamber angle[21].

Schwalbe's line

The anteriormost structure of the anterior chamber angle is the Schwalbe's line and it is the point where descemet's membrane ends, it also marks the point where corneal endothelium continues as trabecular endothelium.

Trabecular meshwork

The trabecular meshwork is an important structure of the anterior chamber angle playing a vital role in the maintainence of IOP, it is triangular in outline with its apex directed towards Schwalbe's line and base towards sclera spur.

Parts of trabecular meshwork

- Uveal meshwork
- Corneoscleral meshwork
- Juxta-canalicular meshwork

The inner layers of trabecular meshwork are formed by uveal meshwork, the next layer is formed by corneoscleral meshwork and it extends from anterior part of sclera sulcus to sclera spur. Juxtacanalicular meshwork is considered as the main site of resistance for aqueous outflow and it is made up of proteoglycan matrix with embedded collagen and endothelial cells connected by gap junctions[22].

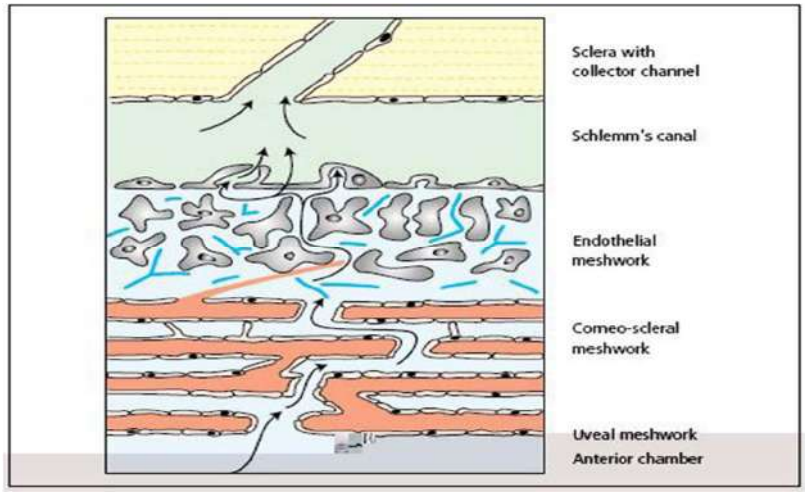


Fig 1. Anatomy of angle of anterior chamber

Schlemn's canal

Schlemn's canal is a vascular channel lying within the sclera sulcus. It consists of two layer of endothelial cells. The outer layer is attached to sclera. The inner wall plays an important role in IOP maintenance by its invaginations called 'giant vacuoles'. These giant vacuoles increase in size and number when IOP increases facilitating aqueous outflow thus reducing IOP. [23]

Scleral spur

Scleral spur is a wedge shaped projection from the anterior sclera and attached anteriorly onto the trabecular meshwork. Posteriorly is attached to sclera and longitudinal ciliary muscle fibres. As sclera spur is attached posteriorly to ciliary muscle, when ciliary muscle contracts it pulls the sclera spur posteriorly which in turn pulls the trabecular lamellae attached to it. This results in widening of intertrabecular spaces and also prevents the schlemn's canal to get narrowed or collapsed, in this way they help in aqueous humor outflow[23].

AQUEOUS HUMOUR DYNAMICS AND DRAINAGE

Aqueous humour is formed in the ciliary process by three important mechanisms namely simple diffusion, ultrafiltration and active secretion. Active secretion of ions across the epithelium of the ciliary body is considered as the primary mechanism for the formation of aqueous these days. Active secretion creates an osmotic gradient which in turn leads to passive flow of water into the posterior chamber. This process is decreased by hypoxia, hypothermia and any inhibitor of active metabolism[24].

Aqueous humor exits the eye via two pathways. The trabecular meshwork is considered the conventional, pressure-dependent pathway, while the uveoscleral pathway is considered the unconventional, pressure-independent pathway.

Mean outflow facility is 0.22 to 0.33 microlitre/min/mmHg.

Trabecular outflow accounts for 5 to 95% of aqueous drainage. This flow is pressure dependent, that is the flow is proportional to the difference between IOP and the hydrostatic pressure in the canal.

In Trabecular outflow, aqueous at the angle of the anterior chamber flows through the trabecular meshwork (TM) of the sclera, into Schlemm's canal and then via its efferent channels, carried to the episcleral vessels, where aqueous mixes with blood[25].

In the uveoscleral outflow pathway, aqueous humor exits the eye through the interstitial spaces between the ciliary muscle fibres into the supraciliary and suprachoroidal space where it is absorbed into the venous system. There are age-related changes to both the TM and the uveoscleral outflow pathways, including decreased TM cellularity with age and increased extracellular depositions in both the TM and uveoscleral pathways that are associated with age-dependent decreased aqueous outflow[25].

GONIOSCOPY AND GRADING OF ANGLE

Gonioscopy refers to the technique used to view and define the structures and abnormalities of anterior chamber angle or iridocorneal angle[26].

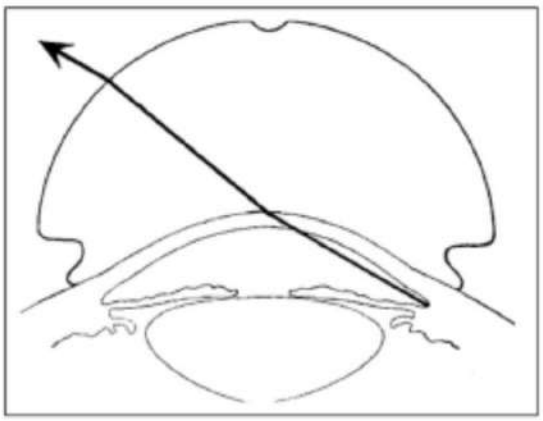
PRINCIPLE

As the light emitted from angle structures undergo total internal reflection, it is not possible to visualize the angle through intact cornea. Contact lenses have an index of refraction similar to that of the cornea, allowing light to enter the lens and then be refracted (goniolens) or reflected (gonioprism) beyond the contact lens-air interface. Gonioscopy can be done by two methods - direct and indirect gonioscopy[27].

Direct gonioscopy:

In this method,anterior curve of the contact lens i.e configuration of the contact lens is such that exiting light rays strike the contact lens/air interface at a steeper angle than critical angle

so that they will pass directly to the observer without reflection inside the lens. Eg. koeppe lens.

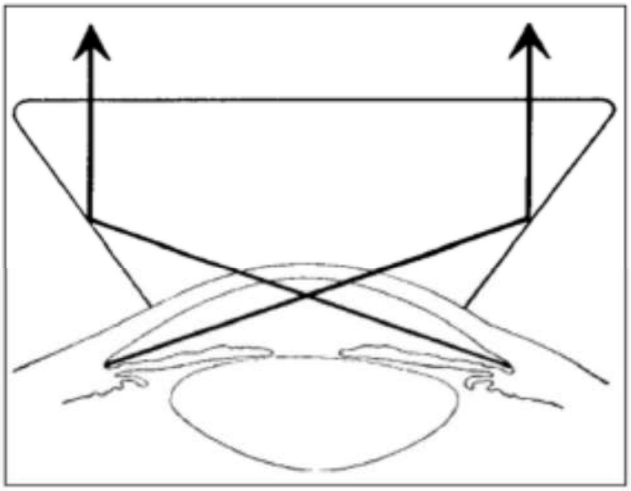


Rays of light from the angle, emerging through a Koeppe lens.

Fig 2. Direct gonioscopy

Indirect gonioscopy :

In indirect gonioscopy light rays from the angle are reflected by a mirror such that they exit the lens at an angle much less than the critical angle. So the angle viewed is the angle exactly opposite to the mirror. Indirect gonioscopy is done with the help of a slit lamp. Eg. Goldmann and Zeiss types of lenses.



Rays of light emerging through a Zeiss indirect gonioscopic lens.

Fig 3. Indirect gonioscopy

ANGLE GRADING:

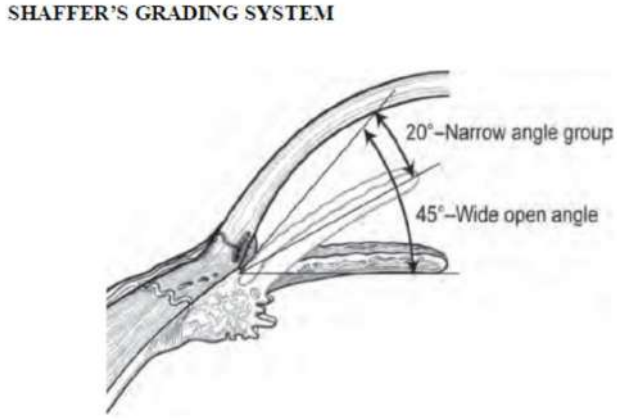
In an open angle structures seen from posterior to anterior are the root of the iris,ciliary body, scleral spur, trabecular meshwork and the Schwalbe's line[28]

Anterior chamber angle is graded by various systems namely,

- SHAFFER GRADING SYSTEM
- SCHEIE GRADING SYSTEM
- SPAETH GRADING SYSTEM

1) SHAFFER'S GRADING SYSTEM

In Shaffer's grading system, anterior chamber angle is graded, considering that the angle is formed between the anterior iris surface and posterior corneal wall.



Grade	Angle Width	Description
4	45-35	Wide open
3	35-20	Wide open
2	20	Narrow
1	10	Extremely narrow
S	<10	Slit
0	O	Closed

Fig 4. Shaffers grading of angle

2) SPAETH GRADING SYSTEM

Spaeth grading of anterior chamber angle is based on three variables

a) Insertion of iris root

b) Angular width

c) Configuration of iris

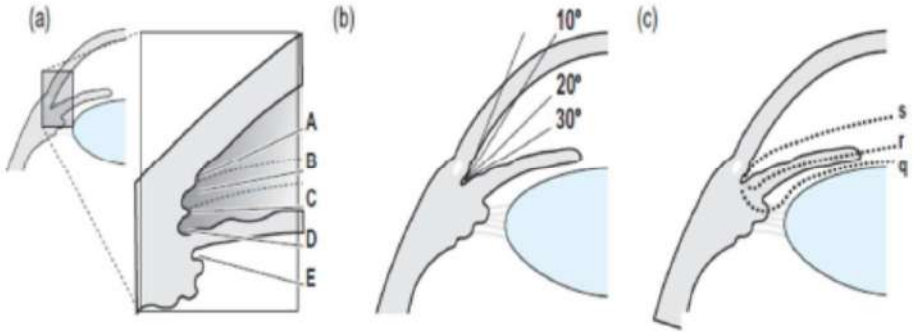


Fig 5. Insertion of iris root

A - Anterior to Schwalbe's line.

B - Behind Schwalbe's line.

C - Centered at scleral spur

D - Deep to scleral spur

E - Extremely deep in ciliary body.

Configuration of iris

s - 'Steep' or convexly configured

r - 'Regular' or flat

q - 'Queer' for deeply concave

3) SCHEIE GRADING SYSTEM

This system is based on the extent of angle structures visualized

All structures seen -Wide open

Iris root not seen - Grade I narrow

Ciliary body band not seen - Grade II narrow

Posterior trabeculum obscured - Grade III narrow

Only Schwalbe's line visible - Grade IV narrow

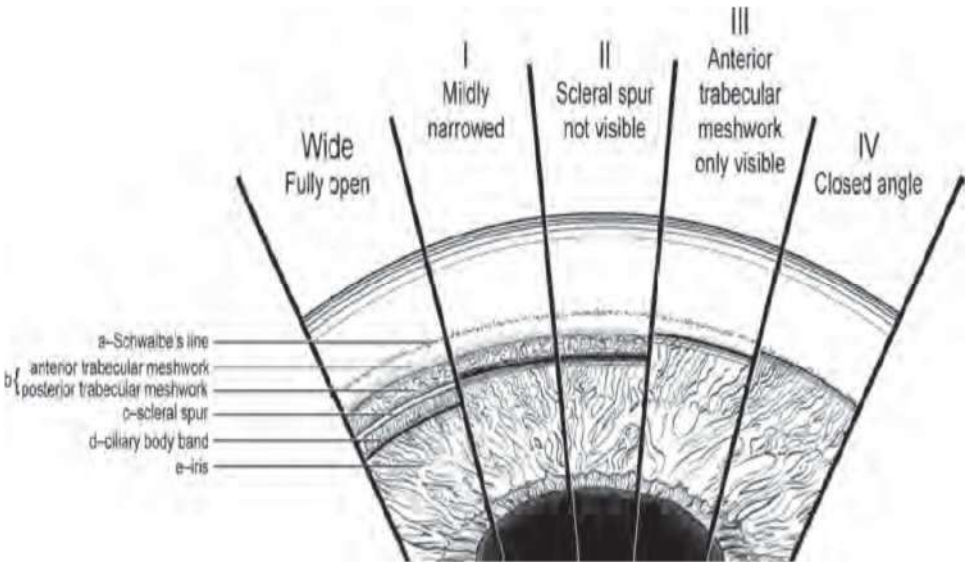


Fig 6. Scheie grading system

INTRAOCULAR PRESSURE

Reduction of IOP remains the only proven and approved means of glaucoma management and is the single most important modifiable risk factor. To cause glaucomatous optic neuropathy, there is a complex interaction between IOP and other risk factors.

Determinants of Intraocular Pressure

Intraocular pressure (IOP) is determined by three factors namely aqueous formation (F), facility of outflow (C), and episcleral venous pressure (Pv).

Goldmann equation relates these factors by the formula

$$Po = F/C + Pv ,$$

or if solving for F then

$$F = (Po - Pv) C$$

in which Po represents the IOP in the undisturbed eye in mmHg, F represents aqueous formation in ul/min, C is aqueous outflow facility in ul/min/mmHg and Pv stands for episcleral venous pressure in mmHg.

From the equation, it is clear increase in IOP occurs when the aqueous formation rate increases the episcleral venous pressure increases or the outflow facility decreases.

Normal intraocular pressure:

The range of intraocular pressures in the normal population is fairly wide; the average intraocular pressure is approximately 16 mmHg with a standard deviation of 2.5. The 'statistical' normal range, defined as the mean two standard deviations, would therefore be approximately 11–21 mmHg.

IOP is influenced by number of factors like age, sex, race, heredity, obesity, posture, exercise etc. IOP is also altered by cholinergic and adrenergic inputs Corticosteroids raise IOP; diabetes associated with increased IOP; myopic individuals have higher IOP.

Diurnal variation -Most people have a diurnal pattern, IOP varies withan average of 3–6 mmHg in normal individuals.

TONOMETER:

The tonometer is a device that measures intraocular pressure by causing deformation of the globe to the force that caused it to distort. Tonometers are divided based on the shape of the deformation: indentation and applanation (flattening).

Applanation tonometry

Principle: Imbert- fick law

In applanation tonometry the intraocular pressure (IOP) is measured from the force required to flatten (applanate) a constant area of the cornea.

A standard portion of the cornea is flattened and the force needed to flatten it is measured. The Goldmann applanation tonometer is the prototype in this category.

Eg. Goldmann applanation tonometer (GAT), Perkins tonometer,

Draeger tonometer, Mackay – Marg and Tono – pen tonometers, Pneumatic tonometer, Non-contact tonometer (NCT) / Ocular Response Analyzer and Ocuton tonometer

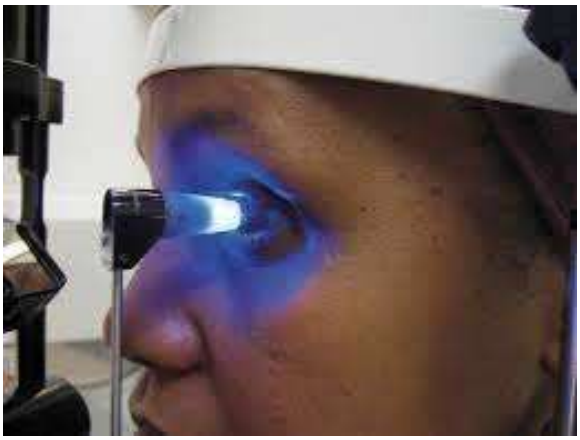


Fig 7. Goldmann applanation tonometer

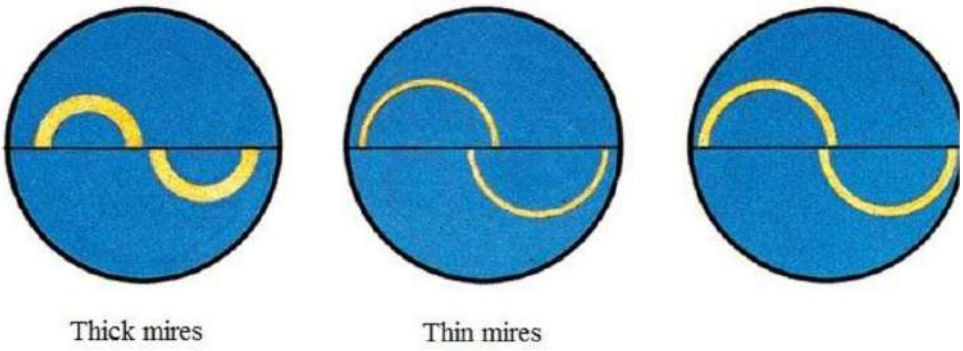


Fig 8: Fluoresceine-stained mires during applanation tonometry

(ii) Indentation instruments

The truncated cone is the shape of deformation. To calculate the IOP, conversion tables must be employed. Schiotz tonometer and Impact rebound tonometer[29]. The Schiotz tonometer is the prototype.

	APPLANATION	NON-CONTACT	SCHIOTZ
ADVANTAGES	1) not affected by ocular rigidity or stretchability of the globe	1) quick and may be delegated 2) no anaesthetic required 3) minimal risk of infection so safely used in post operative eyes[23]	1)Cheap and portable 2) Can be done on supine position 3) Screening purposes
DISADVANTAGES	1) need for anaesthetic 2) cannot be delegated 3) contact with cornea (slight chance of abrasion) 4) affected by central corneal thickness	1) uncomfortable to some patients 2) expensive 3) difficult to obtain reading on scarred corneas	1)Heavy(total weight 16.5g) 2) Corneal abrasions more likely 3) Risk of infection 4) Effect of scleral rigidity on reading 5)steep or thick cornea cause increased displacement fluid, hence it causes false high reading

CENTRAL CORNEAL THICKNESS

Central corneal thickness acts as an intrinsic risk factor for glaucoma damage. There is increased risk of conversion from ocular hypertension to open-angle glaucoma in patients with thin corneas. People of African ancestry had thinner corneas and this is explained as the factor responsible for increased risk for conversion from ocular hypertension to open-angle glaucoma among black population[30].

Ocular Hypertension Treatment Study (OHTS) found the impact of CCT on the development of glaucoma . It determined that a thinner CCT measurement was a strong, independent predictive factor for the development of POAG. Participants with a CCT

<555 µm had three times the risk of developing POAG compared with patients with CCT >588 µm. The risk of developing POAG doubled for every 40 µm decrease in CCT from the overall mean of 573.3 µm in the OHTS and EGPS pooled sample. Other studies have confirmed that a thinner CCT is a risk factor for glaucoma outcomes, such as the presence of advanced glaucoma damage, SWAP visual field defects, and further visual field progression[31]. Intraocular pressure measurement is influenced by corneal thickness, among other factors. Eyes with thin corneas tend to have pressures that are under-estimated by tonometry, while eyes with thick corneas tend to have pressures that are over-estimated.

Manufacturers of devices that measure corneal thickness (such as the corneal pachymeter) often supply clinicians with tables, which help to convert corneal thickness measurements into an "adjustment factor." This adjustment factor is applied to the measured IOP, in order to determine more closely the "true" IOP for a given eye.

Table 1. Correction factors for IOP based on CCT measurements

Central Corneal Thickness (Microns)	Adjustment in IOP (mm Hg)
445	+7
455	+6
465	+6
475	+5
485	+4
495	+4
505	+3
515	+2
525	+1
535	+1
545	0
555	-1
565	-1
575	-2
585	-3
595	-4
605	-4
615	-5
625	-6
635	-6
645	-7

Corneal pachymetry:

Corneal pachymetry is the measurement of the cornea's thickness. It is used before refractive surgery, keratoconus screening, cataract surgery as well as screening for people who may develop glaucoma[32].

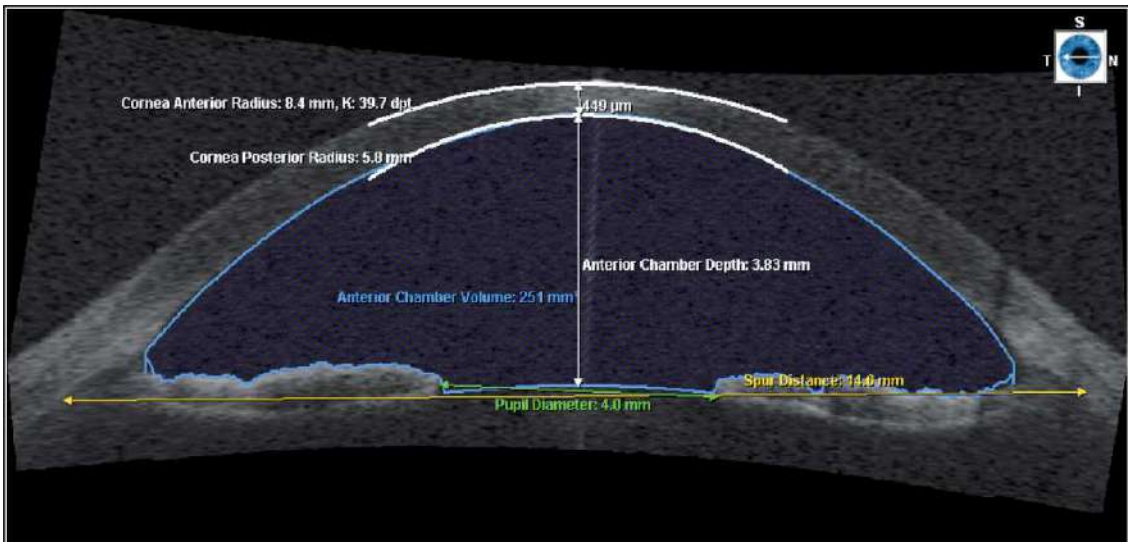


Fig 8. Assessement of Central Corneal Thickness using as OCT

CLASSIFICATION OF GLAUCOMAS

Glaucomas are classified based on[33]:

The etiology (i.e. the underlying pathology that causes alteration of aqueous humour dynamics)

(i) Primary (no obvious systemic or other ocular disorders)

(ii)Secondary (associated with ocular or systemic abnormalities)

The mechanism (i.e. increase in IOP caused by alteration in the anterior chamber angle)

(i) Open angle glaucoma

(ii) Angle closure glaucoma

PRIMARY OPEN ANGLE GLAUCOMA

Chronic open angle glaucoma and chronic simple glaucoma are other names used for POAG. At least two of the three requirements must be met for POAG[34]:

1) Intraocular pressure (IOP) of greater than 21 mm Hg on many occasions, IOP asymmetry of more than 5 mm Hg between the two eyes, and a circadian change in IOP of more than 8 mm Hg

2) Changes in the optic nerve head indicative of glaucoma

3) Typical glaucomatous visual field

A. PATHOGENESIS

Increased IOP is primarily caused by increased resistance to aqueous circulation at the pupil and/or drainage via the anterior chamber angle. The amount of uveoscleral outflow is 20%, which is inadequate to maintain normal IOP.

1) CHANGES IN TRABECULAR MESHWORK[35]

I) Foreign elements obstructing trabecular mesh work include glycosaminoglycans, amorphous material, extracellular lysosomes, plaque-like materials, and proteins.

II) Phagocytosis, as well as the production and breakdown of macromolecules, are disrupted in trabecular endothelial cells.

III) The endothelium of Schlemm's canal has lost its giant vacuoles. The Schlemm's canal is accessed by these vacuoles, which allow fluid to drain from the meshwork.

IV) Endothelial cells are underactive or overwhelmed by foreign material, resulting in cell death and the lack of normal phagocytic activity, i.e. the meshwork's self-clearing filter feature.

V) Decreased permeability of trabecular meshwork due to

a) Increased sensitivity to adrenergic agonists

b) Increased gamma-globulin and plasma cell levels, as well as antinuclear antibodies, in the trabecular meshwork.

c) Altered corticosteroid metabolism

- Elevated plasma levels of cortisol
- Different dosages of exogenous dexamethasone increased plasma cortisol suppression.
- Disturbed pituitary adrenal function
- Glucocorticoids suppress mitogen-stimulated lymphocyte transformation more effectively.
- The steroid responsiveness in POAG patients is controlled by the myocilin (TIGR-trabecular meshwork-inducible glucocorticoid response) gene[36].

2) CHANGES IN OPTIC NERVE HEAD

The local characteristics of the nerve head that play a role in resistance against increased IOP

-

Diameter of scleral ring

Strength of lamina cribrosa

Integrity of vascular supply

<u>Vasogenic theory of nerve damage[37]</u>

This theory implies that structural and functional defects occurring in optic nerve head with glaucoma are due to ischemia. Increased IOP leads to reduced capillary blood flow due to

a) Mechanical compression of vessels at lamina cribrosa

b) Reduced flow in annulus of Zinn which supplies nutrition to laminar and post laminar optic nerve head

Recently, Anderson put forth the hypothesis that inhibition of autoregulation of blood supply to optic nerve can cause increased susceptibility of disc to pressure induced ischemia[38].

Mechanical theory of nerve damage

Initially, increased IOP causes mechanical pressure on the lamina cribrosa, changing capillary blood flow and reducing axoplasmic flow. Later, severe rearward displacement and compaction of the laminar plates narrows the holes through which the axons travel, harming the nerve fibre bundles directly and causing atrophy.

Biochemical theory

Decrease in neurotropic factors / increased levels of neurotoxins.

Genetics (39)

25 loci have been linked with POAG but only three genes have been identified –Myocilin, 21 Optineurin and WDR36.

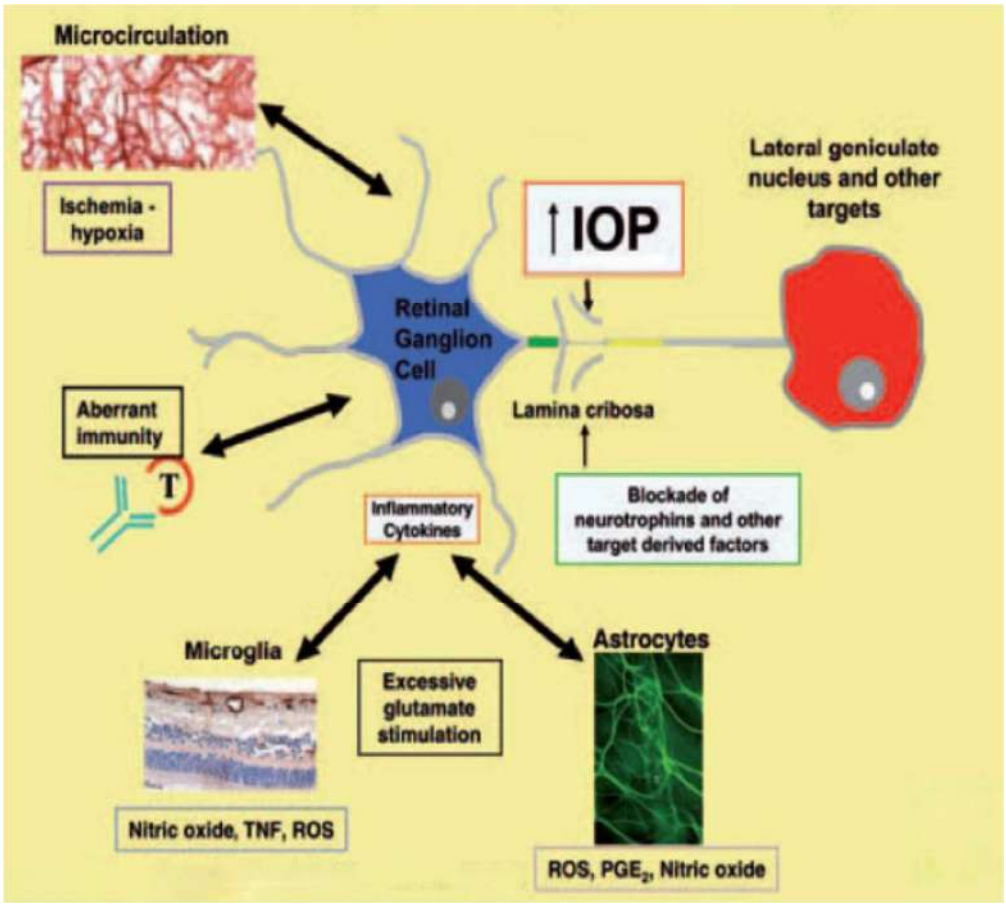


Fig 9. Factors damaging optic nerve in glaucoma

CLINICAL FEATURES

Symptoms

POAG is a slowly progressing, bilateral disorder with an insidious beginning that is generally asymptomatic until the latter stages of the disease. When doing a monocular visual activity, the patient may detect a scotoma or have frequent changes of glasses. As glaucoma progresses, people may have symptoms such as loss of fixation in one or both eyes, as well as loss of peripheral vision and tubular vision, which may make tasks like as driving difficult.

Signs are:

1) Elevated IOP

Elevated IOP may range from 22 to 40 mmHg, with 60 or 80 mmHg being possible in rare occasions. Diurnal fluctuations of less than 5 mm Hg are considered normal, whereas those of greater than 8 mm Hg are considered abnormal. Early in the morning, IOP is highest, and late at night, it is lowest. Diagnosing POAG and explaining increasing damage despite apparent acceptable IOP management may be done using diurnal intraocular pressure readings. It aids in determining the success of treatment and differentiating POAG from normal tension glaucoma[40].

2) **Optic disc changes**

a) Focal atrophy – When the vertical cup-disc ratio exceeds the horizontal cup-disc ratio, the vertical cup-disc ratio becomes greater. The chronological sequence has shifted:

- Polar notching (focal notching or pit like change (pseudopit) - Sharpened polar neural edge, rim, and notching up to the disc border, commonly in the inferior temporal quadrant.
- Bayonetting sign - Sharp bend of the retinal vessels at the disc edge in the areas of sharpened rim.

b) Concentric atrophy: Cup enlargement in concentric rings, most typically inferotemporally or superotemporally directed.

- Temporal unfolding – The loss of neural rim tissue starts at the poles and continues circumferentially[41].

- The thinned out neural rim is seen as cresentic shadow adjacent to the disc margin.

c) Cup deepening: causes increased cupping and exposes the underlying lamina cribrosa (Laminar dot sign)

d) Pallor/Cup discrepancy: Glaucomatous optic atrophy is indicated by cupping greater than pallor, whereas non-glaucomatous optic atrophy is indicated by pallor greater than cupping.

e) Advanced glaucomatous cupping – loss of all neural tissue,

Bean-pot cupping – The vessels bend at the disc's border, and the white disc has lost all of its neural rim tissue[42].

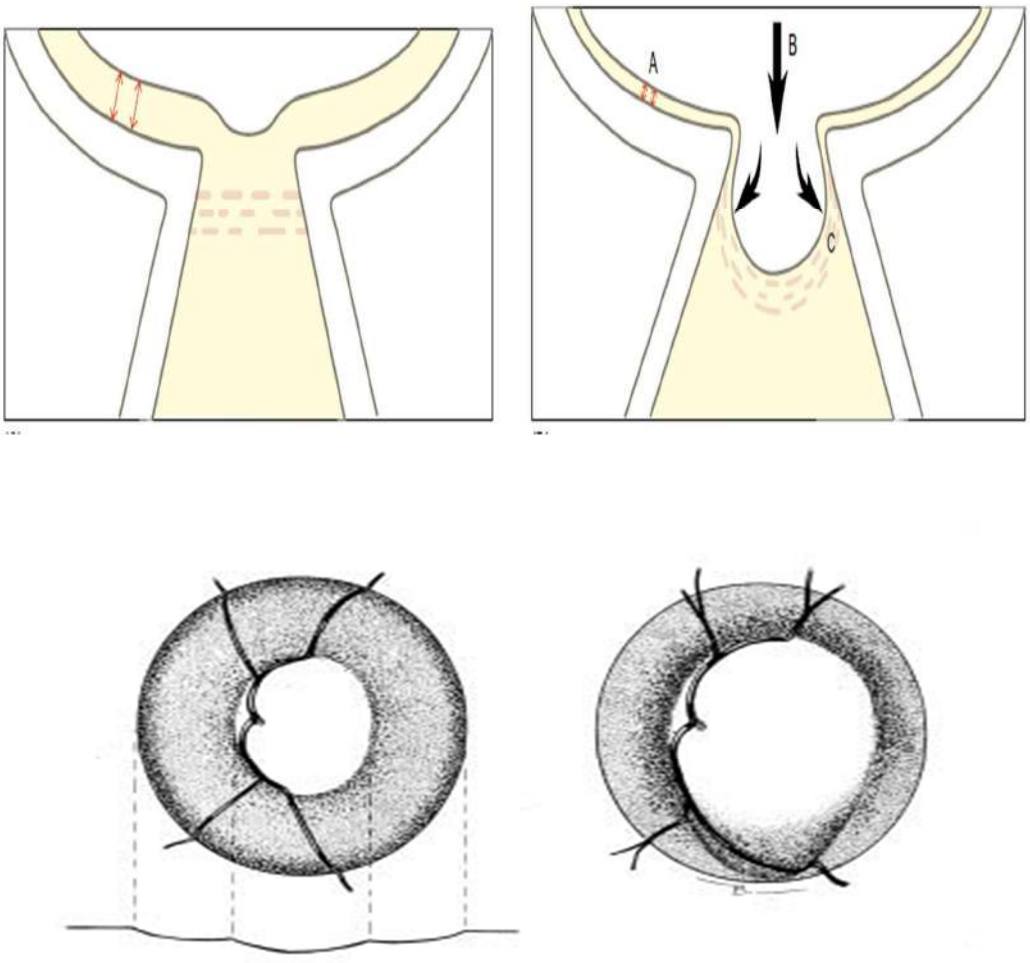


Fig 10. Optic disc cupping

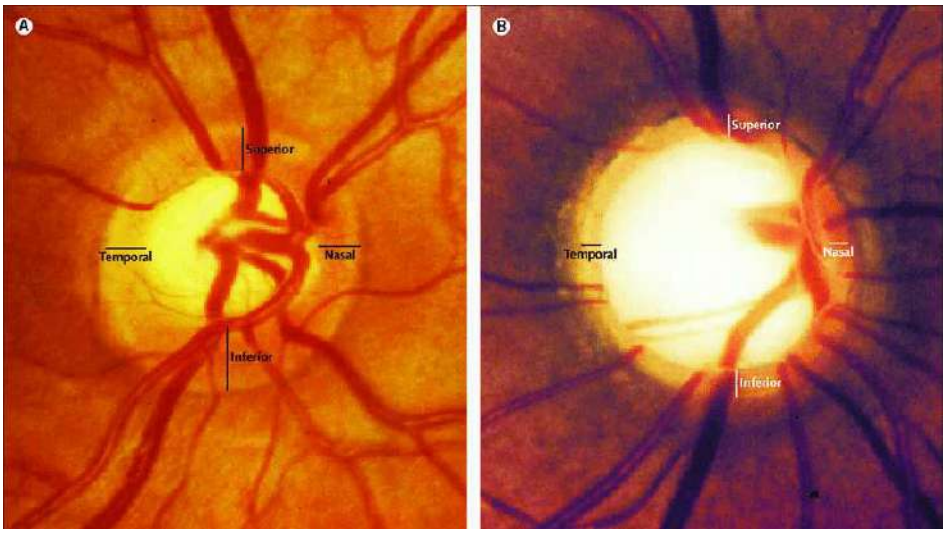


Fig 11. Optic disc (a) Healthy and (b) Glaucotomas

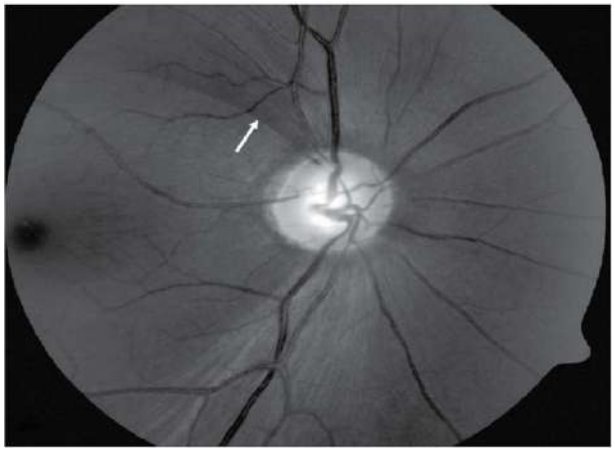


Fig 12. Retinal nerve fiber layer defect

Vascular signs[43]

i) Optic disc haemorrhages

- Splinter haemorrhages near the margin of the optic disc

- Common location is in the inferior quadrant

- Prior to retinal nerve fibre layer problems, notches in the neural rim, and field defects, this might be the earliest indicator of glaucoma.

ii) Tortuosity of retinal vessels is seen in advanced glaucomatous optic atrophy

iii) Location of retinal vessels in relation to the cup

- Overpass cupping
- Baring of the circumlinear vessels

iv) The retinal vascular displacement in the nasal cavity is not a relevant diagnostic measure.

Peripapillary changes

Inner zone beta, which is a depigmented chorio scleral crescent, and outer zone alpha, which has enhanced pigmentation, are the two zones of peripapillary atrophy. Zone beta is more closely linked to glaucoma, and it grows in size as the disease progresses[44].

Nerve fibre bundle defects

Dark stripes, wedge-shaped flaws, or a generalized loss of striations may all be seen. Glaucoma patients are more likely than ocular hypertensives to have diffuse loss[45].

3) **Gonioscopy:**

- This is performed using an indirect goniolens of either the Goldmann or Zeiss 4 mirror type.
- In POAG, anterior chamber angle is open.
- Have more iris processes, a deeper insertion of the iris root, more pigmentation in the trabecular meshwork, and a greater degree of segmentation in the meshwork pigmentation than usual[46].

4) **Visual field abnormalities**

It is originally discovered in the Bjerrum region. Later on, it progresses from paracentral scotomas, nasal step, Seidel scotoma, arcuate or Bjerrum scotoma, ring or double arucate scotoma, tubular vision, to end-stage or near-total deficiency with just a remnant temporal island of vision. The nonspecific alterations include generalized visual field depression, concentric contraction of the visual field, which is more pronounced in the nasal field and is referred to as "crowding of the peripheral nasal isoptres," blind spot enlargement, and

angioscotoma. Progressive visual field loss is the most helpful indicator of POAG in terms of diagnosis, therapy, and follow-up[47].

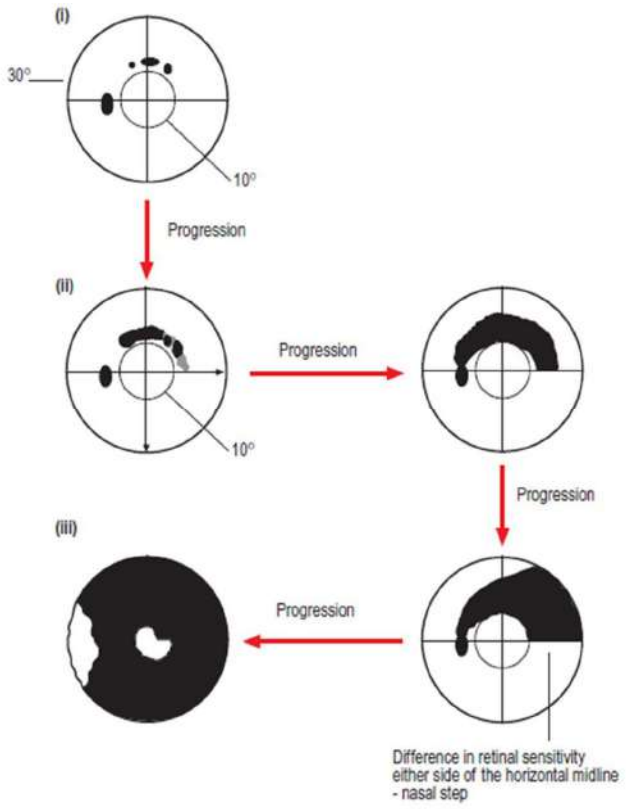


Fig.13. Progression of visual field defect

C.DIAGNOSIS

A diagnosis of POAG can be made after performing the following tests:

1. Intraocular pressure recording

2. Optic nerve head / retinal nerve fibre layer (RNFL) assessment

3. Gonioscopy

4. Visual field analysis

Optic Nerve Head Assessment is done using

- An auxiliary fundus lens and a slitlamp are used to examine the fundus (Goldmann 3 mirror contact lens, the handheld 78 D or 90D lens, Hruby lens slitlamp attachment)

- At each follow-up, a diagrammatic depiction of the disc, neuroretinal rim, vascular changes, and nerve layer abnormalities is drawn.
- Stereo photography of the optic nerve head (ONH) to ascertain small changes sequentially

Analysis of Optic nerve head and Retinal nerve fibre layer is done using

- Direct ophthalmoscope with a red-free filter (ophthalmoscopy)
- Slitlamp and an auxiliary fundus lens with a red-free filter
- The Confocal Scanning Laser Polarimetry concept is used by the RNFL analyzer for glaucoma diagnosis (GDx). It is used to determine the thickness of the RNFL at the peripapillary level.
- In Optical Coherence Topography the ONH, RNFL, and macula can all be imaged using high resolution cross section. The axial resolution is the finest. The macular imaging programme identifies early signs of glaucoma.
- The concept of confocal scanning laser ophthalmoscopy is used in Heidelberg Retina Tomography. It is used to create three-dimensional pictures of the optic disc in order to diagnose glaucomatous damage and track glaucoma development.

PERIMETRY

Perimetry is the technique employed to examine and quantify the visual field using targets of various sizes and colours. It is of two types

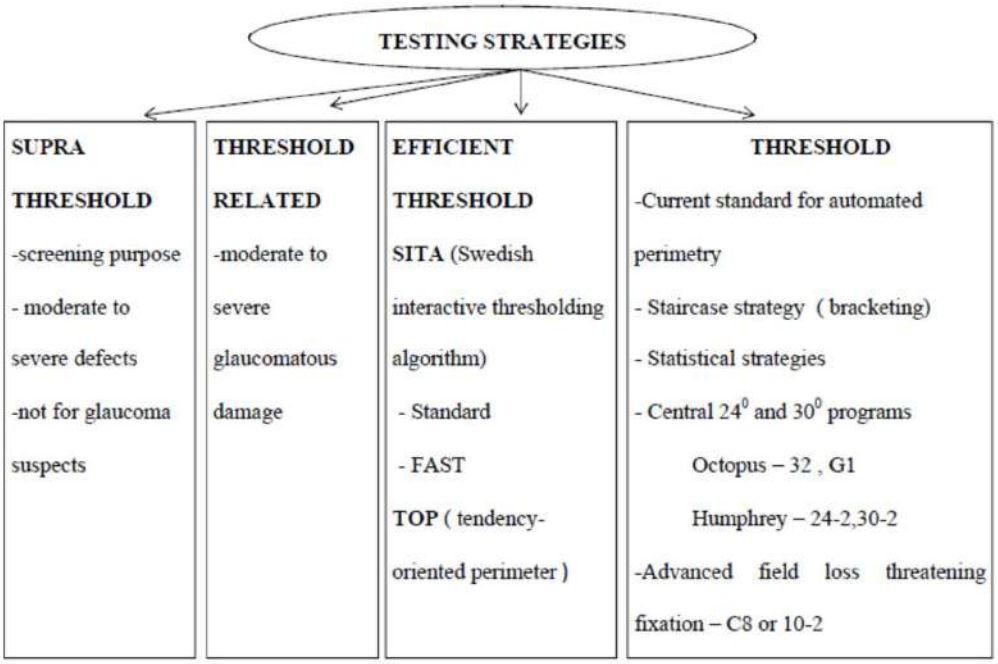
1) Kinetic
2) Static

Static techniques

Static approaches include automated (Humphrey Field Analyzers HFA, Octopus) and manual (Goldmann perimetry). It is the predominant technique of testing in the field, and it employs a

number of different testing methodologies[48]. Automated static perimetry detects and quantifies damage, and in follow-up of a diagnosed patient, it detects stability or progression of loss.

- **HUMPHREY FIELD ANALYZER (HFA)**



SITA-Fast is a quicker method of testing.

There are numerous testing protocols to select, based on the purpose. The first number denotes the extent of the field measured on the temporal side, from the centre of fixation, in degrees. The '-2' represents the pattern of the points tested. They include:

- **10-2:** Measures 10 degrees temporally and nasally and tests 68 points. Used for macula, retinal and neuro-ophthalmic conditions and advanced glaucoma.
- **24-2:** Measures 24 degrees temporally and 30 degrees nasally and tests 54 points. Used for neuro-ophthalmic conditions and general screening as well as early detection of glaucoma

.

- **30-2**: Measures 30 degrees temporally and nasally and tests 76 points. Used for general screening, early glaucoma and neurological conditions.

Properties:

a. **Stimulus size and intensity:** The Humphrey machine can produce stimuli that can vary in brightness in the range over 5.1 log units between 0.08 and 10000 apostilbs. With the standard size 3 stimulus, the dimmest stimulus that can be perceived by a well trained observer is a little less than 40 dB. Hence the dimmest 10 dB range is beyond the perception of the human eye. The standard automated perimetry almost exclusively uses size 3 and in advanced field loss size 5. The other available sizes 1, 3, 4 are rarely used.

b. **Background illumination:** It is an internationally accepted standard to choose 31.5 apostilb as the uniform background illumination for the bowl, the one that was started with then Goldmann machine. The rationale is that it is the minimum intensity for photopic (daylight) cone-related vision. The advantage of testing the photopic system is that it is more contrast than brightness oriented.

c. **Stimulus duration:** Standard stimulus duration is 200 ms, long enough for visibility to be affected by slight changes in duration and adequately short for latency of eye movement.

d. **Stimulus location and disease:** The more peripheral is the location of the stimulus, the more is the intensity required to perceive it, in normal individuals. In glaucoma, however, depending on the location of the defect the retinal sensitivity decreases and the abnormal points require more than the normal intensity for them to "be seen". This results in decrease in the dB value of that point as compared with normals.

INTERPRETATION OF HUMPHERY PERIMETRY CHART

Reliability parameters

There are a set of reliability parameters that are printed at the left upper corner of the printout, which indicate the test performance reliability of the patient. They are fixation losses, false positives, and the false negatives.

Fixation losses: They are calculated using the Heijl - Krakau method. After the localization of the blind spot in the initial part of the test; the machine on multiple occasions, lands a stimulus (5% of total stimuli), on the location of the blind spot. If the patient still responds to such stimulation it is considered as a fixation loss. Fixation losses of greater than 20% are indicative of unreliable field tests.

The gaze tracking graph is a continuous dynamic monitoring of the fixation .It has ups and downs depending on blinks.

False positive error: This is a positive response by the patient even in absence of stimulus . Up to 20 % false positives are acceptable and defects can get masked in cases of high false positivity.

False negative error: Some of the previously thresholded "seen" points are again presented with brighter stimuli and absence of response is considered as a false negative. Conventionally acceptable limit was 20% and the false negatives can be indicator of fatigue as well as disease .

Global indices

There are certain indices that are calculated after the completion of the threshold testing.

Mean deviation (MD): This is the average deviation from the normative data at all the tested points. It has a negative (-) sign. A small localized defect will show a small MD, whereas a generalized or an advanced defect will show a high MD. The value does not differentiate a generalized and a localized field loss. It also does not give the location of the defect.

Pattern standard deviation (PSD): This index gives an idea about the resemblance of the patients' field to the shape of hill of vision. It has a positive sign. Low PSD indicates a normal shape of the hill, whereas a high value indicates a disturbed shape of the hill. Localized defect will give a high PSD, whereas a generalized defect will give a low PSD.

Short - term Fluctuation (SF): Also referred to as the intra-test variability. It is available only with the full threshold printouts. Ten preselected points are thresholded twice and the variation in the thresholds is represented as a number. SF > 3 is considered as an indicator of unreliable result, but is also seen in advanced disease.

Long-term fluctuation: Also known as inter-test variability, should be kept in mind while interpreting the multiple tests over time, however, no machine provides any measure for long-term fluctuation.

Corrected pattern standard deviation (CPSD) : It is the PSD corrected for the SF in the Humphrey and the Octopus, respectively.

***P*-value (probability value):** All the global indices are supplemented with a probability (P < x%) on the side, indicating that less than x% of the normal population has figure like this or in other words there is an x% chance that the index would be seen in normal. Lower the P value beside the global index the higher chance of it being abnormal.

INTERPRETATION OF THE GLOBAL INDICES : All the indices are considered together for interpretation and are inter-related. MD is used for determination of the stage of glaucoma damage. PSD and the CPSD are important for the diagnosis of early glaucoma.

Glaucoma hemifield test

This is useful in the diagnosis of early glaucoma and is available only on the Humphrey. It basically relies on the fact that the glaucomatous defect occurs on either side of the horizontal midline never crossing it and is unlikely to be symmetrical across the horizontal meridian. Thresholds derived at the five sets of points, which are mirror image along the horizontal meridian as shown are compared and the results are displayed as follows: Glaucoma hemifield test (GHT) within normal limits

- GHT borderline
- GHT outside normal limits

The positive GHT does not mean always glaucoma hence, a clinical correlation is required. However, in case of very early glaucoma, a negative GHT can definitely rule out any glaucomatous field defect.

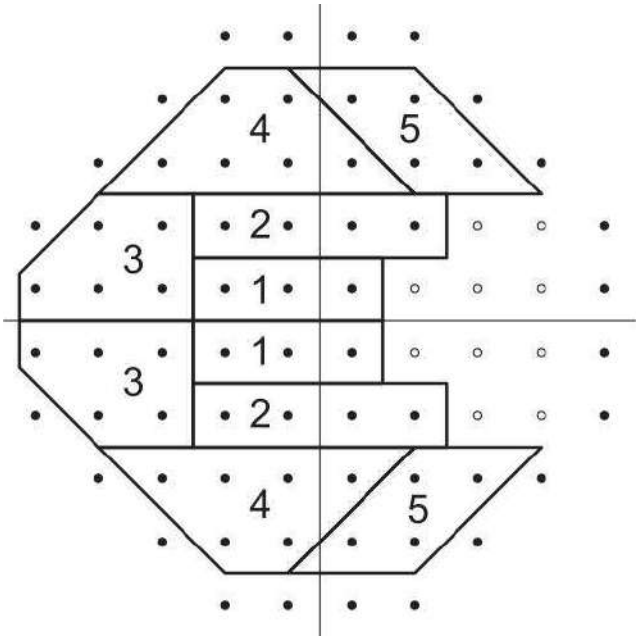


Fig 14. GLAUCOMA HEMIFIELD TEST POINTS

Visual field index: Visual field index (VFI) is a single number that summarizes each patient's visual field status as a percentage of the normal age-corrected sensitivity. It was originally designed to approximately reflect the rate of ganglion cell loss. It is derived from the pattern deviation plot and is centre weighted, considering the high density of the retinal ganglion cells in the central retina.

ANALYSIS OF PERIMETRY PRINTOUT:

General information Reliability indices → Grayscale→ total deviation → pattern deviation → global indices → hemifield test result → RAW DATA → VFI.

Andersen's criteria for glaucomatous field defect: These criteria (in relation to a Humphrey printout only) are helpful in the diagnosis of early glaucoma and are as follows: Abnormal GHT

1. Three or more nonedge points of the 30-2 printout, contiguous and with a $P < 5\%$, out of which at least 1 has a $P < 1\%$ (edge points valid in 24-2 program)
2. CPSD or PSD (in SITA) should be abnormal and should have a $P < 5\%$.

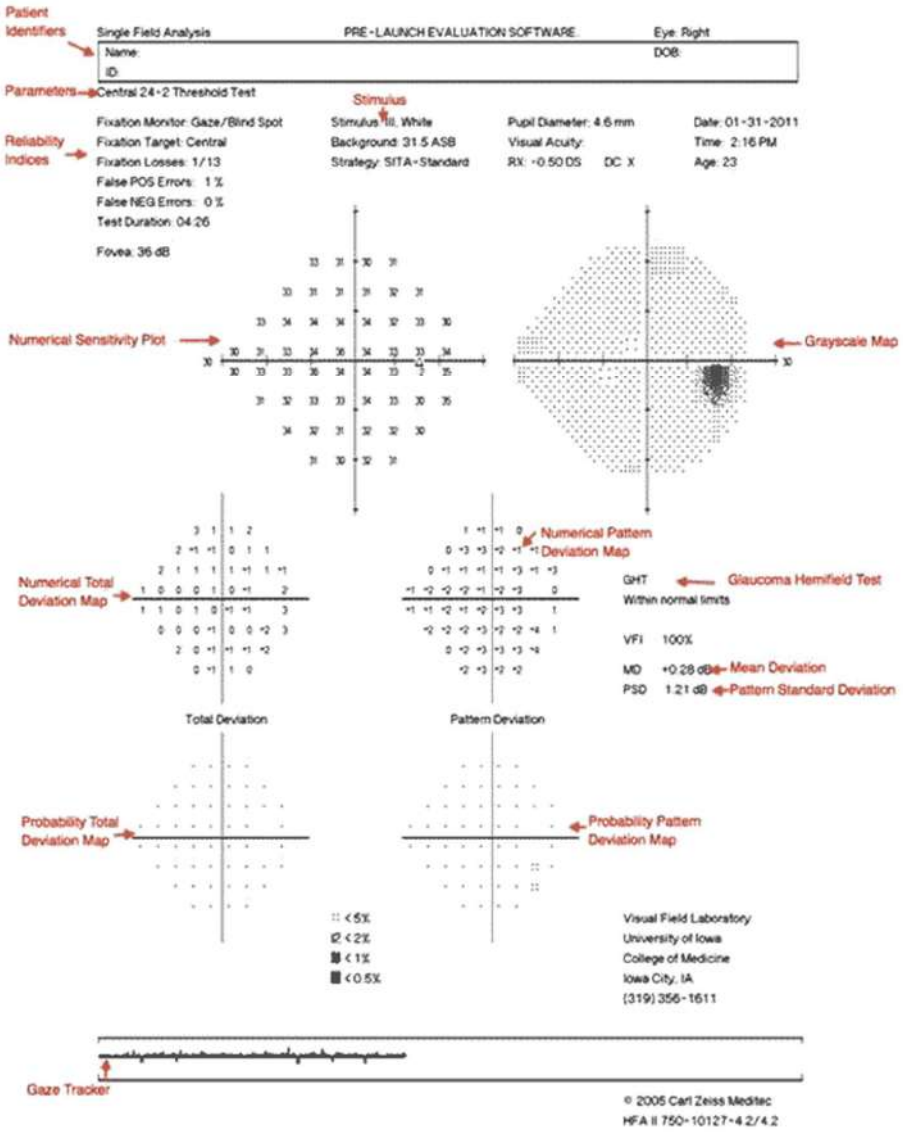


Fig 15. PARAMETERS IN HUMPHREY PERIMETRY CHART

Kinetic techniques

Kinetic perimetry includes techniques such as confrontation, Tangent screen, Lister perimeter, and Goldmann perimeter, in which the stimulus intensity and size are maintained constant but the stimulus position is altered (non seeing to a seeing area).

Newer perimetric techniques[49]

1) Short-wavelength automated perimetry (SWAP)

- blue on yellow perimetry
- helps in early identification of glaucomatous damage by testing small ganglion cells, called bistratified blue-yellow ganglion cells - available on HFA II (700 series) and Octopus 1-2-332

2) Frequency – doubling technology (FDT) perimetry[50]

- A sinusoidal grating with a low spatial frequency undergoes fast phase reversal flicker.
- Early detection of glaucomatous injury should preferentially activate M cells.
- older instrument using 16 to 18 large test fields.
- screening programmes
- new instrument Matrix with 54 smaller test fields

3) HRP – High Pass Resolution Perimetry, also known as ring perimetry

4) Flicker perimetry in the Octopus perimeter

For assessing possible progression

Delta program with the Octopus perimeter

Humphrey Field Analyzer

- STATPAC 2 (incorporates glaucoma change probability and linear regression analysis)
- Progressor Program for analysis of serial fields
- Glaucoma Change Probability (GCP)

- Glaucoma Probability Analysis (GPA)

OCULAR HYPERTENSION

Patients who have an IOP above 21 mm Hg for which there is no apparent cause but whose optic nerve heads and visual fields are normal are commonly said to have ocular hypertension[51]

GLAUCOMA SUSPECT

Open angle by gonioscopy and one of the following in at least one eye[52]:

1) IOP consistently >21 mm Hg by applanation tonometry

2) Glaucomatous injury is indicated by the appearance of the optic disc or the retinal nerve fibre layer.

3) Diffuse or focal narrowing or sloping of the disc rim

4) RNFL anomalies that are widespread or isolated, notably at the superior and inferior poles

5) Disc haemorrhage

6) Asymmetry in the disc or rim between the eyes, indicating the loss of neural tissue

7) Visual fields suggestive of glaucomatous damage in its early stages.

A person suspected to have glaucoma may show signs of optic disc asymmetry or a large physiological cup or may have a presumed predisposition to glaucoma because of risk factors like rise in IOP, positive family history of glaucoma,myopia.

Enlarged optic disc - Persons said to have enlarged optic cups when the ophthalmologist noted excessive excavation of one or both discs during the initial survey examination. Persons with such excessive excavation can be considered as glaucoma suspects.

NORMAL TENSION GLAUCOMA

Normal tension glaucoma (NTG) is a progressive disease

- On diurnal testing, IOP was continuously equal to or less than 21mm, with no single reading over 24mm Hg and no therapy.
- Open drainage angles on gonioscopy
- Absence of any secondary cause for a glaucomatous optic neuropathy
- Typical glaucomatous cupping and loss of neuroretinal rim injury to the optic disc
- Visual field deficiency associated with glaucomatous cupping and neuroretinal rim loss.
- It is a condition that affects the elderly and is more common among women. NTG41's key genetic marker is the OPA gene[53].

REVIEW OF LITERATURE

Quantification of glaucomatous visual field defects with automated perimetry (1985)

A method to quantify different glaucomatous visual field defects is presented in the study. Three visual field indices are calculated: the short-term fluctuation, the mean defect, and the corrected loss variation. The method was applied to visual fields tested with program JO on the Octopus automated perimeter. The indices of 130 glaucoma suspects and 50 glaucoma patients were compared with 100 normal controls. The indices provide good detectability of visual field defects and easy follow-up[54].

A case-control study of risk factors in open angle glaucoma (1987)

In a case-control study using an exploratory health questionnaire, study examined the relationship between POAG and a variety of personal characteristics and potential toxic exposures in patients in a general eye service. There were 83 patients with definite POAG, 121 POAG suspects, and 237 controls. Using multiple logistic regression analysis for simultaneous evaluation of potential risk factors, study found that black race (rate ratio = 6.8; 95%) and untreated systolic hypertension (rate ratio = 5.8; 95%) were the most important risk factors. Current cigarette smoking was also associated with glaucoma (rate ratio = 2.9; 95%). Suggestive associations were found with family history of glaucoma, definite or borderline diabetes, and myopia. The effects of many of these personal characteristics and exposures as risk factors were also noted for the glaucoma suspect group, though not as strongly as for the definite glaucoma cases[55].

Family History and Risk of Primary Open Angle Glaucoma: The Baltimore Eye Survey (1994)

POAG has been previously associated with a positive family history of glaucoma. The current study used data from the Baltimore Eye Survey to examine this association. A population-based prevalence survey identified 161 cases of primary open angle glaucoma among 5308 black and white residents of east Baltimore, Md, who were 40 years of age or older. Family history was ascertained by interview and included all first-degree relatives (parents, siblings, and children. The study concluded that family history as an important risk factor for POAG, although clinic-based studies are likely to overstate its impact[56].

Analysis of risk factors that may be associated with progression from ocular hypertension to primary open angle glaucoma (2002)

As a multifactorial disease, glaucoma may be associated with pressure-dependent and pressure independent factors. Ocular hypertension (OHT) may develop into POAG for many patients. Groups with OHT and POAG were compared for pressure-dependent and independent risk factors. A high prevalence of any factor(s) could indicate a contribution to progression from OHT to POAG sample of patients with POAG ($n = 438$) and with OHT ($n = 301$) were selected from those attending a tertiary referral private glaucoma practice, and data were collected regarding age and intraocular pressure at the time of diagnosis, sex, family history of glaucoma, systemic hypertension, diabetes, Raynaud's phenomenon, migraine and myopia. Patients who had OHT were observed at higher risk of developing POAG if they also have myopia, a family history of glaucoma or are of older age[57].

Diabetes mellitus as a risk factor for primary open-angle glaucoma: a meta-analysis (2004)

The association of diabetes mellitus with primary open-angle glaucoma has been controversial. This study aimed to examine the strength of this association through a detailed meta-analysis of studies published in peer-reviewed journals. A comprehensive search for articles published through 2002 was performed and data were abstracted. Prior to meta-analysis, all studies were evaluated for publication bias and heterogeneity. Pooled odds ratio (OR) was calculated using the random and the fixed-effects model. The present meta-analysis results suggest that diabetic patients are at significantly increased risk of developing primary open-angle glaucoma. Clinicians should be aware of this possibility[58].

Primary open angle glaucoma and intraocular pressure in patients with systemic hypertension (2009)

Study investigated the association between POAG, Intraocular Pressure (IOP) and systemic hypertension. New patients with documented history of hypertension medication of over three months and current attendees at hypertension clinic of the same hospital were enrolled into the study. A total of 150 hypertensive patients and 50 age-sex matched controls were studied. Age range of hypertensives was 34-54 years and controls 31-71 years. The mean age for hypertensive group was 56 years (±12.95) and controls 54.76 years (±9.65). The mean IOP was 28.45mmHg (±10.3) in hypertensive group and 15.2mmHg (±5.09) in controls. POAG was present in 58 (38.7%) of the hypertensive patients whilst only nine (18.0%) had POAG in control group. IOP> 21mmHg was found in 28 (18.7)) of hypertensive group and two (4.0%) of the control. Systemic hypertension showed a modest positive association with elevated intraocular pressure. The strong relationship with IOP in part supports the association with POAG[59].

High myopia as a risk factor in primary open angle glaucoma (2012)

Glaucoma, one of the leading causes of irreversible blindness in the adult population worldwide, is a progressive optic neuropathy. POAG is the most commonly reported type of glaucoma in population based prevalence studies worldwide. Elevated intraocular pressure is a well-known major risk factor for POAG. In addition, there is growing evidence that other risk factors like age, gender, race, refractive error, heredity and systemic factors may play a role in glaucoma pathogenesis. Many studies found that high myopia has been associated with POAG, however, direct and convincing evidences are still lacking. This study summarized the evidences implicating high myopia as a risk factor in the pathogenesis of POAG[60].

Five-year incidence of primary open-angle glaucoma and rate of progression in health center-based Korean population: the Gangnam eye study (2014)

Study investigated the 5-year incidence and progression rate of POAG in a health-center-based Korean population. The study population involves 5,021 subjects who participated in standardized health screening (including non-contact tonometry and fundus photography) at the Gangnam Healthcare Center during the period from January 2005 to December 2006 and again from January 2010 to December 2011. Among these subjects, 948 (18.9%) with findings suggestive of glaucoma were subjected to a comprehensive glaucoma evaluation, which included applanation tonometry and standard automated perimetry. Based on the results, the subjects were diagnosed as POAG suspect or definite POAG. In the health-center-based Korean population, the 5-year incidence of POAG was 0.72%, and the rate of progression from POAG suspect to definite POAG was 4.75% per year. This study identified old age, high baseline IOP, high BMI, high level of education, and high hematocrit level as significant risk factors for incident POAG[61].

Risk factors for POAG Progression: A Study Ruled in Torino (2016):

The study was aimed to describe features of a population sample, affected by POAG in order to evaluate damage progression on the basis of the emerged individual risk factors. Study included 190 caucasian patients (377 eyes), evaluating relationship between individual risk factors (explicative variables) and MD (Mean Deviation) of standard automated perimetry. POAG progression was evaluated through a statistic General Linear Model on four follow up steps (mean follow up 79 months). Results of the study confirm that IOP as the main risk factor for glaucoma progression; age and familiarity are great risk factors; female sex can be important risk factors; arterial hypertension should always be evaluated in timing of our clinic follow up[62].

AIM OF THE STUDY

To analyse the visual field using Static Humphrey Automated Perimetry in cases of glaucoma suspect and to identify the prevalence of POAG among glaucoma suspects .

OBJECTIVES OF THE STUDY

1. To assess the prevalence of POAG among glaucoma suspects using Humphrey Automated Perimetry as confirming tool.
2. To assess the visual field changes in Glaucoma suspects.
3. To assess the predictability of various risk factors associated with suspicion of glaucoma.

STUDY DESIGN: A hospital based Cross sectional study

SETTING : Department of Ophthalmology , Government Stanley Medical College and Hospital, Chennai.

TARGET POPULATION : Patients attending ophthalmology OPD in Government Stanley Medical College Hospital, Chennai.

STUDY PERIOD: March 2020 - November 2021

STUDY POPULATION: All cases of glaucoma suspect attending ophthalmology OPD.

SAMPLING METHOD : Simple random sampling.

SAMPLE SIZE

Previously P. Dahal et al ,Lecturer, Department of Ophthalmology, College of Medical Sciences, Bharatpur, Chitwan district ,Nepal, had did this study on **GLAUCOMA SUSPECT AND HUMPHERY FIELD ANALYZER A CORRELATION.** In this study the prevalence was 22%.Based on this value sample size was calculated.

Using the Formula

n= 4pq/d2

n= 4x22x78/ 100

n= 100

After adding 10% non response rate,

n= 110.

The values taken were,

p =22

q =(100-p)

d – absolute precision (10)

Sample size obtained -110.

MATERIALS AND METHODS

INCLUSION CRITERIA:

Most common risk factors are considered in the study

- Age >45 years
- Family history of glaucoma
- High myopia >-6D
- Increase in IOP
- Diabetes mellites
- Hypertension
- Suspicious optic nerve defect (symmetric and asymmetric increase in CD ratio).

EXCLUSION CRITERIA:

- Known cases of glaucoma
- Glaucoma in one eye
- Angle closure
- Patients with predisposing factors to Secondary Open Angle Glaucomas
- Migraine
- Pseudoexfoliation
- Pigment dispersion syndrome .

DATA COLLECTION

Glaucoma suspects were identified among the OPD patients and 110 glaucoma suspects fulfilling the inclusion criteria were included in the study after getting informed consent **.** Each patient was subjected to a detailed history taking followed by detailed ocular examination as per the enclosed proforma.

Refraction was done in all patients, the patients with high myope (>-6D) were identified and included in the study.

Blood pressure was measured in all patients by sphygmomanometer.

All patients were subjected to slit lamp examination for anterior segment evaluation.

IOP was measured in all patients by Goldman Applanation tonometry .

Central corneal thickness was measured in all patients using AS OCT .

Gonioscopy done for all patients using Goldmann 3 mirror gonio lens and graded according to modified Shaffers grading .

Fundus examination was done by direct and indirect ophthalmoscope followed by +90 D lens under slit lamp for assessment of optic nerve head.

Finally all patients were subjected to perimetry analysis by Humphrey - under strategy of standard SITA, full threshold ,central 24* with Goldmann stimulus size III to assess the visual field defect .

The glaucoma suspect with risk factors like high myope (>-6D) , borderline IOP (≥ 21 mmhg) , increased CD ratio (symmetrical /asymmetrical) and family history of POAG were listed and an automated perimetery with Humphrey was done for all the participants during their visit to the hospital and results were analysed. . An abnormal test result was defined as a glaucoma hemifield test “outside normal limits.” Test results were included only if they were reliable; a test result was considered unreliable if both false positives and false negatives exceeded 33 % and fixation losses exceeded 20 %, respectively.

To quantify visual field deterioration in cases, we monitored P values (probability) of global indices in visual field chart.

Blood pressure, IOP measurement, Gonioscopy, fundus examination and Humphery automated perimetry were repeated for another 2 visits at an interval of 4 to 5 months. The perimetry results were recorded, analysed and compared.

Laboratory investigations – Fasting blood sugar levels.

DATA ANALYSIS

Data are presented as percentages and the number of cases. Categorical data were analyzed with Pearson chi-square tests. Significance was defined by P values less than 0.05 using a two-tailed test. Data analysis was performed using IBM-SPSS version 21.0 (IBM-SPSS Science Inc., Chicago, IL).

OBSERVATION AND RESULTS

OBSERVATION:

VISIT 1 (V1)

Among 110 patients subjected to Humphrey visual field analysis,

22 patients had abnormal visual fields in V1.

- GHT outside normal limits and abnormal Global indices .

Table 2. No. of patients showed abnormal visual field in v1

RISK FACTORS	NO. OF PATIENTS	PERCENTAGE
Increased IOP	10	45.5%
Family history	1	4.5%
Myopia > -6D	0	0%
Increased CD ratio	9	41.0%
Family history + increased IOP	2	9.0%
TOTAL	22	100%

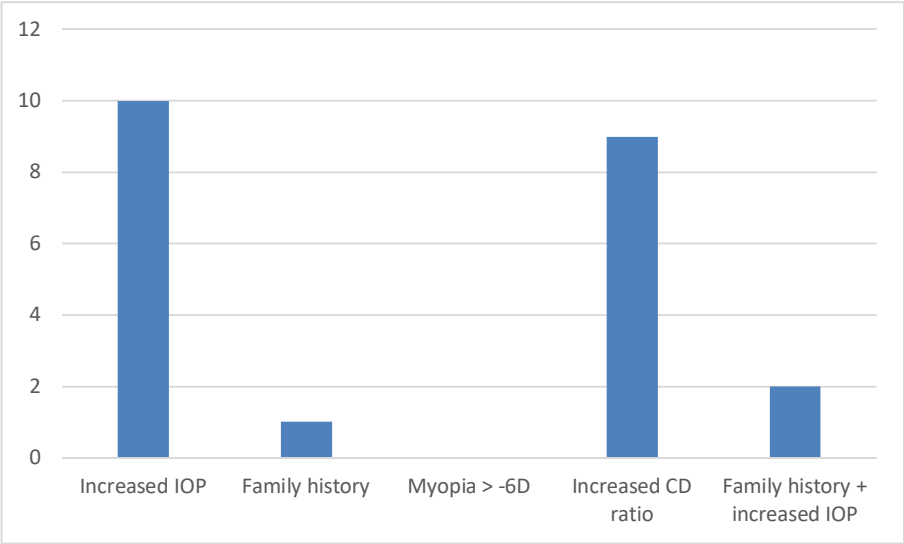


Fig 16. No. of patients showed abnormal visual field in v1

VISIT 2 (V2)

Among 22 patients who had abnormal fields in visit 1, 9 patients showed GHT outside normal limits and increase in significance of P values in global indices , in visit 2 .

Remaining 13 patients who showed abnormal fields in visit 1, did not show any significant change of P values in global indices (mentioned as static) in perimetry as compared to previous visit.

The 88 patients, who showed no changes in visit 1, did not show any further change in visual field in visit 2 also.

Table 3. No. of patients showed abnormal visual field in v2

RISK FACTORS	NO. OF PATIENTS	PERCENTAGE
Increased IOP	7	77.8%
Family history	0	0%
Myopia > -6D	0	0%
Increased CD ratio	1	11.1%
Family history + increased IOP	1	11.1%
TOTAL	9	100%

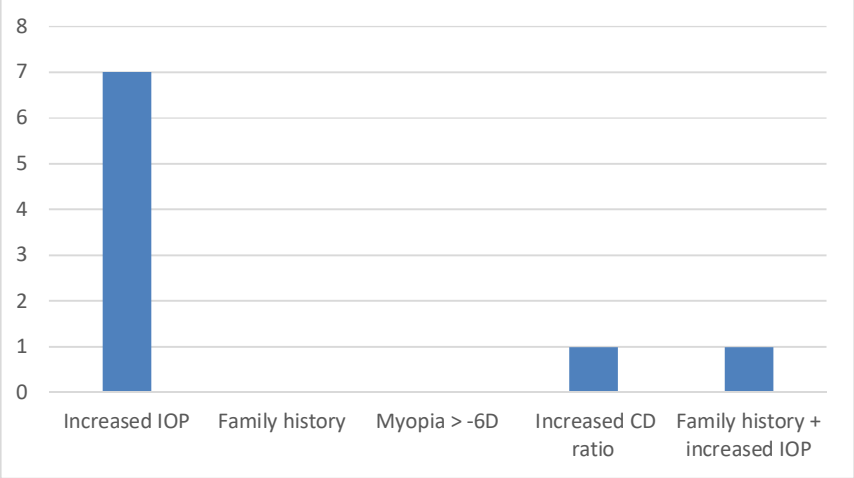


Fig 17 . No. of patients showed abnormal visual field in v2

VISIT 3 (V3)

Among the 9 patients, who had abnormal GHT and significant increase in P values of global indices in V2 , 2 patients showed further significant increase in P values of global indices in visit 3 and they fulfilled the Anderson criteria.

The remaining 7 patients did not show any significant increase in P values of global indices (mentioned as static).

13 patients who remained static in visit 2, continued to remained static (did not show any significant increase in P values of global indices) in visit 3 also.

The 88 patients ,who showed normal fields in V1 and V2 showed normal field in visit 3 also.

Table 4. No. of patients showed abnormal visual field in v3

RISK FACTORS	NO. OF PATIENTS	PERCENTAGE
Increased IOP	1	50%
Family history	0	0%
Myopia > -6D	0	0%
Increased CD ratio	0	0%
Family history + increased IOP	1	50%
TOTAL	2	100%

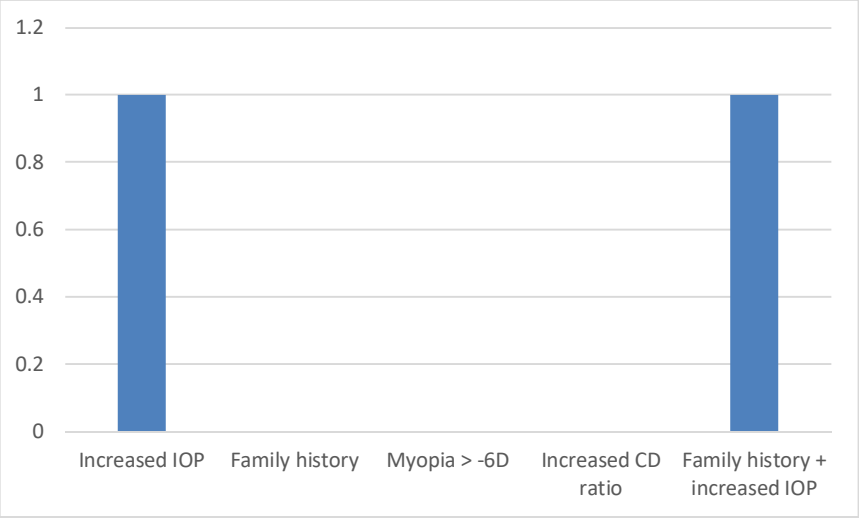


Fig 18. No. of patients showed abnormal visual field in v3

RESULTS

In this study , all 110 glaucoma suspect patients fulfilling the inclusion criteria are followed up for 3 visits at a gap of 4 to 5 months with Humphrey visual fields and results were compared.

All the 110 patients in the study are assessed for any risk factors like increased IOP, Family history , myopia > - 6 D, symmetrical and asymmetrical increase in CD ratio.It was observed that increased IOP in 38 (34.5%) patients, positive family history of glaucoma in 32 (29%) patients, myopia >- 6 D in 13 (11.8%) patients, symmetrical and asymmetrical increase in CD ratio in 11(10%) and 14 (12.8%) respectively. There are overlapping of more than one risk factors in 2 (1.8%) patients especially family history with increased intraocular pressure. (Table:5, Fig.19)

TABLE 5: Distribution of risk factors among glaucoma suspect patients

RISK FACTORS	NO. OF PERSONS	PERCENTAGE
Increased IOP	38	34.5%
Family history	13	11.9%
Myopia > -6D	14	12.8%
Increased CD ratio	43	39.0%
Family history + increased IOP	2	1.8%
TOTAL	110	100%

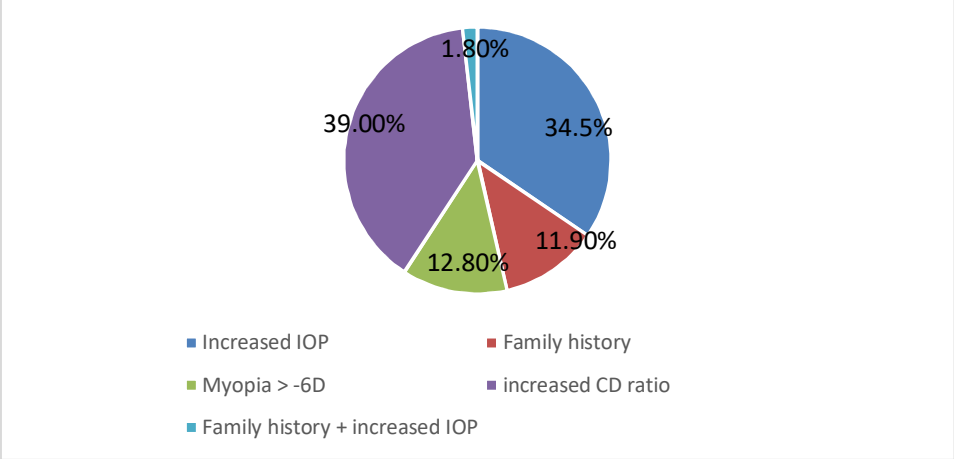


Fig 19 : Distribution of risk factors among glaucoma suspect patients

In the present study total of 110 patients were enrolled, maximum of patients 60 (54.5%) were reported in the age group of 51 to 60 years, followed by an age group of < 50 years with 29 (26.4%) patients and minimum patients 21 (19.1%) were observed in the age group of > 61 years. (Table 6, Fig 20).

Table 6: Age group distribution of enrolled patients.

AGE GROUP	Frequency	Percent
<50	29	26.4
51-60	60	54.5
>61	21	19.1
Total	110	100

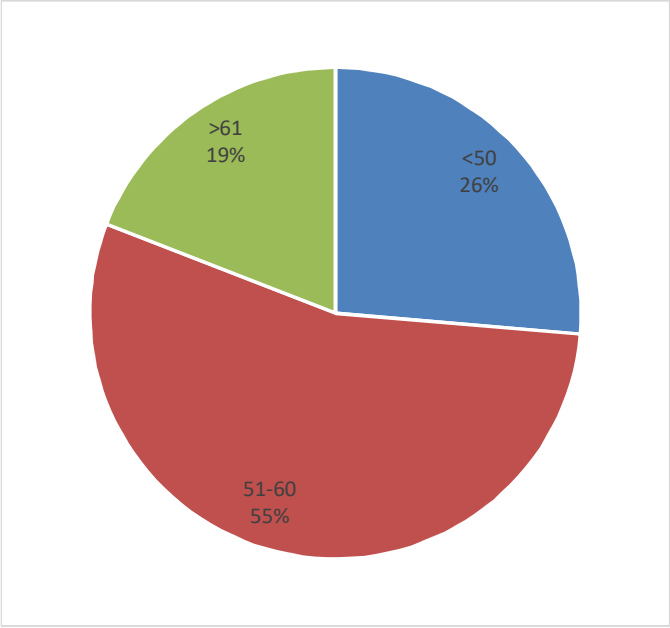


Fig 20: Age group distribution of enrolled patients.

Of all 110 patients, males were found to be predominant than females. The male patients were 63 (57.3%), whereas female patients were reported 47 (42.7%) in the present study (Table 7, Fig 21).

Table 7: Gender distribution of all patients.

GENDER	Frequency	Percent
F	47	42.7
M	63	57.3
Total	110	100.0

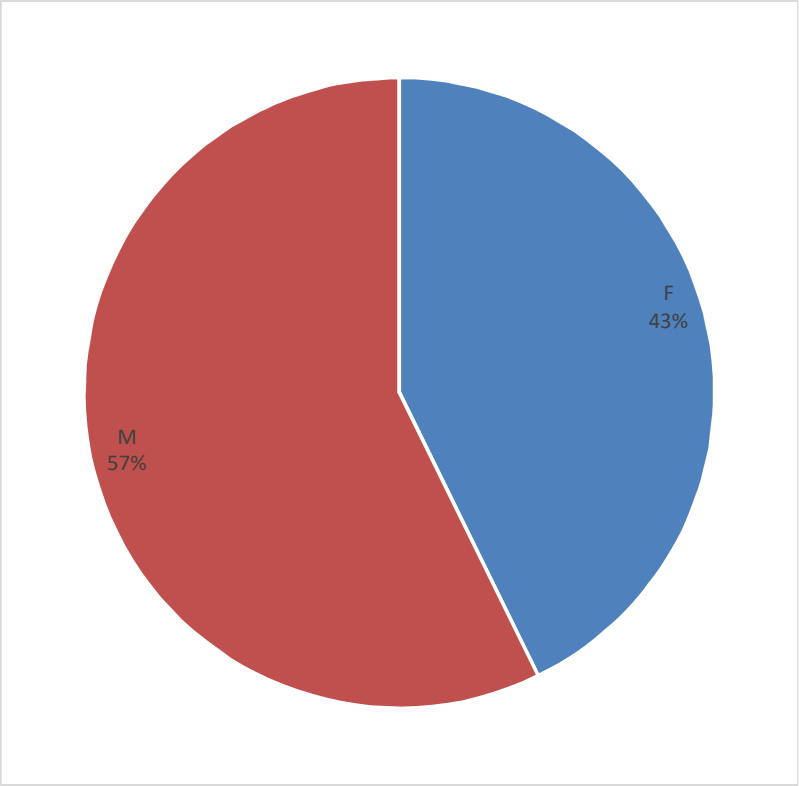


Fig 21: Gender distribution of all patients.

Presence or absence of family history of open angle recorded in all patients . Total 13 (11.8%) patients were observed to have a family history of open angle glaucoma, whereas 97 (88.2%) patients were found without any family history of open-angle glaucoma (Table 8, Fig 22).

Table 8: Distribution of family history of glaucoma among patients

FAMILY HISTORY	Frequency	Percent
NO	97	88.2
YES	13	11.8
Total	110	100.0

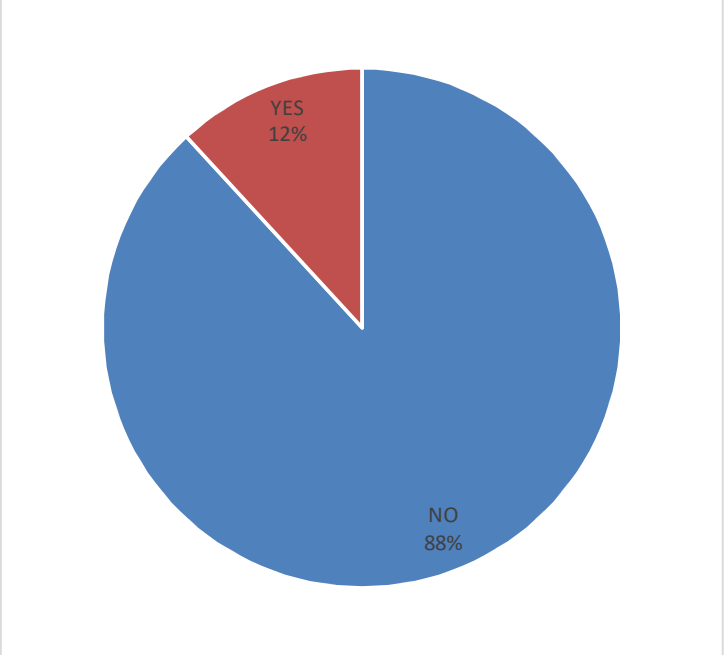


Fig 22: Distribution of family history of glaucoma among patients

The presence of myopia(>- 6 D) , a predisposing refractive error to open angle glaucoma was recorded and included as a risk factor in the present study, and it was found that 14 (12.8%) patients were observed with myopia, whereas 95 (87.2%) patients were observed with non myopia. (Table 9, Fig 23).

Table 9: Distribution of myopia among patients

MYOPIA	Frequency	Percent
NO	96	87.2
YES	14	12.8
Total	110	100.0

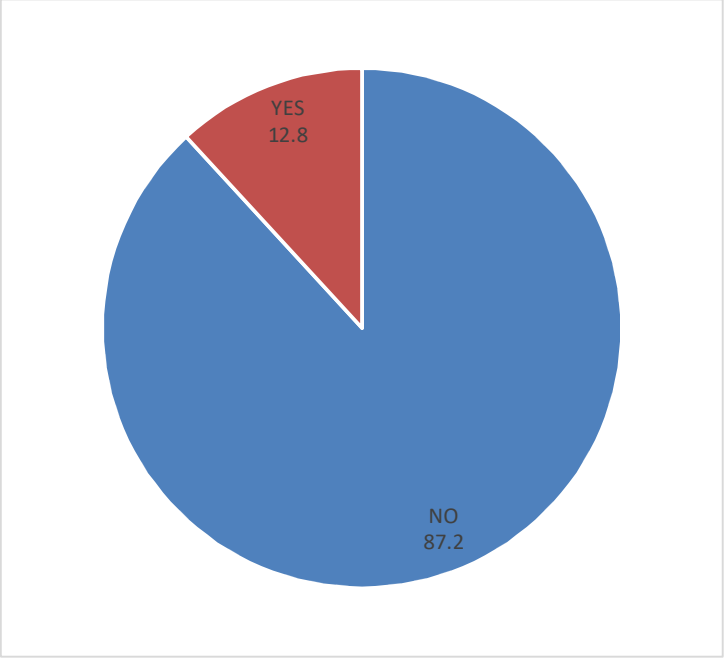


Fig 23: Distribution of myopia among patients

All patients were examined for any co-morbidity like DM, and it was observed that 43 (39.1%) patients had DM , whereas 67 (60.9%) patients were reported free from DM (Table 10, Fig 24)

Table 10: Observation of DM in all patients

DM	Frequency	Percent
NO	67	60.9
YES	43	39.1
Total	110	100.0

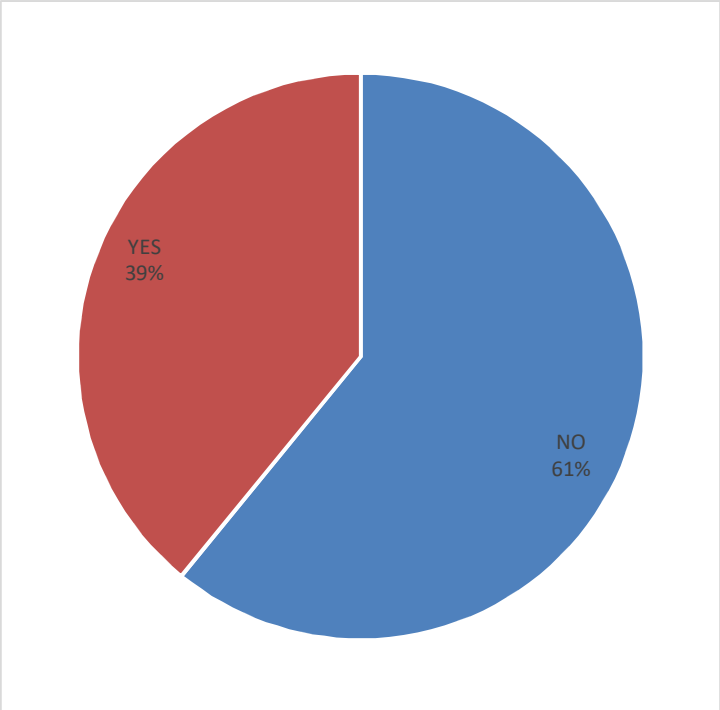


Fig 24: Observation of DM in all patients

Another metabolic abnormality / comorbidity like systemic hypertension (SHTN) was also recorded among all patients, SHTN was observed in 44 (40%) patients, whereas 66 (60%) patients were found without SHTN (Table 11, Fig 25)

Table 11: Observation of SHTN in enrolled patients

SHTN	Frequency	Percent
NO	66	60.0
YES	44	40.0
Total	110	100.0

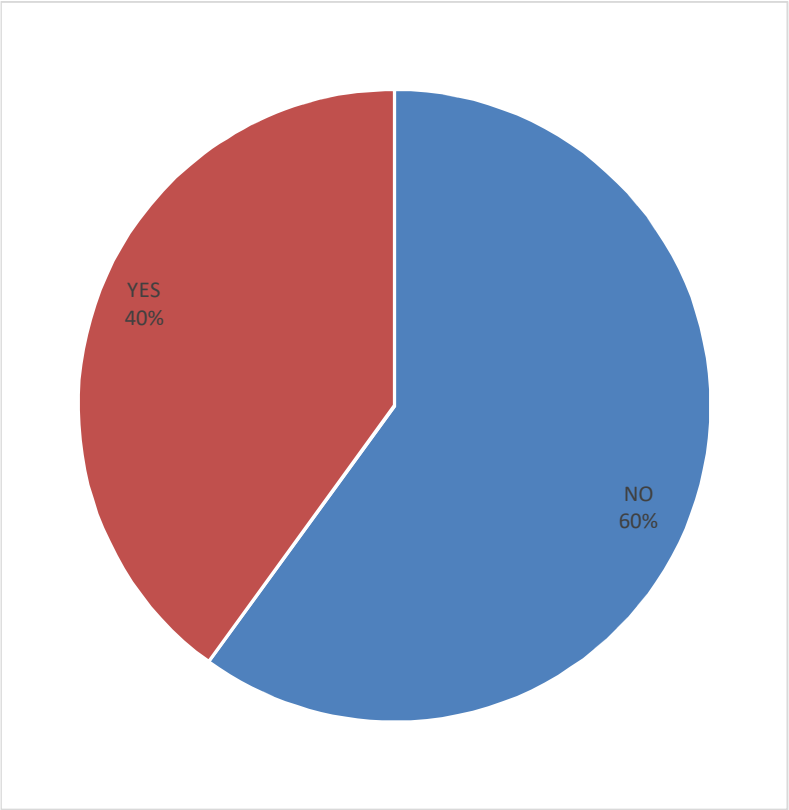


Fig 25: Observation of SHTN in enrolled patients

The visual fields done for the study population at first ,second and third visits were named as V1,V2 and V3 respectively.

Visual field V1 parameters were compared with visual field V2 in all the patients. In the present study it was found that, among 22 patients with abnormal visual field in V1, 9 (40.9%) patients had abnormal visual field in V2 and 13 (59.1%) patients had static visual field in V2. Whereas, 88 (100%) patients who had normal visual field in V1 were showed to have normal visual field in V2 also (Table 12, Fig 26).

Table 12: Comparison of Visual field V1 to Visual field V2

			VISUAL FIELD V2			Total	P value
			ABNORMAL	NORMAL	STATIC		
VISUAL FIELD V1	ABNORMAL	Count	9	0	13	22	<0.0001
		% within VISUAL FIELD V1	40.9%	0.0%	59.1%	100.0%	
	NORMAL	Count	0	88	0	88	
		% within VISUAL FIELD V1	0.0%	100.0%	0.0%	100.0%	
Total		Count	9	88	13	110	
		% within VISUAL FIELD V1	8.2%	80.0%	11.8%	100.0%	

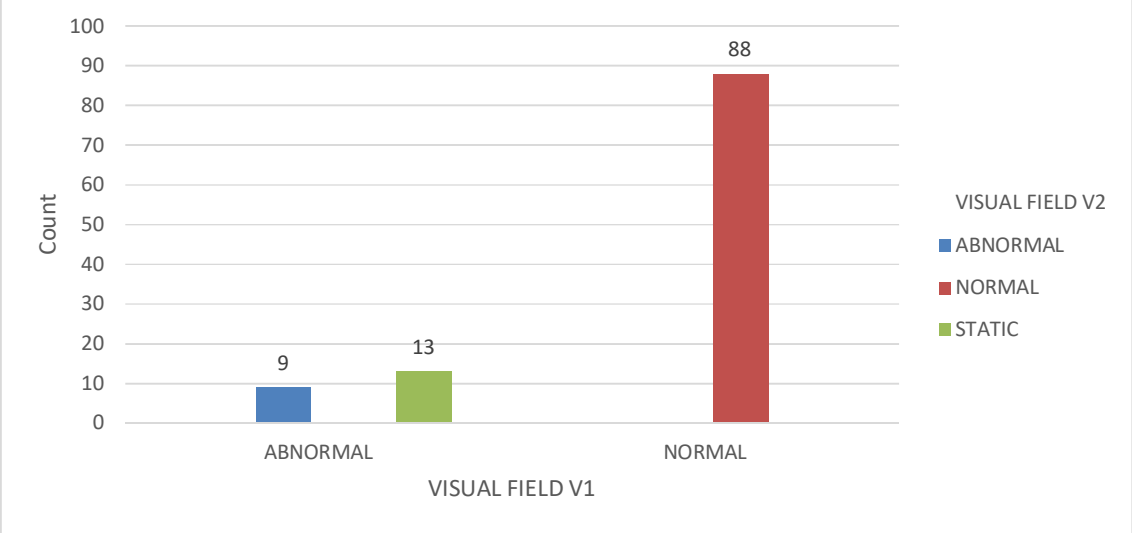


Fig 26: Comparison of Visual field V1 to Visual field V2

Visual field V2 parameters were compared with V3 in all patients. In the present study, it was found that among the 9 patients with abnormal visual field inV2, 2 (22.2%) patients were observed to have abnormal visual field in V3 and 7 (77.8%) patients had static visual field in V3. 88 (100%) patients with normal visual field in V2 were observed to have normal visual field in V3 also.(Table 13, Fig 27).

Table 13 : Comparison of Visual field V2 to Visual field V3

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
VISUAL FIELD V2	ABNORMAL	Count	2	0	7	9	<0.0001
		% within VISUAL FIELD V2	22.2%	0.0%	77.8%	100.0%	
	NORMAL	Count	0	88	0	88	
		% within VISUAL FIELD V2	0.0%	100.0%	0.0%	100.0%	
	STATIC	Count	0	0	13	13	
		% within VISUAL FIELD V2	0.0%	0.0%	100.0%	100.0%	
Total		Count	2	88	20	110	
		% within VISUAL FIELD V2	1.8%	80.0%	18.2%	100.0%	

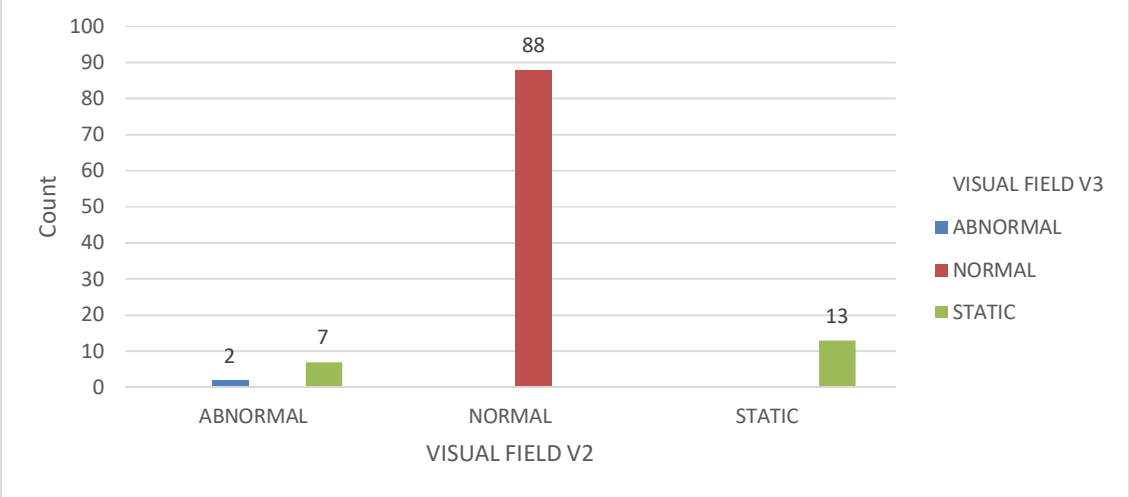


Fig 27: Comparison of Visual field V2 to Visual field V3

Visual field V1 parameters were evaluated with visual field V3. In the present study, it was found that among 22 patients with abnormal visual field in V1, 2 (9.1%) patients had abnormal visual field in V3 and 20 (90.9%) patients had static visual field in V3. Whereas patients who showed normal visual field in V1, showed no abnormal and static visual field in V3 (Table 14, Fig 28).

Table 14 : Comparison of Visual field V1 to Visual field V3

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
VISUAL FIELD V1	ABNORMAL	Count	2	0	20	22	<0.0001
		% within VISUAL FIELD V1	9.1%	0.0%	90.9%	100.0%	
	NORMAL	Count	0	88	0	88	
		% within VISUAL FIELD V1	0.0%	100.0%	0.0%	100.0%	
Total		Count	2	88	20	110	
		% within VISUAL FIELD V1	1.8%	80.0%	18.2%	100.0%	

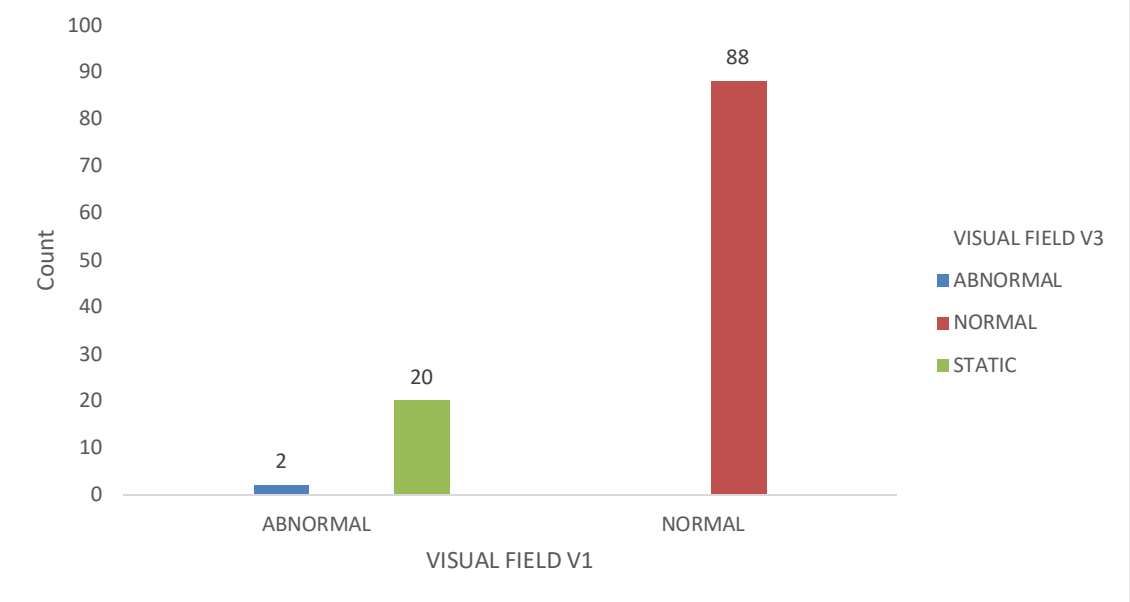


Fig 28 : Comparison of Visual field V1 to Visual field V3

Since the study ended with V3 as a last visit , its results were taken as significant and analysed.

Different age groups were correlated with visual field V3; it was found that the age group of 51 to 60 years did not have abnormal visual field. The age group of less than 50 years and more than 60 years were found to have abnormal visual field with 1 (3.4%) and 1 (4.8%) respectively. In all age groups, many patients showed normal visual field in V3 (Table 15, Fig 29)

Table 15: Observation of different age groups of patients in V3

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
AGE GROUP	<50	Count	1	26	2	29	0.027
		% within AGE GROUP	3.4%	89.7%	6.9%	100.0%	
	51-60	Count	0	50	10	60	
		% within AGE GROUP	0.0%	83.3%	16.7%	100.0%	
	>61	Count	1	12	8	21	
		% within AGE GROUP	4.8%	57.1%	38.1%	100.0%	
Total		Count	2	88	20	110	
		% within AGE GROUP	1.8%	80.0%	18.2%	100.0%	

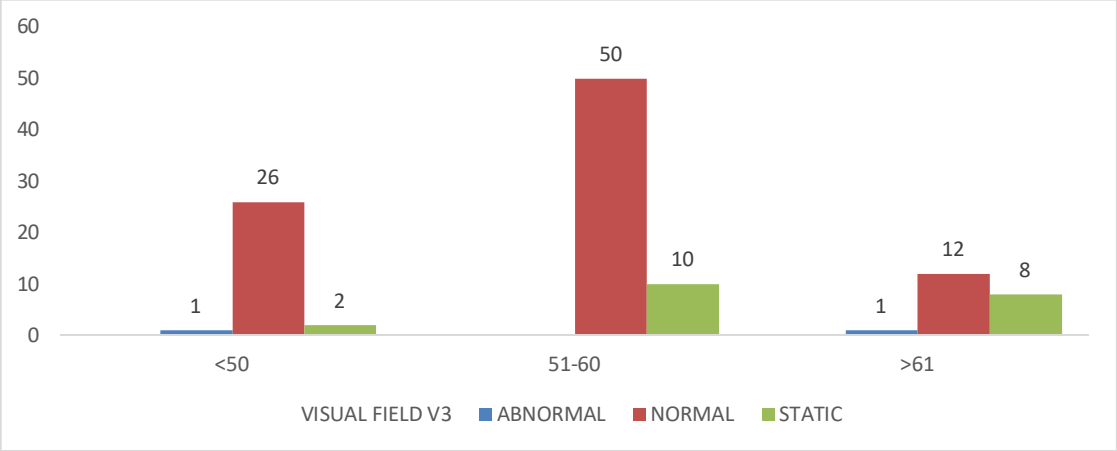


Fig 29: Observation of different age groups of patients in V3

In the present study, no female was reported with abnormal visual field in V3, whereas 2 (3.2%) males were observed with abnormal visual field in V3. However, most males and females were observed with normal visual fields with 49 (77.8%) and 39 (83%), respectively (Table 16, Fig 30).

Table 16: Observation of patient's gender in V3.

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
GENDER	F	Count	0	39	8	47	0.44
		% within GENDER	0.0%	83.0%	17.0%	100.0%	
	M	Count	2	49	12	63	
		% within GENDER	3.2%	77.8%	19.0%	100.0%	
Total		Count	2	88	20	110	
		% within GENDER	1.8%	80.0%	18.2%	100.0%	

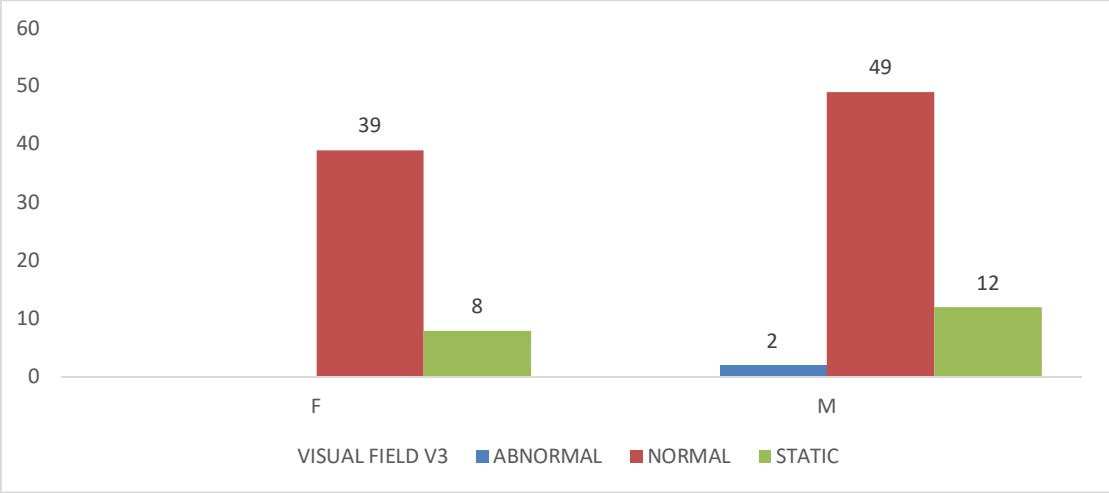


Fig 30: Observation of patient's gender in V3

The family history of open angle glaucoma was evaluated in visual field V3 in all patients. The abnormal visual field was observed in both the groups of with and without a family history of glaucoma with 1 (7.7%) and 1 (1%) patients.18 (18.6%) patients with no family history of glaucoma and 2 (15.5%) patients with family history of glaucoma showed static visual field in V3. (Table 17. Fig. 31).

Table 17: Observation of patient's family history of glaucoma in V3

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
FAMILY HISTORY	NO	Count	1	78	18	97	0.237
		% within FAMILY H	1.0%	80.4%	18.6%	100.0%	
	YES	Count	1	10	2	13	
		% within FAMILY H	7.7%	76.9%	15.4%	100.0%	
Total		Count	2	88	20	110	
		% within FAMILY H	1.8%	80.0%	18.2%	100.0%	

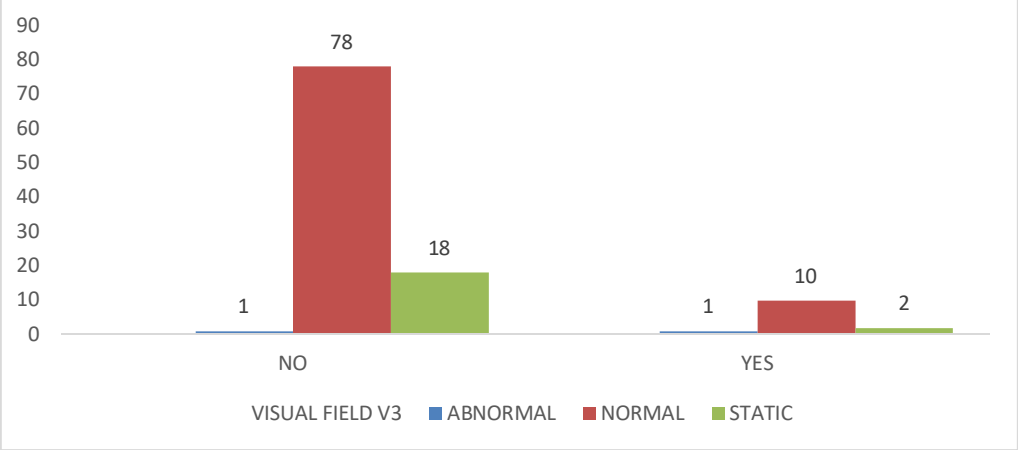


Fig 31 : Observation of patient's family history of glaucoma in V3

All Myopia patients (>- 6D)were evaluated in visual field V 3; it was found that 2 (2.1%) patients were observed with abnormal visual field in V3 in the non-myopia group, whereas no patients were found with the abnormal visual field in myopia group .Most of the patients in both groups 14 (100%) in the myopia and 73 (76.8%) in non myopia has showed normal visual field in V3 , respectively. (Table 18, Fig 32).

Table 18: Observation of myopia patients in V3.

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
MYOPIA	NO	Count	2	73	20	95	0.114
		% within MYOPIA	2.1%	76.8%	21.1%	100.0%	
	YES	Count	0	15	0	15	
		% within MYOPIA	0.0%	100.0%	0.0%	100.0%	
Total		Count	2	88	20	110	
		% within MYOPIA	1.8%	80.0%	18.2%	100.0%	

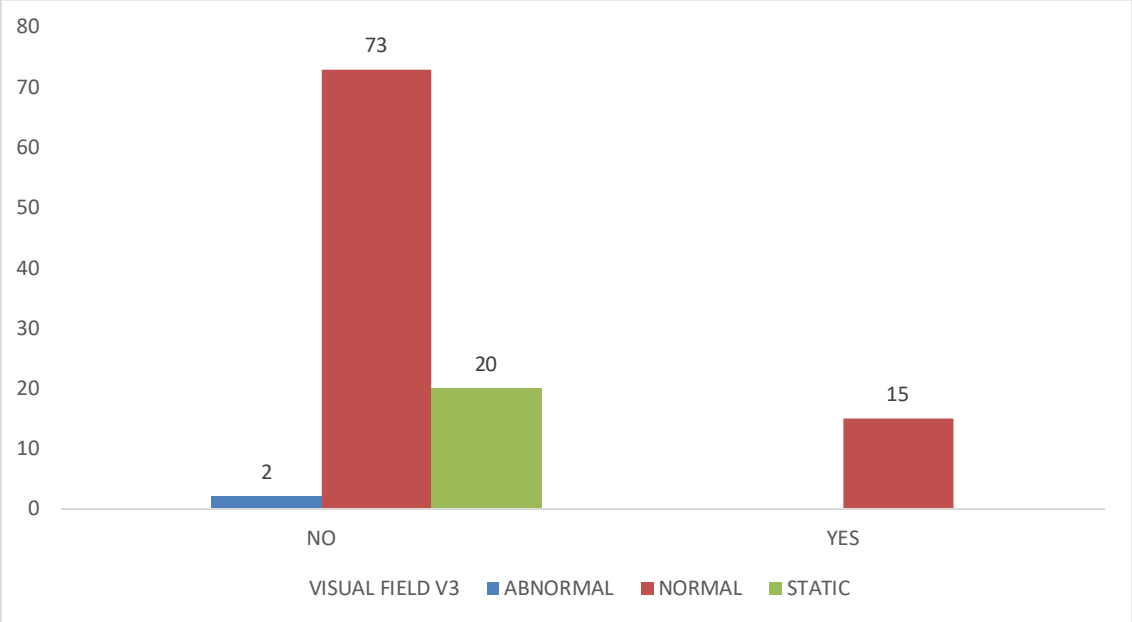


Fig 32: Observation of myopia patients in V3.

The patients with Increased IOP was evaluated with visual field V3. 2 (4.2%) patients with increased IOP showed abnormal visual field in V3 whereas, patients without increase in IOP had no abnormal visual field in V3. 35 (72.9%) patients with increased IOP and 53(85.5%) patients without increase in IOP had normal visual field in V3. 11(22.9%) patients with increase IOP and 9 (14.5%) patients without increase in IOP had static visual field in V3. (Table 19, Fig 33).

Table 19. Observation of increased IOP patients in V3 .

			VISUAL FIELD V3			Total	P value
			ABNORMAL	NORMAL	STATIC		
IOP V3	ABNORMAL	Count	2	35	11	48	0.124
		% within IOP V3	4.2%	72.9%	22.9%	100.0%	
	NORMAL	Count	0	53	9	62	
		% within IOP V3	0.0%	85.5%	14.5%	100.0%	
Total		Count	2	88	20	110	
		% within IOP V3	1.8%	80.0%	18.2%	100.0%	

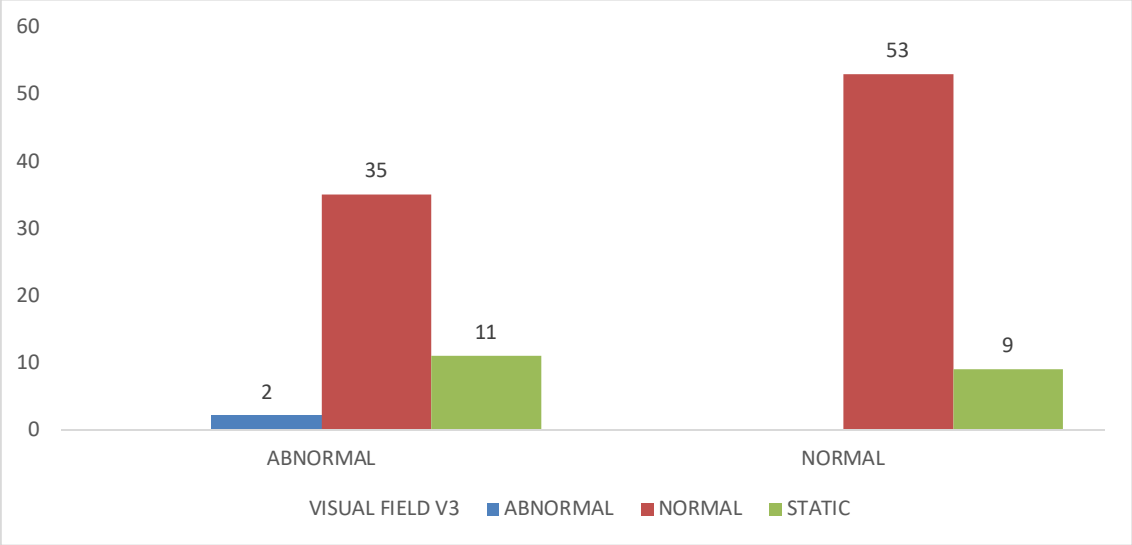


Fig 33: Observation of increased IOP patients in V3 .

The patients with Increased CD ratio was evaluated with visual field V3, it was noticed that the right and left eyes CD ratio mean were 0.45 and 0.50 in the patients with abnormal visual fields in V3. The right and left eyes CD ratio mean of the patients with normal visual fields were 0.40 and 0.52 respectively. The patients with static visual field in V3 had mean of 0.39 and 0.43 in the right and left eyes. (Table 20, Fig 34).

Table 20. Observation of increased CD ratio patients in V3.

		RE CDR		LE CDR	
		Mean	Standard Deviation	Mean	Standard Deviation
VISUAL FIELD V3	ABNORMAL	0.45	0.00	0.50	0.00
	NORMAL	0.40	0.11	0.42	0.13
	STATIC	0.39	0.11	0.43	0.16

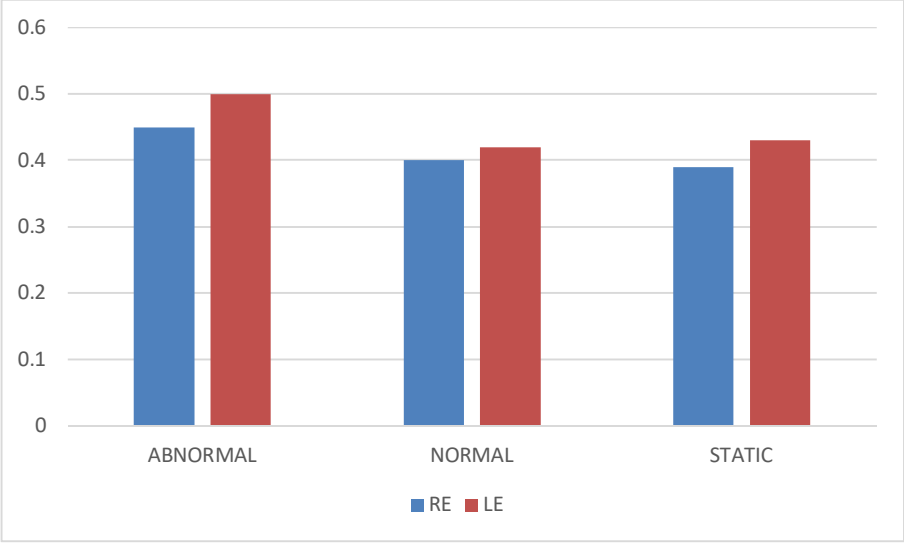


Fig.34. Observation of increased CD ratio patients in V3.

All patients with DM were also evaluated in visual field V3; it was observed that, both the groups patients with DM and without DM, had abnormal visual field i.e 1 (2.3%) patient in DM group and 1 (1.5%) patient in without DM group. However, 16 (37.2%) patients with DM and 4 (6%) patients without DM were observed to have static visual field in V 3 . (Table 21, Fig 35).

Table 21: Observation of DM patients in V3.

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
DM	NO	Count	1	62	4	67	<0.0001
		% within DM	1.5%	92.5%	6.0%	100.0%	
	YES	Count	1	26	16	43	
		% within DM	2.3%	60.5%	37.2%	100.0%	
Total		Count	2	88	20	110	
		% within DM	1.8%	80.0%	18.2%	100.0%	

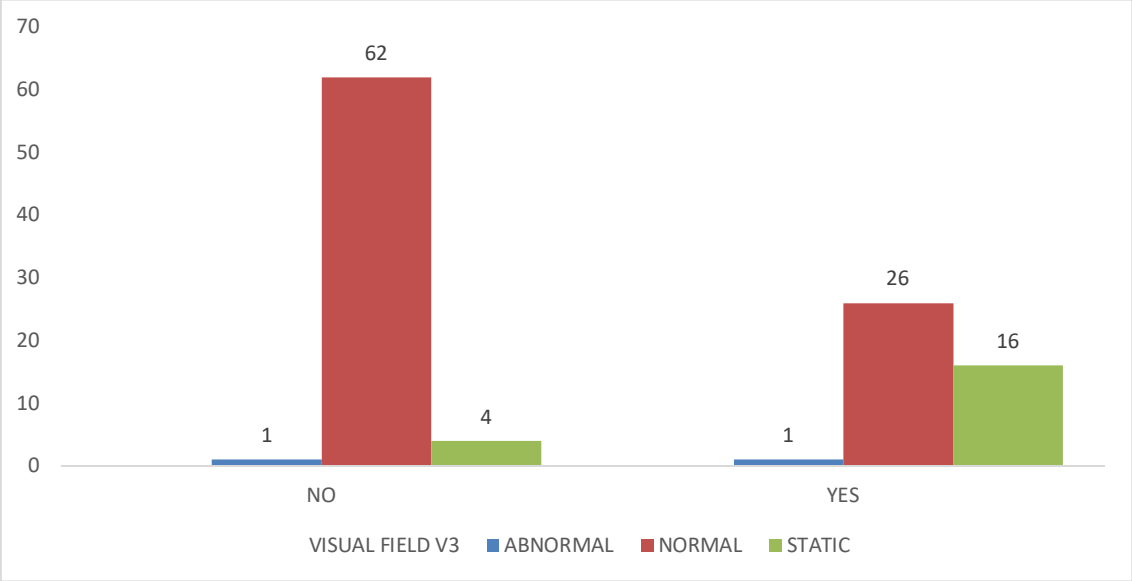


Fig 35: Observation of DM patients in V3.

The patients with SHTN were also evaluated in visual field V 3. It was found that 1 (2.3%) patient had abnormal visual field V3 in non SHTN group and 1 (1.5%) patient had abnormal visual field V3 in SHTN group . However, most of the patients in both groups were observed to have normal visual field in V3 with 29 (65.9%) and 59 (89.4%) patients in SHTN and non-SHTN groups, respectively (Table 22, Fig 36).

Table 22: Observation of SHTN patients in V3

			VISUAL FIELD V3			Total	P-value
			ABNORMAL	NORMAL	STATIC		
SHTN	NO	Count	1	59	6	66	0.009
		% within SHTN	1.5%	89.4%	9.1%	100.0%	
	YES	Count	1	29	14	44	
		% within SHTN	2.3%	65.9%	31.8%	100.0%	
Total		Count	2	88	20	110	
		% within SHTN	1.8%	80.0%	18.2%	100.0%	

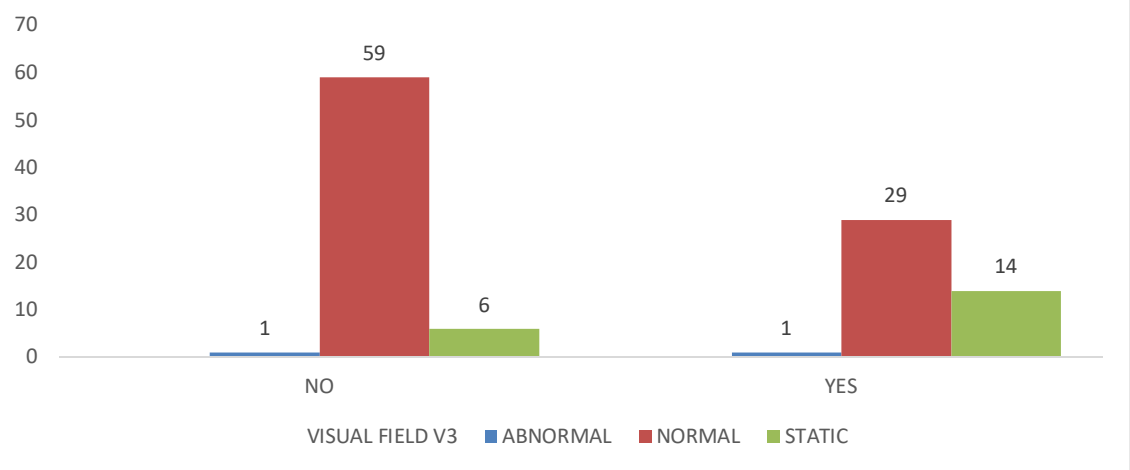


Fig 36: Observation of SHTN patients in V3

DISCUSSION

Glaucoma is one of the leading cause of irreversible blindness globally [63] and it is fast emerging as a major cause of blindness in India. Thereby diagnosing POAG at early stage is vital for the prevention of blindness .Therefore the earliest we diagnose glaucoma the better we will serve in the battle against blindness due to glaucoma. The timing of diagnosing is pivotal in glaucoma management. The field analysis is one of the effective tool in diagnosing POAG.

In this study , to estimate the prevalence of primary open angle glaucoma (POAG) , 110 glaucoma suspect individuals were examined .They underwent a systemic and complete ocular examination and field analysis. Humphrey Automated perimetry was used to evaluate damage progression on the basis of the emerged individual risk factors.

The age group of the patients included in the study ranged from 45 to 68 years. The majority of the patients were in the age group of 51 to 60 years. The mean age of presentation was 55.5 years.

In this study, males represented 57.3% and females represented 42.7 % which showed a mild male preponderance. This is similar with the study of the Canadian glaucoma study, which showed out of 258 patients, 131 (50.7%) were males and 127(49.0%) were females.[64]

The patient's attributes were correlated with the final visual field of V3 .In this study, it was observed that many patients in the age group of 51 to 60 years showed normal visual fields and some patients showed no change in progression of abnormal visual fields (remained static). One (3.4%) patient had abnormal visual field in the age group of less than 50 years and one (4.8%) patient had abnormal visual field in the age group of more than 60 years. Most patients in all age group found to have normal visual field. The similar observations were noted in the following studies

Claire Imrie et al. also reported that the most common type of glaucoma is POAG, affecting 2% of individuals older than 40 years and 10% of individuals older than 75 years, particularly in African-Caribbean people[65].

In the EMGT, the relative risk of progression of early glaucoma was 1.5 for those in the age group 68 years and above.[66]

In the study done by Quigley HA showed that in blacks with 25% of cases beginning by age 54 years and 50% by age 65 years.[63]

Chauhan [67] identified age as the main predictive independent factor: in this study the subjects older than 60 years had an incidence of glaucomatous perimetrical defects seven times higher than the one surveyed among the individuals under 40 years.

In this study, no female was found with abnormal visual field , whereas 2 (3.2%) males were observed with abnormal visual field in V3. However, more males and females were observed with normal visual fields with 49 (77.8%) and 39 (83%), respectively. These findings in the present study were similar to the study of

Naira Khachatryan et al showed the risk of having POAG was higher for men across all age groups, particularly at age less than 50 years old. However, at the age of 50–55 (age of menopause), men and women had almost the same risk of having POAG [68] . In Yih chung tham et al study, for the European population, the total prevalence of glaucoma was 2.51% with a 36% higher prevalence in males than in females.[69]

In this study , the abnormal visual field was noted in one (7.7%) patient with family history and one (1%) patient without family history of glaucoma. Both groups had the static visual field with 18 (18.6%) patients having family history and 2 (15.5%) patients without family

history of glaucoma. Several studies have demonstrated that the family history of glaucoma is associated with the presence and severity of visual fields[70,71].

Mc Carty CA et al reported that the family history of glaucoma was found to carry a relative risk of 2.1 times for being associated with at least possible OAG [72].

Around half of all primary OAG patients have a positive family history, and their first degree relatives (parents, siblings or children) have an approximately 9-fold increased risk of developing glaucoma[73].

Wolfs and co-authors found that the first degree relatives of glaucoma patients were found to have a 22% lifetime risk of glaucoma themselves in comparison to 2.3% in relatives of normal controls. The prevalence of glaucoma was 10.4% in the siblings of glaucoma patients compared to 0.7% in the siblings of normal controls [74]

The Baltimore Survey, 50% of the suffering patients had a positive familiarity, suggesting the genetic defect as important for the pathology development. [75]

In this study , 2 (2.1 %) patients in the non-myopia group had abnormal visual field . No abnormal visual field was noted in the patients of myopia group . Most of the patients in both groups had normal visual field in V3, with 14 (100%)patients in myopia and 73 (76.8%) patients in non-myopia group. The present study indicated no significant relationship for myopia in developing POAG. The individuals with myopia were not found to have a higher incidence or progression of glaucoma in the studies of OHTS or the EMGT.[76,77]

some studies have indicated that high myopia is an important in the pathogenesis of glaucoma, especially for POAG[78,79].

Marcus MW et al showed that high myopia and increased axial length in certain age groups have both been identified as risk factors suggests that the risk of glaucoma development and progression increases with the degree of myopia[80] .

The Canadian Glaucoma Study [64]stated that factors leading to a worsening of the MD variable in visual field over time are age, female gender and IOP.

In the Barbados Eye Study black population, persons most likely to have open-angle glaucoma were older men and had a family history of open-angle glaucoma, high intraocular pressure[81]

In this study, 2 (4.2%) patients with increased IOP showed abnormal visual field . The patients without increase in IOP had no abnormal visual field in V3. 35 (72.9%) patients with increased IOP and 53(85.5%) patients without increase in IOP had normal visual field . 11(22.9%) patients with increase IOP and 9 (14.5%) patients without increase in IOP had static visual field .

This study showed 2 patients with increased IOP had abnormal visual fields, but it is statistically insignificant in the study which may be probably due to diurnal variation of IOP was not recorded and it was the office hours IOP measured in this study. But increased IOP patients developed POAG which is comparable to the studies like, The Early Manifest Glaucoma Trial, EMGT [66] study showed that each increasing mmHg of the IOP creates a possible growth of 10% in the progression risk. A reduction of the IOP of 25% obtained through a standardized treatment (laser + betaxolol) allowed a slowdown in the disease progression. In the end the study indicated the high IOP as the significant risk factors of the progression.

The Canadian Glaucoma Study[64] confirmed that the average IOP at follow up (before demonstrating a progression) was directly proportional to the progression itself.

The Melbourne Visual Impairment Project, showed that for every 1 mm Hg, the risk for glaucoma increased by 10%.[82]

The OHTS also demonstrated that reducing the IOP by an average of 23% decreased the incidence of COAG by 60% [83].

In this study, the right and left eyes CD ratio mean were 0.45 and 0.50 in the patients with abnormal visual fields . The mean CD ratio of right and left eye of the patients with normal visual fields were 0.40 and 0.52 . The patients with static visual field had mean of 0.39 and 0.43 in the right and left eyes. There is no much change in the mean CD ratio of right and left eye in patients with normal, static and abnormal visual fields. This showed CD ratio was statistically insignificant and similar findings were noted in the other studies like

Wang XH et al , compared both eyes of patients with glaucoma, the eye with the larger optic disc showed neither more nor less damage than the contra-lateral eye, indicating that damage is not associated with optic disc size in a particular individual.[84,85]

J B JONAS et al ,the susceptibility to glaucomatous optic nerve fiber loss may be independent of the optic disc size.[86]

In the Baltimore Eye Survey, optic disc size in patients with glaucoma was slightly larger than in healthy individuals. However, after adjusting for confounding factors in their study, the difference between optic disc areas was not statistically significant [87].

In this study it was found that both the patients with DM and without DM had abnormal visual field (2.3 percent patients DM group and 1.5 percent patients without the DM group). 16 patients (37.2%) with DM showed static visual field , 4 (6%) without DM also showed static visual field . The association of DM with the development of POAG was statistically significant in this study which shows, the individuals with DM has increased risk of development of POAG.

Some studies showed IOP is an important confounder of the association between diabetes and glaucoma because patients with diabetes appear to have a slightly higher IOP and have been

reported to have a higher prevalence of ocular hypertension and incidence of IOP elevation, compared with persons who do not have diabetes[88,89,90]

According to Wei Wang et al study, Individual with DM has increased risk of developing POAG.[91] The study of louis Pasquale et all showed that Type 2 diabetes mellitus is associated with an increased risk of POAG in women [92]. However, Baltimore Eye Survey[87] has shown no relationship between diabetes and the visual field.

In this study, one (2.3%) patient in the non SHTN group had an abnormal visual field , while one (1.5 percent) patient in the SHTN group had an abnormal visual field. The majority of patients in both groups were found to have a normal visual field , with 29 (65.9%) and 59 (89.4%) patients in SHTN and non-SHTN groups, respectively. The incidence of SHTN was significantly associated with the effect POAG.

Some studies like J M Tielsch et all , have shown an association between systemic hypertension and POAG[93]

A study of bono I et al, in 4297 subjects over 40 years of age in a defined predominantly white population found a positive correlation between systemic BP and IOP and an association between POAG and systemic hypertension[94].

The visual field V1 parameters, which deals with the initial step of visual field analysis was compared with visual field V2 in all the patients. In the present study it was found that, among the 22 patients with abnormal visual field in V1, 9 (40.9%) patients had progression in visual fields in V2 and 13 (59.1%) patients had static visual field in V2. Whereas, 88 (100%) patients who had normal visual field in V1 were showed to have normal visual field in V2 also and was statistically significant ($p < 0.001$).

The visual field V2 parameter was compared to the visual field V3 findings in all cases. The current investigation showed that among 9 patients with aberrant visual field V2, 2 (22.2%)

had progression in visual field in V3 and 7 (77.8%) had static visual field V3. Individuals with a normal visual field in V2 were compared to 88 (100%) patients with a normal visual field V3 and statistically significant ($p < 0.001$).

The visual field V1 parameter was compared with visual field V3. The study showed that among 22 patients with aberrant visual field in V1, 2 (9.1%) had abnormal visual field in V3 and 20 (90.9%) had static visual field in V3. Whereas, patients who showed normal visual field in V1, also showed normal visual field in V3 and statistically significant ($p < 0.001$).

This is similar to the study of Chauhan BC et al showed that the visual field indices in perimetry that were used in their study were regressed on follow up time in few patients, but the great majority of their patients appeared to show no change in visual field on follow up of the glaucoma patients.[95]

In this study, among 2 patients who developed POAG , one was the male patient with age of 49 years , had risk factors like positive family history and increased IOP. Another male patient with the age of 68 years ,had increased IOP, diabetes and hypertension as risk factors.

In this study, the risk factors like diabetes and hypertension showed statistical significance .The risk factors like positive family history, increased CD ratio, high myopia , increased IOP were showed statistically insignificant. This is probably due to limitations like short duration of study and small sample size.

SUMMARY

- In the present study, total of 110 patients were enrolled; maximum patients 60 (54.5%) were reported in the age group of 51 to 60 years, male patients were 63 (57.3%) and female patients were 47 (42.7%) .
- Total 15 (13.6%) patients were observed with a family history of glaucoma.
- 14 (12.8%) patients observed with myopia.
- 40 (36.3%) patients observed with increased IOP.
- 43(39.1%) patients observed with increased CD ratio.
- 43 (39.1%) patients had DM co-morbidity.
- 44 (40%) patients had SHTN co-morbidity.
- No female was reported with abnormal visual field in V3, whereas 2 (3.2%) males were observed with abnormal visual fields in V3.
- The patients with abnormal visual field with 1 (3.4%) and 1 (4.8%), respectively were found in the age group of less than 50 years and more than 60 years
- The abnormal visual field was observed in both groups with or without a family history of glaucoma with 1 (7.7%) and 1 (1%) patients.
- 2 (2.1%) patients were observed with abnormal visual field in V3 in the non-myopia group, whereas no patients were found with the abnormal visual field in myopia group patients.
- Patients with DM are observed with significantly higher in static visual field in V 3, 16 (37.2%) ,whereas patients in without DM group showed only 4 (6%) patients in static visual field category.
- Most of the patients in both groups were observed in normal visual field in V3 with 29 (65.9%) and 59 (89.4%) patients in SHTN and non SHTN group groups, respectively.

- In the present study, it was found that in patients with 22 abnormal visual field in V1, 2 (9.1%) patients had abnormal visual field in V3 and 20 (90.9%) patients with static visual field V3. Whereas patients with normal visual field V1, observed with no abnormal and static visual field in V3.And the patients with normal visual field in V2, 88 (100%) observed with normal visual field in V3 .

CONCLUSION

The prevalence of POAG in Glaucoma suspect with or without risk factor was found to be 1.8%.

The risk factors like family history , advancing age , increased IOP and systemic risk factors like diabetes mellitus and hypertension are found to have notifiable relationship with the development of POAG but statistically not significant in this study.

The risk factors like increased CD ratio and high myopia showed no significant relationship with the development of POAG in this study.

So the present study highlights the importance of people with family history of glaucoma, systemic hypertension, diabetes mellitus and those with static visual field must be highly vigilant and any subtle signs of visual disturbance they must periodically undergo ocular examination and visual field evaluation to detect early field changes , which aids in early treatment and monitoring the progression of disease.

This study also emphasizes the need for further extensive studies to evaluate the correlation of risk factors and progression of the glaucomatous damage over time.

To overcome the limitations in usage of visual field analysis , OCT imaging of ONH and RNFL analysis can be utilized. All these investigations when combined can play a role in effective spacing the screening of visual field and diagnosing the early stages of glaucoma. This will pave the way to efficient and successful management of glaucoma cases.

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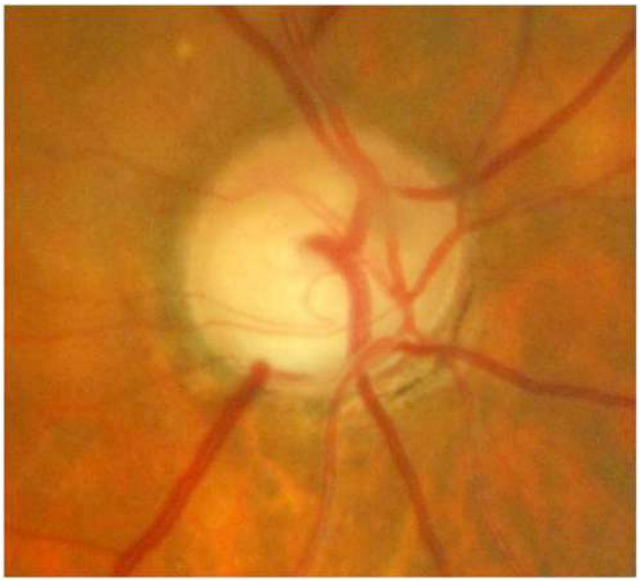
RE



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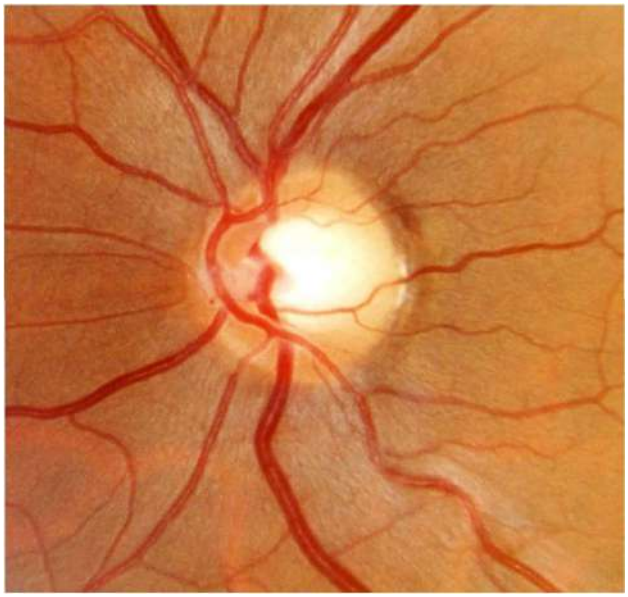

Fig 37. FUNDUS PICTURES OF POAG PATIENTS

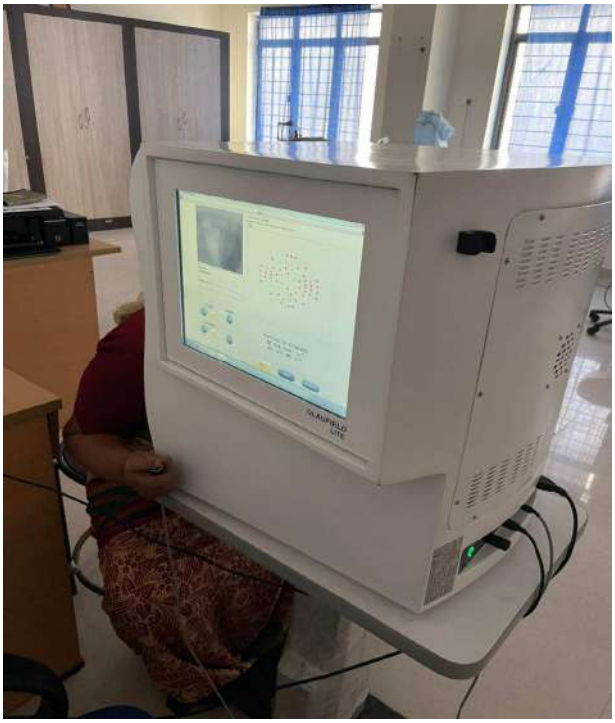

Fig 38. PATIENT UNDERGOING VISUAL FIELD ANALYSIS IN HUMPHREY PERIMETRY

HUMPHERY AUTOMATED PERIMETRY CHARTS

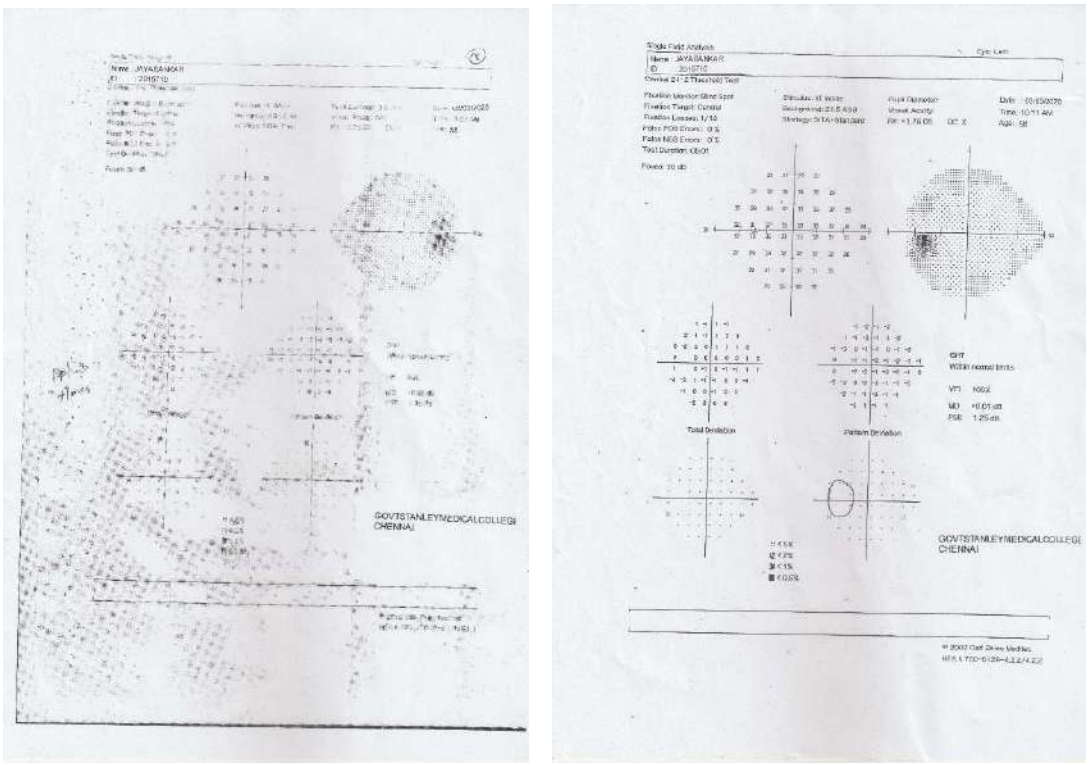

Fig 39. IMAGE OF VISUAL FIELD OF PATIENT -1 AT VISIT 1.

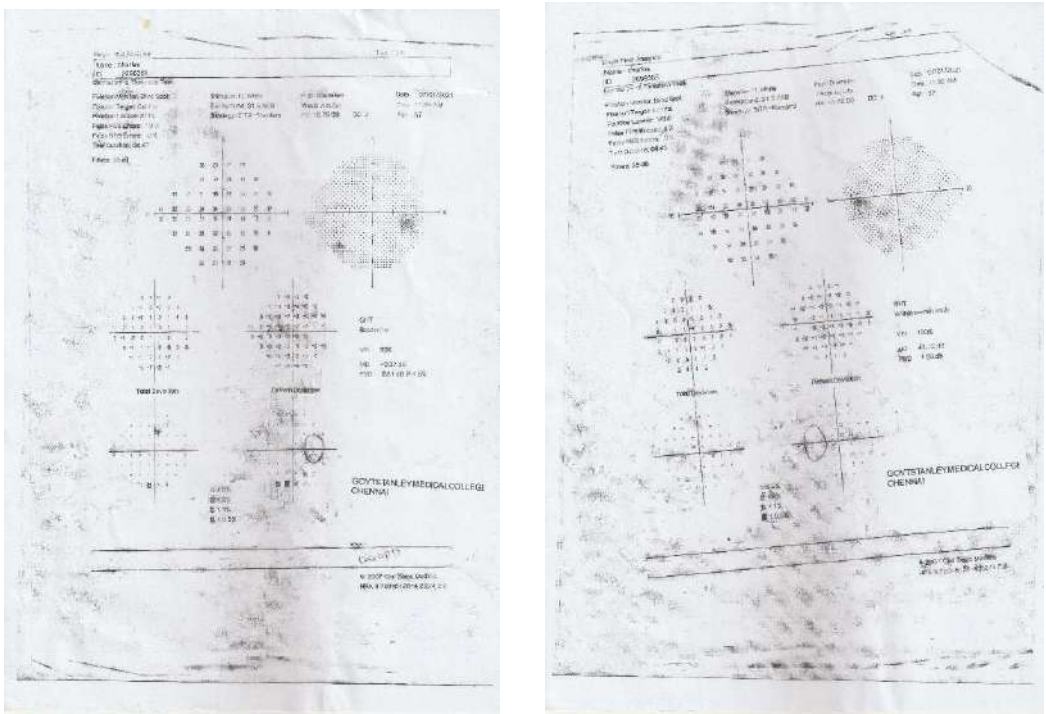

Fig 40. IMAGE OF VISUAL FIELD OF PATIENT -2 AT VISIT 1 .

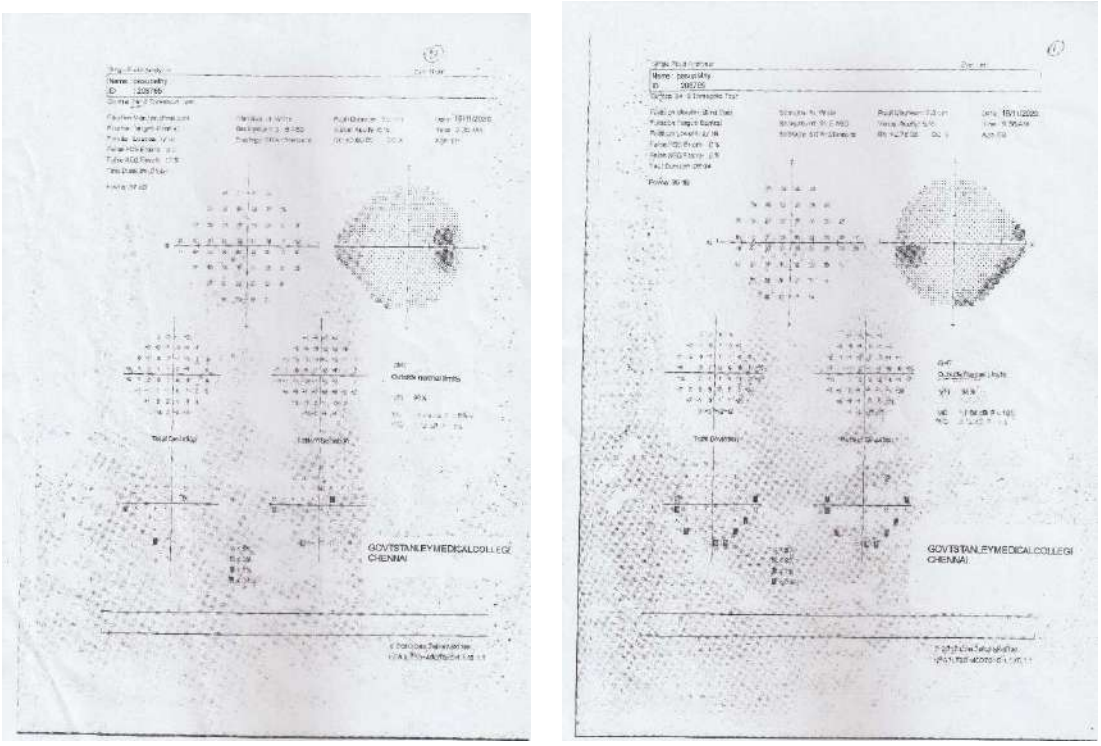

Fig 41. IMAGE OF VISUAL FIELD OF PATIENT -3 AT VISIT 1.

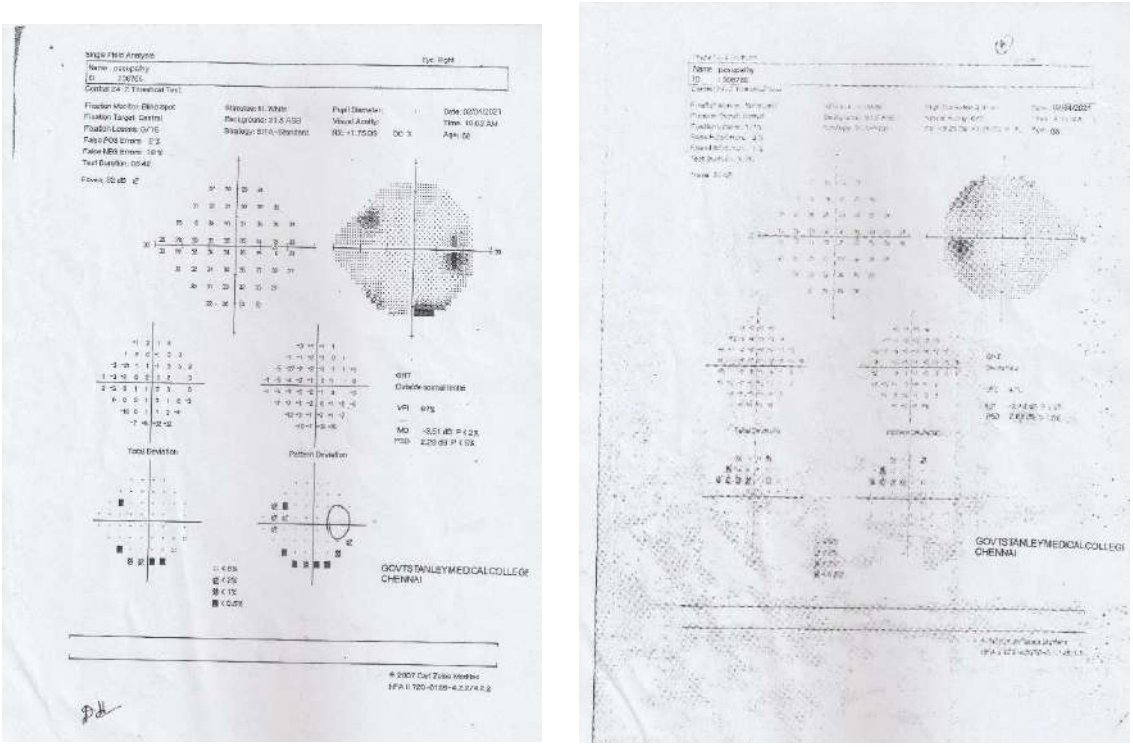

Fig 42. IMAGE OF VISUAL FIELD OF PATIENT -3 AT VISIT 2.

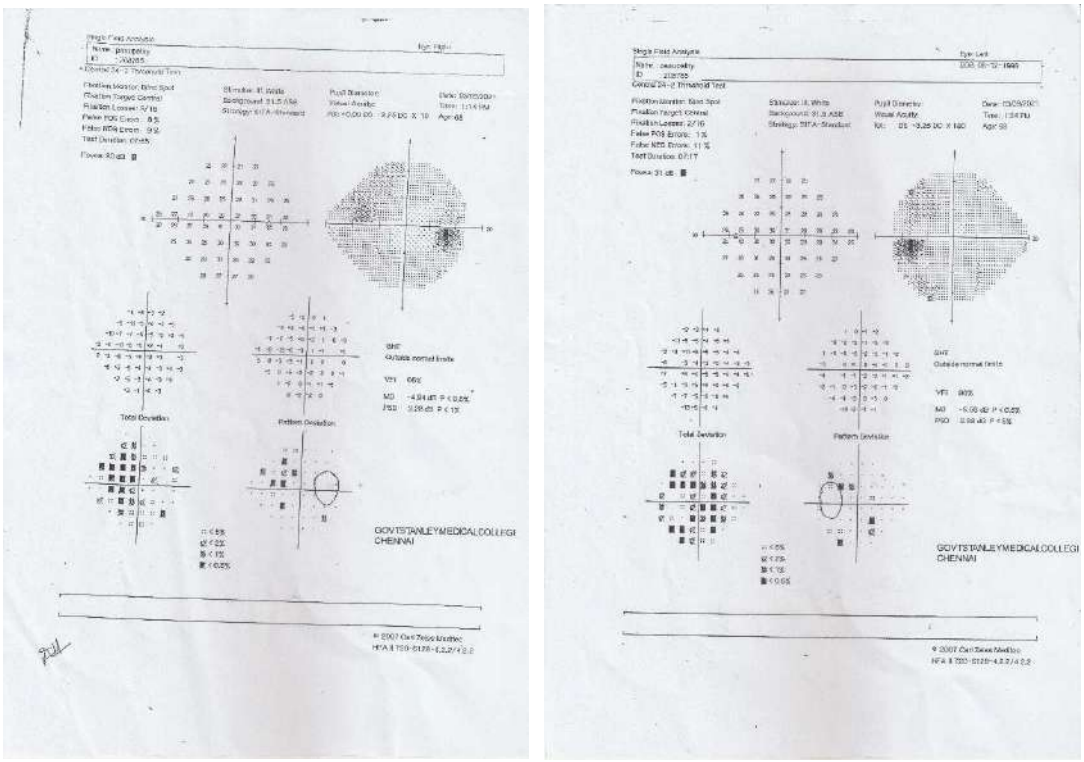

Fig 43. IMAGE OF VISUAL FIELD OF PATIENT -3 AT VISIT 3.

PROFORMA

A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS (PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER)

Serial no : OP NO.:

Name :

Age :

Sex :

Occupation :

Address :

1. HISTORY

Ocular complaints :

Past history :

Personal history :

Treatment history :

Associated systemic illness : Hypertension –

Duration: On treatment (yes/no)

Diabetes mellites-

Duration: On treatment (yes/no)

Family history of glaucoma : yes / no

Known glaucoma patient : yes / no

2. **GENERAL PHYSICAL EXAMINATION**:

Blood Pressure :

Pulse rate :

Cardiovascular system :

Respiratory system :

Per abdomen :

Nervous system :

3. **OCULAR EXAMINATION**

RE LE

Vision (BCVA)

Distance

Near

Head Posture

Facial Asymmetry

Extraocular movements

Slit lamp examination

Ocular Adnexa

Conjunctiva

Cornea

Anterior chamber

Iris

Pupil

Lens

Intraocular pressure by

Applanation tonometry :

Gonioscopy :

Pachymetry :

Fundus Examination :

Fields by Humphery

automated perimetry :

Other investigations :

DIAGNOSIS :

PATIENT INFORMATION SHEET

TITLE: A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS(PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER).

I , Dr. P.Nanthini , post graduate, MS in Ophthalmology ,Government Stanley Medical College is going to undertake the study on the above mentioned topic.

The purpose of this study is to evaluate the prevalence of primary open angle glaucoma in all case of glaucoma suspects with the help of perimetry (visual field analysis) .

If you are willing to participate in this study you will be asked some questions regarding risk factors like family history of glaucoma, hypertension and diabetic history. And you may need to undergo a non invasive tests like IOP measuring, Gonioscopy to evaluate your intraocular pressure and angle grading and fundus examination and then subjected to Humphery perimetry to detect extent of field loss.

This study helps in detecting glaucoma in early stage and preventing the progression of glaucoma to the advanced stage by introducing early intervention as soon as possible.

I assure that all the information provided by you will be kept highly confidential and privacy is assured. Your identity won't be revealed to anyone. The study may be published in scientific journal, but your identity will not be revealed.

Your participation in this study is voluntary and you can withdraw from this at any point of time.

Signature/left thumb impression of the participant

INFORMATION SHEET

தகவல் நகல்

TITLE: A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS(PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER).

இந்த ஆய்வில் உங்களிடம் கேட்கும் கேள்விகளுக்கு முழு மனதுடன் பதில் அளிக்க வேண்டும்.

இந்த ஆய்வில் உங்கள் நாள்பட்ட நோய்கள், உடல்நலம் தேடும் நடத்தை தொடர்பாக விவரங்கள் பற்றி கேட்கப்படும்.

இந்த ஆய்வில் உங்களுக்கு எந்த பின் விளைவும் ஏற்படாது என்பதை நான் உறுதியளிக்கிகேன்.

உங்களிடம் கேட்கும் கேள்விகளில் உங்களின் சுயவிபரம், குடும்பவிபரம், தொழில் விபரம், விபரங்கள் மற்றும் இதர விபரம் அடங்கும்.

உங்களுக்கு பணம் எதுவும் அளிக்கபடாது என்பதை இதன் மூலம் தெறிவிக்கிறேன்.

இந்த ஆய்வின் மூலம், கண் அழுத்தநோயை ஆரம்பக்கட்டத்திலேயே கண்டறியப்பட்டு, அதனை மேலும் வளர்ச்சியடைந்து, சிகிச்சையளிக்க முடியாத நிலைக்கு செல்வத்திலிருந்து தடுத்து முறையான சிகிச்சையளித்து கண் பார்வையை பாதுகாக்கமுடியும்.

இந்த ஆய்வில் நீங்கள் அளிக்கும் தகவல் மூலம் புதுயுக்திகள் வகுக்கப்படலாம். அதன் மூலம் வருங்காலத்தில் உங்களுக்கோ அல்லது உங்களை போன்ற மக்களுக்கோ பயன்படலாம்.

உங்களின் விபரங்கள் எதுவும் மற்றவர்களுக்கு தெரிவிக்கப்படாது என்பதை உறுதியளிக்கிறேன்.

இந்த ஆய்வின் முடிவுகள் பத்திரிக்கைளில் பிரசுரிக்கபடலாம் ஆனால் உங்களின் அடையாளம் எதுவும் காட்டப்படாது.

உங்களுக்கு விருப்பம் இல்லை என்றால் எப்போது வேண்டுமானாலும் இந்த ஆய்வில் இருந்து விலகிக்கொள்ளலாம். அதனால் உங்களுக்கு எந்த பாதிப்பும் இல்லை.

கையொப்பம்/ இடது பெருவிரல் ரேகை

INFORMED CONSENT

TITLE: A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS(PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER).

The content of the information sheet dated _____ that was provided have been read carefully by me/explained in detail to me, in a language that I comprehend and fully understood the contents.

I confirm that I have had the opportunity to ask questions.

The nature and purpose of the study and its potential risks/benefits and expected duration of the study and other relevant details of the study have been explained to me in detail.

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal right being affected.

I agree to take part in the above study.

_____

(Signature/Left thumb impression)

Name of the Participant: _____

Son/Daughter/Spouse of _____

Complete postal address: _____

This is to certify that the above consent has been obtained in my presence.

_____ Date:

Signature of the principal investigator Place:

1)Witness – 1 2) Witness – 2

_____ _____

Signature: Signature:

Name: Name:

Address: Address:

INFORMED CONSENT

தகவல் தொடர்பு ஒப்புதல் படிவம்

TITLE: A HOSPITAL BASED STUDY TO ASSESS THE PREVALANCE OF PRIMARY OPEN ANGLE GLAUCOMA AMONG GLAUCOMA SUSPECT PATIENTS WITH OR WITHOUT RISK FACTORS(PREDISPOSING TO GLAUCOMA) WITH THE HELP OF AUTOMATED PERIMETRY (HUMPHERY FIELD ANALYSER).

நான் தகவல் நகலில் கொடுக்கபட்டுள்ள முழு விவரங்களையும் கவனமாகப் படித்தேன். ஆய்வின் முழு விவரங்களையும் தமிழில் எனக்கு விளக்கமாக எடுத்துக் கூ றப்பட்டது.

நான் இந்த ஆய்வின் விவரங்களை முழுமையாக புரிந்துகொண்டேன்.

ஆய்வில் பங்கு எடுக்கும் போது ஏற்படும் சாத்தியமான அபாயங்கள் மற்றும் பயன்களை நான் அறிந்துள்ளேன்.

மேலும், நான் எந்த ஒரு வேளையிலும் ஆய்வில் இருந்து திரும்ப முடியும் என்றும், அதன் பின்னர் நான் வழக்கம் போல் மருத்துவச் சிகிச்சைப் பெற முடியும் என்றும் புரிந்துக் கொள்கிறேன்.

நான் ஆய்வில் பங்கு கொள்ள பணம் எதையும் பெற முடியாது என்று அறிந்துள்ளேன்.

நான் இந்த ஆய்வில் பங்கு எடுப்பதன் மூலம் நான் என்ன செய்ய போகிறேன் என்று தெரியும்.

நான் இந்த ஆய்வில் என் முழு ஒத்துழைப்பையும் கொடுப்பேன் என்று உறுதியளிக்கின்றேன்.

ஆய்வில் பங்கேற்பவர் பெயர்: *சாட்சி:*

பெயர் மற்றும் முகவரி: *பெயர் மற்றும் முகவரி:*

கையொப்பம்/ விரல் ரேகை *கையொப்பம்/ விரல் ரேகை*

ஆராய்ச்சியாளராக கையொப்பம் மற்றும் தேதி

KEY TO MASTER CHART

M – Male

F – Female

D – Diopter

P- Present

A-Absent

OA-Open Angle

DM – Diabetes Mellitus

SHTN – Systemic Hypertension

RE – Right Eye

LE – Left Eye

IOP – Intraocular Pressure

AT – Applanation Tonometry

V1 – Visit 1

V2 – Visit 2

V3 – Visit3

MASTER CHART

S.NO	NAME	SEX	AGE	RISK FACTORS				COMORBIDITY		ANTERIOR SEGMENT	PACHYMETRY		CORRECTED IOP(AT)		GONIOSCOPY		FUNDUS		VISIT 1 (V1) VISUAL FIELD		CORRECTED IOP(AT)		GONIOSCOPY		FUNDUS		VISIT 2 (V2) VISUAL FIELD		CORRECTED IOP(AT)		GONIOSCOPY		FUNDUS		VISIT 3 (V3) VISUAL FIELD	
				FAMILY	HYPER(>-6	DM	SHTN																											
1	kalaisorthy	M	53	A	A	A	P	NORMAL			505	511	22	20	OA	OA	CD 0.3		NORMAL		20	20	OA	OA	CD 0.3		NORMAL		22	22	OA	OA	CD 0.3		NORMAL	
2	shivagami	F	50	A		A		NORMAL			564	556	20	22	OA	OA	CD 0.3		NORMAL		22	22	OA	OA	CD 0.3		NORMAL		22	24	OA	OA	CD 0.3		NORMAL	
3	prakasam	M	46	P		A		NORMAL			532	574	12	14	OA	OA	CD 0.3		NORMAL		12	12	OA	OA	CD 0.3		NORMAL		14	14	OA	OA	CD 0.3		NORMAL	
4	arunachalam	M	56	A		P	A	NORMAL			543	548	22	20	OA	OA	CD 0.3		NORMAL		22	22	OA	OA	CD 0.3		NORMAL		22	24	OA	OA	CD 0.3		NORMAL	
5	prasenthi	F	59	A	A	P	A	NORMAL			536	514	12	14	OA	OA	CD 0.4		ABNORMAL		22	12	OA	OA	CD 0.4		STATIC		STATIC		16	14	OA	OA	CD 0.6		STATIC	
6	vijaya	F	62	A	A	P	P	NORMAL			497	485	22	22	OA	OA	CD 0.3		ABNORMAL		24	24	OA	OA	CD 0.3		ABNORMAL		24	22	OA	OA	CD 0.3		STATIC	
7	ameruvelhi	F	55	A	A	P	A	NORMAL			544	536	22	24	OA	OA	CD 0.3		NORMAL		22	22	OA	OA	CD 0.3		NORMAL		20	24	OA	OA	CD 0.3		NORMAL	
8	jayabalan	M	60	A	A	A	P	NORMAL			488	473	14	16	OA	OA	CD 0.3		ABNORMAL		14	14	OA	OA	CD 0.8		CD 0.5		STATIC		16	14	OA	OA	CD 0.3		STATIC	
9	viralgam	M	50	A	P	A	A	NORMAL			467	459	12	10	OA	OA	CD 0.5		ABNORMAL		12	12	OA	OA	CD 0.5		NORMAL		STATIC		10	12	OA	OA	CD 0.5		STATIC	
10	muthu	M	46	A	A	P	A	NORMAL			523	514	18	22	OA	OA	CD 0.3		NORMAL		20	22	OA	OA	CD 0.3		NORMAL		NORMAL		20	22	OA	OA	CD 0.3		NORMAL	
11	maalliga	F	66	A	A	P	A	NORMAL			523	514	18	22	OA	OA	CD 0.3		NORMAL		20	22	OA	OA	CD 0.3		NORMAL		NORMAL		20	22	OA	OA	CD 0.3		NORMAL	
12	maanikam	M	56	A	A	P	A	NORMAL			480	476	20	22	OA	OA	CD 0.3		ABNORMAL		22	22	OA	OA	CD 0.3		ABNORMAL		ABNORMAL		22	24	OA	OA	CD 0.3		STATIC	
13	saravanan	M	52	A	A	A	A	NORMAL			499	512	12	14	OA	OA	CD 0.4		CD 0.6		NORMAL		14	16	OA	OA	CD 0.4		CD 0.6		NORMAL		14	14	OA	OA	CD 0.4		CD 0.6	
14	vanaja	F	58	A	A	P	A	NORMAL			532	546	20	22	OA	OA	CD 0.3		NORMAL		20	20	OA	OA	CD 0.3		NORMAL		NORMAL		22	22	OA	OA	CD 0.3		NORMAL	
15	arulmozhi	F	60	A	A	P	A	NORMAL			543	549	16	18	OA	OA	CD 0.4		CD 0.5		NORMAL		16	16	OA	OA	CD 0.4		CD 0.5		NORMAL		18	16	OA	OA	CD 0.4		CD 0.5	
16	meenakshi	F	51	A	A	A	A	NORMAL			567	573	14	16	OA	OA	CD 0.5		NORMAL		14	16	OA	OA	CD 0.4		CD 0.5		NORMAL		16	14	OA	OA	CD 0.4		CD 0.5	
17	mohammad hussain	M	58	A	A	P	P	NORMAL			522	543	12	14	OA	OA	CD 0.5		ABNORMAL		12	14	OA	OA	CD 0.5		STATIC		STATIC		14	14	OA	OA	CD 0.5		STATIC	
18	manjula	F	54	A	A	P	A	NORMAL			539	537	22	20	OA	OA	CD 0.3		NORMAL		24	22	OA	OA	CD 0.3		NORMAL		NORMAL		22	20	OA	OA	CD 0.3		NORMAL	
19	kanikala	F	49	P	A	A	A	NORMAL			526	532	14	12	OA	OA	CD 0.3		CD 0.3		NORMAL		16	14	OA	OA	CD 0.3		NORMAL		NORMAL		14	14	OA	OA	CD 0.3		NORMAL	
20	velu	M	68	A	A	P	P	NORMAL			486	522	14	16	OA	OA	CD 0.5		CD 0.7		ABNORMAL		14	14	OA	OA	CD 0.5		CD 0.7		STATIC		16	14	OA	OA	CD 0.5		CD 0.7		STATIC	
21	kalaivanidhen	M	59	A	A	A	P	NORMAL			462	475	20	18	OA	OA	CD 0.3		CD 0.3		ABNORMAL		20	20	OA	OA	CD 0.3		CD 0.3		NORMAL		24	20	OA	OA	CD 0.3		CD 0.3		NORMAL	
22	radha	F	58	A	A	A	A	NORMAL			462	475	20	18	OA	OA	CD 0.3		ABNORMAL		20	20	OA	OA	CD 0.3		ABNORMAL		STATIC		20	18	OA	OA	CD 0.3		STATIC		NORMAL	
23	rajanagar	M	53	A	A	A	A	NORMAL			567	543	14	12	OA	OA	CD 0.6		CD 0.4		NORMAL		16	14	OA	OA	CD 0.6		CD 0.4		NORMAL		14	16	OA	OA	CD 0.6		CD 0.4		NORMAL	
24	vrisalm	F	58	P	A	A	P	NORMAL			478	496	16	14	OA	OA	CD 0.3		CD 0.3		NORMAL		16	16	OA	OA	CD 0.3		CD 0.3		NORMAL		14	14	OA	OA	CD 0.3		CD 0.3		NORMAL	
25	mohammad hussain	M	59	A	A	P	P	NORMAL			555	543	24	22	OA	OA	CD 0.3		CD0.3		NORMAL		22	24	OA	OA	CD0.3		CD0.3		NORMAL		22	24	OA	OA	CD0.3		CD0.3		NORMAL	
26	bharathan	M	64	A	P	P	P	NORMAL			538	517	22	24	OA	OA	CD 0.3		ABNORMAL		20	22	OA	OA	CD 0.3		ABNORMAL		ABNORMAL		22	22	OA	OA	CD 0.3		STATIC		NORMAL	
27	lakshmi	F	55	P	A	A	A	NORMAL			503	489	12	12	OA	OA	CD 0.3		CD 0.3		NORMAL		14	12	OA	OA	CD 0.3		CD 0.3		NORMAL		12	14	OA	OA	CD 0.3		CD 0.3		NORMAL	
28	aneela	F	58	A	P	P	P	NORMAL			534	550	12	10	OA	OA	CD 0.5		ABNORMAL		12	12	OA	OA	CD 0.5		STATIC		STATIC		14	12	OA	OA	CD 0.5		STATIC		NORMAL	
29	lakshmunan	M	66	A	A	P	P	NORMAL			537	525	14	16	OA	OA	CD 0.5		CD 0.4		NORMAL		16	18	OA	OA	CD 0.5		CD 0.4		NORMAL		16	16	OA	OA	CD 0.5		CD 0.4		NORMAL	
30	pandiaraj	M	50	A	A	A	A	NORMAL			578	543	12	10	OA	OA	CD 0.5		CD 0.7		NORMAL		14	12	OA	OA	CD 0.5		CD 0.7		NORMAL		12	12	OA	OA	CD 0.5		CD 0.7		NORMAL	
31	vanitha	F	62	A	A	P	P	NORMAL			562	539	20	22	OA	OA	CD 0.3		ABNORMAL		22	22	OA	OA	CD 0.8		CD 0.8		STATIC		22	20	OA	OA	CD 0.3		STATIC		NORMAL	
32	muniyammal	F	52	A	A	A	A	NORMAL			503	489	16	16	OA	OA	CD 0.6		CD 0.7		NORMAL		14	16	OA	OA	CD 0.6		CD 0.7		NORMAL		14	14	OA	OA	CD 0.6		CD 0.7		NORMAL	
33	shanthi	M	47	P	A	A	A	NORMAL			543	533	16	18	OA	OA	CD 0.3		CD0.3		NORMAL		18	16	OA	OA	CD 0.3		CD0.3		NORMAL		16	16	OA	OA	CD 0.3		CD0.3		NORMAL	
34	dhanes	M	57	A	A	P	A	NORMAL			544	545	14	12	OA	OA	CD 0.6		CD 0.6		NORMAL		14	16	OA	OA	CD 0.6		CD 0.6		NORMAL		14	14	OA	OA	CD 0.6		CD 0.6		NORMAL	
35	malliga m	F	48	P	A	A	A	NORMAL			552	548	20	22	OA	OA	CD 0.3		CD 0.3		NORMAL		22	22	OA	OA	CD 0.3		CD 0.3		NORMAL		24	22	OA	OA	CD 0.3		CD 0.3		NORMAL	
36	ashokan	M	59	A	A	P	A	NORMAL			549	551	12	12	OA	OA	CD 0.5		CD 0.4		NORMAL		14	14	OA	OA	CD 0.5		CD 0.4		NORMAL		12	14	OA	OA	CD 0.5		CD 0.4		NORMAL	
37	Annakili	F	66	A	P	P	A	NORMAL			542	556	12	14	OA	OA	CD 0.5		CD 0.5		NORMAL		14	12	OA	OA	CD 0.5		CD 0.5		NORMAL		14	12	OA	OA	CD 0.5		CD 0.5		NORMAL	
38	bakyalakmi	F	65	A	A	P	A	NORMAL			485	498	12	12	OA	OA	CD 0.5		CD 0.5		NORMAL		14	14	OA	OA	CD 0.5		CD 0.5		NORMAL		12	14	OA	OA	CD 0.5		CD 0.5		NORMAL	
39	annakili	F	48	P	A	A	A	NORMAL			497	508	16	16	OA	OA	CD 0.3		CD 0.3		NORMAL		16	16	OA	OA	CD 0.3		CD 0.3		NORMAL		16	18	OA	OA	CD 0.3		CD 0.3		NORMAL	
40	jayalakshmi	F	61	A	P	A	A	NORMAL			518	532	12	12	OA	OA	CD 0.6		CD 0.6		NORMAL		14	14	OA	OA	CD 0.6		CD 0.6		NORMAL		12	12	OA	OA	CD 0.6		CD 0.6		NORMAL	
41	kannapiran	M	49	P	A	A	A	NORMAL			545	549	22	24	OA	OA	CD 0.3		ABNORMAL		24	22	OA	OA	CD 0.6		CD 0.3		ABNORMAL		24	24	OA	OA	CD 0.5		CD 0.4		ABNORMAL	
42	kattapoimuse	M	53	A	A	A	A	NORMAL			533	512	16	18	OA	OA	CD 0.5		CD 0.7		NORMAL		16	16	OA	OA	CD 0.5		CD 0.7		NORMAL		14	16	OA	OA	CD 0.5		CD 0.7		NORMAL	
43	ponrayi	M	46	P	A	A	A	NORMAL			487	475	12	14	OA	OA	CD 0.3		NORMAL		14	14	OA	OA	CD 0.3		NORMAL		NORMAL		14	14	OA	OA	CD 0.3		NORMAL		NORMAL	
44	kamatchi	F	59	A	A	P	A	NORMAL			534	546	16	18	OA	OA	CD 0.5		CD 0.5		NORMAL		14	16	OA	OA	CD 0.5		CD 0.5		NORMAL		16	14	OA	OA	CD 0.5		CD 0.5		NORMAL	
45	kaarthiga	F	48	P	A	A	A	NORMAL			476	489	14	14	OA	OA	CD 0.3		CD 0.3		NORMAL		14	14	OA	OA	CD 0.3		CD 0.3		NORMAL		14	14	OA	OA	CD 0.3		CD 0.3		NORMAL	
46	Parthiban	M	45	A	A	A	A	NORMAL			545	546	18	16	OA	OA	CD 0.5		CD 0.5		NORMAL		16	14	OA	OA	CD 0.5		CD 0.5		NORMAL		18	16	OA	OA	CD 0.5		CD 0.5		NORMAL	
47	Thangaraj	M	45	A	P	A	A	NORMAL			527	519	12	10	OA	OA	CD 0.6		CD 0.6		NORMAL		12	12	OA	OA	CD 0.6		CD 0.6		NORMAL		10	12	OA	OA	CD 0.6		CD 0.6		NORMAL	
48	thangarai	M	61	A	A	P	P	NORMAL			479	488	12	14	OA	OA	CD 0.6		CD 0.6		NORMAL		14	12	OA	OA	CD 0.6		CD 0.6		NORMAL		12	14	OA	OA	CD 0.6		CD 0.6		NORMAL	
49	kamalatdas	M	50	A	A	A	A	NORMAL			493	486	20	22	OA	OA	CD 0.3		CD 0.3		NORMAL		14	12	OA	OA	CD 0.3		CD 0.3		NORMAL		22	22	OA	OA	CD 0.3		CD 0.3		NORMAL	
50	gnanasundari	F	54	A	A	A	A	NORMAL			524	507	16	18	OA	OA	CD 0.6		CD 0.6		NORMAL		16	16	OA	OA	CD 0.6		CD 0.6		NORMAL		18	18	OA	OA	CD 0.6		CD 0.6		NORMAL	
51	ruiman	F	52	A	P	A	A	NORMAL			572	563	12	14	OA	OA	CD 0.4		CD 0.4		NORMAL		14	12	OA	OA	CD 0.5		CD 0.4		NORMAL		12	12	OA	OA	CD 0.5		CD 0.4		NORMAL	
52	paramasivan	M	65	A	P	A	P	NORMAL			532	555	16	16	OA	OA	CD 0.4		CD 0.6		NORMAL		16	16	OA	OA	CD 0.4		CD 0.6		NORMAL		16	14	OA	OA	CD 0.4		CD 0.6		NORMAL	
53	malarkodi	F	57	A	A	P	A	NORMAL			582	567	10	12	OA	OA	CD 0.5		CD 0.5		NORMAL		12	12	OA	OA	CD 0.5		CD 0.5		NORMAL		12	14	OA	OA	CD 0.5		CD 0.5		NORMAL	
54	thangathurai	M	46	A	P	A	A	NORMAL			506	489	14	12	OA	OA	CD 0.5		CD 0.6		NORMAL		14	12	OA	OA	CD 0.5		CD 0.6		NORMAL		12	14	OA	OA	CD 0.5		CD 0.6		NORMAL	
55	devi priya	F	49	A	P	A	A	NORMAL			542	555	12	12	OA	OA	CD 0.3		CD0.3		NORMAL		14	12	OA	OA	CD0.3		CD0.3		NORMAL		12	14	OA	OA	CD0.3		CD0.3		NORMAL	
56	balakrishnan	M	60	A	A	P	P	NORMAL			547	534	14	16	OA	OA	CD 0.6		ABNORMAL		14	14	OA	OA	CD 0.6		STATIC		STATIC		14	16	OA	OA	CD 0.6		STATIC		NORMAL	
57	rajadurai	M	58	A	A	P	P	NORMAL			546	532	14	12	OA	OA	CD 0.3		CD 0.4		NORMAL		12	14	OA	OA	CD 0.3		CD 0.4		NORMAL		14	14	OA	OA	CD 0.3		CD 0.4		NORMAL	
58	selvi	F	56	A	A	A	A	NORMAL			543	519	12	10	OA	OA	CD 0.5		CD0.4		NORMAL		10	12	OA	OA	CD 0.5		CD0.4		NORMAL		12	12	OA	OA	CD 0.5		CD0.4		NORMAL	
59	vasudaiammal	F	50	A	P	A	A	NORMAL			562	578	14	16	OA	OA	CD0.4		CD0.4		NORMAL		16	14	OA	OA	CD0.4		CD0.4		NORMAL		14	16	OA	OA	CD0.4		CD0.4		NORMAL	
60	vadivukarasi	F	62	A	A	P	P	NORMAL			538	557	22	20	OA	OA	CD0.3		ABNORMAL		22	20	OA	OA	CD0.3		ABNORMAL		ABNORMAL		20	22	OA	OA	CD0.3		STATIC		NORMAL	
61	chidambaram	M	53	A	A	A	P	NORMAL			467	490	16	16	OA	OA	CD 0.6		CD 0.6		NORMAL		14	14	OA	OA	CD 0.6		CD 0.4		NORMAL		14	16	OA	OA	CD 0.6		CD 0.4		NORMAL	
62	shanthivel	M	50	A	P	A	A	NORMAL			551	553	12	12	OA	OA	CD 0.5		CD 0.6		NORMAL		14	12	OA	OA	CD 0.5		CD 0.6		NORMAL		12	14	OA	OA	CD 0.5		CD 0.6		NORMAL	
63	parameshwari	F	63	A	A	A	A	NORMAL			488	496	12	12	OA	OA	CD 0.5		CD 0.5		NORMAL		10	12	OA	OA	CD 0.5		CD 0.5		NORMAL		12	10	OA	OA	CD 0.5		CD 0.5		NORMAL	
64	perumal	M	56	A	A	P	A	NORMAL			523	506	16	14	OA	OA	CD 0.6		CD 0.6		NORMAL		14	16	OA	OA	CD 0.6		CD 0.6		NORMAL		14	14	OA	OA	CD 0.6		CD 0.6		NORMAL	
65	jayasha sekar	M	58	A	A	P	A	NORMAL			540	536	22	20	OA	OA	CD 0.3		NORMAL		20	22	OA	OA	CD 0.3		NORMAL		NORMAL		20	20	OA	OA	CD 0.3		NORMAL		NORMAL	
66	kavitha	F	50	A	A	A	A	NORMAL			557	548	14	12	OA	OA	CD 0.6		NORMAL		12	14	OA	OA	CD 0.6		NORMAL		NORMAL		14	14	OA	OA	CD 0.5		NORMAL		NORMAL	
67	kalaiarasan	M	45	A	A	A	A	NORMAL			489	467	12	14	OA	OA	CD 0.5		CD 0.5		NORMAL		14	12	OA	OA	CD 0.5		CD 0.5		NORMAL		12	12	OA	OA	CD 0.5		CD 0.5		NORMAL	
68	prabhu	M	47	A	A	A	A	NORMAL			543	537	12	14	OA	OA	CD 0.3		CD 0.3		NORMAL		12	12	OA	OA	CD 0.3		CD 0.3		NORMAL		14	12	OA	OA	CD 0.3		CD 0.3		NORMAL	
69	sethirkhan	M	59	A	A	P	P	NORMAL			569	537	18	16	OA	OA	CD 0.6		ABNORMAL		16	16	OA	OA	CD 0.6		ABNORMAL		ABNORMAL		16	18	OA	OA	CD 0.6		CD 0.6		STATIC	

71	kalavathi	F	52	P	A	A	A	NORMAL	565	574	12	14	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	14	16	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	16	14	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
72	kannamma	F	48	A	A	A	A	NORMAL	534	554	12	14	OA	0A	CD 0.5	CD 0.7	NORMAL	NORMAL	14	12	0A	OA	CD 0.5	CD 0.7	NORMAL	NORMAL	12	12	OA	0A	CD 0.5	CD 0.7	NORMAL	NORMAL
73	rajeshwari	F	60	A	A	A	P	NORMAL	567	564	14	14	OA	0A	CD 0.6	CD 0.6	NORMAL	NORMAL	14	12	0A	OA	CD 0.6	CD 0.6	NORMAL	NORMAL	14	16	OA	0A	CD 0.6	CD 0.6	NORMAL	NORMAL
74	manikandan	M	63	A	A	P	P	NORMAL	554	547	16	18	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL	16	16	0A	OA	CD 0.5	CD 0.5	NORMAL	NORMAL	18	16	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL
75	Sudharshan	M	56	A	P	P	A	NORMAL	546	553	12	14	OA	0A	CD 0.4	CD 0.4	NORMAL	NORMAL	14	12	0A	OA	CD 0.4	CD 0.4	NORMAL	NORMAL	14	14	OA	0A	CD 0.4	CD 0.4	NORMAL	NORMAL
76	shivan	M	51	A	P	A	A	NORMAL	556	549	12	10	OA	0A	CD 0.4	CD 0.4	NORMAL	NORMAL	12	12	0A	OA	CD 0.4	CD 0.4	NORMAL	NORMAL	10	12	OA	0A	CD 0.4	CD 0.4	NORMAL	NORMAL
77	ganesan	M	56	A	A	A	P	NORMAL	567	558	16	18	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL	14	16	0A	OA	CD 0.5	CD 0.5	NORMAL	NORMAL	16	14	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL
78	venkatesan	M	53	A	P	A	A	NORMAL	546	551	16	16	OA	0A	CD 0.5	CD 0.4	NORMAL	NORMAL	14	16	0A	OA	CD 0.5	CD 0.4	NORMAL	NORMAL	16	14	OA	0A	CD 0.5	CD 0.4	NORMAL	NORMAL
79	sundararaj	M	64	A	A	P	P	NORMAL	507	531	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	20	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
80	dhanapal	M	57	A	A	A	P	NORMAL	491	498	14	16	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL	14	14	0A	OA	CD 0.5	CD 0.5	NORMAL	NORMAL	16	14	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL
81	**Suseela**	**F**	**66**	**A**	**A**	**P**	**P**	NORMAL	569	554	**12**	**12**	**OA**	**0A**	**CD 0.5**	**CD 0.7**	**ABNORMAL**	**ABNORMAL**	**14**	**12**	**0A**	**OA**	**CD 0.5**	**CD 0.7**	**STATIC**	**STATIC**	**12**	**14**	**OA**	**0A**	**CD 0.5**	**CD 0.7**	**STATIC**	**STATIC**
82	Palanivel	M	50	A	A	A	A	NORMAL	551	568	22	24	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	24	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
83	balakrishnan	M	46	A	P	A	A	NORMAL	534	532	14	14	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL	14	12	0A	OA	CD 0.5	CD 0.5	NORMAL	NORMAL	12	14	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL
84	rajina	F	49	A	P	A	A	NORMAL	565	576	14	14	OA	0A	CD 0.5	CD 0.6	NORMAL	NORMAL	16	14	0A	OA	CD 0.4	CD 0.6	NORMAL	NORMAL	22	22	OA	0A	CD 0.4	CD 0.6	NORMAL	NORMAL
85	Komala	F	60	A	A	P	P	NORMAL	479	498	14	14	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL	14	16	0A	OA	CD 0.5	CD 0.5	NORMAL	NORMAL	16	14	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL
86	vasunthara	F	47	A	A	A	A	NORMAL	457	466	12	14	OA	0A	CD 0.4	CD 0.6	NORMAL	NORMAL	16	18	0A	OA	CD 0.4	CD 0.6	NORMAL	NORMAL	14	16	OA	0A	CD 0.4	CD 0.6	NORMAL	NORMAL
87	dhanasekar	M	56	A	A	A	A	NORMAL	552	546	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	26	24	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
88	rajagopal	M	60	A	A	A	A	NORMAL	556	532	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	22	24	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
89	paneer selvam	M	57	A	A	P	A	NORMAL	546	554	22	20	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	20	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
90	sumathi	F	59	A	A	P	P	NORMAL	574	567	22	24	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	24	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	24	24	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
91	**muthukumar**	**M**	**60**	**A**	**A**	**P**	**A**	NORMAL	572	579	**22**	**24**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**ABNORMAL**	**ABNORMAL**	**20**	**22**	**0A**	**OA**	**CD 0.3**	**CD 0.3**	**STATIC**	**STATIC**	**22**	**22**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**STATIC**	**STATIC**
92	**selvam**	**M**	**61**	**A**	**A**	**P**	**P**	NORMAL	483	478	**20**	**20**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**ABNORMAL**	**ABNORMAL**	**22**	**20**	**0A**	**OA**	**CD 0.3**	**CD 0.3**	**ABNORMAL**	**ABNORMAL**	**20**	**22**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**STATIC**	**STATIC**
93	azhagappan	M	65	A	A	A	A	NORMAL	546	543	20	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	20	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
94	arul	M	55	A	A	A	A	NORMAL	532	545	14	16	OA	0A	CD 0.3	CD 0.5	NORMAL	NORMAL	14	14	0A	OA	CD 0.3	CD 0.5	NORMAL	NORMAL	12	14	OA	0A	CD 0.3	CD 0.5	NORMAL	NORMAL
95	arivazhagan	M	61	A	A	A	P	NORMAL	553	557	20	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	22	20	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
96	murugesan	M	51	A	A	A	A	NORMAL	546	554	14	12	OA	0A	CD 0.5	CD 0.6	NORMAL	NORMAL	12	14	0A	OA	CD 0.5	CD 0.6	NORMAL	NORMAL	14	14	OA	0A	CD 0.5	CD 0.6	NORMAL	NORMAL
97	bharani	M	50	A	A	A	A	NORMAL	549	543	22	20	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	20	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
98	arunaimozhi	F	56	A	A	A	P	NORMAL	556	558	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	24	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
99	visalatchi	F	59	A	A	P	P	NORMAL	538	524	22	20	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	20	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
100	sundaram	M	55	P	A	A	A	NORMAL	529	537	14	12	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	14	14	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	12	14	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
101	**pasupathy**	**M**	**68**	**A**	**A**	**p**	**P**	NORMAL	552	555	**22**	**24**	**OA**	**0A**	**CD 0.4**	**CD 0.4**	**ABNORMAL**	**ABNORMAL**	**24**	**24**	**0A**	**OA**	**CD 0.4**	**CD 0.4**	**ABNORMAL**	**ABNORMAL**	**24**	**22**	**OA**	**0A**	**CD 0.5**	**CD 0.5**	**ABNORMAL**	**ABNORMAL**
102	**govindhan**	**M**	**63**	**A**	**A**	**P**	**P**	NORMAL	566	557	**22**	**22**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**ABNORMAL**	**ABNORMAL**	**22**	**20**	**0A**	**OA**	**CD 0.3**	**CD 0.3**	**ABNORMAL**	**ABNORMAL**	**24**	**22**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**STATIC**	**STATIC**
103	amaravathi	F	53	A	A	A	A	NORMAL	553	546	22	20	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	20	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
104	jeevanandhan	M	48	A	P	A	A	NORMAL	554	563	16	18	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL	18	18	0A	OA	CD 0.5	CD 0.5	NORMAL	NORMAL	16	18	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL
105	**dhanam**	**F**	**52**	**A**	**A**	**A**	**A**	NORMAL	550	544	**14**	**12**	**OA**	**0A**	**CD 0.5**	**CD 0.6**	**ABNORMAL**	**ABNORMAL**	**12**	**12**	**0A**	**OA**	**CD 0.5**	**CD 0.6**	**STATIC**	**STATIC**	**14**	**14**	**OA**	**0A**	**CD 0.5**	**CD 0.6**	**STATIC**	**STATIC**
106	sandhanam	M	57	A	A	P	A	NORMAL	479	488	20	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	20	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	20	20	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
107	muthammal	F	53	A	A	A	A	NORMAL	565	546	22	24	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL	22	22	0A	OA	CD 0.3	CD 0.3	NORMAL	NORMAL	24	22	OA	0A	CD 0.3	CD 0.3	NORMAL	NORMAL
108	kanaga	F	55	A	A	A	P	NORMAL	562	566	14	16	OA	0A	CD 0.5	CD 0.4	NORMAL	NORMAL	16	14	0A	OA	CD 0.5	CD 0.4	NORMAL	NORMAL	14	14	OA	0A	CD 0.5	CD 0.4	NORMAL	NORMAL
109	**thangadurai**	**M**	**56**	**A**	**A**	**P**	**P**	NORMAL	498	487	**22**	**20**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**ABNORMAL**	**ABNORMAL**	**20**	**20**	**0A**	**OA**	**CD 0.3**	**CD 0.3**	**STATIC**	**STATIC**	**22**	**22**	**OA**	**0A**	**CD 0.3**	**CD 0.3**	**STATIC**	**STATIC**
110	prasanth	M	58	A	A	A	P	NORMAL	547	554	12	14	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL	14	16	0A	OA	CD 0.5	CD 0.5	NORMAL	NORMAL	14	12	OA	0A	CD 0.5	CD 0.5	NORMAL	NORMAL