"THE PROFILE OF PRIMARY GLAUCOMA IN A TERTIARY CARE CENTRE AT KOLAR"

By

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In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

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Under the guidance of

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DEPARTMENT OF OPHTHALMOLOGY

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LIST OF ABBREVATIONS

SL NO	ABBREVATIONS	FULL FORM
1	IOP	INTRAOCULAR PRESSURE
2	PAOG	PRIMARY OPEN ANGLE GLAUCOMA
3	PACG	PRIMARY ANGLE CLOSURE GLAUCOMA
4	PACS	PRIMARY ANGLE CLOSURE SUSPECTS
5	POAGS	PRIMARY OPEN ANGLE GLAUCOMA SUSPECT.
6	CNTGS	COLLABORATIVE NORMAL TENSION STUDY
7	AGIS	ADVANCED GLAUCOMA INTERVENTION STUDY
8	CIGTS	COLLABORATIVE INITIAL GLAUCOMA TREATMENT STUDY.
9	OHTS	OCULAR HYPERTENSION TREATMENT STUDY.
10	EMGT	EARLY MANIFEST GLAUCOMA TRIAL
11	CCT	CENTRAL CORNEAL THICKNESS
12	GAT	GOLDMANN APPLANATION TONOMETRY

13	APEDS	ANDHRA PRADESH EYE DISEASE STUDY.
14	VES	VELLORE EYE SURVEY
15	CGS	CHENNAI GLAUCOMA SURVEY
16	ACES	ARAVIND COMPREHENSIVE EYE SURVEY.
17	WBGS	WEST BENGAL GLAUCOMA SURVEY

ABSTRACT

BACKGROUND: Glaucoma afflicts almost 67 million people worldwide of which 6.6 million are blind. It remains the leading cause of irreversible blindness after cataract. According to the World Health Organization model, densely populated countries should be encouraged to carry out periodic surveys of the magnitude and causes of visual impairment of the disease.

METHODS: This is an observational study where 3500 patients visiting the ophthalmology outpatient department was screened for established and suspected primary glaucoma cases and their risk factors were studied.

OBJECTIVES: To determine the proportions of primary open angle glaucoma and primary angle closure glaucoma and study the risk factors.

RESULTS. 72 cases (2.09%) were in the primary open angle group comprising of POAG (0.94%) cases and POAG suspects (1.11%). 27 cases (0.77%) were PACG, 85 cases (2.42%) were diagnosed as PACS. Mean age of POAG and PACG was 64.50 ± 13.06 68.60 ± 10.94 years, respectively. Male to Female ratio in PAOG and PACG was 2:1 and 1:2.4 respectively.10 (41.7%) myopic patients had POAG, 8 (29.6%) hyperopic patients had PACG.10 cases (30.3%) of POAG and 15 cases (33.33%) of PACG had a positive family history.

INTERPRETATION & CONCLUSION: From the observations of this study, all subjects above forty years should be screened for glaucoma, and thorough evaluation of patients with additional risk factors like refractive error and strong family history of glaucoma should be done.

KEYWORDS: Primary Open Angle Glaucoma, Primary open angle glaucoma suspects, Primary Angle Closure glaucoma, Primary angle closure suspects, , Risk factors.

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INTRODUCTION

Glaucoma is a leading cause of irreversible blindness throughout the world. World Health Organization statistics, published in 1995, indicate that glaucoma accounts for blindness in more than 5.1 million people, or 13.5% of global blindness (behind cataracts and trachoma at 15.8 million and 5.9 million persons, or 41.8% and 15.5% of global blindness, respectively).[1] Worldwide, it has become the second most common cause of bilateral blindness.[2]

Currently, we define the glaucoma as a group of optic neuropathy characterized by recognizable patterns of optic disc and retinal nerve fiber structural and visual field functional damage, which may or may not be associated with a raised IOP.[3]

Glaucomatous optic neuropathy is not the disease; it is the end-result of several as yet unidentified cellular disease processes. The contour changes of the optic nerve head ("cupping") with progressive loss of the retinal nerve fiber layer and associated functional deficits are hallmark of the disease; which is due to accelerated retinal ganglion cell apoptosis.

Thus, the definition varies with the perspective of the definer: retinal ganglion cell apoptosis to a scientist, optic neuropathy to a clinician, and fear of blindness for a patient.

Clinico-etiologically glaucoma may be classified as follows:

(A) Congenital and developmental glaucoma

1. Primary congenital glaucoma (without associated anomalies).

2. Developmental glaucoma (with associated anomalies).

(B) Primary adult glaucoma

1. Primary open angle glaucoma (POAG)

2. Primary angle closure glaucoma (PACG)

3. Primary mixed mechanism glaucoma

(C) Secondary glaucoma.

ANATOMICAL CONSIDERATIONS

Maintenance of intraocular pressure and pathophysiology of glaucoma revolves around the aqueous humour dynamics. Aqueous humour is produced by the pars plicata of the ciliary body (which is forward continuation of the choroid at the ora serrata), from which it enters the posterior chamber, passes through the pupil, enters the anterior chamber and drains into the peripheral recess or angle of the anterior chamber.

The angle plays an important role in the process of aqueous drainage. Clinically, the angle structures can be visualized by gonioscope examination. Starting from posterior to anterior, the angle recess is formed by the following structures:

- **THE CILIARY BAND**

It is the posterior most landmark in the angle recess. It is formed by the anterior most part of the ciliary body between its attachment to the scleral spur and insertion of iris.

- **SCLERAL SPUR**

It is the posterior portion of the scleral sulcus, which usually appears as a prominent white line on gonioscopy. On it are attached ciliary body posteriorly and corneoscleral meshwork anteriorly. The scleral spur-roll is composed of 75% to 80% collagen and 5% elastic tissue.

- **TRABECULAR MESHWORK**

It is seen as a band just anterior to scleral spur. Its appearance varies considerably since it has no pigments at birth and develops increasing pigments with age.

- **SCHWALBE'S LINE**

It is a fine ridge seen just in front of the trabecular meshwork. It is formed by the oblique insertion of uveal trabeculae into limbal stroma. It marks the anterior limit of the structures forming the angle of the anterior chamber.

- **THE AQUEOUS OUTFLOW SYSTEM**

It includes the trabecular meshwork, Schlemm's canal, collector channels, aqueous veins and the episcleral veins.

TRABECULAR MESHWORK

It is seen as a band just anterior to the scleral spur.

It is a sieve – like structure through which aqueous humour leaves the eye. It bridges the scleral sulcus and converts it into a tube, which accommodates Schlemm's canal. This tissue consists of a connective tissue core surrounded by endothelium and may be divided into three portions: (a) uveal meshwork, (b) corneoscleral meshwork, and (c) juxtacanalicular tissue, which is sometimes referred to as the cribriform layer

a) Uveal meshwork

It is the innermost part of the trabecular meshwork and extends from the iris root and ciliary body to the Schwalbe's line. The arrangement of uveal trabecular bands creates irregular openings, which vary in size from 25 microns to 75 microns.

b) Corneoscleral meshwork

It forms the larger middle portion and extends from the scleral spur to the lateral wall of the scleral sulcus. It consists of flat sheets of trabeculae, which are perforated by elliptical openings, which are smaller than those in the uveal meshwork (5-50 microns).

c) Juxtacanalicular (endothelial) meshwork

It forms the outermost portion of the trabecular meshwork. This part of the trabecular meshwork offers the normal resistance to aqueous outflow.

- **SCHLEMM'S CANAL**

This is an endothelial lined oval channel present circumferentially in the sclera sulcus. The outer wall of the canal contains the numerous openings of the collector channels.

- **COLLECTOR CHANNELS**

These intrascleral aqueous vessels are about 25 - 35 in number and leave the Schlemm's canal at oblique angles to terminate ultimately into episcleral veins.

- **EPISCLERAL VEINS**

Most of the aqueous vessels drain into the episcleral veins. The episcleral veins ultimately drain into the cavernous sinus via the anterior ciliary and superior ophthalmic veins.

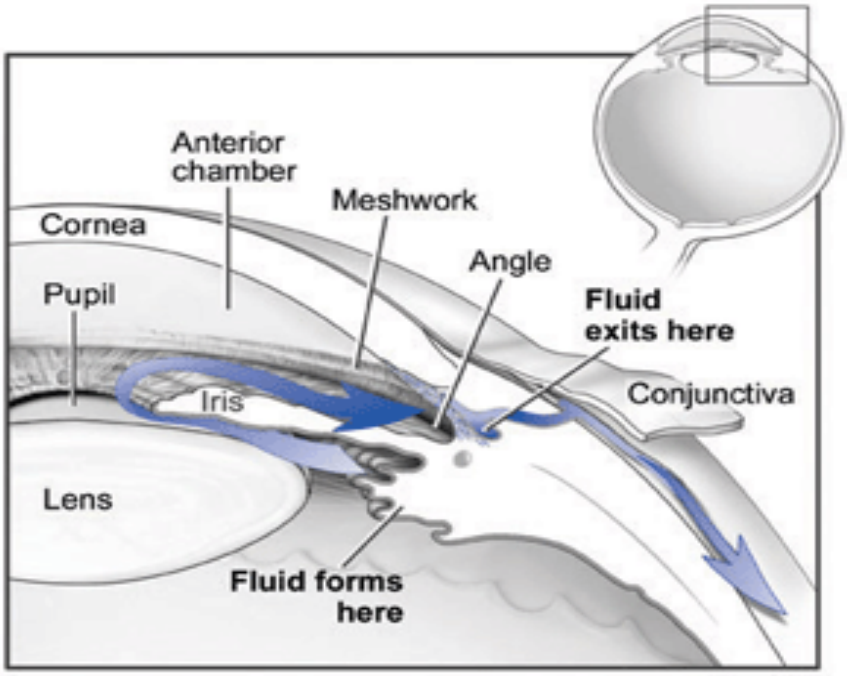

FIGURE 1: ANGLE OF ANTERIOR CHAMBER

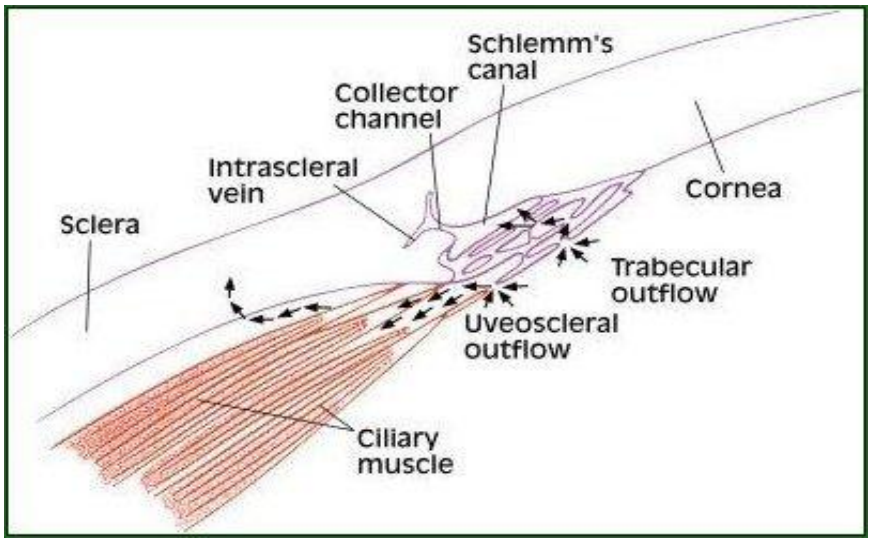

FIGURE 2: UVEOSCLERAL OUTFLOW SYSTEM

FORMATION OF AQUEOUS HUMOUR

The aqueous humour is primarily derived from the plasma within the capillary network of the ciliary processes. The various constituents of the aqueous humour have to traverse the three tissue layers viz., the capillary wall, the stroma and the two layers of the epithelium; for reaching the posterior chamber from the plasma within the capillary network of the ciliary processes. The various substances appear to pass through these layers by the following processes[: 4]

I. Diffusion

Diffusion is a biophysical process by which molecules of a gas or solution distribute themselves uniformly throughout the space in which they are contained, by motion of its particles. In this process, there occurs a net flux of the particles from areas of high concentration to areas of low concentration. In the process of aqueous production, the lipid soluble substances are transported by diffusion through the lipid portions of the cell membrane of the ciliary processes, proportional to a concentration gradient across the membrane.

II. Ultrafiltration

When a solution of protein and salt is separated from plain water or a less concentrated salt solution by a membrane permeable to salt and water and not the protein, there will be a net movement of water on the protein side by diffusion and a movement of the salt away from the protein side. This process is called dialysis. Ultrafiltration refers to occurrence of dialysis under hydrostatic

pressure. In the process of aqueous formation, water and water soluble substances (limited by size and charge) flow through the theoretical micro pores in the protein part of the cell membrane in response to osmotic gradient or hydrostatic pressure.

III. Secretion (active transfer)

Secretion implies an active process that selectively transports some substances across the cell membrane. With the energy consumed, substances can be moved across a concentration gradient in a direction opposite to what would be expected by passive mechanism alone. In the process of aqueous formation, water soluble substances of larger size or greater charge are actively transported across the cell membrane. This mechanism is probably mediated by globular proteins in the membrane and requires the expenditure of energy.

CELLULAR ORGANIZATION OF THE UVEOSCLERAL PATHWAY

The unconventional outflow for aqueous humor outflow has not been studied as extensively as the trabecular outflow pathway. Historically, two unconventional pathways have been discriminated:

a. Through the anterior uvea at the iris root and anterior surface of the ciliary body, which is referred to the uveoscleral pathway, and
b. Through transfer of fluid into the iris vessels and vortex veins, which has been described as uveovortex outflow.

UVEOSCLERAL OUTFLOW

Tracer studies in human [5] and animal [6, 7] eyes have shown that aqueous humor passes through the root of the iris and interstitial spaces of the ciliary muscle to reach the suprachoroidal space. From there it passes to episcleral tissue via scleral pores surrounding ciliary blood vessels and nerves, vessels of optic nerve membranes, or directly through the collagen substance of the sclera. The extracellular matrix of normal human ciliary muscle contains collagen types I, III, and IV, fibronectin, and laminin in association with muscle fibers and blood vessels, and it has been suggested that the biosynthesis and turnover of these glycoproteins may play an important role in resistance to flow within the unconventional pathways and in mediating the action of certain pharmacologic agents.

UVEOVORTEX OUTFLOW

Tracer studies in primates have also demonstrated unidirectional flow into the lumen of iris vessel by vesicular transport, which is not energy dependent. The tracer can penetrate vessels of the iris, ciliary muscle, and anterior choroid to eventually reach the vortex veins. However, the role of net fluid movement into the iris vasculature is probably not clinically significant.

PRIMARY OPEN ANGLE GLAUCOMA

POAG is clearly the most common single form of glaucoma. A proposed definition of POAG is a multifactorial optic neuropathy in which there is characteristic atrophy of the optic nerve (modified from the American Academy of Ophthalmology Preferred Practice Guidelines, 2000). Within this large group of glaucoma, however, the most common form (or forms) is typically characterized by the following three criteria: (a) an intraocular pressure (IOP) consistently above 21 mm Hg in at least one eye; (b) an open, normal-appearing anterior chamber angle with no apparent ocular or systemic abnormality that might account for the elevated IOP; and (c) typical optic nerve head damage and/or glaucomatous visual field damage.

IDENTIFYING PATIENTS AND THOSE AT RISK

POAG has no associated symptoms or other warning signs before the development of advanced visual field loss. It is for this reason that public and family physician awareness programs are needed to ensure that high-risk patients receive glaucoma assessment examinations by ophthalmologists and optometrists. Once a patient has been found to have persistent IOP elevation but no apparent optic nerve head or visual field damage, the additional risk factors must be considered by the physician when trying to decide which of these individuals require closer observation or the initiation of therapy before definite damage occurs.

TABLE 1: RISK FACTORS WITH RELATIVE RISK

GOOD EVIDENCE

RISK FACTOR	RELATIVE RISK
Age (per decade over 40)	2
Blacks v/s whites	4
Family history (first-degree relative)	2 – 4
Myopia	1.5 – 3
Pseudo exfoliation	5 – 10
Diastolic perfusion pressure (<55 mm Hg)	3

FAIR EVIDENCE	WEAK EVIDENCE
• Large C/D ratio • Diabetes mellitus • Optic disc hemorrhage	• Systolic blood pressure • Peripapillary atrophy • Migraine (for NTG) • Hypothyroidism • Sleep apnea • Autoimmune

CLINICAL FEATURES

PRIMARY OPEN-ANGLE GLAUCOMA

Visual Abnormalities

Central visual acuity, as measured by standard clinical tests, typically remains normal until peripheral visual field loss is advanced and is, therefore, of no value in the early detection of POAG. Once typical glaucomatous damage to the visual field has been documented in one eye, there is a high incidence of subsequent field loss in the fellow eye. The latter was reported to be 29% in 31 patients followed up for 3 to 7 years [9], and 25% of 104 individuals after 5 years of follow-up in another series.[10]

ANTERIOR SEGMENT SIGNS.

Cornea

The cornea is typically normal in POAG. In one study, patients with cornea guttata were reported to frequently have abnormal tonometric values, [11] and preliminary specular microscopic studies of patients with chronic glaucoma suggested abnormal corneal endothelium.[12] However, a subsequent study comparing individuals with normal IOP, untreated ocular hypertension, treated ocular hypertension, or POAG revealed no significant difference in the central corneal endothelial density or central corneal thickness.[13] Most studies, however, suggest that patients diagnosed with ocular hypertension have a higher incidence of increased central corneal thickness than glaucoma or control subjects, which may cause an artifactual elevation of the IOP measurements.

Of greater significance, thinner central corneal thickness, implying artifactualy lower IOP measurements, was shown to be a significant risk factor for development of COAG in the Ocular Hypertension Treatment Study.[14]

Optic Nerve Head

The mean area of the optic disc in whites is 2.1–2.8 mm^2 and is independent of age after the first decade of life. Disc size is related to race – it tends to be smaller in whites, intermediate among Asians, and largest among blacks.

When the smallest 5° aperture of the direct ophthalmoscope is projected onto the retina in eyes with a refractive error within 4D of emmetropia, the circular spot has an area of 1.7 mm^2 – and thus provides a reliable estimation of whether an optic disc is clinically larger or smaller than normal. Another technique for clinical-research use is the calculation of the disc area based on measuring the horizontal and vertical diameters of the ONH through a slit beam.
A distinctive category of disc appearance that is commonly seen in POAG with high IOPs includes a disc of normal size with diffusely enlarged round cups. Localized rim defects are uncommon, so abnormality or progression of cup enlargement necessitates comparison with the fellow eye, with serial photographs, or drawings. The cup increase is often biased toward the temporal rim, with gradual attenuation of the neural rim.

The appearance of the optic nerve head and peripapillary retina is the single most important clinical feature in establishing the presence of

glaucomatous damage. A helpful early finding is defects in the retinal nerve fiber layer, which may be a sign of glaucomatous optic atrophy before apparent changes are seen in the nerve head. Other early findings include enlargement of the optic dose cup, thinning or saucerizing of the neural rim, disc hemorrhages, and peripapillary atrophy.

INVESTIGATIONS

Intraocular Pressure

As previously noted, part of the definition of POAG is an IOP greater than 21 mm Hg before treatment. Even though an elevated IOP is only one of several risk factors for the development of POAG, it is a causative risk factor and most studies agree that it is the single most important risk factor. In the initial stages the IOP may not be raised permanently, but there is an exaggeration of the normal diurnal variation. In most patients IOP falls during the evening, contrary to closed angle glaucoma.

Gonioscopy

By traditional definition, the anterior chamber angle in eyes with POAG is open and grossly normal. Preliminary studies, however, suggest that these patients may have more iris processes, a higher insertion of the iris root, more trabecular meshwork pigmentation [15], and a greater than normal degree of segmentation in the pigmentation of the meshwork.

Visual Field Test

The current generation of computerized perimeters allows placement of stimuli of varying sizes, intensities, and colors into backgrounds of varying intensities, and they accurately chart the patient's responses. Other technologies are available and being developed in the hope that formal visual field testing will become easier, more reliable, more affordable, and more widespread – using equipment that can detect glaucoma earlier than standard perimetry. These emerging technologies include:

- Short-wavelength (blue–yellow) automated perimetry (SWAP)
- Frequency doubling technology (FDT) perimetry
- Motion displacement perimetry (MDP).

ANALYSIS OF VISUAL FIELD LOSS PRIMARY OPEN-ANGLE GLAUCOMA

In the early stages there may be a generalized depression that progresses gradually or sometimes in steps from paracentral scotomata to arcuate to altitudinal to end-stage defects. Defects usually become denser and then increase in area in one hemi field before progressing to the next hemi field.

It has been speculated that increased intraocular pressure (IOP) may cause diffuse loss but have less influence on the development of localized defects. The association was stronger for patients with primary angle-closure glaucoma (PACG) than those with primary open-angle glaucoma (POAG). This supports the concept that increased IOP is the proximal cause of damage in PACG, but

that other factors may predominate in at least some patients with POAG. In both circumstances, the amount of field loss correlated well with the amount of optic nerve damage.[9]

PRIMARY ANGLE-CLOSURE GLAUCOMA

Angle-closure glaucoma is characterized by apposition of the peripheral iris against the trabecular meshwork, resulting in obstruction of aqueous outflow .Traditionally, some forms of angle-closure glaucoma have been referred to as primary angle-closure glaucoma (PACG) because the mechanisms of angle closure were not thought to be associated with other ocular or systemic abnormalities or because the mechanisms were not well understood. Conditions that have been included in this group are pupillary-block glaucoma, plateau iris, and combined mechanism glaucoma.

PUPILLARY-BLOCK GLAUCOMA

Pupillary-block glaucoma, a category of angle-closure glaucoma, is the most common form of these conditions. The initiating event is thought to result from increased resistance to flow of aqueous humor between the pupillary portion of the iris and the anterior lens surface, which is associated with mid-dilatation of the pupil. The functional block produces increased fluid pressure in the posterior chamber, causing a forward shift of the iris. Anterior movement of the peripheral iris can result in closure of the anterior chamber angle .Four forms

of pupillary-block glaucoma may be distinguished on the basis of symptoms and clinical findings:

- Acute angle-closure glaucoma,
- Sub-acute angle-closure glaucoma,
- Chronic angle-closure glaucoma, and
- Combined-mechanism glaucoma.

ACUTE ANGLE-CLOSURE GLAUCOMA

In acute angle-closure glaucoma, the symptoms are sudden and severe, with marked pain, blurred vision, and a red eye. The patient may also have nausea and vomiting.

SUBACUTE ANGLE-CLOSURE GLAUCOMA

Sub-acute angle-closure glaucoma is thought to have the same pupillary-block mechanism as the acute form, but symptoms are mild or absent. The condition has also been called intermittent, prodromal, or subclinical. Patients with sub-acute angle-closure glaucoma may have repeated sub-acute or subclinical attacks before finally having an acute attack or developing peripheral anterior synechiae with chronic pressure elevation.

CHRONIC ANGLE-CLOSURE GLAUCOMA

In chronic angle-closure glaucoma, portions of the anterior chamber angle are permanently closed by peripheral anterior synechiae, and the intraocular pressure (IOP) is chronically elevated. The synechial closure may result from a prolonged acute attack or repeated sub-acute attacks of angle-closure glaucoma.

TWENTY-FIRST CENTURY CONSENSUS CLASSIFICATION

The new classification of primary angle-closure (PAC) disease relies on three simple categories: IOP measurement, gonioscopy, and disc and visual field evaluation.

1. Primary angle closure SUSPECT (PACS): greater than 270° of irido-trabecular contact plus absence of peripheral anterior synechiae (PAS) plus normal IOP, disc, and visual field. The angle is at risk.
2. Primary angle CLOSURE (PAC): greater than 270° of irido-trabecular contact with either elevated IOP and/or PAS plus normal disc and visual field examinations.
3. Primary angle-closure GLAUCOMA (PACG): greater than 270° of irido-trabecular contact plus elevated IOP plus optic nerve and visual field damage. In other words, angle closure glaucoma manifests the criteria of closure above, plus demonstrable disc and/or visual field changes. The angle is abnormal in structure and function, with optic neuropathy.

RISK FACTORS

General Features of Patients

Several factors influence the configuration of the anterior chamber angle and the risk of developing pupillary block glaucoma.

Age

The depth and volume of the anterior chamber diminish with age, which may result from a thickening and forward displacement of the lens. Consequently, the percent of individuals with critically narrow angles is higher in older age groups. However, it can occur at any age, including rare cases in childhood.

Race

The relative prevalence of pupillary-block glaucoma among all forms of glaucoma is increased in various populations of Inuit[17, 18] and individuals with far Eastern Asian extraction[19]. Acute angle-closure glaucoma is less common among blacks, but sub-acute or chronic angle-closure glaucoma is not uncommon and appears to be a regularly missed diagnosis.

Gender

There is a statistically significant predominance of females in populations with pupillary-block glaucoma, which is probably caused by the shallower anterior chamber among women in general[17, 18].

Refractive Error

The depth and volume of the anterior chamber are related to the degree of emmetropia, with smaller dimensions occurring in hypermetropia.

Family History

The potential for pupillary-block glaucoma is generally believed to be inherited [20]. In one study, 20% of 95 relatives of angle-closure glaucoma patients were thought to have potentially occludable angles[21]. However, aside from a few reported families in which many members developed angle-closure glaucoma, the family history is not very useful in predicting a future angle- closure attack.

PRECIPITATING FACTORS

In an eye that is anatomically predisposed to develop angle-closure, several factors may precipitate an attack.

Dim illumination

A common history for the development of pupillary-block glaucoma is the onset of an acute attack when the patient is in a dark room, such as a theater or restaurant. It has been reported that the incidence of angle-closure increases in the winter and autumn.

Emotional stress

Occasionally, an acute angle-closure attack follows severe emotional stress. This may be related to the mydriasis due to increased sympathetic tone.

Drugs

Mydriatics may also precipitate an angle-closure attack in an anatomically predisposed eye.

Anticholinergics (e.g., atropine, cyclopentolate, tropicamide) are particularly high risk when administered topically. In one study, 0.5% cyclopentolate precipitated attacks in 9 (43%) of 21 high-risk eyes, and 0.5% tropicamide did the same in 19 (33%) of 58 eyes [22]. However, in a population-based screening study of 4,870 subjects whose eyes were dilated with 1% tropicamide and 2.5% phenylephrine after penlight examination of the anterior chamber depth, none developed an acute angle-closure attack. Other systemic drugs with weaker anticholinergic properties (e.g., antihistaminic, anti-parkinsonian, antipsychotic, and gastrointestinal spasmolytic drugs) also present a risk proportional to their pupillary effect.

Adrenergics (e.g., topical epinephrine) may precipitate an angle-closure attack in the predisposed eye. Phenylephrine can also precipitate an attack, although it was found to be safer than cyclopentolate or tropicamide for dilating high-risk eyes.

THEORIES OF MECHANISM

RELATIVE PUPILLARY BLOCK

The most common mechanism leading to angle-closure glaucoma appears to be increased resistance to aqueous flow from the posterior to the anterior chamber between the iris and lens. This concept was suggested by Curran[16] and Banziger [24] in the early 1920s and was advanced by the teachings of Chandler [23], who observed that an eye with a shallow anterior chamber has a wider zone of contact between the surfaces of the iris and lens. It has been suggested on the basis of gonioscopic studies that the angle closure occurs in two stages: iridocorneal contact anterior to the trabecular meshwork, followed by apposition of the iris to the meshwork as the pressure rises [25, 26]

Anatomic Factors Predisposing to Pupillary Block

Several anatomic aspects of the eye combine to produce a shallow anterior chamber. These include a thicker, more anteriorly placed lens, a smaller diameter and shorter posterior curvature of the cornea, and a shorter axial length of the globe. The ratio of the lens thickness to the axial length appears to correlate best with the predisposition to angle closure[,27].All of these parameters are variably influenced by hyperopia, increasing age, and genetics. Another factor predisposing to a pupillary block mechanism may be a forward displacement to the lens due to loose zonules.

SYMPTOMS OF ANGLE-CLOSURE ATTACK

Angle-closure glaucoma, in marked contrast to chronic open-angle glaucoma, is characterized by profound symptoms, although the severity of these symptoms varies considerably in different forms of the disorder.

Acute Angle-Closure Glaucoma

Acute angle-closure glaucoma is characterized by pain, redness, and blurred vision. The pain is typically a severe, deep ache that follows the trigeminal distribution and may be associated with nausea, vomiting, bradycardia, and profuse sweating. The marked conjunctival hyperemia usually consists of a ciliary flush and peripheral conjunctival congestion. The blurred vision, which is typically marked, may be caused by stretching of the corneal lamellae initially and later edema of the cornea, as well as a direct effect of the IOP on the optic nerve head. Rarely, the corneal decompensation may persist, requiring penetrating keratoplasty.

Sub-acute Angle-Closure Glaucoma

Sub-acute angle-closure glaucoma, a form of pupillary-block glaucoma, may have no recognizable symptoms. In other cases, the patient may notice a dull ache behind the eye or slight blurring of vision. A symptom that is especially typical of the sub-acute attack is colored halos around lights. This is thought to result from corneal epithelial edema, which causes it to act as a diffraction grating, producing a blue-green central and yellow-red peripheral

halo. These symptoms, which more often occur at night after the patient has been in a dark room, often spontaneously clear by the next morning, presumably because of the meiosis of sleep.

Chronic Angle-Closure Glaucoma

Another form of pupillary-block glaucoma, chronic angle-closure glaucoma, is typically asymptomatic until advanced visual field loss develops, although the patient may give a history suggestive of one or more episodes of sub-acute or acute angle-closure glaucoma. Peripheral anterior synechiae may eventually develop with prolonged or recurrent acute or sub-acute attacks, leading to chronic angle-closure glaucoma. The peripheral anterior synechiae in patients after acute angle-closure attacks tend to be broad based, are most commonly seen in the superior quadrant. The synechial closure usually begins superiorly, where the angle is normally narrowest, and progresses inferiorly. This condition has been referred to as shortening of the angle or creeping angle closure.

EXAMINATION OF ANTERIOR CHAMBER

Penlight Examination

The anterior chamber depth can be estimated with oblique penlight illumination across the surface of the iris. With the light coming from the temporal side of the eye, a relatively flat iris is illuminated on the temporal and nasal sides of the pupil, whereas an iris that is bowed forward has a shadow on the nasal side.

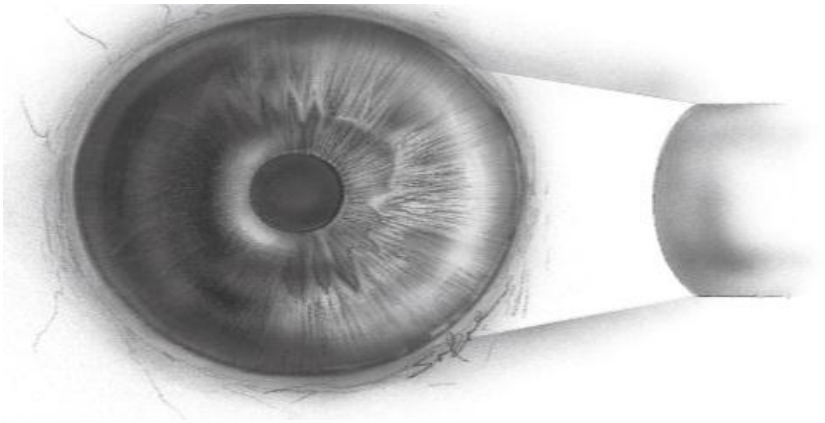

FIGURE 3: ILLUMINATION FROM THE TEMPORAL SIDE CASTS SHADOW ON IRIS IF THERE IS CONSIDERABLE BOMBÉ.

Slit-Lamp Examination

The central anterior chamber depth may be estimated during examination with the slit-lamp, and several techniques for quantitating this parameter have been proposed. Van Herick ET al.[28] developed a technique for making this estimation with the slit-lamp by comparing the peripheral anterior chamber depth to the thickness of the adjacent cornea. This is commonly referred to as the Van Herick technique. When the peripheral anterior chamber depth is less than one fourth of the corneal thickness, the anterior chamber angle may be potentially occludable.

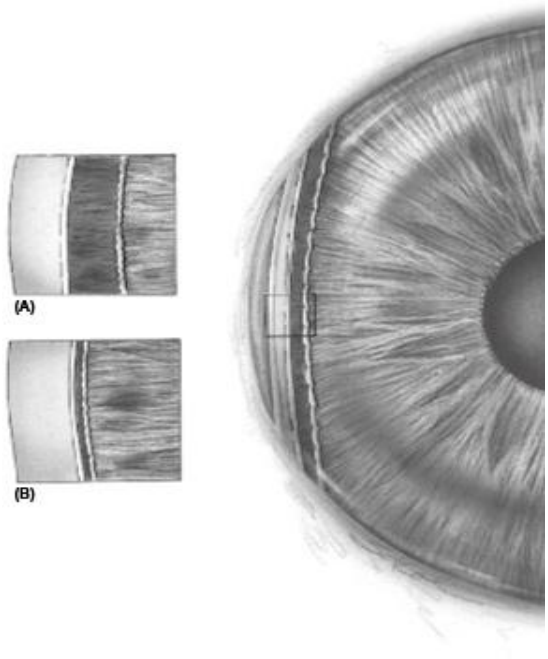

FIGURE 4: SLIT-LAMP EXAMINATION OF THE PERIPHERAL ANTERIOR CHAMBER. (A) If the distance between the iris surface and the corneal endothelium is equal to the corneal thickness, the angles are likely to be deep. (B) Conversely, if the distance is less than one-fourth of the corneal thickness, the angles are likely to be narrow.

Gonioscopy

When the peripheral anterior chamber depth is thought to be shallow (i.e., less than one fourth of the corneal thickness by Van Herick slit-lamp examination), careful gonioscopic examination of the angle is required. This is best accomplished with a Zeiss four-mirror lens or similar goniolens. If 180 degrees or more of the angle is closed (i.e., trabecular meshwork is not visible), it constitutes the definition of an occludable angle, and it is important to use compression gonioscopy to determine whether the closure is appositional or synechial.

Numerous grading systems have been suggested in an attempt to correlate gonioscopic appearance with the potential for angle-closure. Scheie [29] proposed a system based on the extent of the anterior chamber angle structures that can be visualized .He observed a high risk of angle closure in eyes with grade III or IV angles. Shaffer[30] suggested using the angular width of the angle recess as the criterion for grading the angle and attempted to correlate this with the potential for angle-closure.

SHAFFER'S GONIOSCOPIC CLASSIFICATION

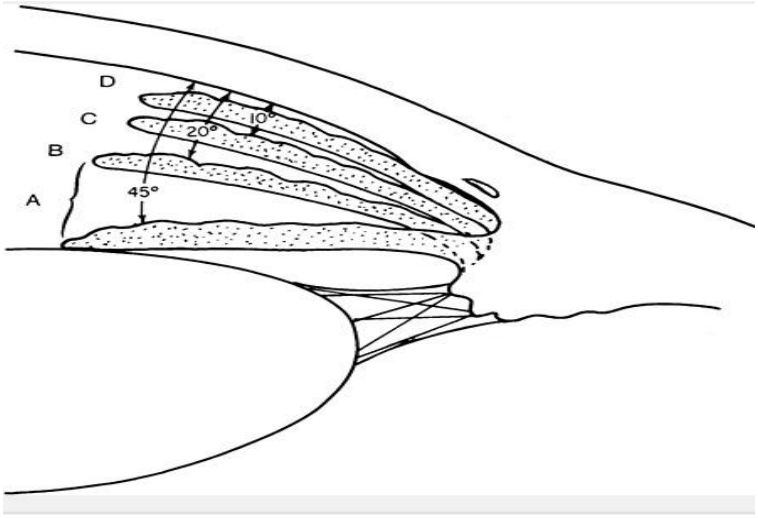

FIGURE 5: SHAFFER'S GONIOSCOPIC CLASSIFICATION OF THE ANTERIOR CHAMBER ANGLE IS BASED ON THE ANGULAR WIDTH OF THE ANGLE RECESS. The angular width and clinical interpretation are given for each of the examples. A: Wide open (20 to 45 degrees): closure improbable. B: Moderately narrow (10 to 20 degrees): closure possible. C: Extremely narrow: closure possible. D: Partially or totally closed: closure present

CLINICAL FINDINGS DURING AN ACUTE ATTACK

The patient who presents during an acute angle-closure attack will typically have marked IOP elevation in the range of 40 to 60 mm Hg or more, with a profound reduction in central visual acuity. Digital palpation can reveal a very firm (i.e., rock-hard consistency) eye compared with the fellow eye, which feels much softer.

External Examination

Characteristic findings include conjunctival hyperemia, a cloudy cornea, and an irregular (usually vertically oval), mid-dilated, fixed pupil.

The pupillary change is thought to result from paralysis of the sphincter, which apparently is caused by a reduction in circulation induced by the elevated IOP and possibly by degeneration of the ciliary ganglion.

Slit-Lamp Examination

This step of the evaluation confirms the presence of the corneal edema. The corneal edema usually clears after the pressure is normalized, although this is not always the case. Specular microscopic examination has revealed significant corneal endothelial cell loss in these cases, which correlates with the duration of IOP elevation the degree of visual field loss, a large cup-disc ratio, and previous intraocular surgery.

The anterior chamber is shallow, but it typically is formed centrally with anterior bowing of the mid-peripheral iris, often making contact with peripheral cornea.

Aqueous flare is often present. Other findings may include pigment dispersion, sector atrophy of the iris, posterior synechiae, and glaukomflecken.

Gonioscopy

It is essential to confirm the diagnosis of angle-closure glaucoma by demonstrating a closed anterior chamber angle. If gonioscopy is not possible because of persistent corneal edema, gonioscopy of the fellow eye may provide useful information if it reveals an extremely narrow angle. Peripheral anterior synechiae may also be present, and documenting the presence and extent of the synechiae is important in establishing the nature of the angle-closure glaucoma and in selecting the appropriate treatment

Fundus Examination

The optic nerve head may be hyperemic and edematous in the early stages of the attack. In a study of human eyes with a history of angle-closure glaucoma, pallor without cupping was seen in eyes after acute attacks, but pallor and cupping occurred in chronic cases [31]. Central retinal vein occlusion may also occur during acute angle-closure glaucoma [32].

Visual Fields

Visual field changes associated with an acute elevation of IOP most often show nonspecific constriction. In one study of 25 patients with acute angle-closure glaucoma that had been surgically corrected, the most common field defect was constriction of the upper field [33], whereas another revealed nerve fiber bundle defects in 7 of 18 acute and 9 of 11 chronic cases [32].

OBJECTVES OF STUDY

OBJECTVES OF STUDY

The objective of the study is to:

1. Determine the proportions of primary open angle glaucoma and primary angle closure glaucoma.

2. Study the risk factors of primary open angle glaucoma and primary angle closure glaucoma.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Since the time of Hippocrates, the glaucoma have mystified physicians. Hippocrates is credited for the term "glaucosis," which translates to "sea green eye. It was not until the beginning of the nineteenth century that Antoinne-Pierre Demours (1818) gave the first excellent description of glaucoma with a raised ocular tension in a treatise. The clinical picture was fully detailed and he described for the first time the appearance of the colours of a rainbow around the lights. In London, Guthrie GJ recognized hardness of the eye as a characteristic of a disease, which he termed 'Glaucoma'.

The link with disc cupping followed Hermann von Helmholtz's 1860s invention of the ophthalmoscope and Albrecht von Graefe's observations. Thus, there arose the structural nerve head-based definitions: Glaucoma was considered a neurological disease. The association with raised intraocular pressure (IOP) occurred over several centuries but was boosted by improvements in tonometers between 1880 and 1910.

With the advent of the gonioscope, various others pointed out that glaucoma could be divided into two types, one with an open angle and other with closed angle.

EPIDOMOLOGY:

Population studies have yielded a wealth of data about glaucoma prevalence, incidence, and risk factors in Caucasian, Latino, African-American, Afro-Caribbean, Indian sub-continental, and Oriental population. Second only to cataract, globally glaucoma is the leading cause of visual disability. As the damage caused is irreversible, but mostly avoidable by treatment, glaucoma is the leading cause of preventable blindness. As prevalence increases exponentially with increasing age, glaucoma is set to become increasingly and relentlessly a worldwide public health issue as populations gray.

The percentage undiagnosed is far greater in underprivileged communities up to 90% of glaucoma patients are not diagnosed.[34, 35]

POPULATION-BASED ("UNIVERSAL") SCREENING

Screening is the use of a test or tests on a target population to find cases of disease. There are different types of screening: targeted and mass population screening. Tools used for screening should fulfill certain criteria. There is no one good mass screening test for glaucoma. As stated by the World Health Organization in 1968, the principles of screening are [36]:

1. The condition should be an important health problem.
2. There should be an accepted treatment for the patients with recognized disease.
3. Facilities for diagnosis and treatment should be available.
4. There should be a recognizable latent or early symptomatic stage.
5. There should be a suitable test or examination.
6. The test should be acceptable to the population.
7. The natural history of the condition, including development from latent to declared

disease should be adequately understood.

8. There should be an agreed policy on whom to treat as patients.
9. The cost of case finding (including diagnosis and treatment of patients diagnosed) should be economically balanced in relation to possible expenditure on medical care as a whole.
10. Case-finding should be a continuing process and not a "once and for all" project.

GLAUCOMA RISK FACTORS: INTRAOCULAR PRESSURE

For the first time, glaucoma was described as a blinding disease associated with high intraocular pressure (IOP) by the Persian physician Ali ibn Rabban at-Tabari (810–861 C.E.) in the writings Firdaws al Hikma (Paradise of Wisdom).[37] In the 1800s, the Dutch ophthalmologist Franciscus C. Donders coined the expression "simple glaucoma" for increased IOP occurring without any inflammatory symptoms. In population-based surveys, intraocular pressure (IOP) has been consistently shown to be a continuous, positive risk factor for the prevalence of glaucoma, even though the normal range of IOP. The association of IOP and open angle glaucoma was first confirmed in studies in the 1990s, although still not in the form of a cause-effect relationship. In the Baltimore Eye Survey [38] and the Barbados Eye Study [39], IOP was found to be an important factor in glaucoma that correlated with increased prevalence and incidence [40]. Population-based screening for glaucoma for long has been handicapped by an undue reliance on IOP levels. The number 21 mmHg as the borderline between normal and abnormal began with Leydhecker's groundbreaking study in 1958 when the IOP of 20,000 eyes was measured with Schiotz tonometry, and a mean of 15.5 mmHg with a standard deviation of 2.57 mmHg was found.[41]

In their population (40–75years), the IOP was 15.9 (SD~3) mmHg in men and 16.6 (SD~3) mmHg in women. The distribution of IOP was non-Gaussian with a skew to the right (i.e., more individuals with higher IOP than predicted) in those older than 60 years.

What is the current role of IOP in diagnosis and treatment of glaucoma?

Although IOP is not part of the definition of glaucoma, its reduction remains the only proven and approved means of treatment, 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.

The 2005 report by the US Agency for Healthcare Research & Quality, US Preventative Services Task Force [42] stated that there is no good single test at present to conduct population screening. However, in 2008, the World Glaucoma Association (WGA) devoted their fifth Consensus meeting to glaucoma screening [43] for open-angle and angle-closure glaucoma.

Statistically, elevated IOP does not equate with the diagnosis of glaucoma, and conversely, normal IOP does not exclude the diagnosis of glaucoma. From the standpoint of the management of individual patients, the significance of this is that the diagnosis of glaucoma must be based primarily on the examination of the optic discs and retinal nerve fiber layer and the evaluation of visual function.

The two most noteworthy trials in this regard are the Ocular Hypertension Treatment Study (OHTS) [44] which demonstrated that IOP reduction reduces the risk of conversion to glaucoma among ocular hypertensive, and the Early Manifest Glaucoma Trial (EMGT) [45] which demonstrated that IOP reduction lowers the risk of glaucoma progression.

IMPORTANT RANDOMIZED CLINICAL TRIALS

- **COLLABORATIVE NORMAL TENSION GLAUCOMA STUDY**

Before the Collaborative Normal Tension Glaucoma Study (CNTGS), [46] it was not known whether IOP that was in the normal statistical range was at all involved in glaucomatous optic nerve damage and visual field loss. During the study it became apparent that – similar to simple POAG – participating patients had a slower glaucoma progression (no progression in 5 years) when IOP was lowered by 30%. The most treatment benefits were observed in patients of female gender, with family history of glaucoma, without family history of stroke, without personal history of cardiovascular disease, and with mild disk excavation. Risk factors were female gender, migraine headaches, and optic disc hemorrhages.[46]

- **ADVANCED GLAUCOMA INTERVENTION STUDY**

The Advanced Glaucoma Intervention Study (AGIS) [47] has to be seen in its historical context. The goal of AGIS was to compare trabeculectomy with argon laser trabeculoplasty (ALT) in eyes that had failed medical management at a time when it was not established that ALT was less effective than trabeculectomy. Eyes with early average intraocular pressure greater than 17.5 mmHg had more progression than eyes with average intraocular pressure less than 14 mmHg. When IOP was less than 18 mmHg during all visits over 6 years in a separate analysis, mean changes from baseline in visual field defect scores were close to zero. A shortcoming is that AGIS was not originally designed to detect prevention of glaucoma progression but that preventative effects noted were detected in an after analysis.

- **COLLABORATIVE INITIAL GLAUCOMA TREATMENT STUDY**

The Collaborative Initial Glaucoma Treatment Study (CIGTS) [48] compared treatment of newly diagnosed POAG with standard medical treatment (typically initial beta-blocker) versus filtration surgery. Surprisingly, despite the lower IOP, the surgical group had more visual field and more visual acuity loss than the medical group in the first 3 years, but this difference disappeared in the follow-up. There were 3.8-fold more cataract extractions in the surgical group [49] in the initial 5 years after trabeculectomy but not thereafter. Results from CIGTS did not support altering treatment practices of initial medical management of patients with primary open-angle glaucoma.[50]

- **OCULAR HYPERTENSION TREATMENT STUDY AND THE EUROPEAN GLAUCOMA PREVENTION STUDY**

The purpose of the Ocular Hypertension Treatment Study (OHTS) [44] was to determine whether the pharmacological reduction of elevated IOP can prevent glaucoma and to define risk factors for glaucoma development. Topical ocular hypotensive medication was effective in delaying or preventing onset of POAG in individuals with elevated IOP by about 50%. Results to date have shown an approximate 50% reduction in conversion from OHT to POAG, with a 20% reduction in intraocular pressure.[51] OHTS demonstrated that medical treatment of people with intraocular pressure of 24 mmHg reduces the risk of the development of primary open-angle glaucoma (POAG) by 60%.[52]

Factors that predicted the development of POAG included older age, race (African-American), sex (male), larger vertical cup–disc ratio, larger horizontal cup–disc ratio, higher intraocular pressure, greater Humphrey visual field (VF) pattern standard

deviation, heart disease, and thin central corneal thickness.[53] At 60 months, the overall probability of developing POAG was 4.4% in the medication group and 9.5% in the observation group [51]. Of African-American participants, 8.4% developed POAG in the medication group when compared with 16.1% in the observation group.[54] The same predictors for the development of POAG were identified independently in both the OHTS observation group and the European Glaucoma Prevention Study (EGPS) [55] i.e. placebo group-baseline age, intraocular pressure, central corneal thickness, vertical cup-to-disc ratio, and Humphrey VF pattern standard deviation.

- **EARLY MANIFEST GLAUCOMA TRIAL**

The Early Manifest Glaucoma Trial (EMGT) [45] compared how glaucoma progression was affected by immediate combined medical (betaxolol) and laser therapy for newly diagnosed OAG with normal or moderately elevated IOP versus late or no treatment. Treatment caused an average reduction of IOP of about 5 mmHg (25%), which reduced glaucoma progression to 45% when compared with 62% in the control group and occurred later. These benefits were preserved after stratifying for age, ethnicity, and POAG stage and type. The percent of patient follow-up visits with disc hemorrhages was also related to progression (hazard ratio = 1.02 per percent higher).[56] Progression risk decreased by about 10% with each millimeter of mercury of IOP reduction from baseline to the first follow-up visit (HR = 0.90 per millimeter of mercury decrease).[56] Elevated IOP was a strong factor for glaucoma progression, but intraocular pressure fluctuation was not.[57] Higher IOP, exfoliation, bilateral disease, and older age were progression factors previously known. New baseline predictors were lower ocular systolic perfusion pressure, cardiovascular disease history (HR 2.75) in patients with higher baseline IOP,

and lower systolic blood pressure (BP) in patients with lower baseline IOP. Thinner central corneal thickness (CCT) (HR 1.25 per 40 micrometer lower) was a new significant factor, a result observed in patients with higher baseline IOP.

CONCLUSION FROM RANDOMIZED TRIALS

Lower intraocular pressure does delay or prevent progression of POAG as evidenced by delay or prevention of development of optic nerve damage from ocular hypertension, visual field defects from existing optic nerve changes, and reduced progression of existing visual field defects. However, another conclusion is that despite good IOP control in many actively treated patients, POAG can still progress and can result in blindness in an unacceptably large number of patients.

Necessity of Continuous IOP Monitoring

Circadian variations in IOP were first recorded by Sidler- Huguenin in 1898. These variations typically range from 3 to 6 mmHg in normal eyes and can exceed 30 mmHg in glaucomatous eyes. Understanding these variations in IOP are important for two reasons. First, they raise the possibility that a single reading taken in the physician's office is not an accurate representation of the patient's normal pressure level. Second, the magnitude of the variation itself is believed to be important for the diagnosis, treatment, and prognosis of glaucoma.

IOP fluctuation during follow-up is associated with a higher probability of VF progression. Large diurnal fluctuations in intraocular pressure are an independent risk factor in patients with glaucoma. Hence, the care of patients with advancing glaucoma

should include measurement of long term IOP fluctuation. Even short-term IOP fluctuation is not accurately measured by three IOP measurements during typical office hours (8 am to 5 pm). A continuous monitoring technique is necessary to accurately document a patient's baseline IOP and the extent of inter visit fluctuations. The continuous monitoring technique must be able to sample IOP quickly enough to capture its dynamic behavior.

GLAUCOMA RISK FACTORS: THE CORNEA

- **CENTRAL CORNEAL THICKNESS**

It has long been accepted that elevated intraocular pressure (IOP) is a key parameter in the diagnosis of glaucoma. IOP is used in the assessment of disease progression and response to treatment. In fact, it is the only modifiable risk factor for glaucoma. Therefore, its accurate measurement is critical. For over half a century, Goldmann applanation tonometry (GAT) has been the most widely used method of measuring IOP. In their article, Goldmann and Schmidt determined that, in accordance with the Imbert-Fick Law, IOP is equal to the force necessary to flatten a spherical surface, divided by the surface area of that flattened surface (the cornea). GAT utilizes a cone that applanates a circular area of the corneal surface with a diameter of 3.06 mm. With this circular applanation area, the force necessary to overcome the resistance of the cornea to flattening is equal to the surface tension of the tear film. This allows the force applied to equal the IOP. Goldmann and Schmidt believed that central corneal thickness (CCT) was very similar among individuals in the normal population. However, they acknowledged that when large variations in CCT did occur, the accuracy of the GAT readings could be

affected. Corneas that were thicker than normal would require greater force to flatten and thinner corneas would require less. This meant that thicker corneas yielded an overestimation of IOP, whereas thinner corneas gave an underestimation. It has since been demonstrated definitely that significant variations do occur in a normal population.

Across populations, patients with normal eyes have a CCT of approximately 540 µm. Patients with ocular hypertension have been found to have CCT values that are increased about 50 µm compared to glaucoma patients or controls [12, 58]. This means that many individuals have been falsely labeled as having ocular hypertension, when in fact they have normal IOP once CCT is taken into account. Several studies have shown that some patients with normal tension glaucoma have thinner corneas than patients with normal eyes.[59] Therefore, if CCT is taken into account, these patients may have elevated IOP with "high pressure" glaucoma. One longitudinal study showed up to a 23 µm decrease over an 8-year period in glaucoma patients.[60] This underscores the importance of taking CCT measurements in all patients who have ocular hypertension or glaucoma.

LaRosa et al [61] reported thinner CCT values among normal black male veterans (mean thickness 530 µm) compared to their white counterparts (545 µm). In another study of patients at a large refractive surgery center, black patients also had thinner CCT values than white patients seeking surgery.[62]

GLAUCOMA RISK FACTORS: FAMILY HISTORY – THE GENETICS OF GLAUCOMA

Kass and Becker [63] were among the first to observe a relationship between family history and glaucoma, and focused their research on the cup-to-disc ratio, elevated pressure, and the glucocorticoid response. Their early investigations into hereditary aspects of glaucoma noted that there was a relationship between family history and the presence of glaucoma, but that it seemed to defy simple genetic analysis.

Becker, and later Armaly, [64] found that glucocorticoid treatment re-elevated IOP more often in glaucoma patients than in other individuals. Testing of family members showed that this response was usually inherited as an autosomal recessive trait.

Subsequently, Polansky[65] theorized that the mutations of these genes in the trabecular meshwork were associated with a corticoid steroid response. He called the specific induced proteins "Trabecular meshwork Inducible Glucocortoid Response protein" (TIGR), the same protein later being termed "Myocilin". Since then, a number of additional genetic loci have been identified that are associated with open angle glaucoma. They all appear to be autosomal dominant.

Pigmentary, pseudoexofoliation, angle closure, and mixed-mechanism glaucoma have been associated with myocilin mutations.[66, 67]. The report of a family with mixed-mechanism glaucoma having a myocilin mutation is intriguing since it suggests that two gene interactions, one for open angle glaucoma and one for narrow angle glaucoma, can both lead to glaucoma. One mutation (Gly399Val MYOC) led to adult POAG with a mean age of onset of 51.

Optineurin is located on GLC1E at chromosome 10. Optineurin may have a role in TNF alpha signaling pathways and has also been implicated in modulation of cellular cytokeleoin of TM cells.

Estimates on the frequency of optineurin have varied substantially, with the original report identifying 16 open angle glaucoma patients as having the disease.[67] GLC1E has numerous mutations. It has been suggested that those with the mutation appear to have more advanced cupping and require more frequent surgical intervention.[68] A synonymous variant Thr34Thr was associated with glaucoma in Japan.[69]

GLAUCOMA RISK FACTORS: ETHNICITY AND GLAUCOMA

Asians comprise many ethnicities, and findings in one group of Asians do not necessarily generalize to another group of Asians. For example, a nationwide Japanese glaucoma survey reported a prevalence of POAG that is intermediate between that of black-Americans and white-Americans, and a POAG to primary angle-closure glaucoma (PACG) proportion that is similar to that in western populations.[70] In contrast, PACG is the predominant type of glaucoma in some Asian populations such as the Chinese.[71]

Japanese have been found to have a much higher prevalence of POAG, with intraocular pressures 21 mmHg or lower compared to other populations. In addition, the population sample in this study had a higher prevalence of POAG with pressures 21 mmHg or lower (3.6%) compared to that of POAG with an intraocular pressure greater than 21 mmHg (0.3%). This higher prevalence of POAG with lower intraocular pressures might be accounted for by the known thinner central corneal thickness in the Japanese population.

Several studies have reported that Asian patients have intermediate CCT values when compared to white and black- Americans.[70] Given this intermediate value of CCT, one can conclude that corneal thickness would not account for the higher prevalence of NTG in Japanese patients. It is also not known with certainty if a thinner CCT value in Asians with ocular hypertension is considered a risk factor as the Ocular Hypertension Study had a very small Asian patient population.

REFRACTIVE ERROR

Population-based surveys in white, [72, 73] black and Japanese populations have provided some evidence to the commonly held belief that myopia is associated with an increased risk for POAG, although other studies did not lend support to this notion. The Early Manifest Glaucoma Trial (EMGT) did not find myopic patients with ocular hypertension to have a higher incidence of glaucoma nor did myopic patients have a greater risk of progression when suffering from glaucoma.[56]

VASCULAR RISK FACTORS

SYSTEMIC HYPERTENSION AND HYPOTENSION

Chronic hypertension may cause microvascular damage, interference with auto regulatory mechanisms, and atherosclerotic changes, while hypotension may reduce local perfusion and lead to ischemic injury. Both of these mechanisms lead glaucomatous progression in the face of IOP elevation or poor vascular autoregulation of blood flow.

Although many studies show an association between blood pressure and IOP, the information available on high blood pressure and OAG continues to be complex and inconclusive.[74,75] In the Egna–Neumarkt study,[76] a positive correlation was found between systemic blood pressure and diagnosis of OAG as well as to IOP, unrelated to age. The Baltimore Eye Study demonstrated that patients with systolic blood pressure higher than 130 mmHg have greater risk for OAG.[77]

Contrasting this, however, the Barbados Incidence Study of Eye Diseases (BISED) [78] researched a large, predominantly African-origin population finding that persons with hypertension had a statistically significant decreased risk of OAG. One explanation is that higher blood pressure in the early stages can protect the optic nerve by maintaining the ocular perfusion pressure, while at later stages, chronic systemic hypertension may lead to dysfunctional regulation.

DIABETES

Diabetes, a disease with many vascular complications, has been reported to be related to glaucoma. The prevalence of OAG was 40% higher in participants of the Los Angeles Latino Eye Study with type 2 diabetes mellitus than in those without .(Age/gender/intraocular pressure-adjusted odds ratio, 1.4; 95% confidence interval, 1.03–1.8; P = 0.03).[79]

Population-based studies, such as the Baltimore eye survey, [80] the Barbados eye study, [81] and the Rotterdam study[82] have failed to support an association between diabetes and glaucoma.

PREVALENCE OF GLAUCOMA IN INDIA

Glaucoma is estimated to affect 60.5 million persons worldwide by the year 2010. The estimated prevalence of glaucoma for India is 11.9 million. These estimates have been derived from population based studies conducted worldwide. The availability of population based data from India is relatively recent as compared to Western countries. There are five populations based studies, three from the state of Tamil Nadu, one from Andhra Pradesh and one from West Bengal. These studies have been carried out from 1993 to 2003. All these studies have used differing methodology and diagnostic criteria for glaucoma.

TABLE: 2 INDIAN POPULATION BASED STUDIES.

STUDY	STUDY PERIOD	SETTING	AGE GROUP	Number examined
VES	1994	Urban	30-60	972
APEDS	1996-2000	Rural , Urban	All ages	10,273
ACES	1995-97	Rural	>40	5150
CGS	2001-03	Rural , Urban	>40	7774
WBGS	1998-99	Rural	>50	1324

****VES**: VELLORE EYE STUDY, **APEDS**: ANDHRA PRADESH EYE DIDESE STUDY, **ACES**: ARAVIND COMPREHENSIVE EYE SURVEY, **CGS**: CHENNAI GLAUCOMA STUDY, **WBGS**: WEST BENGAL GLAUCOMA STUDY

PRIMARY OPEN ANGLE GLAUCOMA:

PREVELENCE IN INDIA

There are again wide variations in the prevalence of POAG between the five studies (Table 2). The Vellore Eye Study (VES) reported the lowest rates of 0.41%. , there are a number of reasons for this, the prime cause being that the study was limited to the age group of 30-60 years. The prevalence of POAG increased with age in all the studies discussed here. On comparing the prevalence of POAG in those is the 30-60 age group from Andhra Pradesh Eye Disease Survey (APEDS) (2.56%) [83] the VES reported prevalence is not dissimilar (0.41%, 95% CI: 0.08, 0.81%). The other possible reason is the low rate of visual field performance in the VES. Since a visual field defect was essential for diagnosis, those who did not perform visual fields would not be classified as glaucoma, the high (51.5 %) non response rates could affect case detection.

With the exception of the VES the reported prevalence rates for POAG appeared to be higher in urban population (CGS (Urban) [84] and APEDS[83]). While the urban populations were older than the rural population this is not the reason for the increased prevalence as the age standardized rates (to the Indian Population 2001) show the same trend. Urban India is known to have higher reported rates of diabetes and cardio-vascular diseases. We speculate that lifestyle changes and their related cardio-vascular influences may account in part of this increase in prevalence.

The proportion of persons with POAG who presented with a "normal" IOP (defined as two standard deviations above the population mean) was significant in all the prevalence studies. Sixty five percent of those with POAG in APEDS,[87] 45% in Aravind Comprehensive Eye Study (ACES) [85] 67% in Chennai Glaucoma Study (CGS)(rural)[96] and 82% in CGS (urban)[84] had a normal presenting intraocular pressure. This is again one of the contributory factors for the large proportion of undiagnosed disease – a single normal IOP does not rule out disease. Optic disc evaluation is mandatory in order to identify those with glaucoma.

Increasing age was the consistent risk factor for all studies. Males were at greater risk of POAG in the ACES.[85] No gender difference was reported by any of the other studies. Myopia was also a risk factor for POAG in the ACES. No association with refractive error was reported by any of the other studies.

PRIMARY ANGLE CLOSURE GLAUCOMA PREVELENCE IN INDIA

The VES was the first to report that PACG comprised a significant proportion of those with glaucoma. The VES definition of PACG encompassed those now classified as PAC in addition to those with PACG. Five persons (0.51%) were reported to have angle closure disease with disc and field damage. These would correspond to a diagnosis of PACG; this prevalence is similar to other reported rates of PACG by studies in South India.[83,84,85,89,90] Subsequent reports from the APEDS[83] did report a lower prevalence of angle closure disease. The difference was most marked in the lower prevalence of PACS. The prevalence of angle closure disease in the CGS [84, 86] is again similar to that reported by the VES. The urban area of the CGS had higher rates of PAC, PACS and PACG than the rural cohort.[84, 85] The VES and the CGS used similar gonioscopic criteria to diagnose a narrow angle (needing 180° of the angle to be closed as defined) unlike other studies that needed three fourths of the angle to be narrow before grading as occludable. The CGS did report that of those with PACG who were previously diagnosed to have glaucoma 40% were misdiagnosed as POAG.

RISK FACTORS:

Increasing age was a common risk factor for PACG in all studies. Female gender (CGS) was a risk factor for PACG and PAC [84, 86] more women in the APEDS had PACG but this difference was not statistically significant.[83] Hyperopia was also a risk factor in the Chennai glaucoma a study (Urban), [86] a trend of increased risk with hyperopia was noted both in APEDS and CGS (Rural).[83, 87] Among systemic conditions diabetes mellitus was reported to be a risk factor. Biometric data at the baseline visit was available only from

the Chennai Glaucoma Study. Eyes with angle closure disease had been reported to have shorter axial length and a shallower anterior chamber and thicker lenses than normal by the CGS.[87]

The population based studies that report glaucoma prevalence provide important information.[83, 84, 85, 86]. The Indian population has substantial risk for POAG with the additional risk of PACG and large burden of angle closure disease. This assumes greater importance as the risk of all forms of glaucoma increases substantially with age and moreover the vast majority of glaucoma in India is undiagnosed; there is an urgent need to adopt comprehensive eye examinations, which include IOP measurement, gonioscopy and optic disc evaluation, as a routine.

MATERIALS AND METHODS

MATERIALS AND METHODS

This is an observational study where 3500 patients visiting the ophthalmology outpatient department was screened and cases fulfilling the inclusion criteria was selected for this study. The study was conducted at ophthalmology outpatient department at R.L.J. HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR attached to SRI DEVARAJ URS MEDICAL COLLEGE between December 2012 to May 2014.

INCLUSION CRITERA: All Patients visiting outpatient clinic above 30 years of age, who are diagnosed as,

1. Primary open angle glaucoma.
2. Primary angle closure glaucoma.
3. Primary open angle glaucoma suspect.
4. Primary angle closure suspect.

EXCLUSION CRITERIA:

Patients with**:**

1. Secondary Glaucoma.
2. Congenital glaucoma / Developmental Glaucoma.

METHOD OF COLLECTION OF DATA

- A detailed history was taken including optical history, family history followed by detailed clinical examinations. Visual acuity was recorded using a Snellen distance vision chart and near vision chart.
- Retinoscopy and recording the best corrected visual acuity was done.
- Examinations under slit lamp was done routinely and the anterior chamber angle was graded with Van Herrick's grading.
- Tonometry was performed using Applanation tonometer; three readings was taken and the mean (the nearest whole number) was recorded as the IOP.
- Examination of fundus with Direct Ophthalmoscope and with +90D lens was done.
- Gonioscopy was carried out using a Goldmann two mirror gonio lens in the Established glaucoma and suspected glaucoma cases.
- The angle of the anterior chamber was graded according to Shaffer's angle grading system.
- Visual field examination using Humphrey field analyzer was carried out and documented in all the selected case.

SELECTION CRITERIA

Patients were diagnosed as Primary open angle glaucoma **(POAG)** on these following criteria

- Optic disc abnormalities
    - Diffuse thinning, focal narrowing, or notching of the optic disc rim, especially at the inferior or superior poles
    - Thinning of the neuroretinal rim with an associated increase in cupping of the optic disc
    - Optic disc neural rim asymmetry of the two eyes
- Open anterior chamber angles
- Visual field abnormality
    - Visual field damage consistent with retinal nerve fiber layer damage (e.g., nasal step, arcuate field defect, or paracentral depression)
- Adult onset

Any secondary glaucoma was excluded from the study as per the exclusion criteria,

Patients with clinical findings and/or a constellation of risk factors that indicate an increased likelihood of developing POAG were diagnosed as primary open angle glaucoma suspect **(POAG Suspect)**

The clinical findings that define a glaucoma suspect patient are characterized by one of the following in at least one eye in an individual with open anterior chamber angles by gonioscopy:

- Appearance of the optic disc or retinal nerve fiber layer that is suspicious for glaucomatous damage
    - Enlarged cup-disc ratio
    - Asymmetric cup-disc ratio
    - Notching or narrowing of the neuroretinal rim
- A visual field suspicious for glaucomatous damage in the absence of clinical signs of other optic neuropathies
- Consistently elevated intraocular pressure (IOP).

Any secondary glaucoma was excluded from the study as per the exclusion criteria.

Patients with angle closure and those at risk was categorized as follows:

Patients with Irido trabecular contact (ITC), as observed on compressive gonioscopy with No PAS, and normal IOP was diagnosed as Primary angle-closure suspect **(PACS).**

Patients with least 180 degrees of ITC and an elevated IOP and glaucomatous optic disc abnormalities, optic neuropathy with visual field defect is the eye was diagnosed as Primary angle closure glaucoma **(PACG).**

STATISTICAL ANALYSIS:

Data was compiled in Microsoft excel after coding and was analyzed using SPSS 20 version software. Qualitative data was represented by frequencies and proportions and quantitative data by mean and standard deviation. Chi-square test was used as test of significance for qualitative data. Independent t test to measure the mean difference between two groups and ANOVA test to measure the mean difference between more than two groups was used as test of significance for quantitative data. P value < 0.05 was considered as statistically significant.

RESULTS

RESULTS

In the study 3500 subjects were screened during the study period of which total of 184 cases (5.26%) were having suspected and established primary glaucoma,

72 cases (2.09%) of them were in the primary open angle group comprising of both POAG (0.94%) cases and POAG suspects. (1.11%)

27 cases (0.77%) were diagnosed having PACG, whereas 85 cases (2.42%) were diagnosed as PACS.

TABLE 3: DIAGNOSIS AT PRESENTATION.

		Frequency
Diagnosis in Glaucoma	**Established POAG**	33
	POAG Suspect	39
	Established PACG	27
	PAGS	85
	Total	

In the study among 72 primary open angle cases 33 (45.8%) were established POAG and 39 (54.2%) were Suspect of POAG.

Similarly among 112 primary angle closure cases 27 (24.10%) were established PACG and 85 (75.9%) were PACS.

PROPORTION OF PAOG AND PAOG SUSPECTS

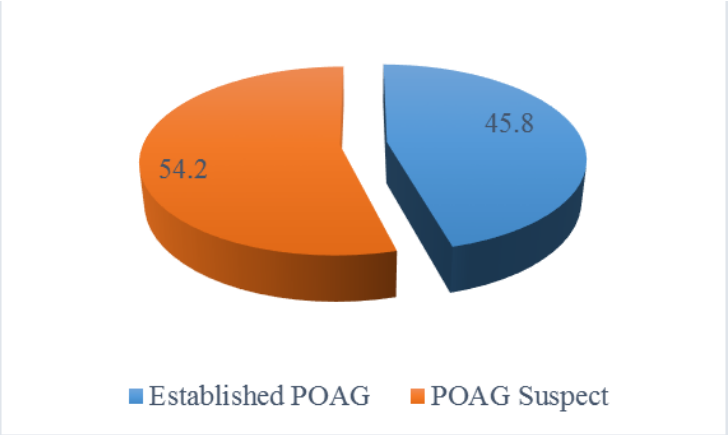

FIGURE 6: PIE DIAGRAM SHOWING PROPORTION OF POAG AND PAOG SUSPECTS.

PROPORTION OF PACG AND PACS

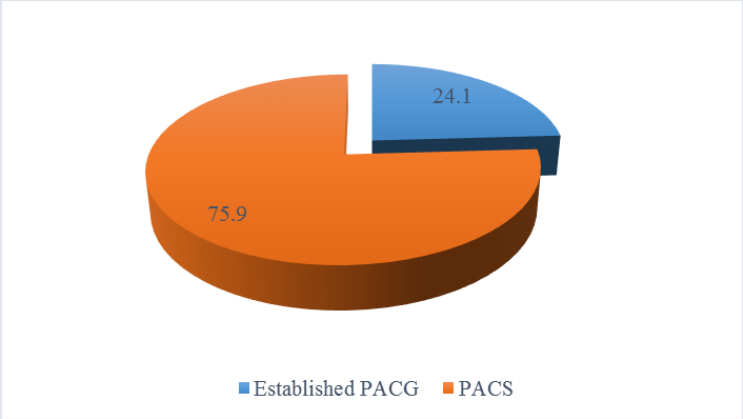

FIGURE 7: PIE DIAGRAM SHOWING PROPORTION OF PACG AND PACS.

TABLE 4: AGE DISTRIBUTION AND MEAN AGE OF PRESENTATION

	Group	N	Mean	Std. Deviation	F value	p value
Age	**POAG**	33	64.50	13.06	7.745	0.0001
	POAG suspect	39	63.00	13.60		
	PACG	27	68.60	10.94		
	PACS	85	55.88	14.65		

In the study the mean age of presentation of POAG subjects was 64.50 ± 13.060, POAG Suspect was 63.00 ± 13.6, among PACG mean age was 68.60 ± 10.94 and among PACG suspect was 55.88 ± 14.65. There was no significant mean difference in age between POAG, POAG suspect, PACG and PACS

TABLE 5: AGE DISTRIBUTION OF PATIENTS WITH RESPECT TO DIAGNOSIS

		Diagnosis				Total	χ 2 , df, p value
		POAG	**POAG Suspect**	**PACG**	**PACS**		
Age	**<40 years**	1(3.03%)	1 (2.56%)	0	15 (17.64%)	19(10.32%)	26.43 15, 0.0337
	40 to 49 years	3 (9.09%)	4(10.26%)	1(3.70%)	14(16.47%)	22(11.96%)	
	50 to 59 years	12(36.36%)	13(33.33%)	5(18.51%)	14(16.47%)	41(22.28%)	
	60 to 69 years	9 (27.27%)	11(28.20%)	11(40.74%)	22(25.90%)	48(26.08%)	
	70 to 79 years	5(15.15%)	6 (15.38%)	9 (33.33%)	16(18.82%)	41(22.28%)	
	>80 years	3(9.09%)	4 (10.26%)	1 (3.7%)	4 (4.7%)	13 (7.06%)	
Total		33	39	27	85	184	

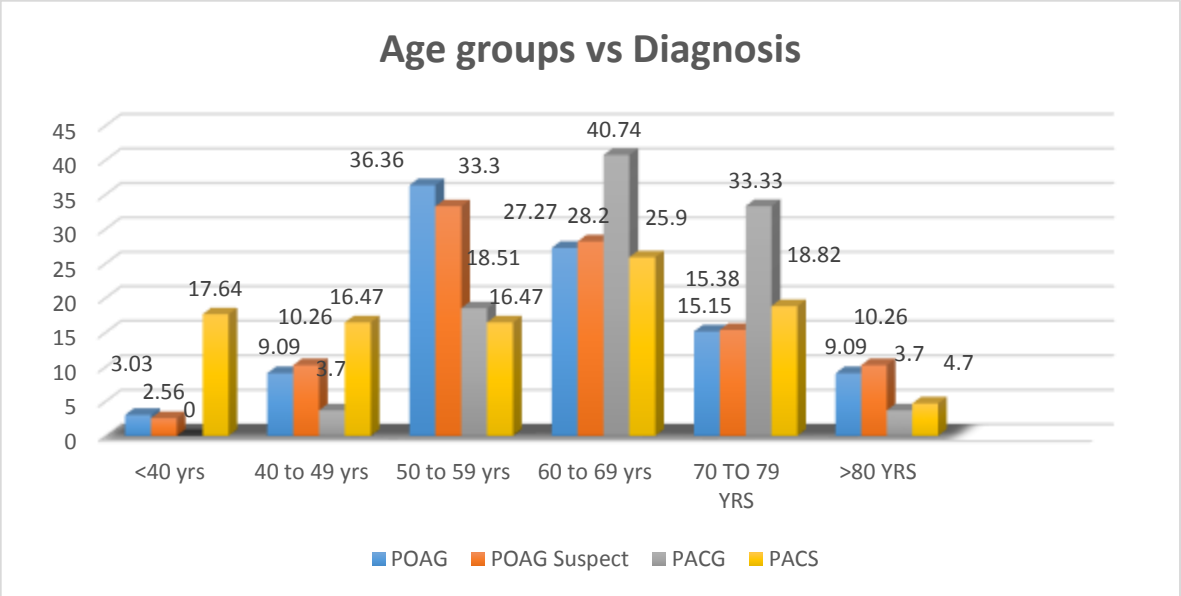

FIGURE 8: BAR DIAGRAM SHOWING AGE GROUPS WITH RESPECT TO DIAGNOSIS

3500 people were screened for glaucoma during the study period and it was observed that proportion of POAG was common after 50 years of age, in the study 29 subjects (87.87%) out of 33 was diagnosed with POAG was more than 50 years of age,

Whereas PACG was more common after 60 years, in the study 21 subjects (77.78%) Subjects diagnosed with PACG was >60 years of age.

This observation was statistically significant.

TABLE 6: SEX DISTRIBUTION OF THE GLAUCOMA PATIENTS WITH RESPECT TO DIAGNOSIS

						Total	χ 2 , df, p value
		PAOG	PAOG Suspect	PACG	PACS		
Sex	Female	11	10	19	65	42	38.32, 3, 0.0001**
	Male	22	29	8	20	63	
Total		33	39	27	85	184	

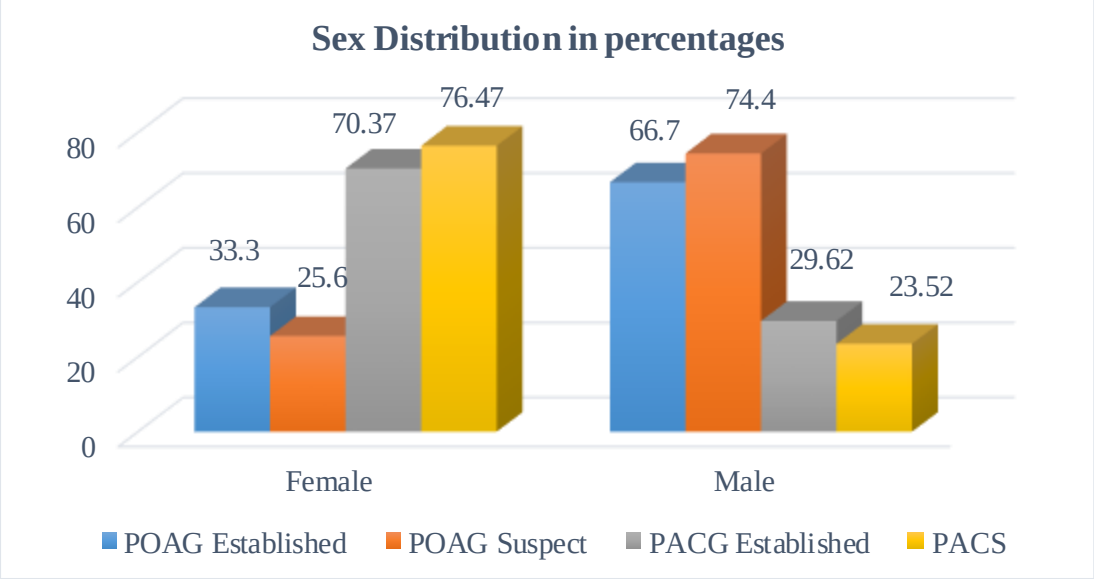

FIGURE 9: BAR DIAGRAM SHOWING SEX DISTRIBUTION OF SUBJECTS AMONG GLAUCOMA PATIENTS

It was observed that POAG was common among Males with male to female ratio was 2:1, whereas in POAG suspects the Male female ratio was 3:1

PACG was common in Females with male to female ratio of 1:2.4,

Whereas male female ratio in PACS was 1:3.2.

This observation was statistically significant.

TABLE 7: VISUAL ACUITY IN PRIMARY OPEN ANGLE GLAUCOMA CASES

VISUAL ACUITY IN ESTABLISHED PRIMARY OPEN ANGLE GLAUCOMA CASES

VISUAL ACUITY	RIGHT EYE	LEFT EYE	TOTAL(RE+LE)
6/6 - 6/12	7 (21.21%)	8 (24.24%)	15
6/18 - 6/24	15 (45.45%)	14 (42.42%)	29
6/36 – 6/60	10 (30.30%)	9 (27.27%)	19
<6/60	2 (6.06%)	2 (6.06%)	4

Most of the POAG cases presented with visual acuity ranging from 6/18 – 6/60 at presentation

One patient presented with PL –VE in one eye.

TABLE 8: VISUAL ACUITY IN SUSPECTED OPEN ANGLE GLAUCOMA CASES

VISUAL ACUITY IN SUSPECTED OPEN ANGLE PATIENTS

VISUAL ACUITY	RIGHT EYE	LEFT EYE	TOTAL
6/6 – 6/12	33 (84.61%)	31 (79.48%)	64
6/18 – 6/24	6 (15%)	8 (20.51%)	12

Most of the primary open angle glaucoma suspect cases presented with visual acuity in the range of 6/6 -6/12.

TABLE 9: VISUAL ACUITY IN PRIMARY ANGLE CLOSURE GLAUCOMA PATIENTS

VISUAL ACUITY	RIGHT EYE	LEFT EYE	TOTAL
6/6 – 6/12	2 (7.41%)	3 (11.11%)	5
6/18 – 6/24	7 (25.9%)	8 (29.6%)	21
6/36 – 6/60	12 (44.44%)	12 (44.44%)	18
<6/60	6 (22.22%)	4 (14.81%)	10

Most Cases with primary angle closure glaucoma presented with visual acuity of 6/36 or less on presentation. One case presented with PL –ve one eye.

TABLE 10: VISUAL ACUITY IN PRIMARY ANGLE CLOSURE SUSPECT

VISUAL ACUITY	RIGHT EYE	LEFT EYE	TOTAL
6/6 – 6/12	74 (87.05%)	76 (89.41%)	148
6/18 – 6/24	10 (11.76%)	9 (10.59%)	19

Majority (90 ~95%) of visual acuity of PACS patients ranged from 6/6 -6/12.

TABLE 11: INTRA OCULAR PRESSURE WITH RESPECT TO DIAGNOSIS

Intra ocular pressure		N	Mean	Std. Deviation	F	p value
Right Eye	POAG	33	24.91	3.778	120.97	0.0001**
	POAG Suspect	39	21.74	3.485		
	PACG	27	32.67	4.497		
	PACS	85	18.47	2.950		
	Total	184	24.44	3.678		
Left eye	POAG	33	24.45	4.236	116.93	0.0001**
	POAG Suspect	39	22.64	3.030		
	PACG	27	32.48	4.610		
	PACS	85	18.63	2.721		
	Total	184	24.55	3.659		

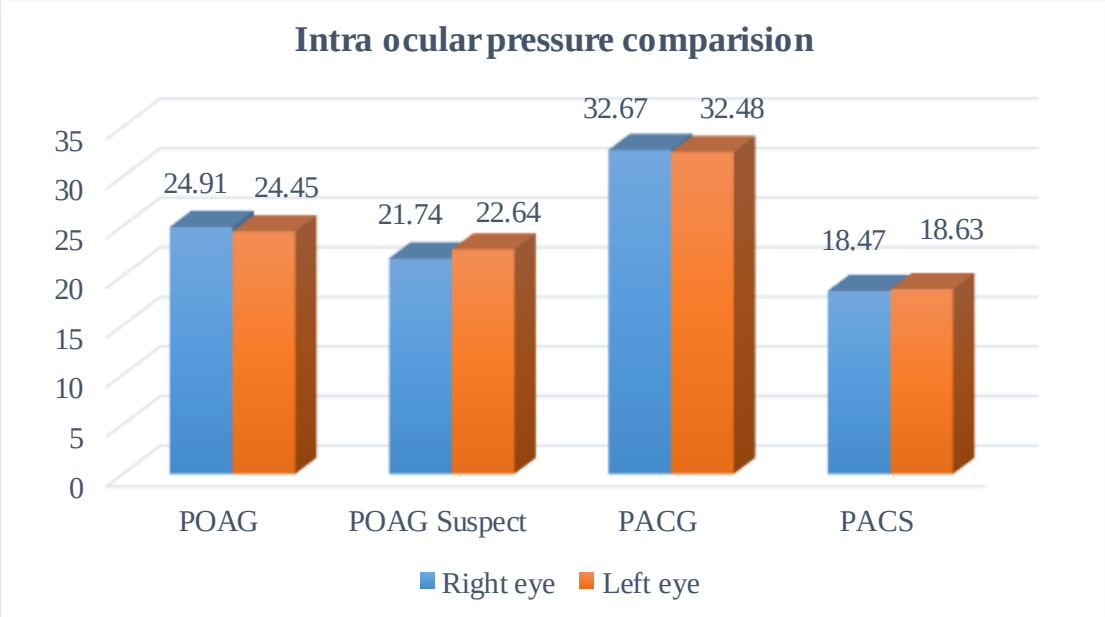

FIGURE 10: BAR DIAGRAM SHOWING INTRA OCULAR PRESSURE WITH RESPECT TO DIAGNOSIS

It was observed that Mean Intra ocular pressure was high in PACG in both eyes (32.67 mmHg in Right eye and 32.48 mmHg in Left eye) followed by POAG, (24.91 in Right eye and 24.45 in Left eye).POAG Suspects had 21.74mmHg in Right eye and 22.74mmHg in Left eye).The mean IOP in PACS subjects were 18.47mmHg in right eye and 18.63mmHg in Left eye. This observation was statistically significant within the groups and between groups in both eyes.

TABLE 12: VAN HERRICKS GRADING WITH RESPECT TO DIAGNOSIS

VAN HERRICKS GRADING		**N**	**Mean**	**Std. Deviation**	**F**	**Sig.**
Right Eye	POAG	33	3.53	0.507	240	0.0001**
	POAG Suspect	39	3.42	0.501		
	PACG	27	0.88	0.667		
	PACS	85	1.52	0.503		
	Total	184	2.33	0.544		
Left eye	POAG	33	3.46	0.501	235	0.0001**
	POAG Suspect	39	3.44	0.502		
	PACG	27	0.76	0.723		
	PACS	85	1.54	0.541		
	Total	184	2.3	0.567		

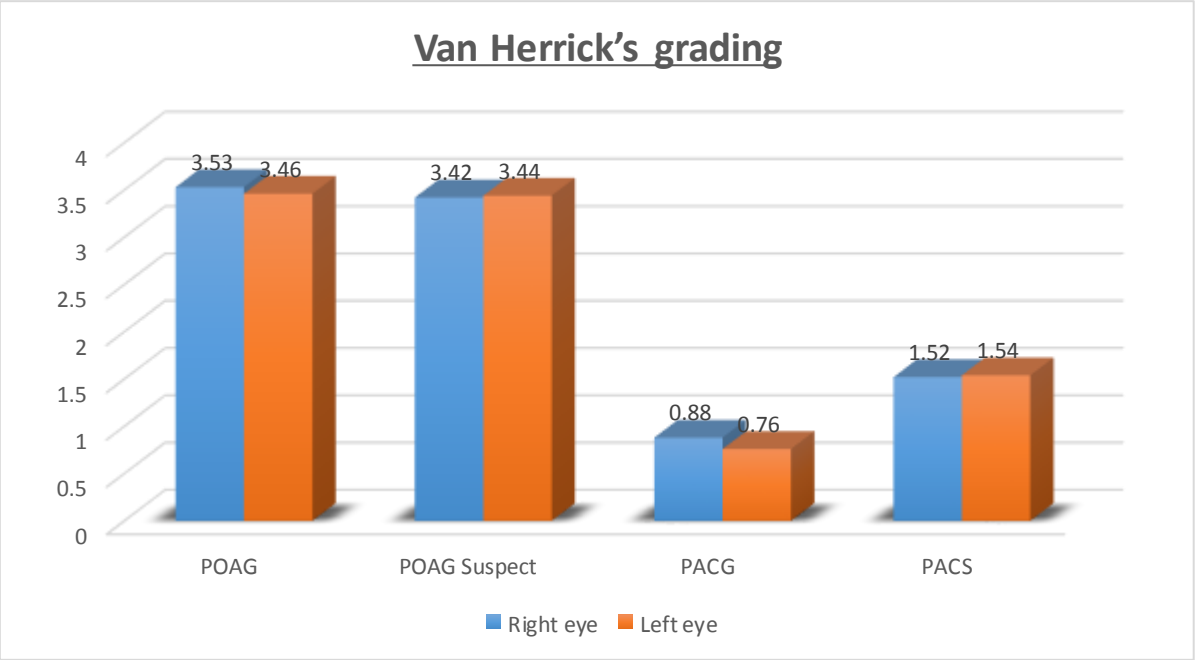

FIGURE 11: BAR DIAGRAM SHOWING DISTRIBUTION OF VAN HERRICK'S GRADING

It is been observed than the mean grading in POAG was 3.53+/- 0.507 (RE) and 3.46 +/- 0.501, POAG suspects had similar mean grading of 3.42 +/- 0.501 (RE) and 3.44+/- 0.502 (LE).

The mean grading in PACG was 0.88+/- 0.667 (RE) and 0.76 +/- 0.723 (LE), in PACS the mean grading was 1.52 +/- 0.503 (RE) and 1.54 +/- 0.541 (LE).

This observation was statistically significant.

TABLE 13: SHAFFER'S GRADE COMPARISON WITH RESPECT TO DIAGNOSIS

Shaffer's Grading		N	Mean	Std. Deviation	F	p value
Right eye	**POAG**	33	3.58	0.502	125.446	0.0001**
	POAG Suspect	39	3.49	0.506		
	PACG	27	1.11	0.863		
	PACS	85	1.99	0.645		
	Total	184	2.54	0.629		
Left eye	**POAG**	33	3.55	0.506	146.305	0.0001**
	POAG Suspect	39	3.54	0.505		
	PACG	27	1.04	0.649		
	PACS	85	1.96	0.663		
	Total	184	2.52	0.580		

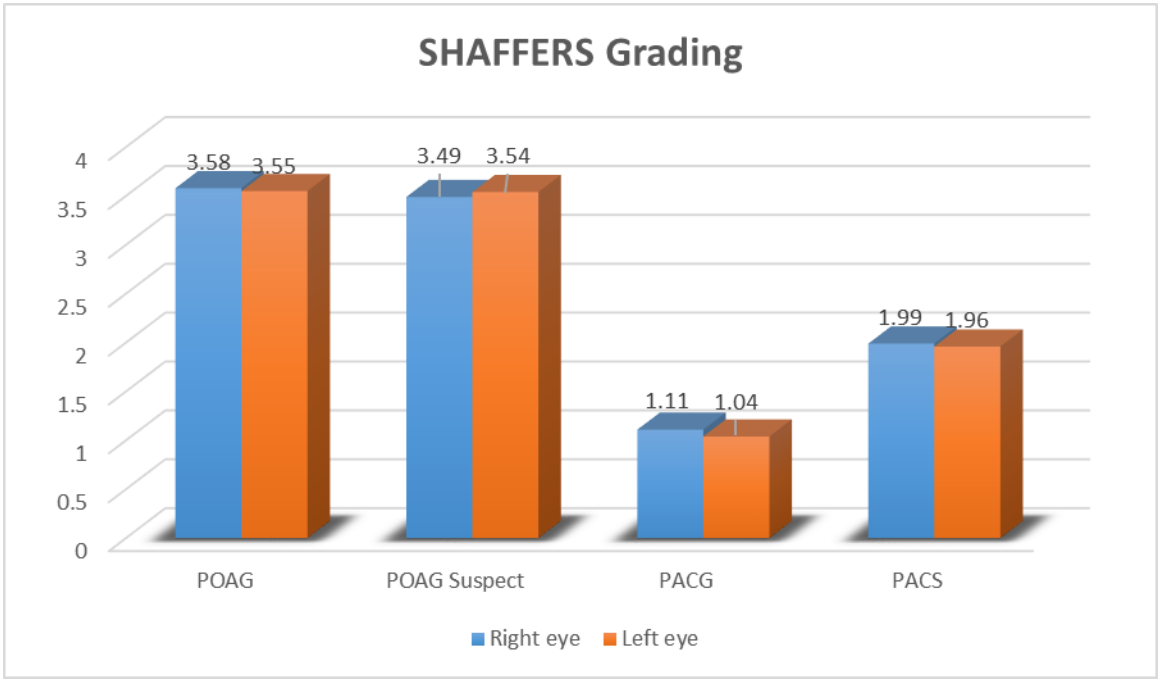

FIGURE 12: BAR DIAGRAM SHOWING SHAFFER'S GRADE IN BOTH EYES WITH RESPECT TO DIAGNOSIS

It was observed that Shaffer's grading POAG was 3.58 +/- 0.502 (RE) and 3.55+/-0.506 (LE) and POAG suspect had similar findings, 3.49 +/- 0.506 (RE) and 3.54+/- 0.506.

For PACG the mean was 1.11+/-0 .863 (RE) and 1.04+/- 0.649 and in PACS cases it was 1.99 +/- 0.645 (RE) and 1.96 +/- 0.663 (LE) .This observation in mean of Shaffer's grade was statistically significant between and within the groups.

TABLE 14: C: D RATIO COMPARISON WITH RESPECT TO DIAGNOSIS

Cup Disc Ratio		N	Mean	Std. Deviation	F	Sig.
Right Eye	POAG	33	0.63	0.15	24.535	0.0001**
	POAG Suspect	39	0.49	0.16		
	PACG	27	0.66	0.13		
	PACS	85	0.46	0.11		
	Total	184	0.56	0.13		
Left eye	POAG	33	0.65	0.11	23.864	0.0001**
	POAG Suspect	39	0.51	0.15		
	PACG	27	0.68	0.12		
	PACS	85	0.49	0.12		
	Total	184	0.58	0.12		

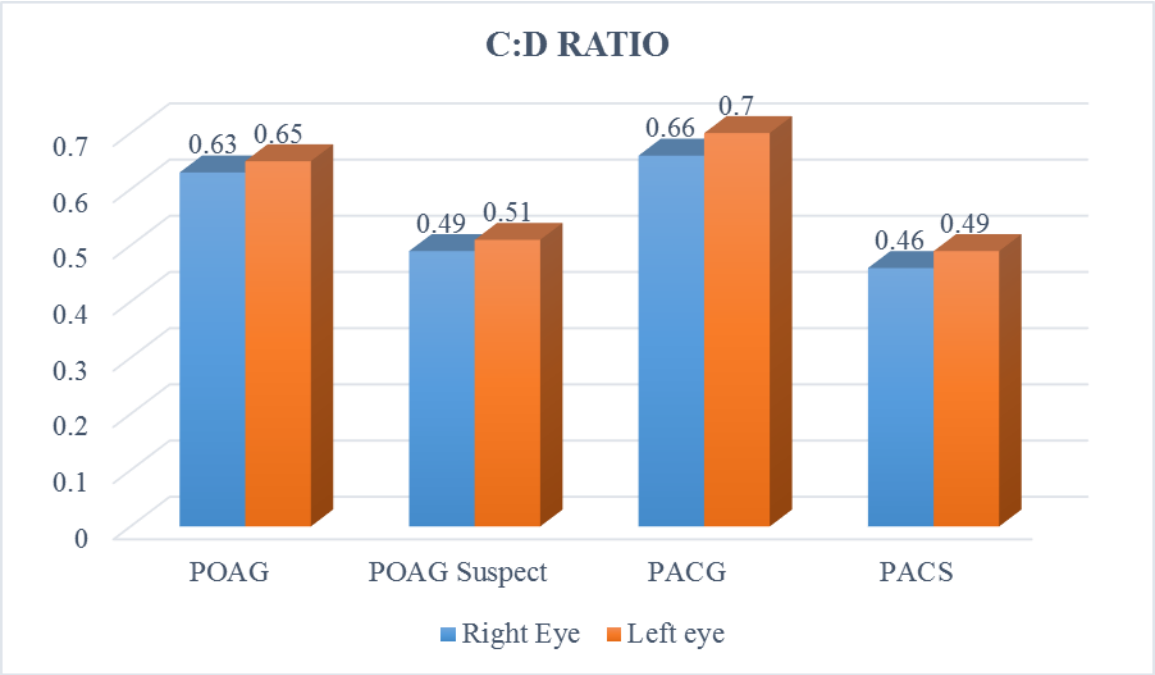

FIGURE 13: BAR DIAGRAM SHOWING CUP DISC RATION WITH RESPECT TO DIAGNOSIS IN BOTH EYES

It was observed that the mean C-D in the POAG was 0.63+/- 0.15 (RE) and 0.65+/- 0.11 (LE), whereas mean C: D ratio in PACG was 0.66 +/- 0.13 (RE) and 0.68+/- 0.12 (LE) Mean C: D ratio for POAG suspects were 0.49 +/- 0.16(RE) and 0.51+/- 0.15 (LE), whereas mean C: D ratio for PACS were 0.46 +/- 0.11 (RE) and 0.49 +/- 0.12 (LE)

The observation was statistically significant.

TABLE 15: GLAUCOMA HEMIFIELD TEST WITH RESPECT TO DIAGNOSIS

Glaucoma Hemifield Test		PAOG	PAOG Suspect	PACG	PACS	Total	χ 2 , df, p value
Right Eye	Borderline	4	19	8	0	31	196.3, 9, 0.0001*
	Not done	1	0	2	0	3	
	Outside normal limits	26	0	17	0	43	
	Within normal limits	2	20	0	85	107	
Left eye	Borderline	6	26	5	0	37	187.2, 9, 0.0001*
	Not done	2	0	2	0	4	
	Outside normal limits	22	0	20	0	42	
	Within normal limits	3	13	0	85	101	

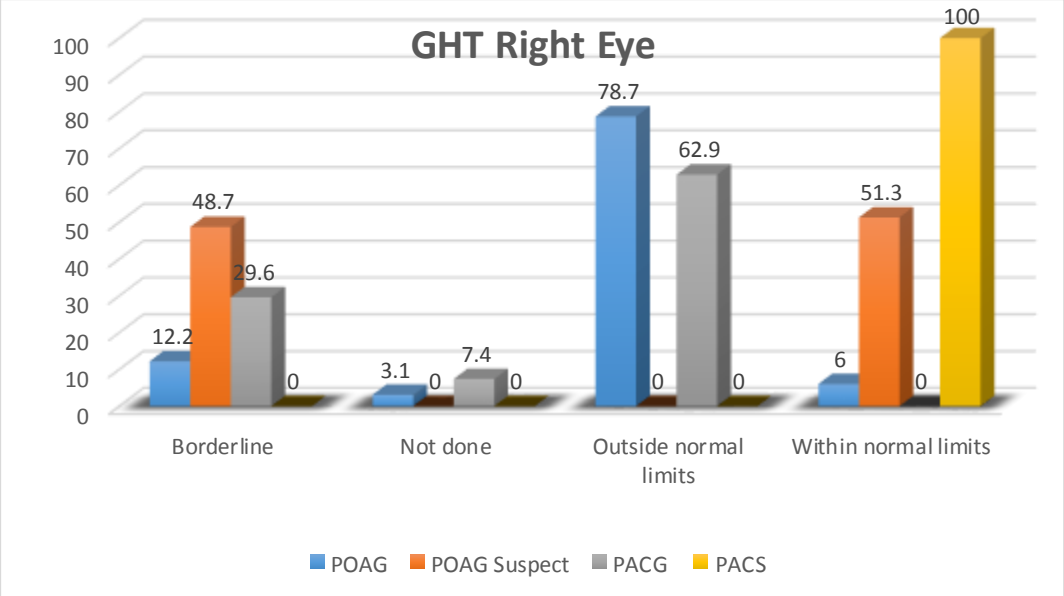

FIGURE 14: BAR DIAGRAM SHOWING GHT IN RIGHT EYE

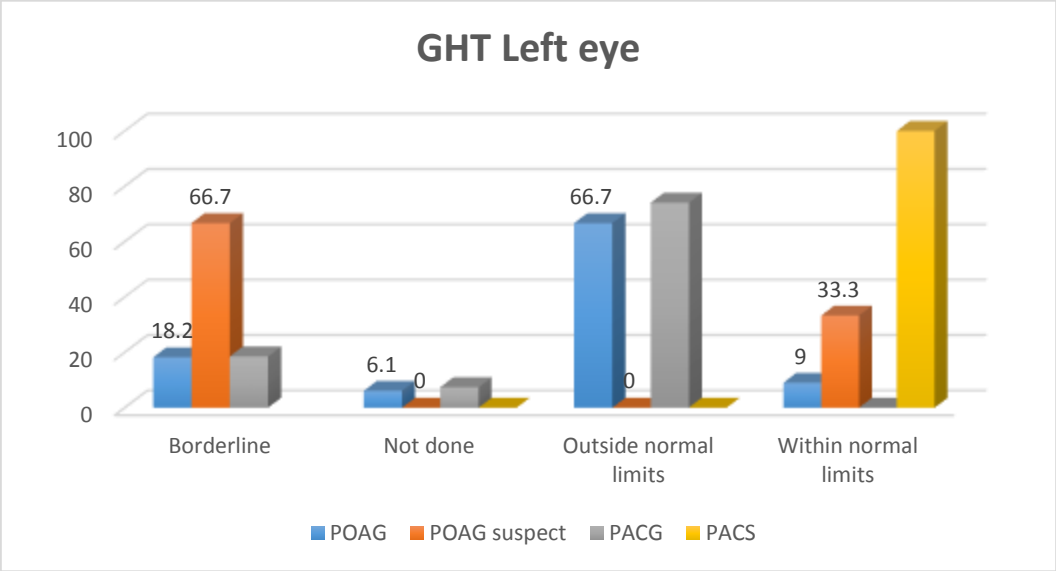

FIGURE 15: BAR DIAGRAM SHOWING GHT IN LEFT EYE WITH RESPECT TO DIAGNOSIS

It was observed that there was significant difference and association between GHT and Type of Glaucoma on Right eye and Left eye. p value <0.05 on both sides.

TABLE 16: ASSOCIATION BETWEEN REFRACTIVE ERROR AND PRIMARY GLAUCOMA

				PACG		Total	χ 2 , df, p value
		PAOG	PAOG Suspect	PACG	PACS		
Refractive Error	Emmetropia	23	25	19	61	128	58.59, 6, 0.0001**
	Hypermetropia	0	0	8	24	32	
	Myopia	10	14	0	0	24	
Total		33	39	27	85	184	

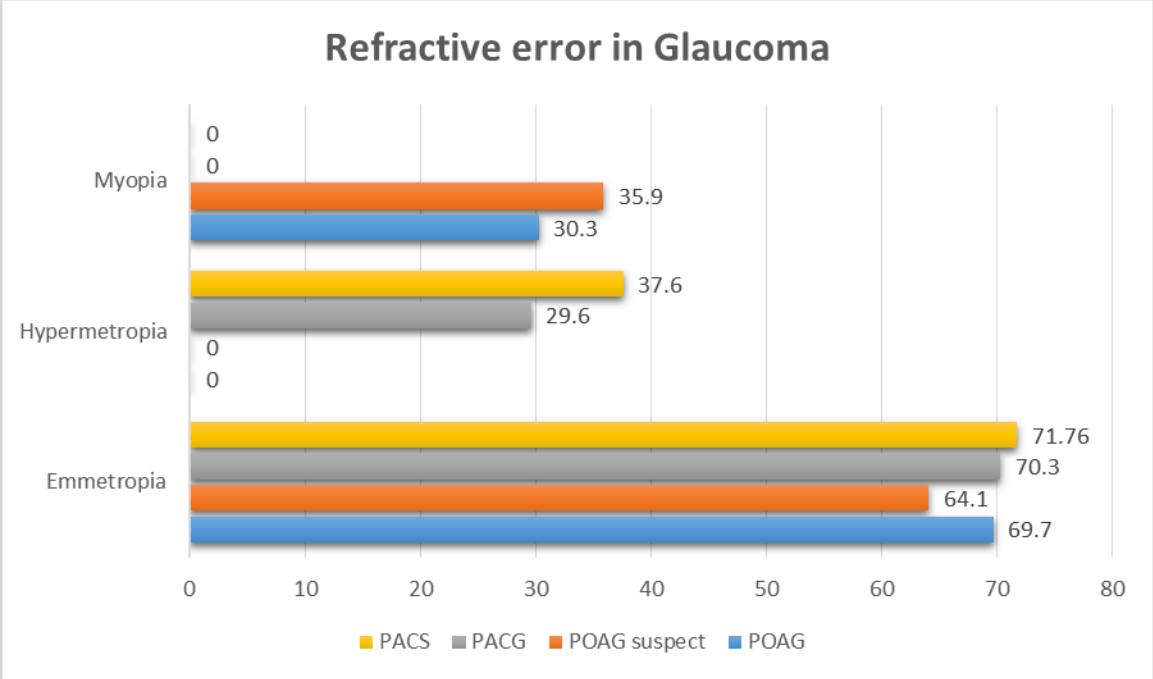

FIGURE 16: BAR DIAGRAM SHOWING ASSOCIATION BETWEEN REFRACTIVE ERROR AND GLAUCOMA IN PERCENTAGES.

It was observed that out of 24 myopic patients 10 (41.7%) patients was diagnosed having POAG, and 14 (58.3%) cases were POAG suspects wherein out of 32 hyperopic patients 8 (29.6%) hyperopic patients were diagnosed as PACG and 24 (75%) were diagnosed as PACS.

TABLE 17: MYOPIA GRADE IN POAG CASES

		POAG	POAG Suspect	TOTAL (%)
Myopia	**<4D**	2	7	9 (37.5)
	>4D	8	7	15 (62.5)
Total		10	14	24 (100)

Among the Myopics in the Primary open angle group 62.5% had > 4D refractive error and 37.5% with <4D refractive error.

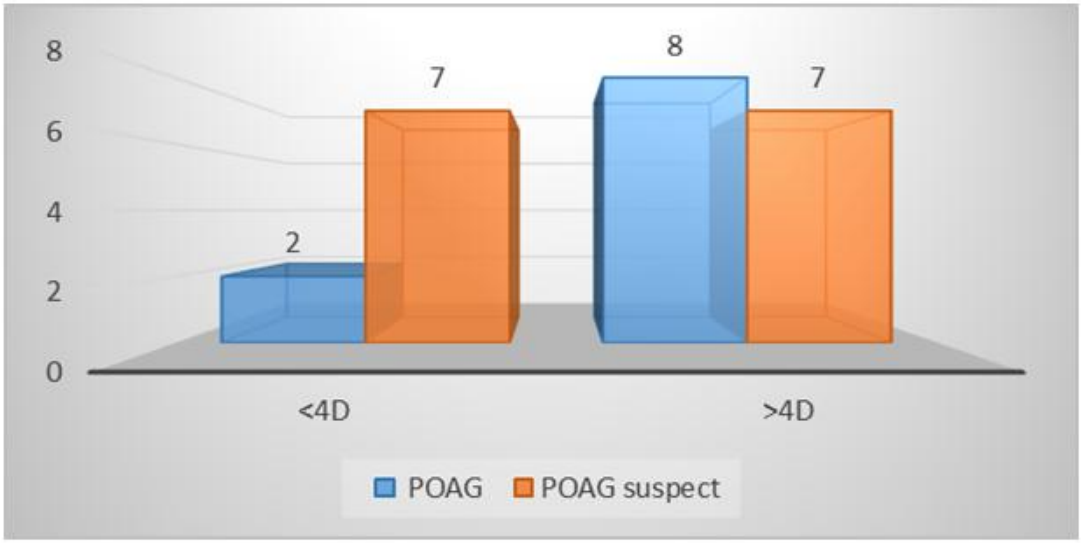

FIGURE 17: BAR DIAGRAM SHOWING MYOPIA IN POAG AND POAG SUSPECT

TABLE 18: ASSOCIATION BETWEEN SMOKING AND TYPE OF GLAUCOMA

						Total	χ 2 , df, p value
		POAG	POAG Suspect	PACG	PACS		
Smoking	Nonsmoker	24	30	23	70	147	2.060, 3, 0.5601
	Smoker	9	9	4	15	37	
Total		33	39	27	85	184	

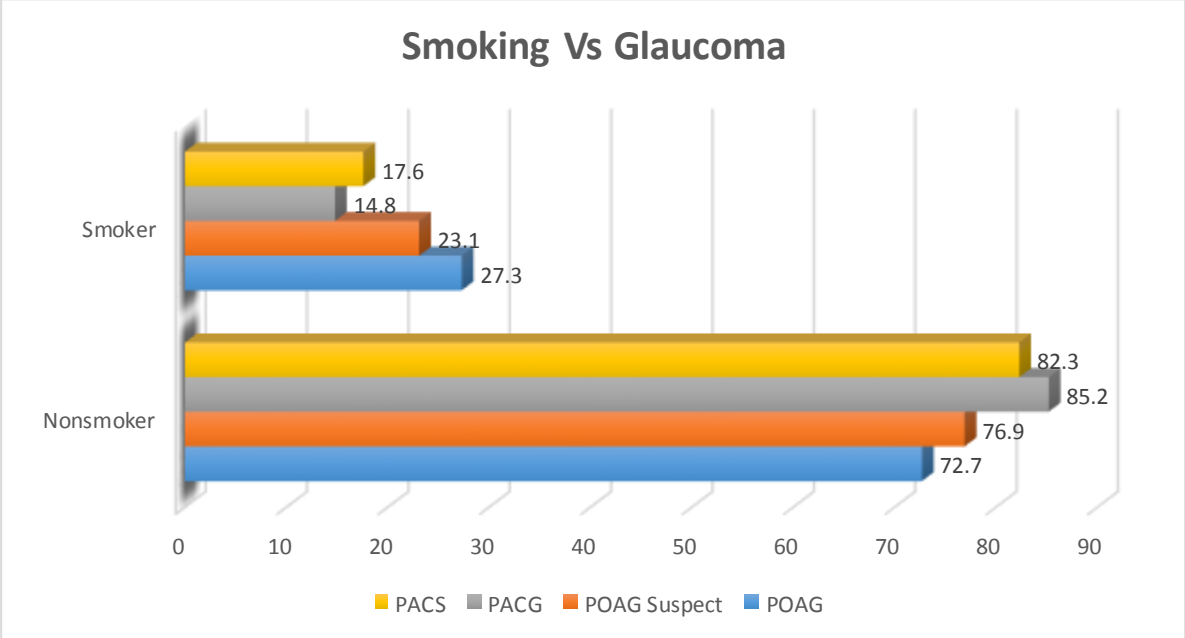

FIGURE 18: BAR DIAGRAM SHOWING ASSOCIATION BETWEEN SMOKING AND GLAUCOMA

It was observed that there was no significant association between smoking and primary glaucoma.

TABLE 19: ASSOCIATION BETWEEN DIABETES MELLITUS AND TYPE OF GLAUCOMA

						Total	χ 2 , df, p value
		PAOG	PAOG Suspect	PACG	PACS		
Diabetes	**Yes**	8	7	9	25	49	2.558, 3, 0.4649
Mellitus	**No**	25	32	18	60	135	
Total		33	39	27	85	184	

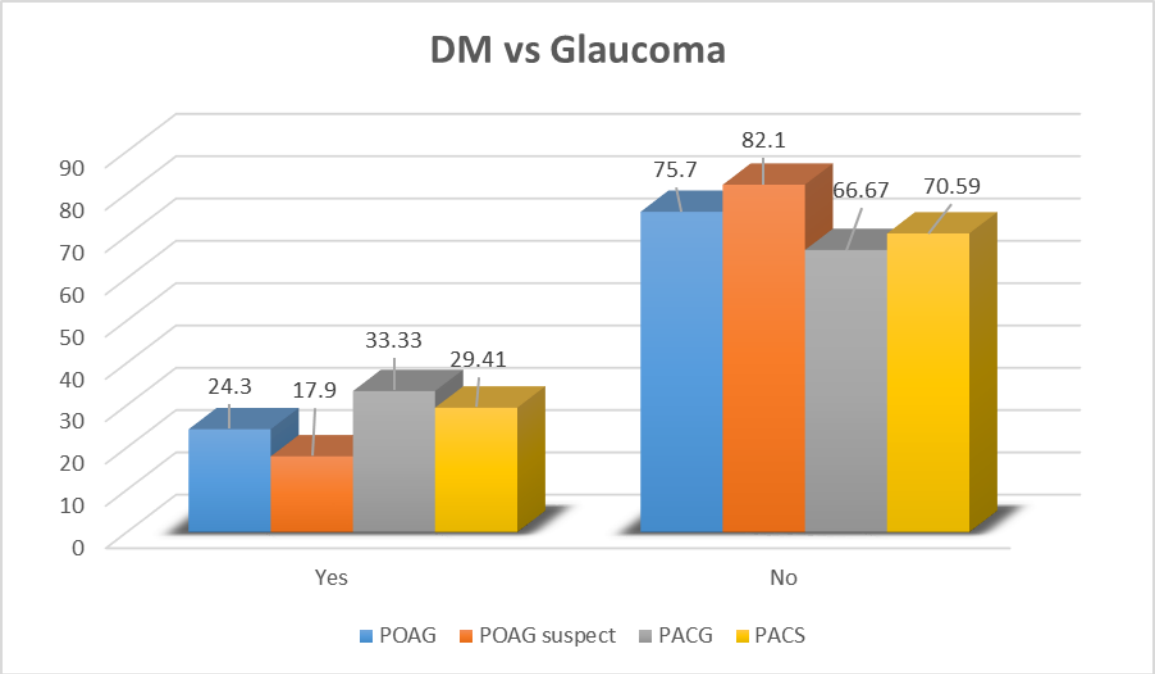

FIGURE 19: BAR DIAGRAM SHOWING ASSOCIATION BETWEEN DIABETES MELLITUS AND GLAUCOMA

It was observed that 8 cases (24.3%) of POAG had diabetes, whereas 7 cases (17.9%) of POAG Suspect had diabetes.

9 cases (33.33%) of PACG had diabetes, whereas 25 cases (29.41%) of PACS had diabetes. It was observed that there was no significant association between diabetes mellitus and type of glaucoma

TABLE 20: ASSOCIATION BETWEEN HYPERTENSION AND TYPE OF GLAUCOMA

						Total	χ 2 , df, p value
		PAOG	PAOG Suspect	PACG	PACS		
Hypertension	**Yes**	5	11	11	27	85	5.162, 3, 0.1603
	No	28	28	16	58	99	
Total		33	39	27	85	184	

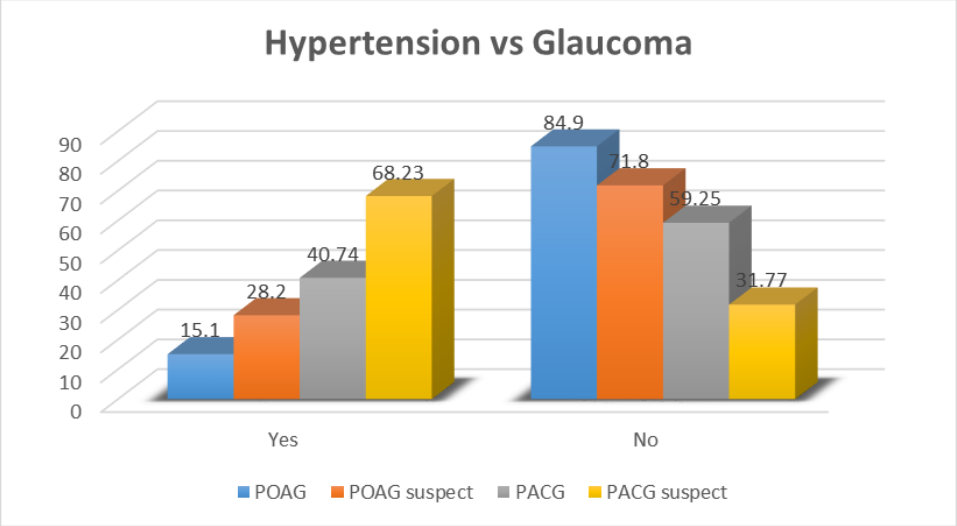

FIGURE 20: BAR DIAGRAM SHOWING ASSOCIATION BETWEEN HYPERTENSION AND GLAUCOMA

It was observed that 5 cases (15%) of POAG and 11 cases (28.20%) of POAG suspect had hypertension.

Similarly 11 cases (40.74%) of PACG and 27 cases (68.23%) of PACS had hypertension.

There was no significant association between hypertension and type of glaucoma.

TABLE 21: ASSOCIATION BETWEEN FAMILY HISTORY AND TYPE OF GLAUCOMA

		PAOG	PAOG Suspect	PACG	PACS	Total	χ 2 , df, p value
Family History	**Absent**	23	20	18	67	128	9.706, 3, 0.0212
	Present	10	19	9	18	56	
Total		33	39	27	85	184	

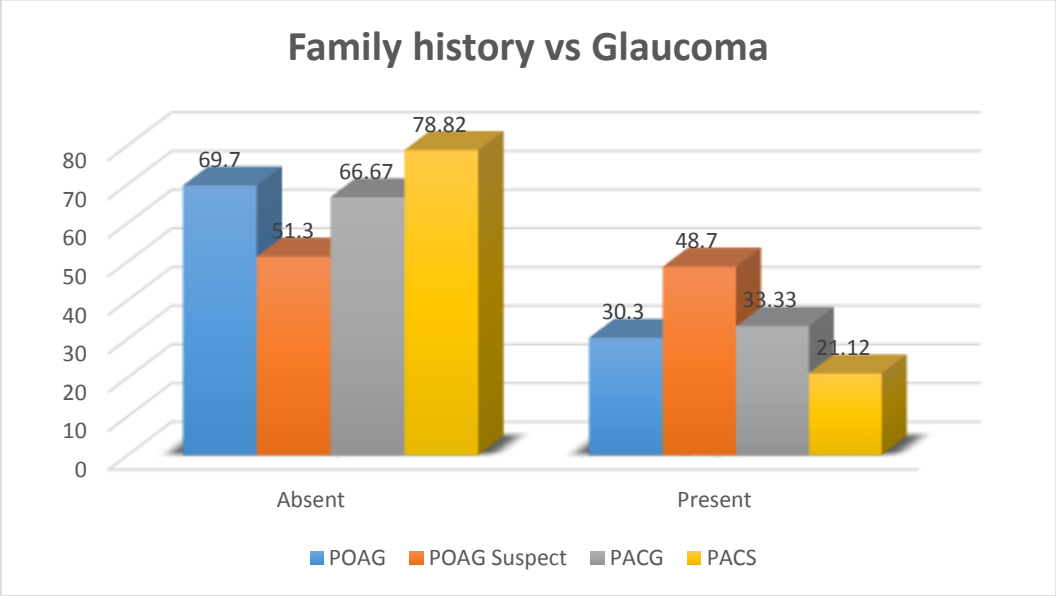

FIGURE 21: BAR DIAGRAM SHOWING ASSOCIATION BETWEEN FAMILY HISTORY AND GLAUCOMA

In the study it was observed that family history of glaucoma was significantly associated with primary glaucoma with 10 cases (30.3%) of POAG had positive family history, and 15 cases (33.33%) of PACG having a positive family history.

The findings were statistically significant.

DISCUSSIONS

DISCUSSIONS

In the study 3500 subjects were screened during the study period of which total of 184 cases (5.26%) were having Established and suspected primary glaucoma,

72 cases (2.09%) of them were in the primary open angle group comprising of both POAG (0.94%) cases and POAG suspects (1.11%). 27 cases (0.77%) were diagnosed having PACG, whereas 85 cases (2.42%) were diagnosed as PACS.

There are again wide variations in the prevalence of POAG between the Indian studies The VES reported the lowest rates of 0.41%. Reasons for this, the prime cause being that the study was limited to the age group of 30-60 years. Comparing the prevalence of POAG in those from APEDS (2.56%) [83], where both POAG and POAG suspects were clubbed together, similar proportions were found in our study.

The VES was the first to report that PACG comprised a significant proportion of those with glaucoma. Subsequent reports from the APEDS[83] did report a lower prevalence of angle closure disease. The difference was most marked in the lower prevalence of PACS (2.21%) in APEDS, similar observation was found in our study.

The CGS (rural) [84] also reported a prevalence % of PACG as 0.87, which is similar to our study.

In our study the mean age **(TABLE 4)** of POAG subjects was 64.50 ± 13.060, POAG Suspect was 63.00 ± 13.6, among PACG mean age was 68.60 ± 10.94 and among PACG suspect was 55.88 ± 14.65.

In **TABLE 5** Proportion of Glaucoma suspects and Established glaucoma with respect to diagnosis is shown, 3500 people were screened for glaucoma during the study period and it was observed that proportion of POAG was common after 50 years of age, in the study 12 cases (36.36%) out of 33 was diagnosed with POAG were 50-59 years of age.

Whereas PACG was more common after 60 years, the study 11 cases (40.74%) diagnosed with PACG was >60 years of age. This observation was statistically significant. Increasing age has been the consistent risk factor for all studies. In India. [83, 84, 85]

The findings compares well with the results of the study from Hyderabad, where out of total glaucoma admissions, only 10.81% were under 40 years of age while the rest were above 40 years, out of these 44% were 41-50 years old, 32% were 51-60 years, 8% were 61-70 years and 4% were more than 70 years of age.[88]

Similar findings were found in a study on POAG patients by Tuck MW where 7% were less than 55 years old, 44% were aged 55-74 years, and 49% were older.[89]

In **TABLE 6** sex distribution with respect to diagnosis is shown, 51 males out of total 72 were POAG and POAG suspects, In POAG male to female ratio was 2:1, whereas in POAG suspects the Male female ratio was 3:1, this it was observed males were likely to present as POAG or POAG suspects.

PACG was common in Females with male to female ratio of 1:2.4, whereas the male female ratio in PACS was 1:3.2.

This observation was statistically significant.

This observation is supported by Barbados[39] and Rotterdam study [82] where males are reported to be 1.5 and 3 times, respectively, more at risk for glaucoma.

In our study 21 females out of 33 were PACG and PACS, it was observed that females were more likely to present with PACG OR PACS.

Similar results from most other studies shows that females were significantly more than males in PACG patients, a study by Hu, in 1989 showed prevalence rate of PACG was 0.13% in males and 0.47% in females, a significant sex difference.[90]

Decreased visual acuity was the commonest presentation with in POAG with more than 75% patients presenting with BCVA of less than 6/18**. (TABLE 7).** One patient had PL – ve, in at least one eye, meaning that the patient had suffered severe optic nerve damage without realizing it.

In **TABLE 8** we observed that in suspected open angle glaucoma cases more than 80% cases had BCVA between 6/6 – 6/12.

In **TABLE 9** we observed that BCVA in PACG cases were less than 6/36 in more than 50 % cases, 1 patient presented with PL –ve, whereas more than 90% cases of PACS had BCVA of 6/6-6/12. (**TABLE 10).**

The BCVA of Glaucoma suspects ranged from 6/6 -6/12 in 80% in case of POAG suspects and 90% in case PACS, this could be an important reason a relatively high number of cases which go undetected .

In **TABLE 11**, it was observed that Mean Intra ocular pressure was high in PACG in both eyes (32.67 mmHg in Right eye and 32.48 mmHg in Left eye) followed by POAG, (24.91 in Right eye and 24.45 in Left eye).POAG Suspects had 21.74 mmHg in Right eye

and 22.74mmHg in Left eye).The mean IOP in PACS subjects were 18.47 mmHg in right eye and 18.63 mmHg in Left eye. This observation was statistically significant within the groups and between groups in both eyes.

Similar findings were cited in the Baltimore Eye Survey[38] and the Barbados Eye Study[39], IOP >21mmHg was found to be an important factor in glaucoma that correlated with increased prevalence and incidence[40].

In **TABLE 12** Van Herrick's grading of anterior chamber depth with respect to diagnosis was observed. It is been observed than the mean grading in POAG was 3.53+/- 0.507 (RE) and 3.46 +/- 0.501, POAG suspects had similar mean grading of 3.42 +/- 0.501 (RE) and 3.44+/- 0.502 (LE).The mean grading in PACG was 0.88+/- 0.667 (RE) and 0.76 +/- 0.723 (LE), in PACS the mean grading was 1.52 +/- 0.503 (RE) and 1.54 +/- 0.541 (LE).This observation was statistically significant.

In **TABLE 13**, it was observed that mean Shaffer's grading for POAG was 3.58 +/- 0.502 (RE) and 3.55+/-0.506 (LE) and POAG suspect had similar findings, 3.49 +/- 0.506 (RE) and 3.54+/- 0.506.For PACG the mean was 1.11+/-0 .863 (RE) and 1.04+/- 0.649 and in PACS cases it was 1.99 +/- 0.645 (RE) and 1.96 +/- 0.663 (LE) .

All the cases of PACG had a mean angle grading of 1.04 ~1.11, and all the cases with PACS had mean angle grading of 1.99~ 1.96. This observation in mean of Shaffer's grade was statistically significant between and within the groups and any angle grading <2 on Van Herrick's scale should warrant glaucoma evaluation,

In **TABLE 14,** It was observed that the mean C-D in the POAG was 0.63+/- 0.15 (RE) and 0.65+/- 0.11 (LE), whereas mean C: D ratio in PACG was 0.66 +/- 0.13 (RE) and 0.68+/- 0.12 (LE). Mean C: D ratio for POAG suspects were 0.49 +/- 0.16(RE) and 0.51+/- 0.15 (LE) , whereas mean C:D ratio for PACS were 0.46 +/- 0.11 (RE) and 0.49 +/- 0.12 (LE). The observation was statistically significant. Studies have documented normal C: D ratio ranging from <0.3 in 66% of normal individuals to greater than 0.5 in only 6% of normal individual.[92] Thus any C: D ratio >0.4 should warrant detailed evaluation for glaucoma.

In **TABLE 15** glaucoma hemifield test with respect to diagnosis it was observed that there was significant difference and association between "outside normal limit" GHT and glaucomatous visual field damage .p value <0.05 on both sides. The GHT is based on the fact that glaucoma damages the superior and inferior fields asymmetrically. The GHT compares mirror-image clusters of points in the superior and inferior fields, and it alerts the clinician when significant differences are found between the two hemifields.

In **TABLE 16** the association between refractive error and primary glaucoma it was observed that out of 24 myopic patients 10 (41.7%) patients was diagnosed having POAG, and 14 (58.3%) cases were POAG suspects wherein out of 32 hyperopic patients 8 (29.6%) hyperopic patients were diagnosed as PACG and 24 (75%) were diagnosed as PACS. There was significant association between refractive error and Primary Glaucoma

In **TABLE 17** among the Myopes in the Primary open angle group 62.5% had > 4D refractive error and 37.5% with <4D refractive error. Similar findings were cited in The Blue Mountains Eye Study, which found a strong relationship between POAG and myopia.[72] In the Barbados Eye Study, a myopic refraction was one of several risk factors for POAG in adult black people [81].

In Asian populations, the Singapore Malays Eye Study showed an association between moderate or high myopia (worse than –4 D) and POAG.[97] In Beijing Eye Study in China, a population study, marked to high myopia with a myopic refractive error exceeding -6 D may be a risk factor associated with glaucomatous optic neuropathy [71]

In **TABLE 18** the association between smoking and type of glaucoma it was observed that there was no significant association between smoking and primary glaucoma. Similar association was citied in a population based cross-sectional study of Hispanic adults over 40 years of age, where smoking was not related to glaucoma.[92]

Two case control studies in france[94] and congo[93] found no correlation between cigarette smoking and glaucoma

Smoking status has a strong association with eye health, especially age-related macular degeneration, but population-based studies found no association in the area of glaucoma.[95]

In **TABLE 19** Association between diabetes mellitus and primary Glaucoma, it was observed that 8 cases (24.3%) of POAG had diabetes, whereas 7 cases (17.9%) of POAG Suspect had diabetes.

9 cases (33.33%) of PACG had diabetes, whereas 25 cases (29.41%) of PACS had diabetes. It was observed that there was no significant association between diabetes mellitus and type of glaucoma.

Similar findings were cited in the Baltimore Eye Survey,[80] a population-based study of 5.308 subjects, was unable to show a higher prevalence of POAG among diabetics1. The Ocular Hypertension Treatment Study (OHTS) of 1.636 ocular hypertensive (OH) subjects originally even found that diabetes is protective against the development of POAG[96]

In **TABLE 20** association between hypertension and primary glaucoma was observed, it was observed that 5 cases (15%) of POAG and 11 cases (28.20%) of POAG suspect had hypertension. Similarly 11 cases (40.74%) of PACG and 27 cases (68.23%) of PACS had hypertension. There was no significant association between hypertension and type of glaucoma.

Vijaya L et al [86] conducted a study in rural south India on the prevalence of POAG.

They also studied some of the associated risk factors for POAG such as age, gender, IOP, myopia and hypertension No association was found between systemic hypertension and POAG in this study.

In another study done by Vijaya L et al known as the Chennai glaucoma study[84] no statistically significant association was found between POAG and systemic hypertension

In **TABLE 21**, association between family history and type of glaucoma study it was observed that family history of glaucoma was significantly associated with primary glaucoma with 10 cases (30.3%) of POAG had positive family history, and 15 cases

(33.33%) of PACG having a positive family history. The findings were statistically significant.

Population based studies have supported an association between a positive family history for glaucoma and POAG. In the Barbados Eye Study[81] undiagnosed subjects were more likely to develop glaucoma if they had a history of glaucoma in one or more siblings.

In Rotterdam the population based familial aggregation study showed that the life time risk of glaucoma in siblings and offspring of glaucoma patients was 9.2 times higher than in controls.[82]

CONCLUSION

CONCLUSION

In our study the proportion of POAG was more than PACG. And increasing age has been the consistent risk factor for primary glaucoma.

POAG was found to be more common in males and is present over 50 years of age, whereas PACG was common after 70 years with a female preponderance.

It was observed than PACG had more visual morbidity than POAG patients. There were a large number of PACS was greater than POAG suspects, and 90% of the glaucoma suspects had good vision.

Myopia >4D was a factor for development of POAG, whereas hyperopic eyes were consistent risk factor for PACG.

Family history was also observed to be a major risk factor for both POAG and PACS.

Systemic diseases like hypertension, diabetes and lifestyle habits like smoking were not significantly associated with primary glaucoma in the present study.

The Indian population has substantial risk for POAG, PACG and large burden of angle closure disease and as the majority of the Indian population is less than 30 years of age and with increasing life expectancy the number of those at risk of glaucoma will grow exponentially over time. Thus, from the observation of this study, all subjects above forty years should be screened for glaucoma, and thorough evaluation of patients with additional risk factors like refractive error and strong family history of glaucoma would serve a long way in improving our glaucoma case detection rates which is of utmost importance if we are to make an impact on the burden of the disease in our country.

SUMMARY

SUMMARY

Glaucoma is a leading cause of irreversible blindness throughout the world. Worldwide, it has become the second most common cause of bilateral blindness, the objective of this study is to determine the proportions of POAG and PACG and study their risk factors,

In this observational study 3500 subjects were screened during the study period of which total of 184 cases (5.26%) were diagnosed as having Primary glaucoma and suspected cases of open angle and angle closure.

72 cases (2.09%) of them were in the primary open angle group comprising of both POAG (0.94%) cases and POAG suspects. (1.11%). 27 cases (0.77%) were diagnosed having PACG, whereas 85 cases (2.42%) were diagnosed as PACS. Mean age of presentation of POAG was 64.50 ± 13.060 years, PACG mean age was 68.60 ± 10.94 years. 29 subjects (87.87%) out of 33 was diagnosed with POAG was more than 50 years of age, PACG was more common after 60 years, 21 (77.78%).

POAG was common among Males with male to female ratio was 2:1, PACG was common in Females with male to female ratio of 1:2.4,

Most of the POAG cases presented with visual acuity ranging from 6/18 – 6/60, PACG cases presented with visual acuity of 6/36 or less on presentation.

Mean IOP was high in PACG in both eyes (32.67 mmHg in Right eye and 32.48 mmHg in Left eye) followed by POAG, (24.91 in Right eye and 24.45 in Left eye).

Among cases of primary open angle glaucoma 10 (41.7%) were myopic. In PACG , 8 (29.6%) cases were found to be hyperopic.10 cases (30.3%) of POAG and 15 cases (33.33%) of PACG had a positive family history.

From the observations of this study, all subjects above forty years should be screened for glaucoma, and thorough evaluation of patients with additional risk factors like refractive error and strong family history of glaucoma would serve a long way in improving our glaucoma case detection rates which is of utmost importance if we are to make an impact on the burden of the disease in our country.

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ANNEXURES

ANNEXURES

PROFORMA

Name: IP no:

Age/Sex:

Address:

Chief Complaints:

History of presenting illness:

Past history:

Family history:

Personal history:

General physical examination:

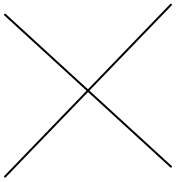
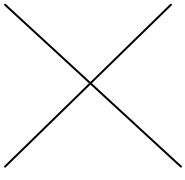
Pallor	Icterus	Cyanosis
Edema	Clubbing	Lymphadenopathy
Pulse:	BP-	

Systemic Evaluation:

Cardiovascular System: Respiratory System:

Per abdomen: Central Nervous System:

OCULAR EXAMINATION

Head Posture:	RE	LE
Ocular Posture		
Eye Lids		
Conjunctiva		
Cornea		
Sclera		
Anterior Chamber		
Van Herick's grading		
Angles (Gonioscopy)		
Iris		
Pupil- Size		
Shape		
Reaction		

Lens

Visual Acuity

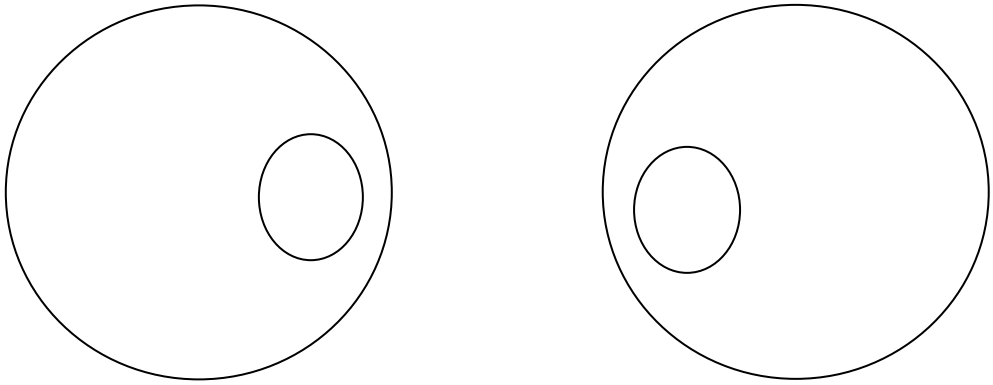
Distant vision-

Near vision-

IOT

Fundus Examination

Optic Disc



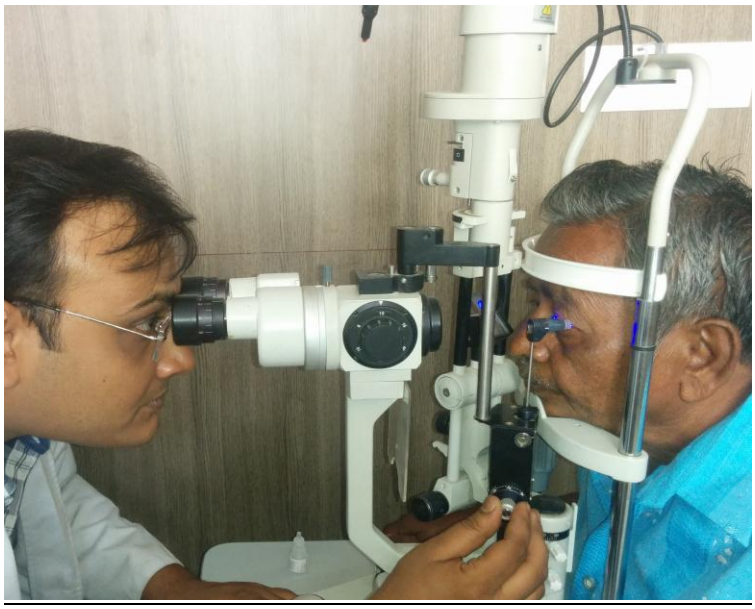
CDR

<u>Visual Field Defect Level</u>

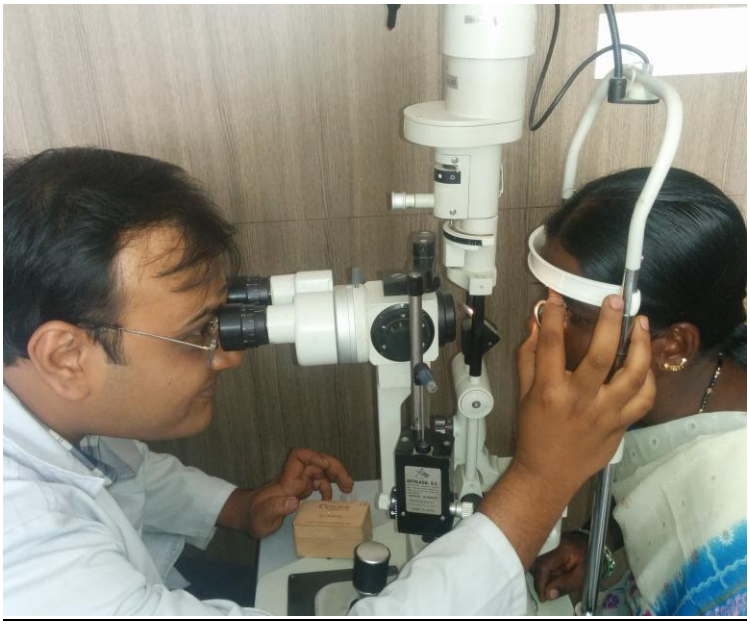
GHT (RE)

GHT (LE)

PHOTOGRAPHS

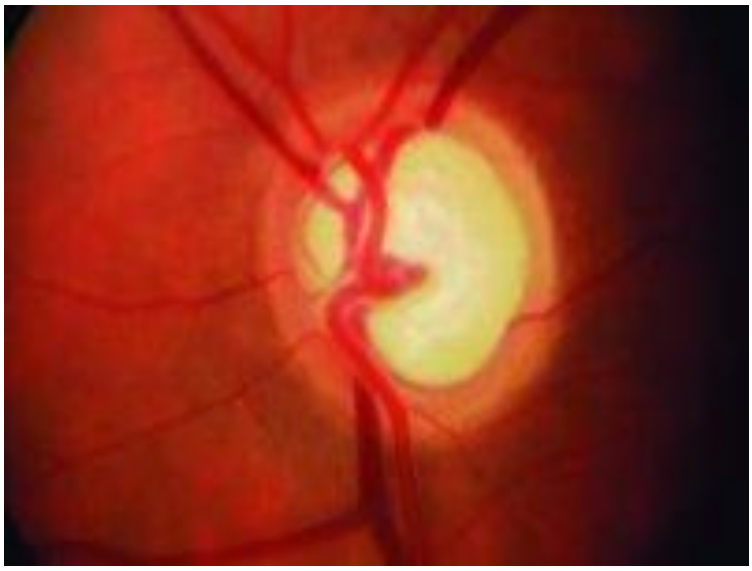


PHOTOGRAPH 1: IOP MEASUREMENT BY GOLDMANN APPLANATION TONOMETER.

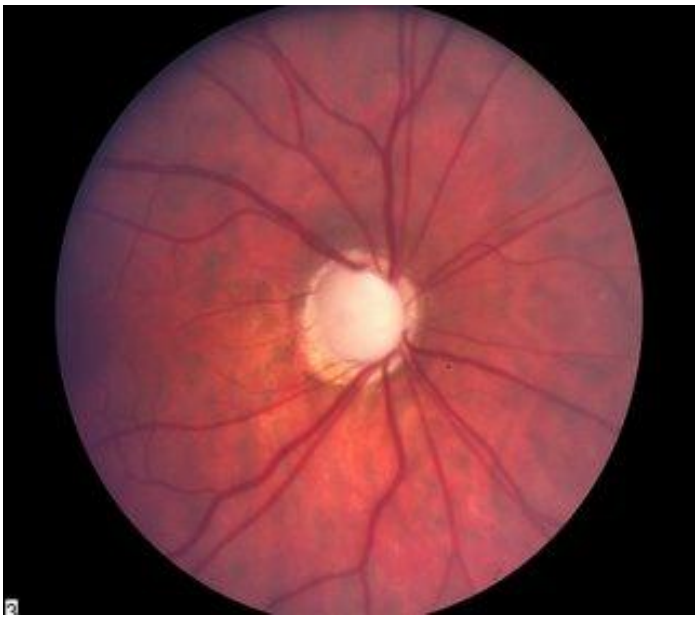


PHOTOGRAPH 2: GOINOSCOPY BY GOLDMANNS TWO MIRROR GONIO LENS.

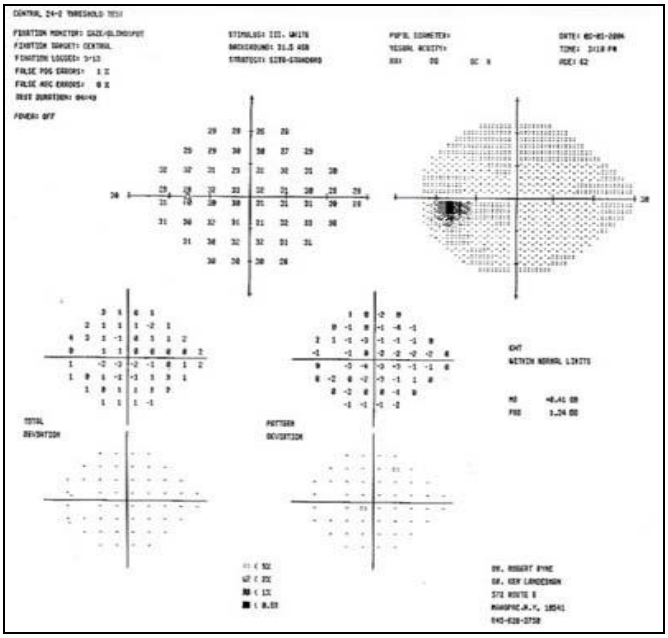
PHOTOGRAPH 3: VISUAL FIELDS TESTING BY HUMPHREYS FIELD ANALYZER.



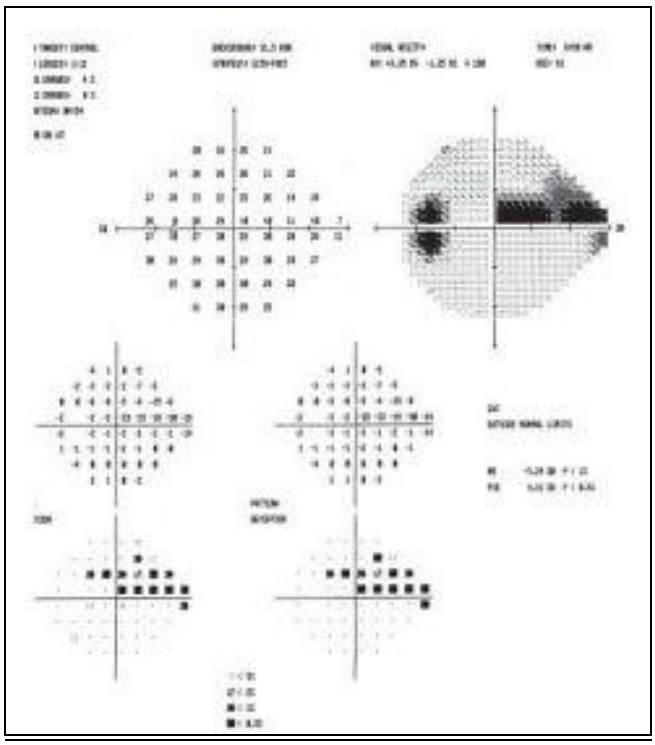
PHOTOGRAPH 4: GLAUCOMATOUS DISC CUPPING



PHOTOGRAPH 5: NEURO-RETINAL RIM THINNING WITH NERVE FIBRE LAYER DEFECT



PHOTOGRAPH 6: NORMAL VISUAL FIELD



PHOTOGRAPH 7: GLAUCOMATOUS VISUAL FIELD DEFECT

KEY TO MASTER CHART

- Sl. No. >> Serial Number.
- POAG >> Primary open angle glaucoma.
- POAG SUSPECT >> Primary open angle glaucoma suspect.
- PACG >> Primary angle closure glaucoma.
- PACS >> Primary angle closure suspect.
- UCDV >> Uncorrected distant vision.
- BCDV >> Best corrected distant vision.
- UCNV >> Uncorrected near vision.
- BCNV >> Best corrected near vision.
- C: D >> Cup and disc ratio.
- GHT >> Glaucoma hemifield test.
- H T N >> Hypertension

MASTER CHART FOR POAG AND POAG SUSPECTS

SL NO	NAME	AGE	SEX	OP NO	UCVA (RE)	UCVA (LE)	BCVA (RE)	BCVA(LE)	UCNV(RE)	UCNV(LE)	BCNV(RE)	BCNV(LE)	IOP(RE)	IOP(LE)	SHAFFERS (RE)	SHAFFERS(LE)	C:D (RE)	C:D(LE)	GHT(RE)	GHT (LE)	DIAGNOSIS	ERROR	>4D	HERICK	HERICK	SMOKING	DIABETIC	HTN	FAMILY HO
1	RAJAMMA	58	F	11592	CF 5 MTS	6/60	6/60	6/36	N8	N8	N6	N6	24	20	3	3	0.5	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
2	P.C RAO	40	M	505828	6/18	6/36	6/18	6/18	N10	N10	N6	N6	21	20	3	4	0.5	0.5	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	SMOKER	DIABETIC	NONHTN	PRESENT
3	VENKATESHAPPA	67	M	524664	6/36	6/60	6/6	6/9	N6	N6	N6	N6	22	28	4	4	0.8	0.6	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	PRESENT
4	P.S VENKATRAMAPPA	48	M	575462	6/32	6/12	6/9	6/6	N6	N6	N6	N6	15	24	4	3	0.3	0.7	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
5	BABU	85	M	590796	6/18	6/9	6/12	6/9	N8	N8	N6	N6	20	24	3	4	0.5	0.6	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	PRESENT
6	SAJJA	59	F	616096	6/36	6/36	6/18	6/24	N8	N12	N6	N8	20	18	3	4	0.5	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	4	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
7	PADMA S	75	F	624677	PL -VE	CF3MTS	PL-VE	CF 5 MTS	N0	N18	N0	N12	24	21	4	4	0.6	0.6	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
8	SRINIVAS	69	M	627474	6/6	6/9	-	-	N12	N12	N8	N8	18	26	4	4	0.3	0.5	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
9	SHOBHA	78	F	629837	6/6	6/6	6/6	6/6	N12	N12	N6	N8	26	21	4	4	0.6	0.5	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	4	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
10	BALAJI	66	M	630447	6/9	6/6	6/6	6/6	N8	N8	N6	N6	17	22	3	4	0.3	0.5	BODERLINE	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
11	MUNNISWAMI	54	M	630509	6/60	6/60	6/9	6/9	N6	N8	N6	N6	18	24	4	4	0.5	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	4	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
12	MUNNISWAMI	60	M	630509	CF 5 MTS	6/60	6/18	6/24	N6	N6	N6	N6	24	24	4	4	0.5	0.7	WITHIN NORMAL LIMITS	BODERLINE	SUSPECT	EMMETROPIA	-	3	4	SMIOKER	DIABETIC	HYPERTENSIVEE	PRESENT
13	P RAO	64	M	631511	6/12	6/9	6/6	6/6	N6	N6	N6	N6	28	26	4	4	0.6	0.6	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
14	NEEJAMANI	89	M	645144	6/6	6/6	-	-	N12	N8	N8	N8	26	28	4	4	0.8	0.8	OUTSIDE NORMAL LIMITS	NOT DONE	POAG	MYOPIA	YES	4	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
15	VEERESH	50	M	671489	6/60	6/36	6/18	6/12	N12	N8	N8	N6	26	24	4	4	0.8	0.7	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	4	3	SMOKER	DIABETIC	NONHTN	ABSENT
16	CHANDRIKA	56	F	674403	6/36	6/36	6/12	6/12	N12	N12	N8	N6	36	20	4	4	0.7	0.7	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
17	ANSAR AHMED	55	M	678758	6/36	6/18	6/18	6/18	N12	N12	N6	N8	21	16	4	4	0.8	0.6	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	PRESENT
18	RAJA REDDY	55	M	692704	6/36	6/60	6/18	6/24	N10	N8	N8	N8	24	18	3	3	0.7	0.7	WITHIN NORMAL LIMITS	BODERLINE	SUSPECT	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
19	JAYARAM	39	M	693806	CF 5 MTS	CF5 MTS	6/36	6/60	N8	N8	N6	N6	24	21	3	3	0.7	0.5	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	4	3	NONSMOKER	DIABETIC	NONHTN	PRESENT
20	ADINARAYANAPPPA	55	M	695399	6/18	6/12	6/18	6/9	N8	N8	N6	N6	28	24	4	4	0.8	0.6	BODERLINE	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	PRESENT
21	KALYAN SAGAR	58	M	700667	6/24	6/9	6/9	6/9	N10	N6	N6	N6	28	31	3	3	0.6	0.8	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	4	SMOKER	DIABETIC	NONHTN	ABSENT
22	SHIVANNA	64	M	707212	6/60	6/36	6/36	6/18	N8	N8	N6	N6	26	24	4	3	0.7	0.8	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	4	SMOKER	DIABETIC	NONHTN	ABSENT
23	SEETHAMMA	72	F	707972	6/36	6/18	6/18	6/12	N12	N12	N8	N6	26	20	4	3	0.7	0.8	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	PRESENT
24	VENKATESH	78	M	714277	6/36	6/36	6/24	6/24	N12	N12	N6	N6	24	26	4	4	0.6	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
25	MUNNICHENNAPPA	59	M	720813	6/12	6/9	6/6	6/6	N6	N6	N6	N6	22	26	4	3	0.8	0.5	BODERLINE	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
26	MUNIYAPPA	50	M	722716	6/6	6/6	6/6	6/6	N10	N10	N6	N6	28	21	4	4	0.6	0.7	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
27	CHOWDAPPA	75	M	722865	6/60	CF 5 MTS	6/12	6/18	N8	N8	N6	N6	28	20	3	3	0.9	0.6	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
28	BHAGYA	82	F	727717	6/60	6/60	6/18	6/12	N6	N6	N6	N6	22	24	4	3	0.3	0.6	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
29	NARAYAN S	42	M	729777	6/6	6/6	6/5	6/6	N8	N8	N6	N6	26	20	3	3	0.6	0.6	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	3	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
30	KONDARAMAN SHETTY	53	M	730563	6/18	6/18	6/18	6/18	N10	N10	N6	N6	24	22	3	3	0.3	0.6	WITHIN NORMAL LIMITS	BODERLINE	SUSPECT	EMMETROPIA	-	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
31	SHAKUNTALA	50	F	735114	6/18	6/18	6/12	6/12	N10	N10	N6	N6	21	21	4	4	0.6	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	NO	3	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
32	NAGARAJU K.M	39	M	749100	6/18	6/12	6/9	6/6	N10	N10	N6	N6	24	18	3	3	0.3	0.6	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
33	MADHUSUDAN	44	M	762751	CF5MTS	6/60	6/60	6/18	N18	N12	N18	N8	28	30	4	4	0.8	0.8	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	4	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
34	MANJULA	36	F	763909	6/18	6/36	6/18	6/18	N12	N12	N6	N6	30	24	4	4	0.3	0.6	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	SMOKER	DIABETIC	NONHTN	ABSENT
35	AFSAR	66	M	765808	6/24	6/32	6/24	6/24	N12	N12	N6	N6	22	25	4	4	0.6	0.5	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	3	4	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
36	KALAPPACHAR	75	M	789461	6/60	CF 5 MTS	6/12	6/60	N8	N12	N8	N12	24	21	3	3	0.3	0.6	WITHIN NORMAL LIMITS	BODERLINE	SUSPECT	MYOPIA	NO	4	4	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
37	RAMACHANDRA RAO	51	M	790678	6/9	6/12	6/9	6/6	N6	N6	N6	N6	20	24	3	3	0.4	0.5	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	NO	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
38	ASHWAT NARAYAN SHETT	78	M	805064	6/12	6/24	6/12	6/18	N10	N10	N6	N6	21	21	4	3	0.5	0.5	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
39	NAGARATHAMMA	62	F	822033	6/9	6/9	6/6	6/6	N6	N6	N6	N6	26	32	3	4	0.6	0.7	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	PRESENT
40	SAMREEN TAJ	87	F	823767	CF 5 MTS	6/60	6/9	6/9	N6	N8	N6	N6	21	28	3	3	0.5	0.8	BODERLINE	WITHIN NORMAL LIMITS	POAG	EMMETROPIA	-	4	4	SMOKER	DIABETIC	NONHTN	ABSENT
41	LAVANYA	38	F	827562	6/60	6/36	6/36	6/12	N12	N8	N8	N6	24	26	3	3	0.3	0.7	WITHIN NORMAL LIMITS	BODERLINE	SUSPECT	EMMETROPIA	-	4	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
42	NEELAAMMA	66	F	837788	6/6	6/6	6/6	6/6	N10	N10	N6	N6	24	28	4	4	0.6	0.5	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	EMMETROPIA	-	3	3	SMOKER	DIABETIC	HYPERTENSIVE	PRESENT
43	DAKSHAYAN	83	M	840604	6/18	6/18	6/18	6/18	N10	N10	N6	N6	21	21	3	3	0.4	0.8	WITHIN NORMAL LIMITS	BODERLINE	SUSPECT	MYOPIA	NO	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
44	LAKSHAMMA	57	F	854584	6/60	6/6	6/36	6/6	N8	N8	N6	N6	24	16	3	3	0.5	0.5	BODERLINE	WITHIN NORMAL LIMITS	POAG	EMMETROPIA	-	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
45	SHANKAR SINGH	56	M	865359	6/12	6/9	6/12	6/6	N10	N10	N6	N8	18	24	3	3	0.5	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	YES	4	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
46	GULAB JAN	53	F	867859	6/12	6/36	6/12	6/18	N18	N18	N6	N8	24	18	3	3	0.7	0.5	BODERLINE	WITHIN NORMAL LIMITS	POAG	MYOPIA	YES	4	4	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
47	VENKATRAMANNA PAO	76	M	879176	6/6	6/6	6/6	6/6	N6	N8	N6	N6	30	24	4	3	0.7	0.5	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	pRESENT
48	VISHWANATH	75	M	879176	6/12	6/9	6/9	6/9	N8	N8	N6	N6	16	24	4	4	0.3	0.5	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	4	4	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
49	SHANTHALAKSHMI	75	F	884861	6/6	6/18	6/6	6/12	N10	N10	N8	N6	21	24	3	4	0.6	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	NO	4	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
50	PARVATHAMMA	64	F	885239	6/36	6/18	6/18	6/18	N10	N10	N8	N8	22	31	3	3	0.4	0.6	BODERLINE	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
51	PARVATHAMMA	52	F	885239	6/6	6/6	6/6	6/6	N10	N10	N6	N6	20	21	4	4	0.6	0.6	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	YES	4	4	SMOKER	DIABETIC	NONHTN	PRESENT
52	M KHAN	59	M	895401	6/36	6/36	6/6	6/6	N6	N6	N6	N6	21	20	3	4	0.7	0.3	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
53	KUMAR	82	M	896619	6/36	6/60	6/36	6/36	N8	N8	N6	N6	30	28	4	4	0.9	0.6	NOT DONE	NOT DONE	POAG	MYOPIA	NO	3	4	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
54	SIRAJUDDIN	57	M	897178	6/12	6/18	6/12	6/18	N8	N8	N6	N6	20	21	3	3	0.6	0.5	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	SMOKER	DIABETIC	NONHTN	PRESENT
55	KARTHIK	48	M	898657	CF 5 MTS	HM + VE	6/60	CF 2 MTS	N18	N0	N18	N0	20	22	3	4	0.5	0.6	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	NO	3	3	SMOKER	DIABETIC	HYPERTENSIVE	PRESENT
56	LAKSHMI B	58	F	899471	6/6	6/6	6/6	6/6	N8	N8	N6	N6	24	24	4	4	0.3	0.5	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	4	3	NONSMOKER	DIABETIC	HYPERTENSSIVE	ABSENT
57	LAXMAN REDDY	56	M	907710	6/36	6/36	6/18	6/36	N8	N8	N6	N6	21	27	3	3	0.3	0.5	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	YES	4	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
58	GOPAL RAO	50	M	922002	6/24	6/24	6/12	6/18	N12	N12	N6	N6	18	24	4	4	0.4	0.7	WITHIN NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	3	SMOKER	DIABETIC	NONHTN	ABSENT
59	NAGAMMA	55	F	928791	6/9	6/36	6/9	6/18	N8	N8	N6	N6	26	33	3	4	0.5	0.7	BODERLINE	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	3	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
60	RAMAPPA	40	M	932641	6/60	CF 5 MTS	6/12	6/18	N8	N8	N6	N6	24	21	3	3	0.3	0.5	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	4	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
61	IMAN KHAN	55	M	948771	6/12	6/36	6/9	6/36	N10	N8	N6	N6	31	25	3	4	0.5	0.7	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	4	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
62	ABDUL REHMAN	72	M	953349	6/60	6/60	6/36	6/60	N12	N12	N6	N6	22	24	3	3	0.5	0.8	BODERLINE	OUTSIDE NORMAL LIMITS	POAG	MYOPIA	YES	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
63	KRISHNAPPA	54	M	967758	6/6	6/6	-6/6	6/6	N6	N6	N6	N6	20	18	4	4	0.7	0.3	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
64	RAMAPPA	60	M	976210	6/6	6/6	6/6	6/6	N8	N8	N6	N6	20	26	4	3	0.4	0.7	BODERLINE	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-	3	4	NONSMOKER	DIABETIC	NONHTN	ABSENT
65	RAMAPPA	59	M	976210	6/60	6/12	6/12	6/6	N12	N12	N6	N6	16	24	4	4	0.8	0.8	BODERLINE	BODERLINE	SUSPECT	MYOPIA	NO	4		NONSMOKER	DIABETIC	NONHTN	PRESENT
66	MUNNPPAIYA	43	M	978593	6/60	CF3 MTS	6/36	CF 5 MTS	N18	N24	N18	N18	26	24	4	4	0.8	0.7	OUTSIDE NORMAL LIMITS	BODERLINE	POAG	MYOPIA	YES	4	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
67	JESEPHINE	54	F	987752	6/6	6/18	6/6	6/12	N10	N10	N8	N6	21	22	3	3	0.5	0.7	WITHIN NORMAL LIMITS	BODERLINE	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
68	KRISHNAMURTHY	82	M	989760	CF 5 MTS	6/36	6/18	6/9	N6	N6	N6	N6	24	24	4	4	0.6	0.6	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
69	RAJA	51	M	1002420	6/9	6/6	6/6	6/6	N8	N8	N8	N6	21	22	4	3	0.5	0.8	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	EMMETROPIA	-		3	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
70	RAJA	77	M	1002420	6/24	6/36	6/18	6/18	N12	N12	N6	N8	17	23	3	3	0.8	0.5	BODERLINE	WITHIN NORMAL LIMITS	SUSPECT	MYOPIA	YES	4	4	NONSMOKER	DIABETIC	NONHTN	PRESENT
71	KAMANTH	40	M	1010651	6/36	6/36	6/9	6/9	N6	N6	N6	N6	27	32	4	4	0.4	0.3	WITHIN NORMAL LIMITS	WITHIN NORMAL LIMITS	SUSPECT	EMMETROPIA	-	3	3	NONSMOKER	DIABETIC	NONHTN	ABSENT
72	MD SARDAR	60	M	15666/12727	6/60	6/36	6/36	6/24	N8	N8	N6	N6	24	22	3	3	0.8	0.8	OUTSIDE NORMAL LIMITS	OUTSIDE NORMAL LIMITS	POAG	MYOPIA	NO	4	3	NONSMOKER	DIABETIC	NONHTN	ABSENT

MASTER CHART FOR PACG AND PACS

SL NO	NAME	AGE	SEX	OP NO	UCVA (RE)	UCVA (LE)	BCVA (RE)	BCVA(LE)	UCNV(RE)	UCNV(LE)	BCNV(RE)	BCNV(LE)	IOP(RE)	IOP(LE)	SHAFFERS (RE)	SHAFFERS(LE)	C:D (RE)	C:D(LE)	GHT(RE)	GHT (LE)	DIAGNOSIS	ERROR	HERICK	HERICK	SMOKING	DIABETIS	HTN	FAMILY HO
1	RAJAMMA	60	F	115920	6/60	6/36	CF 5MTS	6/60	N24	N24	N18	N18	27	32	0	1	0.7	0.7	NORMAL	NORMAL	PACG	EMMETROPIA	1	0	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
2	SHREERAMAIAH	65	M	422300	6/18	6/18	6/18	6/18	N12	N12	N6	N8	16	21	2	3	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	2	SMOKER	NONDIABETIC	HYPERTENSIVE	PRESENT
3	PAPAMMA	50	F	459430	6/36	6/18	6/36	6/18	N12	N6	N8	N8	42	36	2	1	0.8	0.7	NOT DONE	NORMAL	PACG	EMMETROPIA	1	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
4	NATARAJ	71	M	567890	6/36	6/36	6/18	6/36	N18	N8	N12	N8	38	31	2	2	0.6	0.6	NORMAL	BODERLINE	PACG	EMMETROPIA	0	1	NONSMOKER	NONDIABETIC	NONHTN	PRESENT
5	VENKATRAMMAPPA B.S	78	M	575462	6/60	6/36	6/36	6/36	N24	N24	N18	N18	34	28	0	1	0.6	0.6	NORMAL	BODERLINE	PACG	EMMETROPIA	0	0	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
6	PADMA	55	F	624677	6/36	6/18	6/36	6/18	N8	N8	N6	N6	44	32	0	1	0.7	0.6	NORMAL	NORMAL	PACG	EMMETROPIA	2	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
7	PADMAVATHI	67	F	637438	CF 5 MTS	6/36	CF 5MTS	6/36	N8	N8	N12	N8	32	28	1	2	0.7	0.6	NORMAL	NORMAL	PACG	EMMETROPIA	0	2	NONSMOKER	NONDIABETIC	NONHTN	PRESENT
8	BANU K	69	F	669929	6/18	6/60	6/12	6/36	N12	N6	N8	N6	38	32	1	1	0.5	0.7	BODERLINE	NORMAL	PACG	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
9	SEETHAMMA	63	F	707972	6/36	6/12	6/36	6/36	N6	N6	N8	N6	28	30	2	1	0.5	0.6	BODERLINE	NORMAL	PACG	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
10	RAMACHANDRAPPA	70	M	734023	6/24	6/60	6/12	6/36	N6	N12	N8	N8	30	30	0	1	0.8	0.6	NORMAL	BODERLINE	PACG	HYPERMETROPIA	1	0	NONSMOKER	DIABETIC	NONHTN	ABSENT
11	KARIYAPPA	48	M	742207	6/36	6/60	6/36	6/36	N8	N8	N8	N8	35	32	2	1	0.8	0.7	BODERLINE	NORMAL	PACG	EMMETROPIA	1	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
12	FARHANA KAUSAR	75	F	760994	6/60	6/36	6/36	6/18	N12	N12	N8	N8	34	30	1	0	0.7	0.8	NORMAL	NORMAL	PACG	HYPERMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
13	VIJAYLAKSHMI	79	F	783957	6/18	6/18	6/12	6/18	N10	N8	N18	N8	32	35	2	2	0.5	0.7	NORMAL	NORMAL	PACG	EMMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
14	PRABHAVATI	79	F	803362	6/18	6/18	6/18	6/12	N6	N6	N6	N6	30	31	2	1	0.7	0.8	BODERLINE	BODERLINE	PACG	HYPER,ETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	PRESENT
15	NASSERUNIISA	69	F	804615	6/36	6/36	6/24	6/18	N6	N6	N6	N6	36	38	1	0	0.7	0.4	NORMAL	NORMAL	PACG	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	HYPERTENSIVE	PRESENT
16	SHYLA	66	F	818251	6/18	6/36	6/24	6/24	N36	N8	N18	N10	26	27	2	2	0.6	0.7	NORMAL	NORMAL	PACG	HYPERMETROPIA	1	1	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
17	KAMMALAMMA	35	F	830584	6/9	6/6	6/6	6/6	N6	N12	N8	N6	20	17	2	1	0.5	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
18	SRINIVAS	45	M	835014	6/18	6/36	6/18	6/18	N8	N8	N6	N6	17	14	1	2	0.6	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
19	MALLIKA BEGUM	60	F	839820	6/36	6/18	6/36	6/24	N12	N12	N8	N8	27	34	0	1	0.6	0.8	NORMAL	NORMAL	PACG	EMMETROPIA	0	1	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
20	DAKSHYANI	83	F	840604	PL -VE	CF 3 MTS	PL -VE	CF 5 MTS	N0	N36	N0	N24	38	42	0	1	0.9	0.7	NOT DONE	NOT DONE	PACG	HYPERMETROPIA	0	0	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
21	GOVINDAGOWDA	38	M	841861	6/6	6/6	-6/6	6/6	N6	N6	N6	N6	16	20	2	1	0.5	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
22	GOPAL	53	M	848837	6/18	6/9	6/12	6/9	N8	N8	N6	N6	20	21	3	2	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	SMOKER	NONDIABETIC	NONHTN	PRESENT
23	MUNIRAJU	45	M	858825	6/9	6/12	6/9	6/6	N6	N6	N6	N6	14	21	2	3	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	SMOKER	NONDIABETIC	HYPERTENSIVE	PRESENT
24	BYRAPPA	65	M	862638	6/18	6/24	6/18	6/24	N12	N6	N12	N8	31	27	0	1	0.7	0.8	BODERLINE	NORMAL	PACG	EMMETROPIA	2	1	SMOKER	NONDIABETIC	NONHTN	PRESENT
25	SUBBAIAH	80	M	863621	6/9	6/9	6/6	6/6	N6	N6	N6	N6	18	18	2	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
26	VENKATESHAMMA	55	F	865735	6/18	6/12	6/18	6/9	N6	N12	N8	N6	20	21	2	2	0.5	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	2	NONSMOKER	nONDIABETIC	HYPERTENSIVE	PRESENT
27	TIPPAYAAMMA	76	F	870200	6/6	6/6	6/6	6/6	N10	N10	N6	N6	20	20	2	1	0.3	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
28	MUNIYAMMA	70	F	873777	6/6	6/6	6/6	6/6	N10	N10	N6	N6	20	21	3	2	0.5	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
29	LAKSHMI	82	F	874244	6/18	6/12	6/9	6/6	N10	N10	N6	N6	21	20	1	2	0.3	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
30	AMRAVATHI	35	F	874256	6/12	6/9	6/6	6/6	N6	N6	N6	N6	21	21	2	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
31	MUNIAMMA	70	F	877545	6/32	6/12	6/9	6/6	N6	N6	N6	N6	22	20	2	1	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	nONDIABETIC	HYPERTENSIVE	ABSENT
32	SATHYAVATHI	75	F	877915	6/9	6/6	6/6	6/6	N8	N8	N8	N6	16	14	3	1	0.6	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	2	NONSMOKER	DIABETIC	NONHTN	ABSENT
33	BHAGYAMMA	30	F	879508	6/12	6/24	6/9	6/18	N8	N8	N6	N6	21	16	2	3	0.4	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
34	VENKATAMMA	50	F	879508	6/12	6/6	6/12	6/6	N10	N10	N6	N6	21	16	1	2	0.7	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	DIABETIC	NONHTN	ABSENT
35	THIMMUKYA AMMA	68	F	880028	6/6	6/6	6/6	6/6	N6	N6	N6	N6	14	21	2	1	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
36	BESAMMA	65	F	880296	6/9	6/9	6/6	6/6	N6	N6	N6	N6	22	20	3	2	0.4	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
37	REEGBA	55	F	880367	6/6	6/9	-	-	N12	N12	N8	N8	14	12	2	1	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
38	Muniraj	30	F	880372	6/6	6/6	6/6	6/6	N8	N8	N6	N6	14	17	2	1	0.5	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	DIABETIC	NONHTN	ABSENT
39	NAJAMMA	75	F	880397	6/18	6/24	6/12	6/18	N6	N6	N6	N6	17	20	1	3	0.5	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
40	VENKATAMMA	70	F	880912	6/6	6/6	6/6	6/6	N6	N8	N6	N6	18	14	2	2	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
41	RAMAKKA	70	F	882162	6/9	6/12	6/9	6/6	N6	N6	N6	N6	16	15	2	2	0.3	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
42	CHAMUNDESWARI	35	F	882811	6/6	6/6	6/6	6/18	N8	N8	N6	N6	21	17	2	2	0.3	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
43	GANGULAMMA	64	F	883512	6/12	6/12	6/12	6/9	N18	N18	N6	N8	16	21	2	2	0.3	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
44	SASHIKALA	41	F	883905	6/36	6/18	6/18	6/12	N12	N12	N8	N6	21	22	2	2	0.3	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	DIABETIC	NONHTN	ABSENT
45	SUSHEELAMMA	40	F	883908	6/60	6/12	6/18	6/6	N12	N12	N6	N6	14	20	2	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	1	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
46	RAMAKKA	52	F	884022	6/6	6/18	6/6	6/12	N10	N10	N8	N6	21	16	3	2	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
47	SITHAMMA	38	F	884042	6/6	6/6	6/6	6/6	N6	N6	N6	N6	20	14	3	2	0.3	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
48	SEETHAMMA	65	F	884168	6/6	6/6	6/6	6/6	N10	N10	N6	N6	20	21	2	2	0.5	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	PRESENT
49	MD HUSSAIN	37	M	884334	6/24	6/24	6/18	6/18	N6	N6	N6	N6	14	20	1	2	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	1	SMOKER	NONDIABETIC	NONHTN	ABSENT
50	MUNIVENKATAPPA	75	M	884913	6/6	6/6	6/6	6/6	N8	N8	N6	N6	21	21	2	3	0.7	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	SMOKER	NONDIABETIC	NONHTN	PRESENT
51	NARAYANAMMA	65	F	884957	6/32	6/12	6/9	6/6	N6	N6	N6	N6	20	22	3	2	0.3	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
52	RAMYA	35	F	885015	6/6	6/6	6/6	6/6	N8	N8	N6	N6	18	20	1	1	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
53	RAMAKRISHNAPPA	80	M	885022	6/36	6/24	6/18	6/18	N10	N10	N6	N6	16	18	1	3	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	1	sMOKER	NONDIABETIC	NONHTN	ABSENT
54	MUNIYAMMA	70	F	885025	6/9	6/9	6/9	6/9	N12	N12	N6	N8	21	18	2	3	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
55	PARVATHAMMA	30	F	885039	6/9	6/6	6/6	6/6	N6	N6	N6	N6	24	20	3	2	0.4	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
56	RANIAMMA	75	F	885239	6/24	6/12	6/18	6/9	N10	N10	N6	N6	14	21	2	2	0.5	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
57	MALLAMMA	70	F	885242	6/9	6/9	6/9	6/9	N12	N12	N6	N8	20	21	2	3	0.5	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	PRESENT
58	GOPAMMA	48	F	885264	6/18	6/12	6/9	6/6	N10	N10	N6	N6	21	19	2	1	0.5	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
59	MANJULA	60	F	885303	6/6	6/6	6/6	6/6	N6	N6	N6	N6	18	21	2	1	0.3	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
60	RENUKA	70	F	885303	6/6	6/6	6/6	6/6	N10	N10	N6	N6	14	18	3	2	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
61	SONNAMMA	65	F	885305	6/24	6/18	6/18	6/9	N10	N10	N6	N8	20	21	3	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	2	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
62	GOVINDAMMA	65	F	885312	6/9	6/6	6/9	6/6	N8	N8	N6	N6	26	21	1	3	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
63	LAKSHAMMA	70	F	885426	6/9	6/9	6/9	6/9	N12	N12	N6	N8	21	21	1	2	0.5	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
64	RAMMANA	40	F	885512	6/6	6/6	6/6	6/6	N6	N6	N6	N6	15	21	2	2	0.5	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
65	KOMALA	46	F	885529	6/24	6/18	6/18	6/6	N10	N10	N6	N8	17	20	2	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPER,ETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
66	NAGAMMA	80	F	885562	6/9	6/9	6/9	6/9	N10	N6	N6	N6	15	21	2	2	0.3	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
67	KRISHNAPPA	45	F	885563	6/6	6/6	6/6	6/6	N12	N12	N6	N8	21	20	1	1	0.6	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	DIABETIC	NONHTN	ABSENT
68	RAMADEVI	30	F	885630	6/9	6/12	6/9	6/9	N6	N8	N6	N6	18	14	2	3	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	DIABETIC	NONHTN	ABSENT
69	KAMALAMMA	42	F	885871	6/6	6/6	6/6	6/6	N6	N8	N6	N6	16	20	1	2	0.3	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
70	MAHALAKSHMI	78	F	885879	CF 4 MTS	6/24	CF 5MTS	6/18	N12	N6	N18	N12	29	34	2	0	0.7	0.8	NORMAL	NORMAL	PACG	EMMETROPIA	0	0	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
71	SAROJAAMMA	60	F	885948	6/12	6/9	6/9	6/9	N8	N8	N6	N6	20	21	2	2	0.6	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
72	SEETHAMMA	60	F	886080	6/12	6/6	6/6	6/6	N8	N8	N6	N6	21	16	1	3	0.7	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
73	MUNIYAMMA	70	F	886241	6/6	6/6	6/6	6/18	N8	N8	N6	N6	17	21	1	2	0.5	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
74	MANGAMMA	40	F	886247	6/6	6/6	6/6	6/6	N8	N8	N6	N6	26	14	2	2	0.3	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
75	NARAMMA	65	F	886305	6/12	6/6	6/6	6/6	N8	N8	N6	N6	20	16	3	2			MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
76	NARAMMA	65	F	886308	6/6	6/6	6/6	6/6	N8	N8	N6	N6	16	18	2	3	0.5	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
77	MUNIVABEGUM	55	F	886310	6/6	6/6	6/6	6/6	N6	N8	N6	N6	20	16	2	3	0.5	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
78	VENKATAMMA	70	F	886312	6/6	6/6	6/6	6/6	N6	N6	N6	N6	21	18	1	2	0.6	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
79	RAMAPPA	37	M	886313	6/6	6/6	6/6	6/6	N10	N10	N6	N6	14	20	2	3	0.5	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	SMOKER	NONDIABETIC	HYPERTENSIVE	PRESENT
80	SRINATH	45	M	886344	6/6	6/6	-6/6	6/6	N6	N6	N6	N6	17	22	2	1	0.6	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	SMOKER	NONDIABETIC	NONHTN	PRESENT
81	SOMAMAMMA	62	F	886367	6/12	6/9	6/6	6/6	N6	N6	N6	N6	12	14	1	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
82	BAKKAPPA	60	M	886826	6/6	6/9	6/6	6/6	N10	N10	N8	N6	14	20	3	1	0.4	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	SMOKER	NONDIABETIC	NONHTN	PRESENT
83	PRASANNA	43	M	886976	6/9	6/6	6/9	6/6	N8	N8	N6	N6	17	16	2	2	0.6	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
84	MATENULLA SHARIFF	47	M	887186	6/18	6/12	6/9	6/6	N10	N10	N6	N6	16	20	2	2	0.3	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
85	VIJAYKUMAR	56	M	887194	6/9	6/6	6/6	6/6	N6	N6	N6	N6	14	21	1	2	0.3	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	PRESENT
86	SRIDHAR	56	M	887211	6/6	6/9	6/6	6/6	N10	N10	N8	N6	17	21	2	1	0.5		MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	DIABETIC	NONHTN	ABSENT
87	Govindagowda	34	M	887216	6/12	6/9	6/6	6/6	N6	N6	N6	N6	21	20	1	2	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
88	KRISHNAPPA	55	M	887232	6/6	6/6	6/6	6/6	N12	N12	N6	N8	14	17	3	3	0.3	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	sMOKER	NONDIABETIC	NONHTN	ABSENT

89	SHANMUGAM	32	M	887241	6/6	6/9	6/6	6/6	N10	N10	N8	N6	20	14	2	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	NONSMOKER	NONDIABETIC	NONHTN	PRESENT
90	HAIDERSAAB	65	M	887336	6/9	6/12	6/12	6/9	N6	N6	N6	N6	20	16	2	3	0.5	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	DIABETIC	NONHTN	ABSENT
91	LAKSHAMMA	65	M	887483	6/12	6/9	6/6	6/6	N6	N6	N6	N6	18	21	2	1	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	SMOKER	DIABETIC	HYPERTENSIVE	PRESENT
92	NAGAMMA	62	F	887843	6/9	6/6	6/6	6/6	N6	N12	N8	N6	17	15	1	1	0.5	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	1	NONSMOKER	DIABETIC	HYPERTENSIVE	ABSENT
93	VENKATAMMA	56	F	888384	6/9	6/12	6/9	6/6	n10	N10	N6	N6	16	20	2	2	0.4	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	1	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
94	FAMANA	43	F	888926	6/6	6/6	6/6	6/6	N6	N6	N6	N6	19	17	3	1	0.3	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
95	SUBDARAMMA	75	F	889866	6/60	6/12	6/12	6/6	N12	N12	N6	N6	20	21	2	1	0.3	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
96	KADIRAMMA	30	F	889871	6/6	6/6	6/6	6/18	N8	N8	N6	N6	21	12	2	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	DIABETIC	HYPERTENSIVE	PRESENT
97	MUNIVENKATAMMA	65	F	890132	6/6	6/6	6/6	6/6	N6	N8	N6	N6	21	17	3	2	0.4	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
98	KONAMMA	50	F	890325	6/9	6/6	6/6	6/6	N6	N6	N6	N6	22	20	2	3	0.5	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	HYPER,ETROPIA	2	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
99	MUNISA	54	F	890372	6/18	6/12	6/18	6/12	N12	N8	N8	N8	21	14	2	2	0.5	0.5	MAL LIMITS	NORMAL	PACG SUSPECT	HYPERMETROPIA	1	1	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
100	SARDAR	62	M	895109	6/18	6/36	6/18	6/18	N18	N12	N8	N8	31	44	2	1	0.6	0.5	BODERLINE	NORMAL	PACG	EMMETROPIA	1	1	SMOKER	DIABETIC	HYPERTENSIVE	PRESENT
101	MALLIK KHAN	61	M	895401	CF 5 MTS	6/36	CF 5MTS	6/36	N12	N6	N8	N6	32	29	2	1	0.7	0.8	BODERLINE	NORMAL	PACG	EMMETROPIA	0	1	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
102	MUNNILAKSHAMMA	70	F	899208	6/60	6/60	CF 3MTS	CF 5 MTS	N18	N8	N12	N6	30	38	1	2	0.8	0.6	NORMAL	NORMAL	PACG	EMMETROPIA	1	0	NONSMOKER	DIABETIC	NONHTN	PRESENT
103	LAKSHMI	60	F	899948	6/36	6/36	6/36	6/36	N6	N6	N6	N6	30	38	1	0	0.6	0.8	NORMAL	NORMAL	PACG	HYPERMETROPIA	1	2	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
104	MUNIYAPPA	50	M	917982	6/9	6/6	6/6	6/6	N6	N6	N6	N6	21	21	2	1	0.6	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	SMOKER	DIABETIC	HYPERTENSIVE	ABSENT
105	NAGAMMA	72	F	928791	CF 5 MTS	PL -VE	6/60	PL -VE	N36	N0	N18	N0	36	32	1	0	0.8	0.8	NORMAL	NOT DONE	PACG	EMMETROPIA	0	0	NONSMOKER	NONDIABETIC	NONHTN	ABSENT
106	NARAYANAPPA	68	M	932569	6/6	6/6	6/6	6/6	N10	N10	N6	N6	20	20	3	1	0.3	0.6	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	NONDIABETIC	HYPERTENSIVE	ABSENT
107	ANURADHA	55	F	945712	6/60	CF 5 MTS	6/36	CF 5 MTS	N8	N6	N8	N6	30	34	0	1	0.4	0.9	NORMAL	BODERLINE	PACG	EMMETROPIA	1	0	nonSMOKER	NONDIABETIC	NONHTN	ABSENT
108	ANITHA	54	F	949085	6/24	6/36	6/18	6/36	N36	N8	N24	N10	30	25	2	2	0.6	0.8	BODERLINE	NORMAL	PACG	HYPERMETROPIA	1	1	NONSMOKER	NONDIABETIC	HYPERTENSIVE	PRESENT
109	SRINIVASA	65	M	976537	6/18	6/9	6/12	6/9	N8	N8	N6	N6	18	21	3	2	0.3	0.4	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	1	2	NONSMOKER	DIABETIC	NONHTN	ABSENT
110	SONAPPA	60	M	990455	6/6	6/6	6/6	6/6	N10	N10	N6	N6	20	20	1	2	0.5	0.7	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	1	sMOKER	NONDIABETIC	NONHTN	ABSENT
111	SARAT	60	M	990736	6/6	6/6	6/6	6/6	N12	N8	N8	N8	18	14	2	1	0.5	0.3	MAL LIMITS	NORMAL	PACG SUSPECT	EMMETROPIA	2	2	sMOKER	NONDIABETIC	NONHTN	ABSENT
112	KAMANTH	58	M	1010659	CF 4	6/36	6/60	6/36	N18	N6	N12	N8	32	28	2	1	0.5	0.9	NORMAL	NORMAL	PACG	HYPERMETROPIA	1	0	SMOKER	NONDIABETIC	NONHTN	ABSENT