"A CROSS SECTIONAL STUDY TO ASSESS THE CORRELATION AMONGST VISUAL ACUITY, CONTRAST SENSITIVITY AND VISUAL FIELD ANALYSIS IN PATIENTS WITH GLAUCOMA"

By

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Dissertation submitted to

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

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

Under the guidance of

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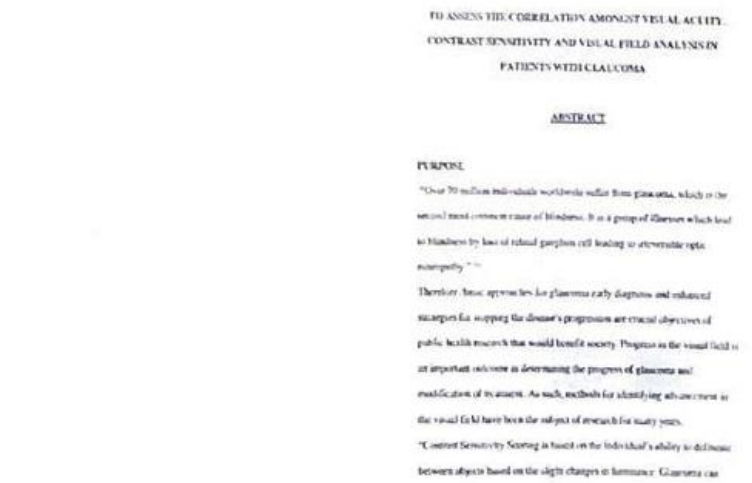

TO ASSESS THE CORRELATION AMONGST VISUAL ACUITY, CONTRAST SENSITIVITY AND VISUAL FIELD ANALYSIS IN PATIENTS WITH GLAUCOMA

ABSTRACT

PURPOSE

"Over 70 million individuals worldwide suffer from glaucoma, which is the second most common cause of blindness. It is a group of illnesses which lead to blindness by loss of retinal ganglion cell leading to irreversible optic neuropathy." "

Therefore, basic approaches for glaucoma early diagnosis and enhanced strategies for stopping the disease's progression are crucial objectives of public health research that would benefit society. Progress in the visual field is an important outcome in determining the progress of glaucoma and modification of its course. As such, methods for identifying advancement in the visual field have been the subject of research for many years.

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LIST OF ABBREVATIONS

Abbreviation	Full Form
CS	Contrast Sensitivity
RGC	Retinal ganglionic cell
HFA	Humphrey Field Analyser
IOP	Intra Ocular Pressure
MD	Mean Deviation
ONH	Optic Nerve Head
PACG	Primary Angle Closure Glaucoma
PEG	Pseudo exfoliation Glaucoma
POAG	Primary Open Angle Glaucoma
OCT	Optical Coherence Tomography
VF	Visual Field

ABSTRACT

PURPOSE

Over 80 million individuals worldwide suffer from glaucoma, which is the second most common cause of blindness. It is a group of illnesses which lead to blindness by loss of retinal ganglion cell leading to irreversible optic neuropathy.

Therefore, basic approaches for glaucoma early diagnosis and enhanced strategies for stopping the disease's progression are crucial objectives of public health research that would benefit society. Progress in the visual field is an important outcome in determining the progress of glaucoma and modification of treatment. As such, methods for identifying advancement in the visual field have been the subject of research for many years.

Contrast Sensitivity Scoring is based on the individual's ability to delineate between objects based on the slight changes in luminance. Glaucoma can reduce contrast sensitivity for all grating sizes, mostly at the middle spatial frequencies.

Visual Acuity testing involves the assessment of central vision hence other modalities of investigation are required in order to identify glaucoma at the earliest and supplement its treatment.

METHODS

At Sri Devaraj Urs Medical College, 41 patients in the ophthalmology department were evaluated as part of the study. Patients who met the requirements for study had their visual acuity assessed by using Snellens visual acuity chart , the obtained value was converted into a LogMAR value. By using Pelli Robson contrast sensitivity chart, the contrast Sensitivity was measured at a distance of 1 Meter.

Goldmann Applanation Tonometry, 4-mirror gonioscopy, and slit-lamp biomicroscopy were

used to evaluate the anterior segment. Fundus lens + 90D used for optic disc inspection. After dilating the pupil, slit lamp biomicroscopy using Fundus lens and indirect ophthalmoscopy was used to determine the extent of damage, type and severity of glaucoma .The Humphrey visual field analyzer 24- 2 program was used to plot the patients' visual fields. SITA standard program was used. A measure called mean deviation, or MD, was employed to assess visual field function. A fault is considered early if its mean deviation is less than -6 dB, moderate if it is less than -12 dB, and severe if it is greater than -12 dB. Patients with the typical changes in the visual field and with optic nerve head changes, were diagnosed with glaucoma .If there are questionable optic nerve alterations but no discernible loss in visual field the patient was suspected of having glaucoma; if intraocular pressure (IOP) is 21 mm Hg or higher but there is no discernible loss in visual field or change in the optic nerve head, the patient is suspected of having ocular hypertension.

RESULTS:

The study in total included 41 people as sample. The age distribution revealed that 51–60 age the group was the most prevalent (48.8%), and that men slightly outnumbered women (56.1% vs. 43.9%). The largest occupational category was made up of clerks (39.0%), followed by tailors (17.1%). There was variation in visual acuity, with the highest acuity of 0.3 in both eyes.

Results of the analysis of the anterior segment data were primarily within normal limits (WNL). Normal tension glaucoma (NTG) (22.0%) was the second most frequent type of glaucoma, after primary open-angle glaucoma (46.3%). The mean values of intraocular pressure (IOP) were found to be 24.71 mmHg for the right eye and 17.71 mmHg for the left, and the mean values of the Humphrey Visual Field Analyzer (HFA) mean deviation (MD) -10.61 dB for the eye right and -8.33 dB for the eye left according to descriptive statistics.

There is no clear relationship between contrast sensitivity and visual acuity for the right eye. The Pearson correlation coefficient between the CS score and the visual field analysis, a high positive link between the two with a P value of less than 0.001, indicating (HFA MD) is 0.613.

Regarding the left eye: There is no direct association seen between the CS score and visual acuity. A statistically significant positive association between the CS score and visual field analysis (HFA MD) in the left eye is provided by the correlation Pearson coefficient of 0.342 and value P of 0.029 between the CS score and HFA MD.

In order to minimize the impact of additional variables, the study was restricted to individuals with an excellent visual acuity score of (6/12) or higher. Eye disorders such as cataracts and other ocular conditions such as retinopathy ,age-related and diabetic maculopathy were excluded.

KEYWORDS:

Optic Nerve Head , Retinal Ganglion cell ,Primary Open Angle Glaucoma , Primary Angle Closure Glaucoma , Pseudoexfoliation Glaucoma ,Intra Ocular Pressure ,Humphrey Field Analyser, Visual Field,Contrast sensitivity.

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INTRODUCTION

INTRODUCTION

Glaucoma, a neurodegenerative disease encompassing a diverse range of age-related disorders, poses a significant global health challenge. Characterized by progressive optic neuropathy, it ranks as the second most common cause of blindness worldwide and the primary cause of irreversible blindness. Despite its widespread prevalence, the precise etiopathogenesis of glaucoma remains incompletely understood, highlighting the complexity of this condition.

Current estimates indicate that approximately 80 million individuals worldwide are affected by glaucoma, with 8.4 million experiencing bilateral blindness. While increased intraocular pressure (IOP) is frequently implicated in retinal ganglion cell degeneration, recent research has unveiled additional contributors to glaucomatous optic neuropathy. These include impaired ocular blood flow, cytokine dysregulation, and structural changes in blood vessels, underscoring the multifactorial nature of the disease.

Although glaucoma-related vision loss is irreversible, early detection and proper management can prevent blindness in most cases. The primary therapeutic approach focuses on reducing intraocular pressure through topical medications, laser therapy, or surgical interventions. However, emerging evidence suggests that targeting various risk factors contributing to glaucomatous neuropathy may offer a more effective treatment strategy.

Addressing factors such as ocular blood flow abnormalities, inflammatory cytokines, and vascular changes could potentially halt or slow the progression of optic nerve damage in glaucoma. This broader approach to treatment aims to preserve visual function and quality of life for affected individuals.

In conclusion, glaucoma presents a complex challenge requiring a multifaceted approach to diagnosis and management. By expanding our understanding of the underlying mechanisms driving the disease and adopting targeted interventions to address these factors, we can improve outcomes and reduce the burden of glaucoma-related blindness on a global scale.

AIMS AND OBJECTIVES

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AIM

To Assess the correlation amongst Visual Acuity, Contrast Sensitivity and Visual Field Analysis in patients with Glaucoma.

OBJECTIVE

To Assess the correlation amongst Visual Acuity, Contrast Sensitivity and Visual Field Analysis in patients with Glaucoma with Snellen visual Acuity Chart, Pelli Robson Contrast Sensitivity chart and Humphrey Visual Field Analyser.

REVIEW OF

LITERATURE

Glaucoma's historical roots trace back to ancient Greece, where it was mentioned in literature as early as 400 BC. However, it wasn't until the 19th century that it was distinctly recognized as a group of ocular disorders. One fundamental aspect of glaucoma is its heterogeneous nature it isn't a singular disease but rather a diverse spectrum of disorders. These conditions exhibit a wide range of clinical and histopathological manifestations, with varying pathophysiological mechanisms ultimately leading to the loss of retinal ganglion cells (RGCs) and optic atrophy (OA). This complexity underscores the challenge in understanding and effectively managing glaucoma, requiring a comprehensive approach tailored to the specific characteristics and needs of each patient.[1]

This condition leads to irreversible loss of retinal ganglion cells and subsequent visual dysfunction. This definition highlights the primary features of glaucoma, emphasizing the progressive nature of the optic nerve damage and the resulting impact on visual function. [1]

Glaucoma ranks as the second most prevalent cause of blindness globally and stands as the leading cause of irreversible visual impairment, making it a significant public health concern. Referred to as the "silent killer of vision," glaucoma often progresses gradually over an extended period without noticeable symptoms, often being identified only when the disease has advanced significantly. This progressive loss of vision is irreversible, highlighting the urgency of early detection and intervention.

While various subtypes of glaucoma exist, damage to the optic nerve serves as the ultimate common pathway, influenced by a diverse array of etiological factors and clinical presentations. [1]

Despite decades of extensive research, the pathogenesis of glaucoma remains incompletely understood to date. While elevated intraocular pressure (IOP) is a major risk factor, the study of glaucoma primarily focuses on its consequences.

. Therefore, a thorough understanding of aqueous humor production and outflow dynamics is crucial. The delicate balance between the secretion of aqueous humor by the ciliary body and its drainage through the trabecular meshwork and uveoscleral outflow pathways determines IOP levels.

In open-angle glaucoma, increased resistance to aqueous outflow through the trabecular meshwork is observed. Conversely, angle-closure glaucoma is characterized by obstruction of access to the drainage pathways. Despite significant advancements, much remains to be elucidated regarding glaucoma, not only in terms of its pathogenesis but also its systemic associations and influences. Continued research efforts are essential to unravel the complexities of this sight-threatening condition and develop more effective management strategies.

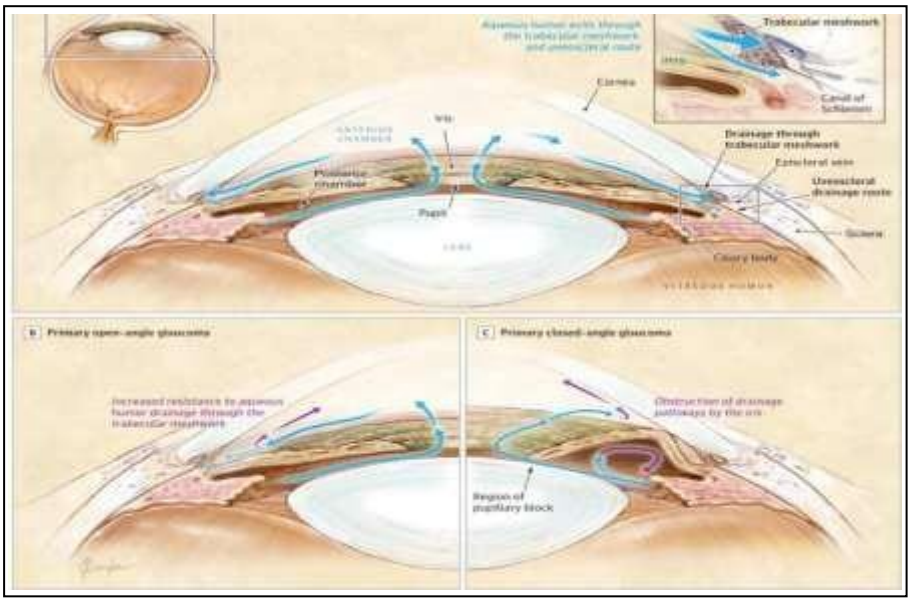


Fig1:Pathway of Aqueous outflow

Elevated intraocular pressure exerts stress and mechanical strain on posterior eye structures, particularly the lamina cribrosa, known as the eye's weakest point under pressure. Situated where retinal ganglion cell axons exit, the lamina cribrosa is perforated in the sclera. Increased pressure may lead to compression, remodeling, and deformation of this structure, causing mechanical damage to axons, disruption of axonal transport, and accelerated death of retinal ganglion cells.

Glaucoma's impact is significant, affecting over 80 million individuals worldwide, with an estimated 6.6 million, or 10%, experiencing blindness.[2]

In 2010, the global incidence of Open Angle Glaucoma (OAG) was recorded at 1.96%. Among various ethnic groups, Europeans exhibited the highest absolute number of individuals affected by OAG. However, the regional prevalence was notably highest among individuals of African descent. Notably, the prevalence of OAG among Indian, European, and Chinese populations was relatively lower and similar to each other.

In contrast, the worldwide incidence of Angle Closure Glaucoma (ACG) was lower, standing at 0.69%. However, the regional prevalence differed, with a higher prevalence observed among the Chinese and Southeast Asian populations, followed by the Indian population.

Understanding these demographic patterns is crucial for targeted screening, diagnosis, and management strategies tailored to the specific needs of diverse populations. [3]

PATHOPHYSIOLOGY OF GLAUCOMA

Since the mid-19th century, the pathogenesis of glaucomatous optic atrophy has been a subject of intense study and debate among researchers and clinicians. Two contrasting concepts have been pivotal in shaping our understanding: In 1985, Muller introduced the

mechanical theory, suggesting that elevated intraocular pressure (IOP) directly compresses and leads to neuronal death. Conversely, Von Jaeger proposed the vascular theory, that states that optic atrophy stems from vascular abnormalities. These divergent theories have underscored the complexity of glaucoma's underlying mechanisms and continue to drive research efforts aimed at unraveling the precise pathophysiology of this sight-threatening condition. [4]

In 1892, presence of empty spaces, "Schnabel" introduced another concept regarding the pathogenesis of glaucomatous optic atrophy, suggesting that occurs due to the atrophy of neural elements.

In 1968, the role of axoplasmic flow in glaucomatous optic atrophy was highlighted. Initially, it was hypothesized that the loss of astroglial supportive tissue precedes neuronal loss, offering an explanation for the early and reversible cupping observed in infants.[5]However, subsequent studies have indicated that glial cells are not selectively lost in early glaucoma but persist as remaining cells after axon loss in advanced cases.[6]

Glaucomatous optic neuropathy develops due to a variety of factors, both intrinsic and extrinsic. Elevated intraocular pressure (IOP) is considered a significant risk factor and plays a pivotal role in the development of glaucomatous optic neuropathy in most cases. [7, 8]

Several factors, primarily acting on cell bodies or their axons, are believed to contribute to the death of retinal ganglion cells (RGCs). According to various proposed theories, factors such as elevated intraocular pressure (IOP) and vascular insults during glaucomatous atrophy

dysregulate the normal cellular processes within the retina, leading to neuronal damage and cell death. [9] This insult manifests as obstruction within the microcirculation of retinal ganglion cell (RGC) axons at the lamina cribrosa. It involves alterations corresponding to lamina level changes, including modifications in laminar glial and connective tissue. [10]

Additionally, secondary insults are caused by excitotoxic damage from glutamate or glycine released from injured neurons, as well as oxidative damage resulting from the overproduction of nitric oxide (NO) and other reactive oxygen species contributes to the complex pathogenesis of glaucomatous optic atrophy, highlighting the multifactorial mechanisms. [11] Further research is essential to elucidate the underlying processes and develop targeted therapeutic interventions. [61]

The development of glaucomatous optic neuropathy:Two hypotheses, the mechanical and ischemic theories, have been proposed to elucidate .[1]

The mechanical theory underscores the direct compression of axonal fibers and adjacent supporting structures of the anterior optic nerve by elevated intraocular pressure (IOP). This compression leads to distortion of the lamina cribrosa plates, impairing axoplasmic flow and ultimately resulting in the death of retinal ganglion cells (RGCs).[12]

In contrast, the ischemic theory emphasizes the role of intraneural ischemia, which occurs due to reduced blood flow to the optic nerve. This decrease in perfusion is attributed to the pressure exerted on the optic nerve's blood supply by elevated intraocular pressure (IOP). Additionally, disruption of vascular autoregulation can worsen the condition, leading to nerve damage. It is noteworthy that glaucomatous optic neuropathy can also occur in

individuals with normal intraocular pressures. This phenomenon may be explained by abnormally low levels of cerebrospinal fluid pressure in the optic nerve's subarachnoid space, creating a significant pressure gradient across the lamina cribrosa.[13]

Other contributing factors to glaucoma include faulty microcirculation, decreased immunity, excitotoxicity, and oxidative stress. These multifaceted mechanisms underscore the complexity of glaucoma pathogenesis and highlight the importance of further research to uncover novel therapeutic targets and interventions. [62, 63]

DIAGNOSIS OF GLAUCOMA

Lowering intraocular pressure (IOP) is the primary treatment objective, as it is the only modifiable key factor that can potentially prevent further progression of optic nerve damage. Goldman's Applanation Tonometry (GAT) stands out as the most accurate method for measuring IOP, unaffected by scleral rigidity. [14]

Gonioscopy serves as the gold standard for assessing the anterior chamber angle, a crucial step in distinguishing between open-angle glaucoma and angle-closure glaucoma. This diagnostic tool provides essential information for guiding treatment decisions.[15]

Additionally, stereoscopic biomicroscopic evaluation of the optic nerve head plays a pivotal role in identifying typical glaucomatous optic disc changes. This meticulous assessment aids clinicians in evaluating disease severity and progression, facilitating timely intervention and management strategies.[16]

PERIMETRY

Perimetry serves as a valuable tool for assessing visual field function. It provides insight into

the area perceived by the steady fixating eye. [17]

Traquair aptly described the visual field as an island of vision amidst darkness, with the peak of this hill located at the fovea, where maximal sensitivity is observed. This analogy vividly captures the central role of the fovea in visual perception and the spatial distribution of visual sensitivity across the field.[18]

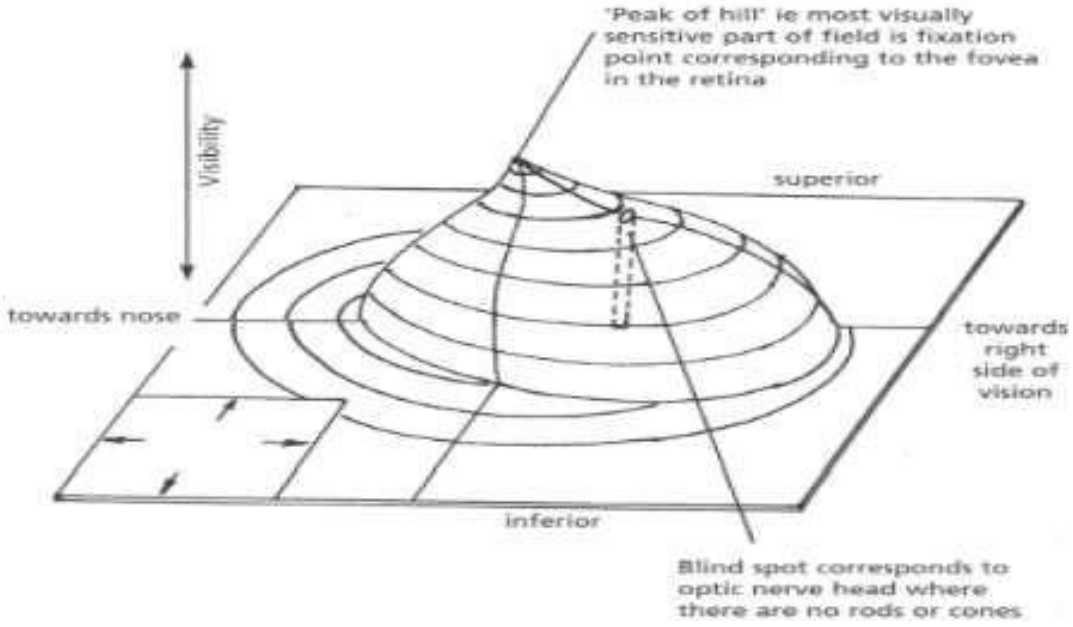


Fig2: Traquair Hill of Vision

Perimetry plays a crucial role in both the diagnosis and management of glaucoma, offering valuable insights into visual field function. It enables clinicians to identify relative defects, indicating decreased sensitivity compared to normal, as well as absolute defects, representing areas where the maximum stimulus is not perceived. These findings provide essential information for assessing disease severity, monitoring progression, and guiding treatment strategies. By accurately characterizing visual field abnormalities, perimetry enhances our understanding of glaucoma and facilitates targeted interventions to preserve visual function and quality of life for affected.[19]

History of Perimetry

In 1889, Bjerrum revolutionized visual field testing by implementing a tangential screen on the back door of his clinic. This innovation, later referred to as the Tangent screen in the USA, remained a cornerstone of testing methodologies for nearly a century.[20]

In 1983, Groenouw's introduction of isopters, delineating points with equal light sensitivity, marked another leap forward in visual field assessment.[21]

In 1909, Dr. Ronne advanced perimetry with the development of kinetic isopters, offering new insights such as the identification of the nasal step in glaucoma.

In 1945, Dr. Goldman's creation of the first cupola perimeter for manual kinetic perimetry further enhanced diagnostic capabilities in ophthalmology.

In 1972, Dr. Fankhauser and colleagues in Bern, Switzerland, laid the groundwork for automated perimetry with their pioneering research. [22] This culminated in the development of the first standard automated perimeter, OCTOPUS 201, in 1976, ushering in a new era of precision and efficiency in visual field testing.[30]

There are two primary types of perimetry: static and kinetic.[25,26]

FAST PAC

It is the reduced threshold time by 40% and using the same bracketing technique as full threshold. Instead of 4 dB, 3 dB steps are used in FAST PAC.

Swedish Interactive Threshold Algorithm (SITA)

It stands as the swiftest and most prevalent approach, slashing testing durations significantly. Offered in two variants: SITA Standard or SITA Fast, it employs an alternative methodology in lieu of the bracketing technique. [27]

It employs an intricate model to gauge threshold values for individual points, drawing from responses to stimuli presented at the specific location as well as from neighboring points.[28]

Refractive correction must precede the test initiation. Ensure proper placement of lenses within the lens holder; misplacement farther from the eye may induce a spurious defect, often manifesting as a circular field loss in the periphery. For evaluating the central field, trial lenses are suitable, whereas for assessing the peripheral field beyond 30 degrees, remove the trial lenses. [26, 27]

Pupil Diameter

While a large pupil size typically doesn't influence results, miotic pupils may generate defects. To prevent false positives, ensure the pupil is at least 3mm in diameter. [28]

RELIABILITY INDICES

This suggests the patient has consistently fixated on the target. If the blind spot serves as the fixation monitor, intermittent stimuli will be projected to the blind spot region. If the individual perceives these stimuli, it's deemed a fixation loss. [26, 27, and 29]. 0/19, the first number shows the number of fixation loss followed by thenumber of stimulus given to blind spot. Fixation loss > 20 % and fixation loss with XX are indicators of poor fixation [29]

False Positive

A false positive occurs when a patient responds in the absence of stimuli.

If the false positive rate exceeds 33%, it suggests unreliability in the field test or indicates patient inattention. [23, 2,25]

False negative

A false negative occurs when the individual fails to perceive presented brighter stimuli. If the false negative rate surpasses 33%, it signifies test unreliability and is also observed in advanced glaucomatous disease. [27]

Numeric data -It's depicted as the patient's response in decibels (dB).

Total Deviation Probability Plots

The total deviation plots highlight regions of the visual field that deviate from the normal range expected for the patient's age. Visual field defects are quantified by the percentage of normal subjects who would typically exhibit such sensitivity. For instance, a symbol with a probability of p< 5% suggests that fewer than 5% of normal subjects would demonstrate similarly low sensitivity [23, 27]

Pattern Deviation Probability Plots

The pattern deviation plots reveal localized defects once the generalized field loss attributed to cataracts or a small pupil has been corrected. They accentuate areas deviating from the expected normal range.

Numerical Plot-contain decibel values corresponding to the total and pattern deviation probability plots.[30]

Gray scale Printouts

The scheme of the visual field reprinted by gray scale colour. The gray scale is

particularly valuable in that caused by trial lens defects and identifying artifact-induced field loss, such as and false positive responses. [.30]Darker shades signify reduced sensitivity within the field. It offers a comprehensive overview of visual field status, including the severity and extent of visual field loss.

Gray scale Printouts

The gray scale represents the color scheme of the visual field. Darker shades signify reduced sensitivity within the field. It offers a comprehensive overview of visual field status, including the severity and extent of visual field loss. The gray scale is particularly valuable in identifying artifact-induced field loss, such as that caused by trial lens defects and false positive responses.[.30]

Visual Field Indices:

It aids in evaluating the average deviation of a patient's overall function from that of age-matched normals. Serving as a general barometer of overall field depression, it reflects the extent of deviation from normal functioning. A generalized depression results in a notable increase in the mean defect. A value of 0 denotes normal functioning with no deviation from the norm, while increased negative values indicate greater deviation from normal functioning.[31]

Pattern standard deviation (PSD) –

It provides a measurement of the extent to which the shape of the patient's field diverges from the normal age-matched field. A high PSD suggests an irregular hill of vision, whereas a low PSD indicates a smooth hill of vision.

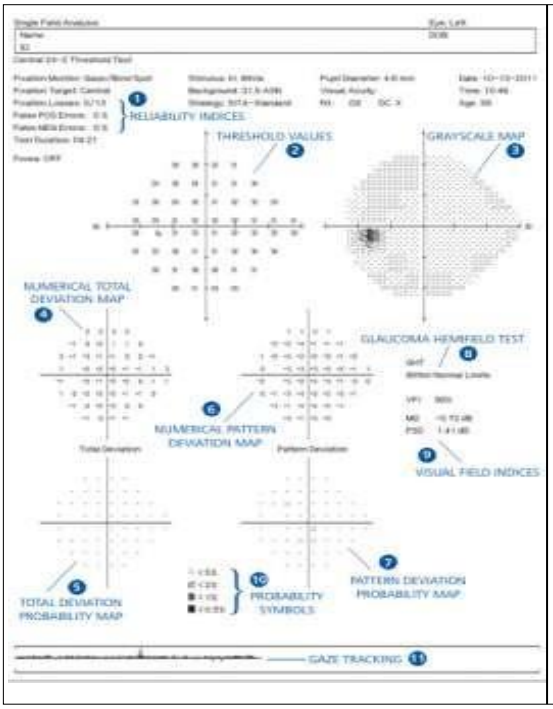


Fig 2: Normal HFA 30-2 of a 65 yr oldpatient

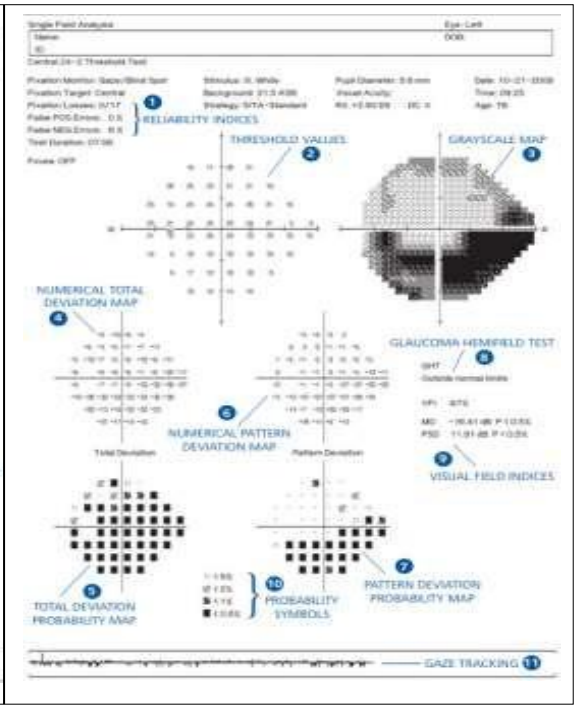


Fig 3:HFA 30-2 with a probable glaucomatous field

The H-P-A classification system is a clinically valuable method that assesses the overall extent of visual field damage using both the MD value and the number of defective points in the Humphrey Statpac-2 pattern deviation probability map of the 24-2, SITA-STANDARD test. This method also considers the proximity of defects to fixation.

This criterion allows for the detection of subtle nerve damage, aiding in the early diagnosis of glaucoma.

It is utilized to gauge the severity of glaucoma based on mean deviation (MD). An MD value less than -6dB is classified as an early defect, MD less than -12dB as moderate, and MD greater than -12dB as severe defect.

HODAPP'S-PARRISH- ANDERSON CLASSIFICATION.

Figure 4- HODAPP'S- PARRISH- ANDERSON CLASSIFICATION

Stage	Humphrey MD score	Additional Criteria at least 1 of the listed criteria must apply)
Stage 0: No or Minimal Defect		
Stage 1: Early Defect	≥ -6.00 dB	- a cluster of ≥ 3 points on the pattern deviation plot in an expected location of the visual field depressed below the 5% level, at least one of which is depressed below the 1% level - CPSD/PSD significant at P<00.5 - GHT Outside Normal Limits""
Stage 2: Moderate Defect	≥ -6.00 to -12.00 dB	- ≥ 25% but <50% of points on the attern deviation plot depressed below the 5% level, and ≥15% but <25% of points deprese below the 1% level - at least 1 point within the central 5° with sensitivity of <15 dB but no points in the central 5° with sensitivity of <0 dB - only 1 hemifield containing a point with sensitivity <15 dB within 5° of fixation
Stage 3: Advanced Defect	≥ -12.01 to -20.00 dB	- ≥ 50% but <75% of points on pattern deviation plot depressed below the 5% level and ≥25% but <50% of points depressed below the 1% level - any point within the central 5° with sensitivity <0 dB - both hemifields containing a point(s) with sensitivity <15 dB within 5° of fixation
Stage 4: Severe Defect	≥ -20.00 dB	- ≥ 75% of points on pattern deviation plot depressed below the 5% level and ≥50% but <50% of points depressed below the 1% level - at least 50% of points within the central 5° with sensitivity <0 dB - both hemifields containing >50% of points with sensitivity <15 dB within 5° of fixation
Stage 5: End-Stage Disease		Unable to perform HVFA in worst eye due to central scotoma or worst eye VA 6/60 or worse due to POAG. Fellow eye may be at any stage

ROLE OF IMAGING IN GLAUCOMA:

Glaucomatous damage is distinguished by a distinct pattern of damage to the optic nerve head (ONH), preceding visual field loss. This sequence implies that structural damage precedes functional loss.[40,41] Structural changes can be analyzed through clinical examination and monitored via serial fundus photographs, albeit with a significant drawback of inter-observer variability. To address this limitation, extensive research has been conducted on imaging modalities.

Advanced imaging devices offer objective quantitative measures of retinal nerve fiber layer thickness, optic nerve head topography, neuroretinal rim thickness, and ganglion cell layer, with high repeatability and low variability. This not only aids in diagnosis but also facilitates the detection of genuine disease-related changes beyond age-related losses, including short-term and long-term fluctuations.[31]

Studies on optical coherence tomography (OCT), confocal scanning laser ophthalmoscopy (CSLO), and scanning laser polarimetry (SLP) have demonstrated the capability of these imaging techniques for early detection and assessment of changes in glaucomatous eyes or eyes of glaucoma suspects over time.[32]

OCT (Optical Coherence Tomography)

Optical Coherence Tomography (OCT) is an advanced imaging technology that produces cross-sectional images of the retina using optical-coherence interferometry. Similar to B ultrasound, OCT employs a near-infrared light beam at a wavelength of 840 nm instead of sound waves, measuring the echo delay time of reflected and backscattered light from the retina. This technique was first reported by Huang et al. in 1991.[32]

This non-contact, non-invasive method measures retinal structures with a precision of 10 microns, compared to the 100-micron scale of ultrasound.[32]It provides both qualitative (morphology and reflectivity) and quantitative (thickness, mapping, and volume) analysis of the retina.[33, 34]

The first and second generations of OCT instruments, known as time-domain (TD) OCT, had an axial resolution of 10–15 μm. The third generation OCT (Stratus; Carl Zeiss

Meditec, Dublin, California, USA) improved this to an axial resolution of 8–10 μm. [73]Spectral-domain OCT (SD-OCT) further enhanced the axial resolution to 5–6 μm.

Three main parameters aid in the detection of glaucomatous loss: the retinal nerve fiber layer, the optic nerve head, and the ganglion cell complex. The numeric values for these parameters are color-coded as white, green, yellow, or red, with yellow and red indicating values less than 5% and less than 1%, respectively, compared to the normative database.[35]

Assessment of progression using OCT:

In these imaging modalities, detecting real structural changes is achieved by improving the signal-to-noise ratio. Many imaging technologies now include progression analysis packages that compile data from multiple visits into trend-based analyses, assisting clinicians in monitoring glaucoma progression.[42]

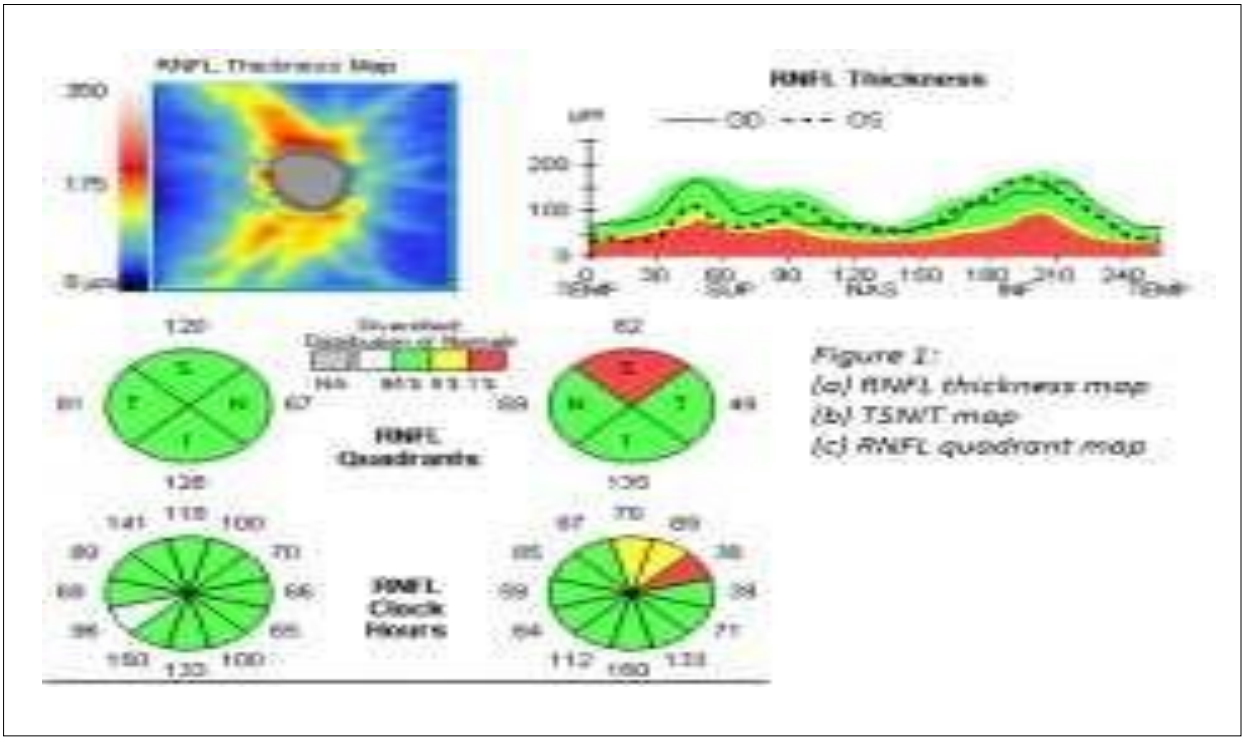
To effectively monitor progression analysis in clinical practice, three requirements must be met: measurements must be reproducible with minimal noise, follow-up images must be accurately registered to each other, and a statistical test must distinguish between true biological change and instrument measurement variability.

RETINAL NERVE FIBRE LAYER

SD-OCT can directly quantify and measure the retinal nerve fiber layer (RNFL) thickness by calculating the area between the internal limiting membrane (ILM) and the RNFL border. [35] The Cirrus RNFL map represents a 6×6 mm cube of A-scan data centered over the optic nerve, from which a 3.4 mm diameter circle of RNFL data is extracted to create the TSNIT map (temporal, superior, nasal, inferior, temporal). This map is depicted using a false color scale, with thickness values referenced to a normative database. The TSNIT map displays RNFL thickness values by quadrants and clock hours, with the RNFL peaks providing an

assessment of the anatomical distribution of nerve fiber axons, represented by the superior and inferior bundles emanating from the optic nerve.[36]

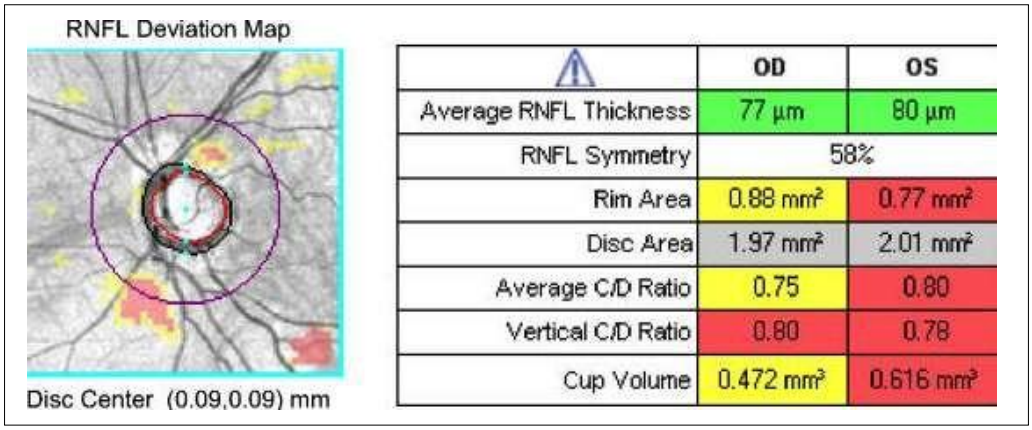
Figure 5: RNFL Analysis



Optic Nerve Head (ONH)

In order to provide objective metrics such optic disc area, neuroretinal rim area, and vertical cup-to-disc ratios, SD-OCT additionally examines the optic nerve head, optic cup, and disc borders. In order to accomplish this, the Cirrus SD-OCT defines the disc's edge as the Bruch's membrane termination. Next, it determines the inner cup margin in each slice of the spiral around the optic disc cube data by calculating the shortest perpendicular distance to the internal limiting membrane (ILM), also referred to as the minimum band distance. This process continues until a center is found. This procedure makes it possible to consistently center the 3.4 mm RNFL circle inside the cube, something that TD-OCT was unable to accomplish. The calculated optic nerve head (ONH) parameters, except for disc area, are then

compared to a normative database.[36] (see Fig.6)



	OD	OS
Average RNFL Thickness	77 µm	80 µm
RNFL Symmetry	58%	
Rim Area	0.88 mm²	0.77 mm²
Disc Area	1.97 mm²	2.01 mm²
Average C/D Ratio	0.75	0.80
Vertical C/D Ratio	0.80	0.78
Cup Volume	0.472 mm³	0.616 mm³

Figure 6- Optic Nerve Head Analysis

Ganglion Cell Analysis (GCA)

The ganglion cell analysis of both eyes measures the combined thickness of the ganglion cell layer (GCL) and inner plexiform layer (IPL) using data from the Macular 200×200 or 512×128 cube scan patterns. These measurements are compared to normative data. The ganglion cell layer is thickest in the perimacular region, and decreased total macular thickness has been observed in glaucomatous eyes, likely due to thinning of the ganglion cell layer in this region. However, segmenting the ganglion cell layer alone is very difficult based on reflectivity, so Cirrus measures its Ganglion Cell Analysis (GCA) by assessing the combined GCL and IPL thickness.[43]

The Ganglion Cell OU Analysis screen contains:

- Thickness Map: Displays the thickness measurements of the GCL + IPL in the 6 mm by 6 mm cube, represented as an elliptical annulus centered at the fovea.
- Deviation Map: Compares the GCL + IPL thickness to normative data, with red indicating areas thinner than 99% of normals and yellow indicating areas thinner than 95% of normals.

- Thickness Table: Shows the average and minimum thickness within the elliptical annulus.
- Sectors: Divides the elliptical annulus of the Thickness Map into six regions: three equally sized sectors in the superior region and three equally sized sectors in the inferior region.

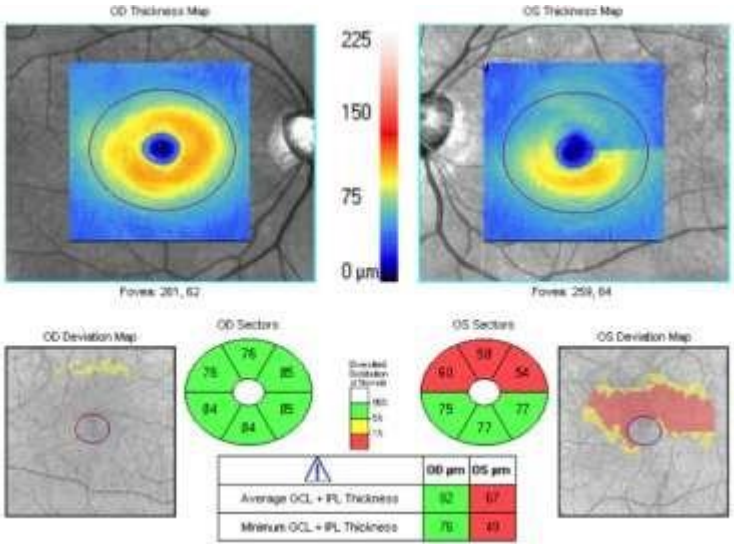


Figure 7 – Ganglion cell complex analysis

Early detection and management of glaucoma are crucial for halting its progression. Current glaucoma therapy primarily focuses on reducing aqueous humor production, increasing fluid drainage, or a combination of both through medical, laser, or surgical interventions. However, this approach addresses only a risk factor, namely elevated intraocular pressure, rather than the underlying disease itself, failing to target the events leading to elevated pressure. Effective management of glaucoma necessitates a deeper understanding of its pathogenic mechanisms.[37]

Treatment options for glaucoma include medical and surgical approaches. Medical management involves the use of topical antiglaucoma medications, which either reduce

aqueous humor production or increase aqueous outflow. These medications typically require lifelong use, with regular follow-up visits to an ophthalmologist for assessing progression and adjusting therapy as needed for optimal efficacy.[38]

Laser therapy, such as laser iridotomy, provides another method of medical management. This procedure involves creating a hole in the peripheral iris to establish an alternate pathway for aqueous humor outflow, particularly in cases of angle-closure glaucoma.[39]

In situations where maximal medical therapy fails to achieve target intraocular pressure levels, such as in cases of non-compliance, surgical management may be considered.

CONTRAST SENSITIVITY

"Contrast sensitivity is the capability of the eye to differentiate between objects based on the difference in luminance"[1].Various charts like "PelliRobson", "Arden gratings", "Vistech", "Reagan charts" can help in measuring this. Possible theories behind this mechanism is the: Neuronal theory, channel theory and the M and P cellular pathways.[72,73,74]

Contrast sensitivity is indicated for identifying disorders like: glaucoma, Visual pathway diseases, Retinal optic nerve diseases, optic neuritis, cataract, and Age related Macular degeneration.

Measurement of contrast sensitivity is given by the following formula: (Lmax - Lmin) / (Lmax + Lmin) ;where " L" is the brightness that photocells scan across the gratings. Main forms of deficiency are low level loss, high level loss and selective loss type.

In Temporal contrast sensitivity: The critical flicker frequency is mediated by

"Magnocellular retinal ganglion" is cells.[75]

In Spatial sensitivity:The "parvocellular" structures senses the spatial contrast.

Patients with glaucoma tend to have decreased: Temporal contrast sensitivity.[77]

PelliRobson chart is mounted at a distance of 1M and the patient is asked to read mono ocularly. Same size letters are present in each row, but the contrast between them decreases by 0.15 log units for every three-letter group.

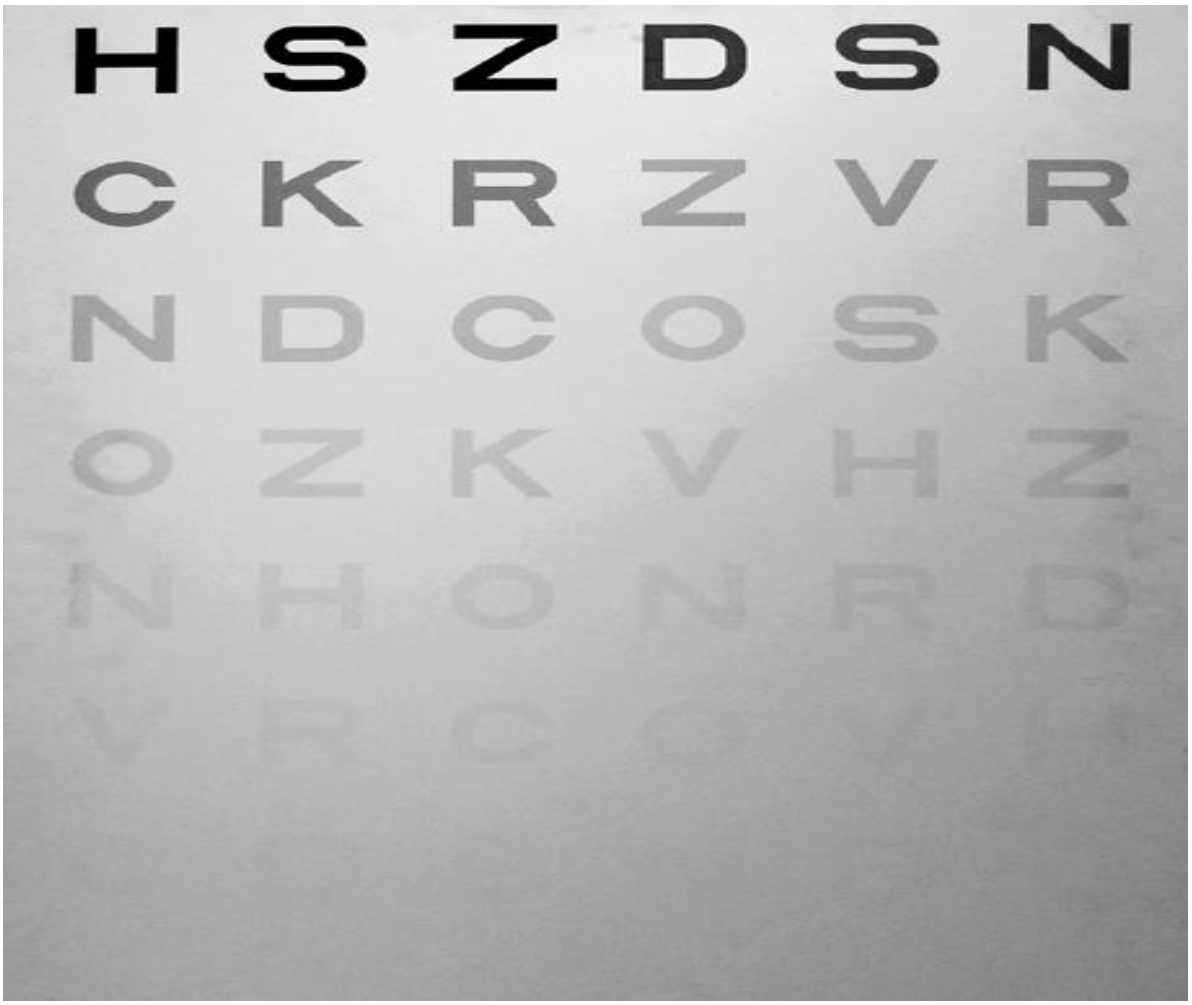


Figure 8:Pelli Robson Contrast Sensitivity Chart

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY AREA: R.L.J Hospital and Research Centre adjoining Sri Devaraj URS Medical College.

STUDY POPULATION: After obtaining approval from Institutional ethics committee, people diagnosed with glaucoma who come under our Inclusion criteria was taken up for the study.

STUDY DESIGN: Cross sectional study

Sample Size: 41 patients

TIME FRAME TO ADDRESS THE STUDY: September 2022 to December 2023

INCLUSION CRITERIA:

Patient with diagnosis of glaucoma (All types of Glaucoma)

Glaucoma suspect

Normal tension glaucoma

Ocular Hypertension

Patients with Visual Acuity 6/12 or better.

EXCLUSION CRITERIA:

Cataract

Posterior capsular opacification

Patients with Pathological Myopia

Media opacities

Diabetic Retinopathy

Age related Macular Degeneration

Any Other Retinal pathology

Methodology:

1)Visual Acuity Assessment done by using Snellen visual acuity chart. The value obtained was converted to LogMAR value.

2)The wall-mounted chart " Pelli Robson Contrast Sensitivity" was used to test the contrast sensitivity. Patient was put through a one-meter distance monocular test.

The ability to read at least two of the triplet's three letters with the same contrast determines the test's outcome.

The result, which is just one integer, represents the subject's log contrast sensitivity.

If a subject receives a score of two, it indicates that they could read at least two of the three letters with a 1% contrast (contrast sensitivity = 100% or log2).

A visual impairment is indicated by a Pelli Robson contrast sensitivity score of less than 1.5, whereas a visual disability is indicated by a score of less than 1.0.

3)Tonometry via Goldmann Applanation Tonometry

4)Gonioscopy by using 4 mirror Gonio Lens

5)Slit lamp Biomicroscopy for assessing the Anterior segment..

6)IDO for Fundus examination and +90 D for optic nerve head examination are performed subsequent to pupil dilation to facilitate the identification of

-Type of Glaucoma

-Severity of glaucoma

-Optic nerve head damage

7) The patients' visual fields were plotted using the 24-2 program on the Humphrey visual field analyzer, utilizing the SITA standard program.

Mean Deviation (MD) was used for the evaluation of visual field function.

MD value less than -6dB is categorized as early defect, MD less than -12dB is moderate and MD more than -12dB as severe defect.[1]

If a patient showed the typical changes to the optic nerve head and visual field loss, they were diagnosed with glaucoma.

If there are questionable alterations in the optic nerve head but no discernible loss in visual field, the patient is suspected of having glaucoma; if intraocular pressure (IOP) is higher than 21 mm Hg but there is no discernible loss in visual field or change in the optic nerve head, the patient is suspected of having ocular hypertension.[1]

In order to minimize the impact of additional variables, such as cataracts and potential coexisting ocular disorders (such as age-related maculopathy and diabetic retinopathy), the study was restricted to participants with an excellent visual acuity score of 6/12 or higher.

Statistical Analysis:

Data was entered into Microsoft excel data sheet and was analysed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions.

Chi-square test or Fischer's exact test (for 2x2 tables only) was used to test the significance for qualitative data.

Yates correction was applied were ever chi-square rules were not fulfilled (for 2x2 tables only). Continuous data was represented as mean and standard deviation.

Independent t test or Mann Whitney U test was used as a test of significance to identify the mean difference between two quantitative variables and qualitative variables respectively. Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram, Pie diagram and Scatter plots.

Pearson correlation or Spearman's correlation done to find the correlation between two quantitative variables and qualitative variables respectively.

P value (Probability that the result is true) of <0.05 will be considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel, SPSS version 22 (IBM SPSS Statistics, Somers NY, USA) used to analyze data. EPI Info (CDC Atlanta), Open Epi, Med calc and Medley's desktop were used to estimate sample size, odds ratio and reference management in the study.

Ethical Issues

1. Patients in the trial were given an explanation of the study's aims as well as its methodology.
2. The provision to withdraw from participation in the study was left unrestrictedly available to participants.
3. During each and every step of the research project, strict secrecy was upheld with regard to the information pertaining to the patients.

‘

RESULTS

RESULTS

Table 1:- Distribution of subjects according to age group

	Frequency	Percent
<30yrs	6	14.6
31-40yrs	2	4.9
41-50yrs	13	31.7
51-60yrs	20	48.8
Total	41	100.0

Figure 1:- Graph showing Distribution of subjects according to age group

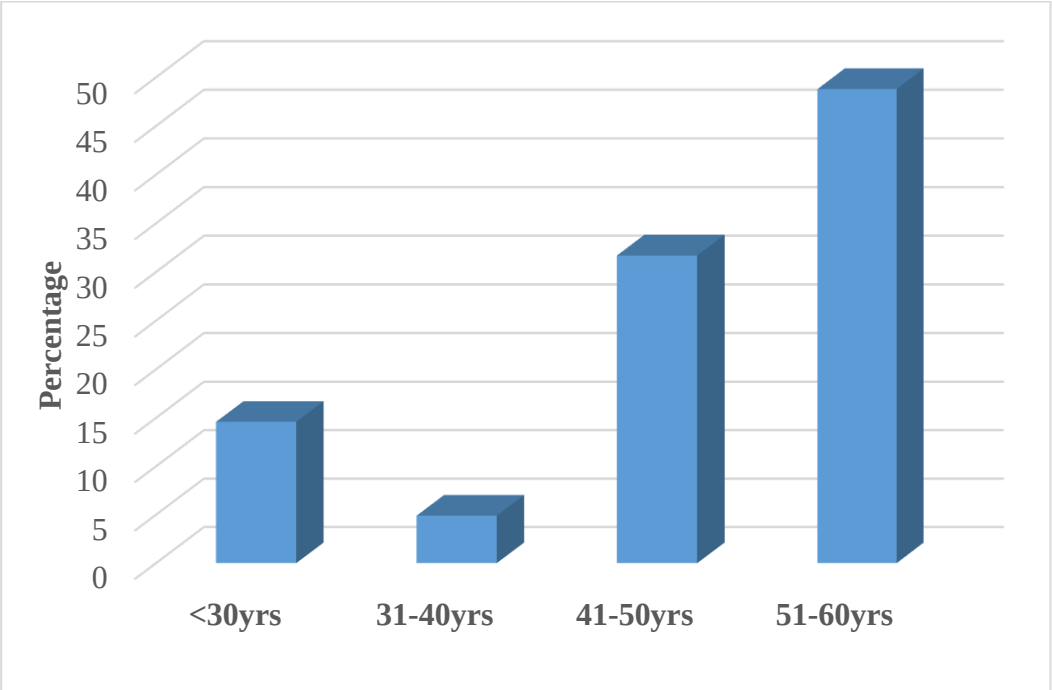


Table 2:- Distribution of subjects according to sex

	Frequency	Percent
Female	18	43.9
Male	23	56.1
Total	41	100.0

Figure 2:- Graph showing Distribution of subjects according to sex

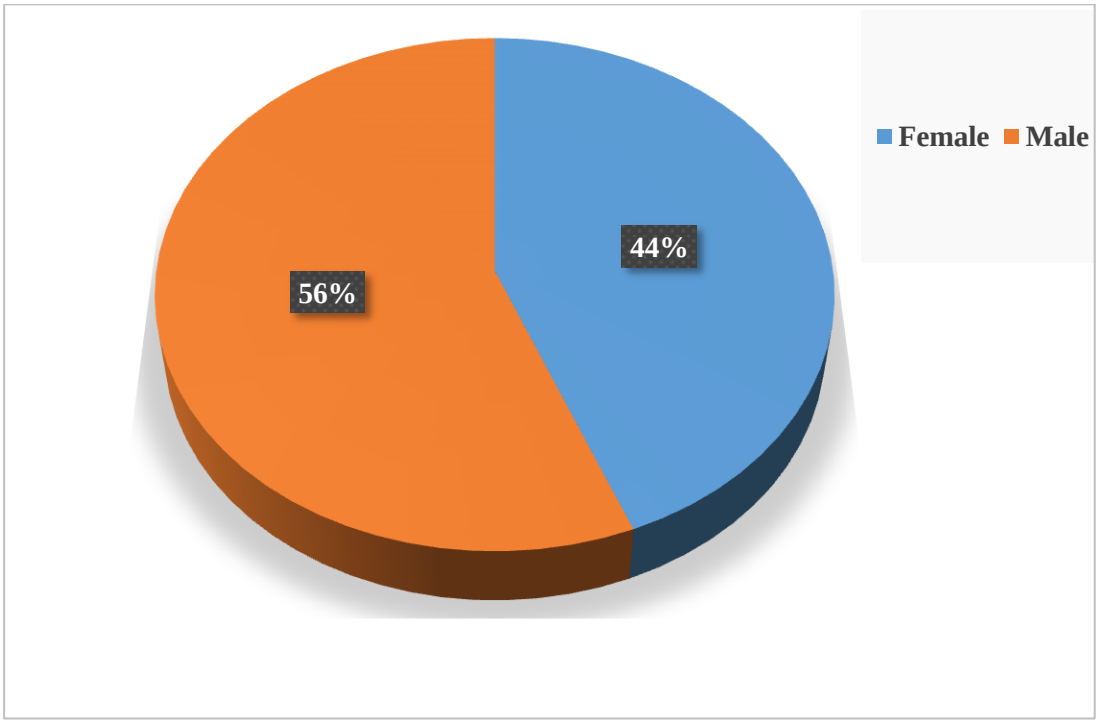


Table 3:- Distribution of subjects according to occupation

	Frequency	Percent
Clerk	16	39.0
Driver	4	9.8
Homemaker	2	4.9
Manager	5	12.2
Student	2	4.9
Tailor	7	17.1
Teacher	5	12.2

Figure 3:- Graph showing Distribution of subjects according to occupation

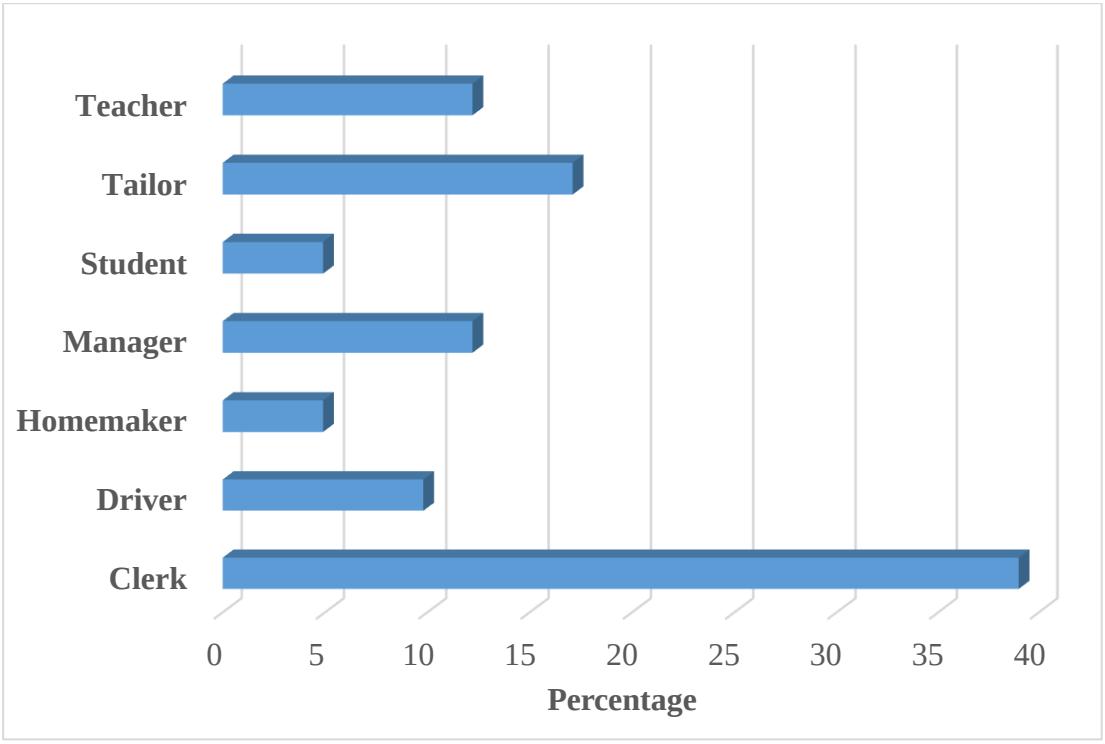


Table 4:- Distribution of subjects according to Visual acuity

	Right eye		Left eye	
	Frequency	Percent	Frequency	Percent
0.0	11	26.8	12	29.3
0.2	10	24.4	22	53.7
0.3	20	48.8	7	17.1

Figure 4a:- Graph showing Distribution of subjects according to right eye Visual acuity

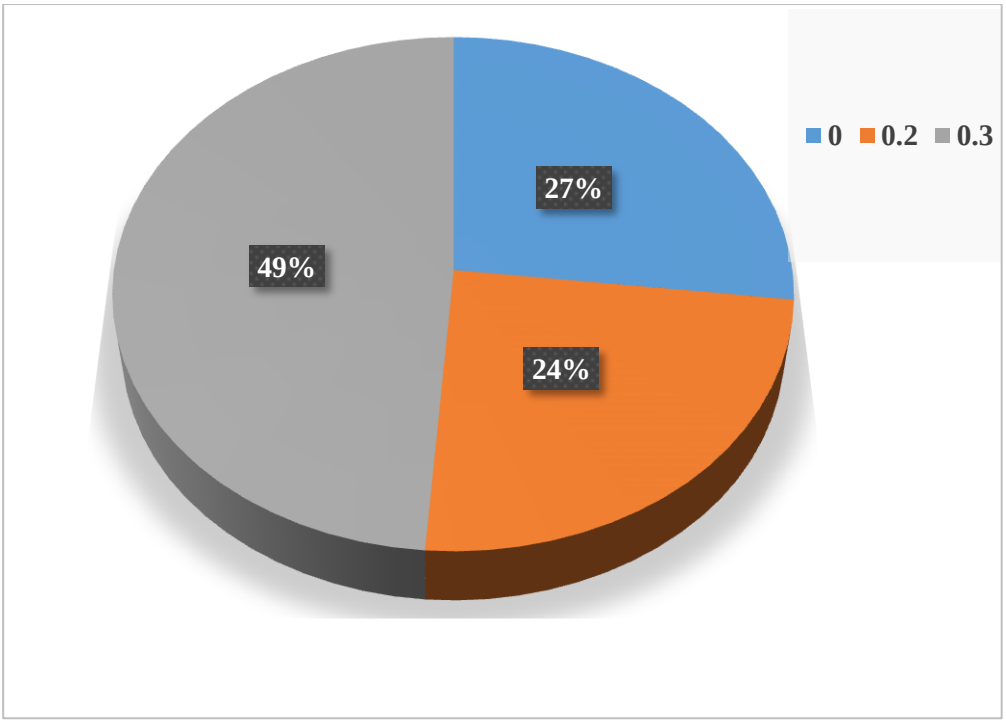


Figure 4b:- Graph showing Distribution of subjects according to left eye Visual acuity

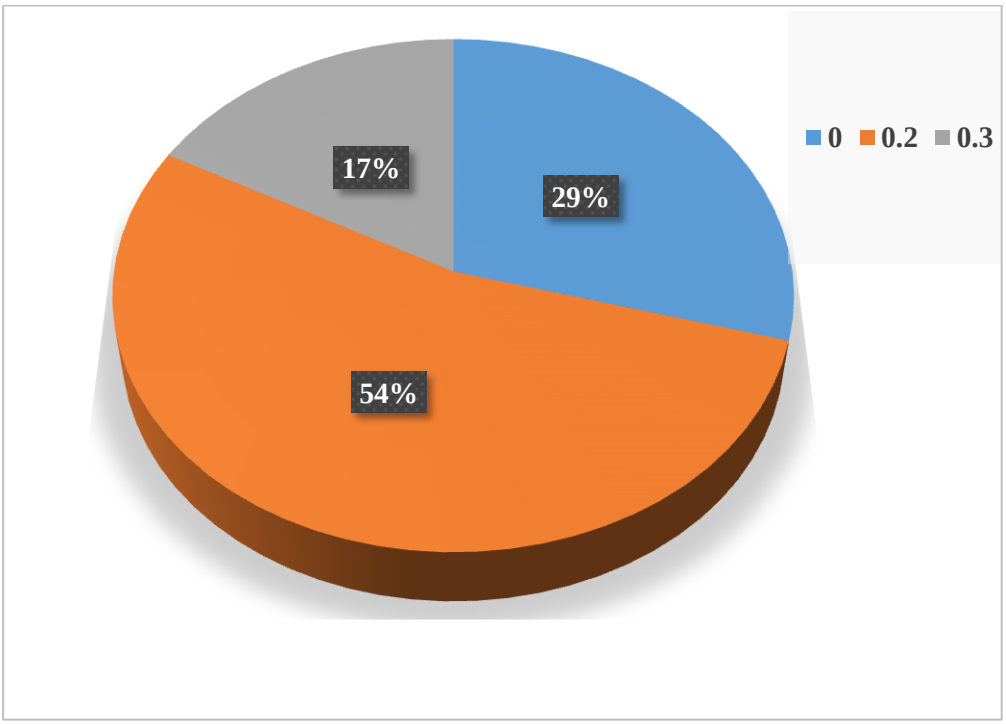


Table 5:- Distribution of subjects according to VH grading

	Right eye		Left eye	
	Frequency	Percent	Frequency	Percent
VH grade 2	8	19.5	5	12.2
WNL	33	80.5	36	87.8

Figure 5a:- Graph showing Distribution of subjects according to right eye anterior segment

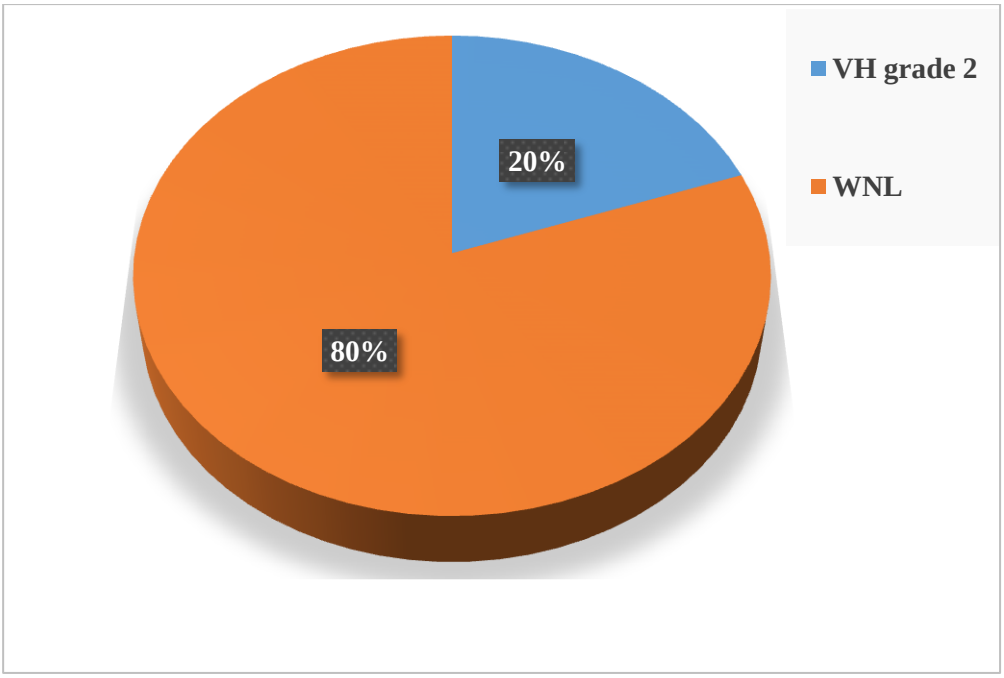


Figure 5b:- Graph showing Distribution of subjects according to left eye anterior segment

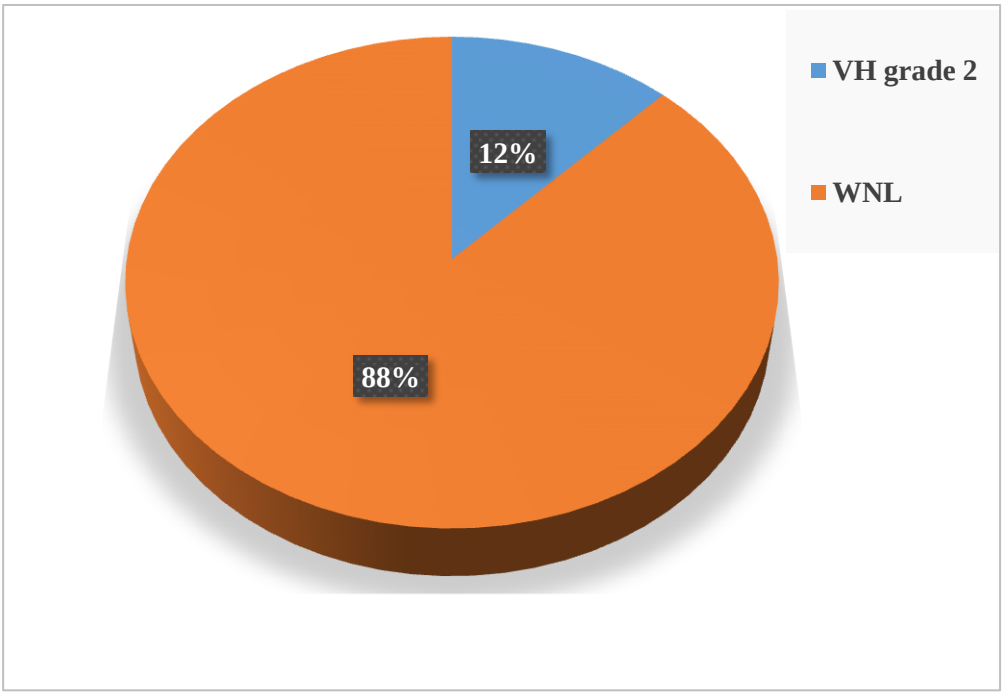


Table 6:- Distribution of subjects according to C:D Ratio

	Right eye		Left eye	
	Frequency	Percent	Frequency	Percent
CD=0.5	3	7.3	3	7.3
CD=0.6	12	29.3	12	29.3
CD=0.7	20	48.8	17	41.5
CD=0.8	6	14.6	6	14.6

Table 7:- Distribution of subjects according to Angle

	Right eye		Left eye	
	Frequency	Percent	Frequency	Percent
Closed	8	19.5	8	19.5
Open	33	80.5	33	80.5

Table 8:- Distribution of subjects according to Type of glaucoma

	Frequency	Percent
BE:NTG	9	22.0
BE:PACG	5	12.2
BE:POAG	19	46.3
LE:POAG	3	7.3
RE:PACG	3	7.3
RE:POAG	2	4.9

Figure 6:- Graph showing Distribution of subjects according to Type of glaucoma

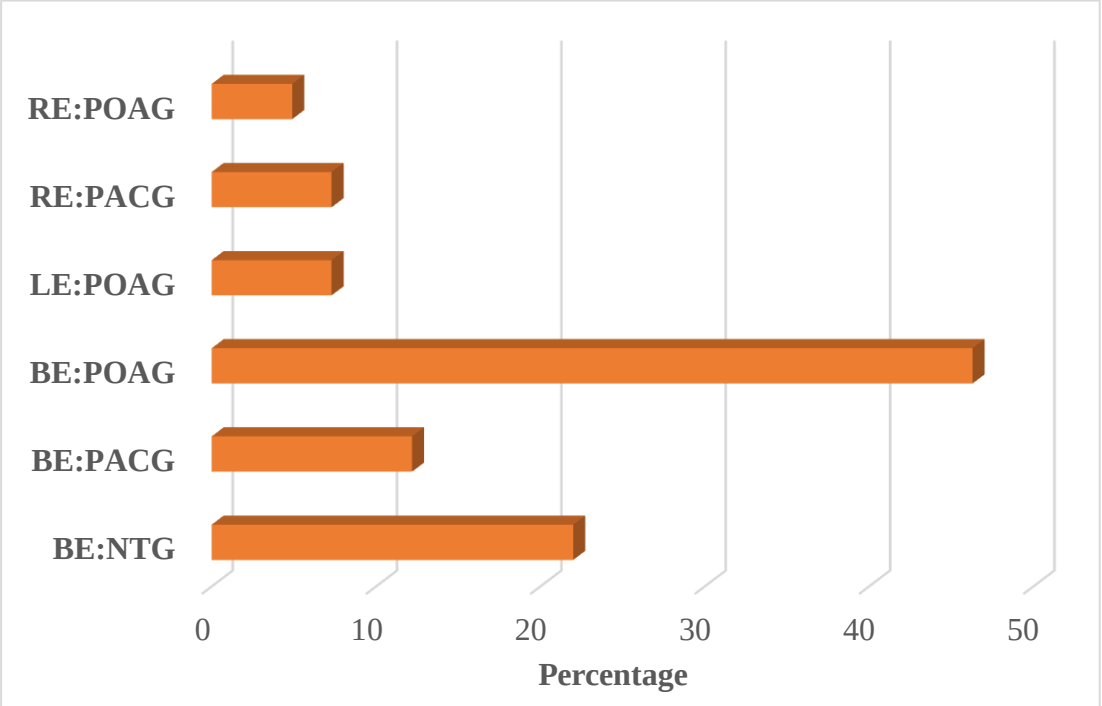


Table 9:- Descriptive statistics of various parameters

	Minimum	Maximum	Mean	Std. Deviation
RE:GAT(mmHg)	12	48	24.71	10.680
LE:GAT(mmHg)	12	36	17.71	5.806
RE:HFA(MD)	-30.4900	-3.2600	-10.611463	8.4265294
LE:HFA(MD)	-28.4400	-3.2600	-8.334634	6.4065412
RE: CS score	.0500	2.3000	1.400000	.9000000
LE:CS score	.0500	2.3000	1.743902	.7924799
AGE	21	60	48.90	10.772

Table 10:- Correlations of Right eye HFA and GAT

		RE:GAT(mmHg)
RE:HFA(MD)	Pearson Correlation	-0.462**
	P value	0.002

There was a negative correlation between RE:HFA(MD) and RE:GAT(mmHg) which was statistically significant .

Figure 10:- Scatter plot showing Correlations of Right eye HFA and GAT

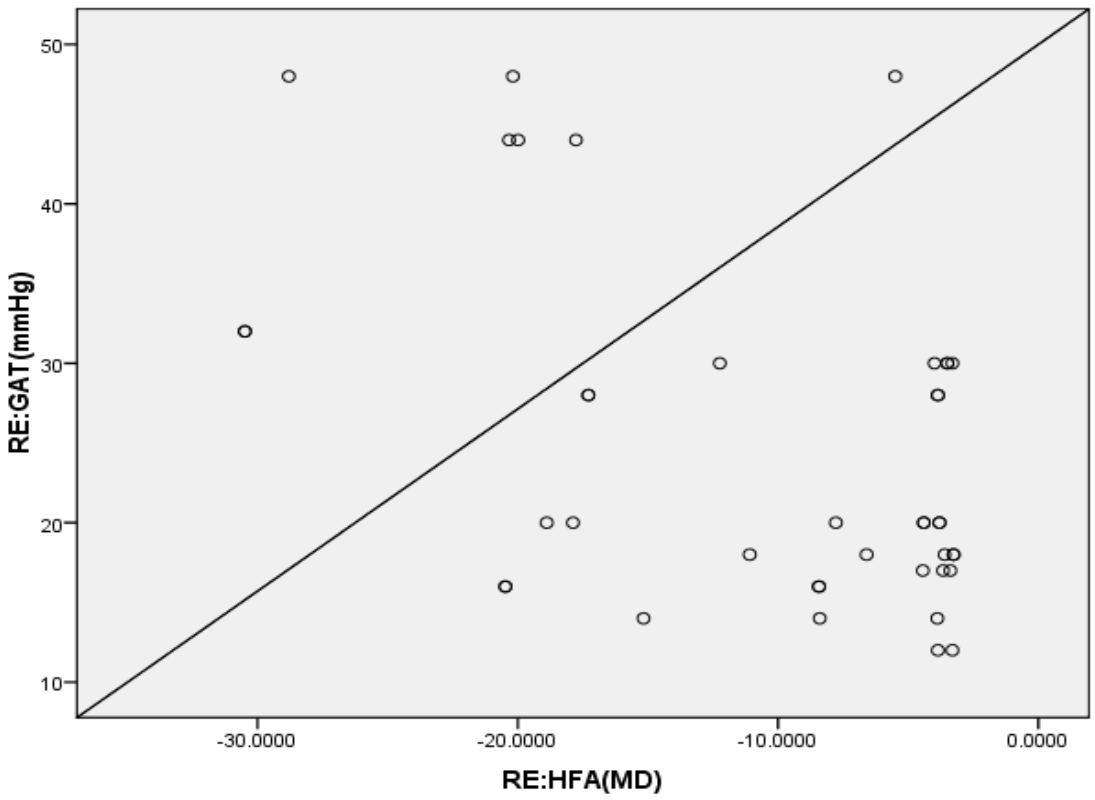


Table 11:- Correlations of Right eye GAT and CS SCORE

		RE:GAT(mmHg)
RE: CS score	Pearson Correlation	-0.254
	P value	0.109

There was a negative correlation between RE: CS score and RE:GAT(mmHg) which was not statistically significant

Figure 11:- Scatter plot showing Correlations of Right eye GAT and CS SCORE

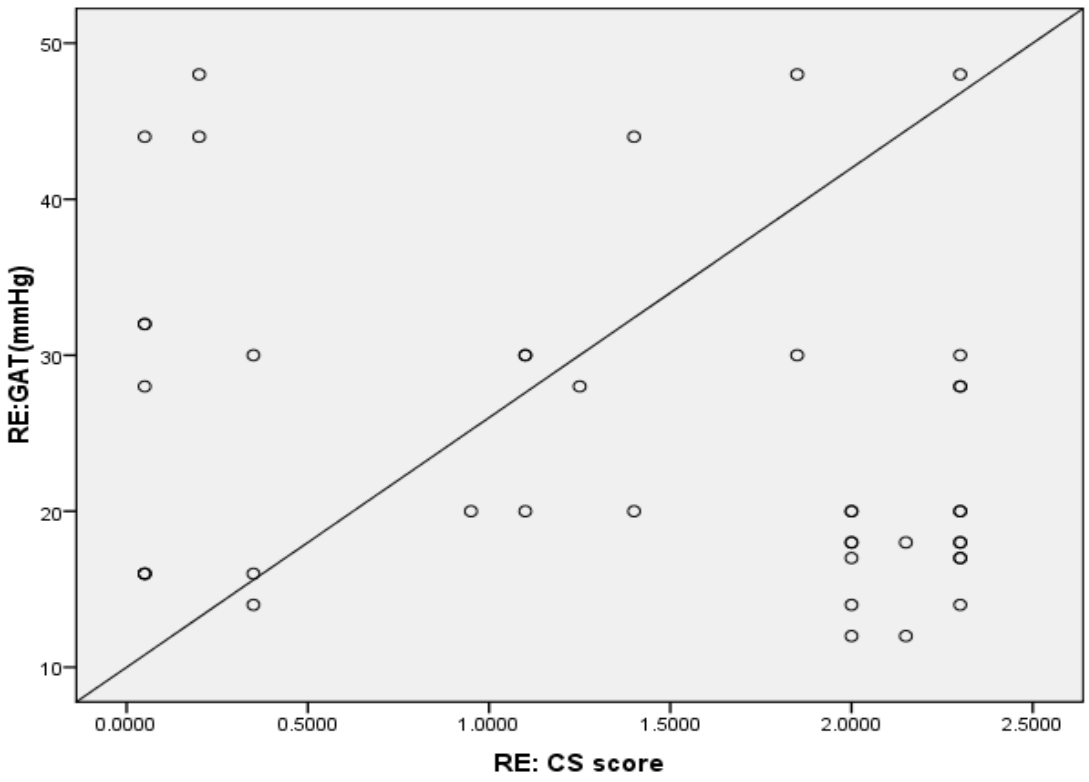


Table 12:- Correlations of Right eye HFA and CS SCORE

		RE: CS score
RE:HFA(MD)	Pearson Correlation	0.613**
	P value	<0.001

There was a positive correlation between RE:HFA(MD) and RE: CS score which was statistically significant

Figure 12:- Scatter plot showing Correlations of Right eye HFA and CS SCORE

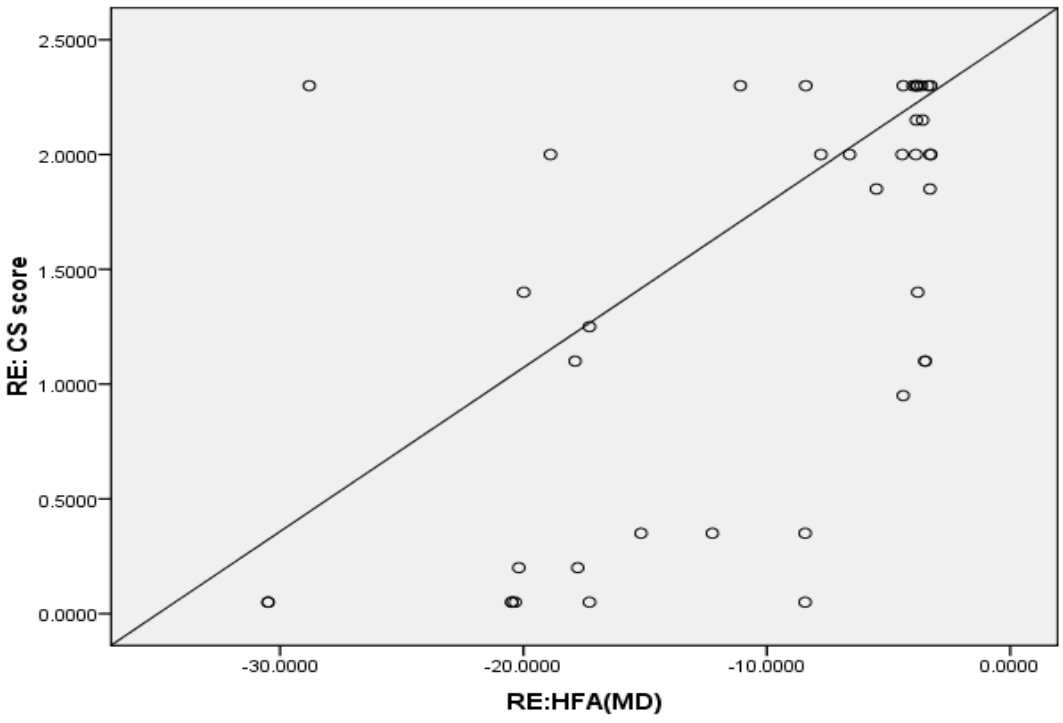


Table 13:- Correlations of Left eye HFA and GAT

		LE:GAT(mmHg)
LE:HFA(MD)	Pearson Correlation	-0.166
	P value	0.301

There was a negative correlation between LE:HFA(MD) and LE:GAT(mmHg) which was not statistically significant .

Figure 13:- Scatter plot showing Correlations of Left eye HFA and GAT

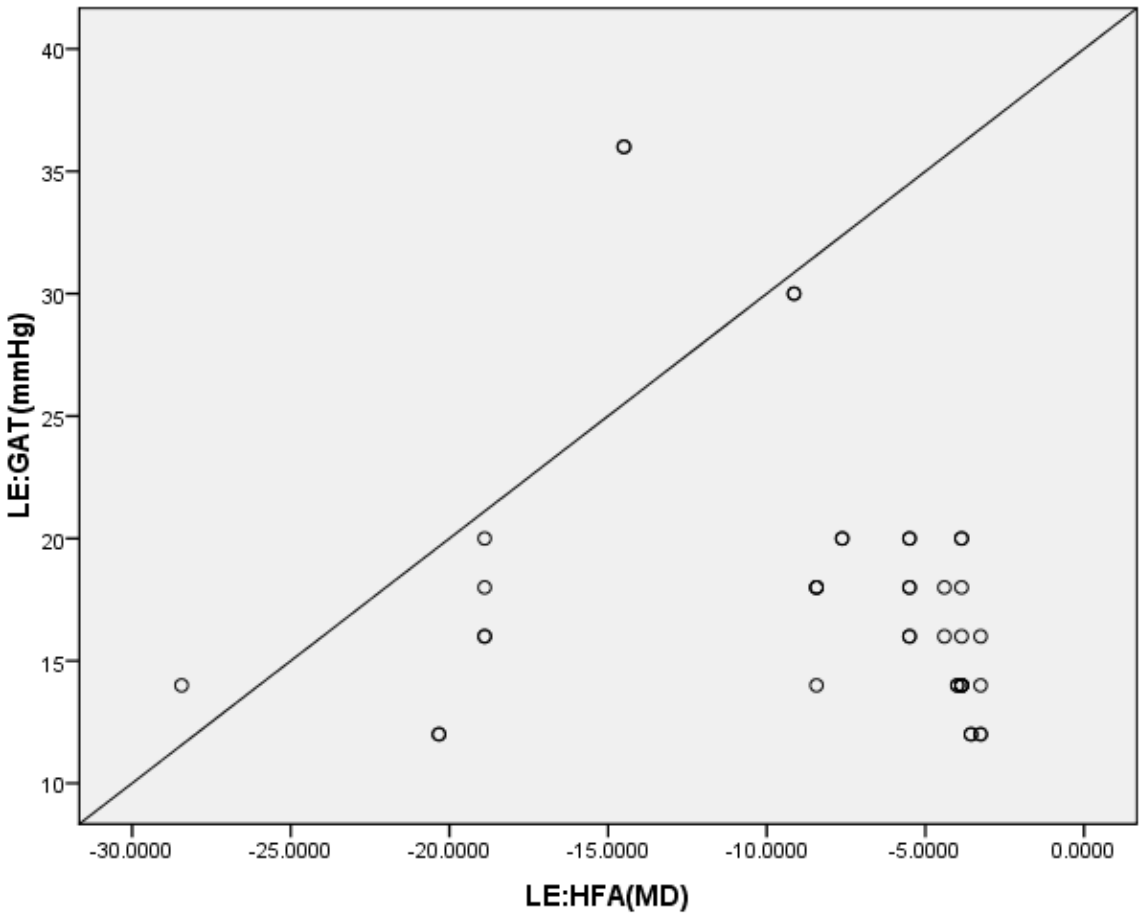


Table 14:- Correlations of Left eye GAT and CS SCORE

		LE:GAT(mmHg)
LE: CS score	Pearson Correlation	-0.344*
	P value	0.027

There was a negative correlation between LE: CS score and LE:GAT(mmHg) which was statistically significant

Figure 14:- Scatter plot showing Correlations of Left eye GAT and CS SCORE

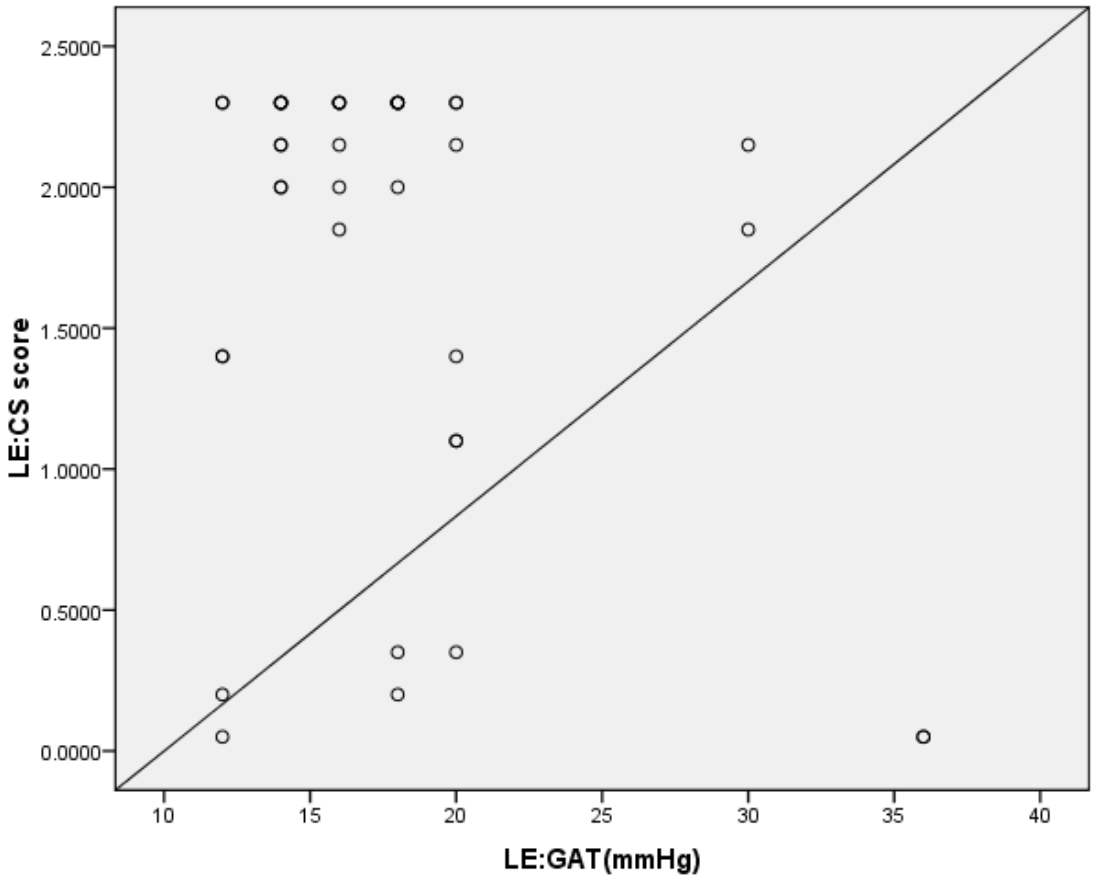


Table 15:- Correlations of Left eye HFA and CS SCORE

		LE: CS score
LE:HFA(MD)	Pearson Correlation	0.342*
	P value	0.029

There was a positive correlation between LE:HFA (MD) and LE: CS score which was statistically significant

Figure 15:- Scatter plot showing Correlations of Left eye HFA and CS SCORE

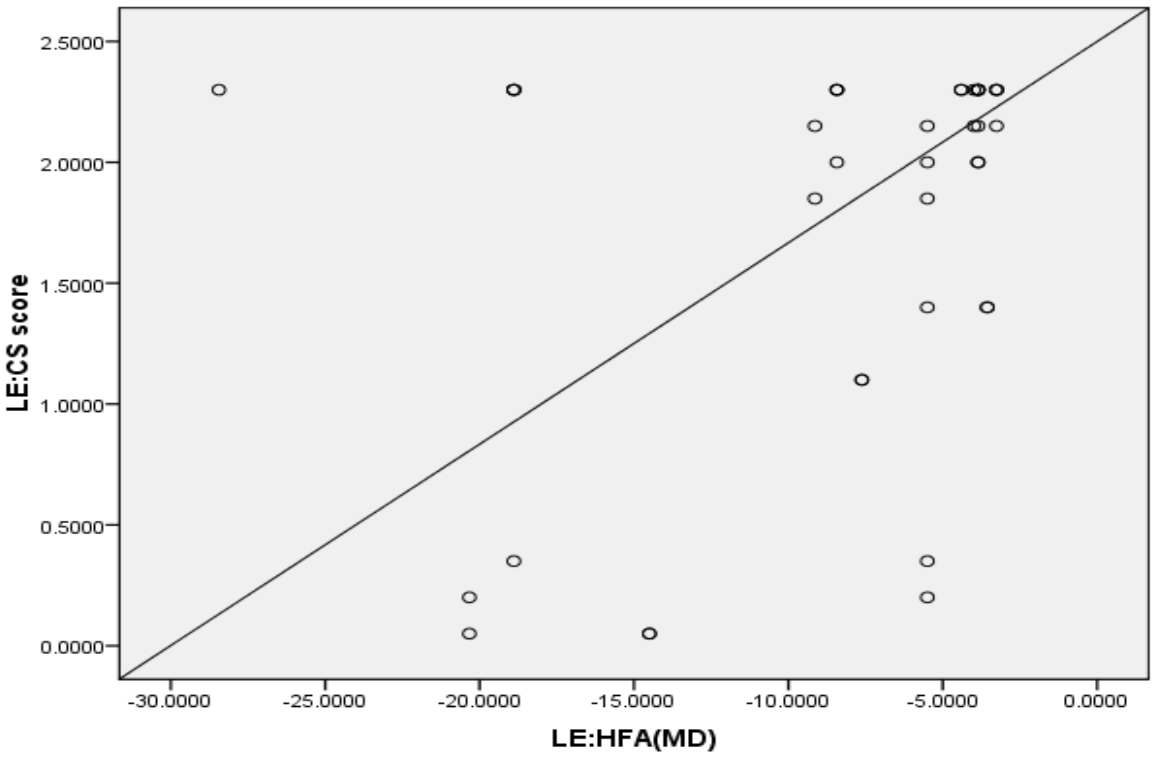


Figure 16:- Scatter plot showing Correlations of Right eye HFA and LogMar SCORE

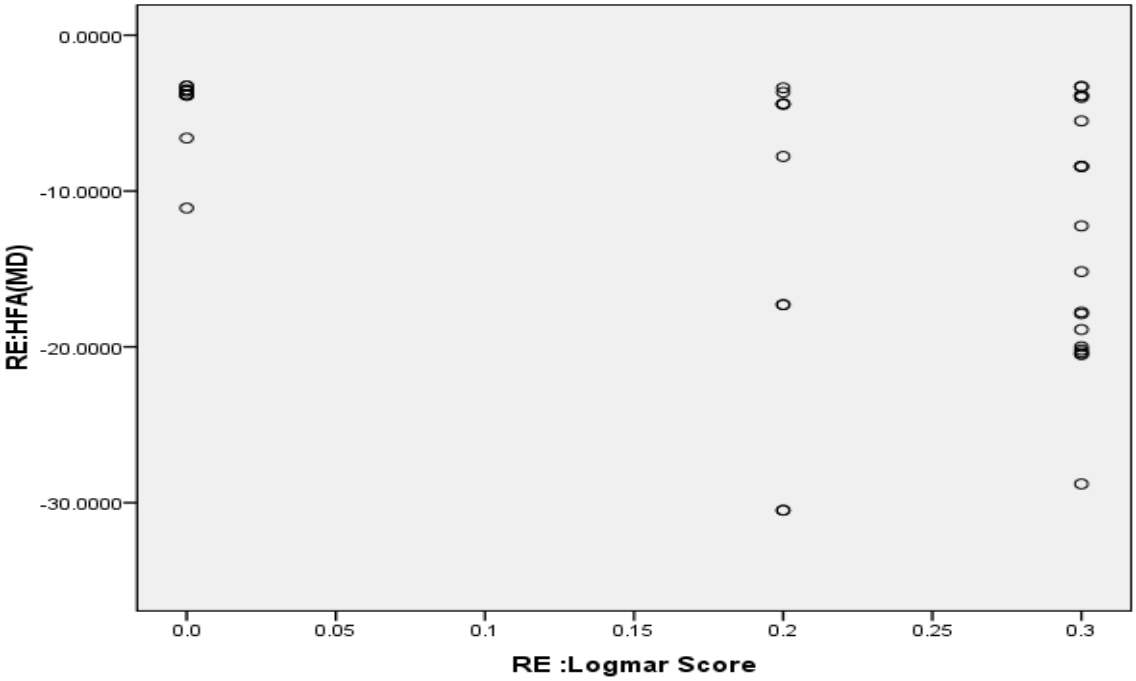


Table 16:- Correlations of Right eye HFA and LogMar SCORE

		RE:HFA(MD)
RE :Logmar Score	Pearson Correlation	-0.428**
	Sig. (2-tailed)	.005
	N	41

Table 16 displays a statistically significant moderate negative correlation (Pearson's r = -0.428, p = 0.005, N = 41) between Right Eye Humphrey Visual Field Analyzer (HFA) Mean Deviation (MD) and Right Eye LogMAR Score.

Figure 17:- Scatter plot showing Correlations of Left eye HFA and LogMar SCORE

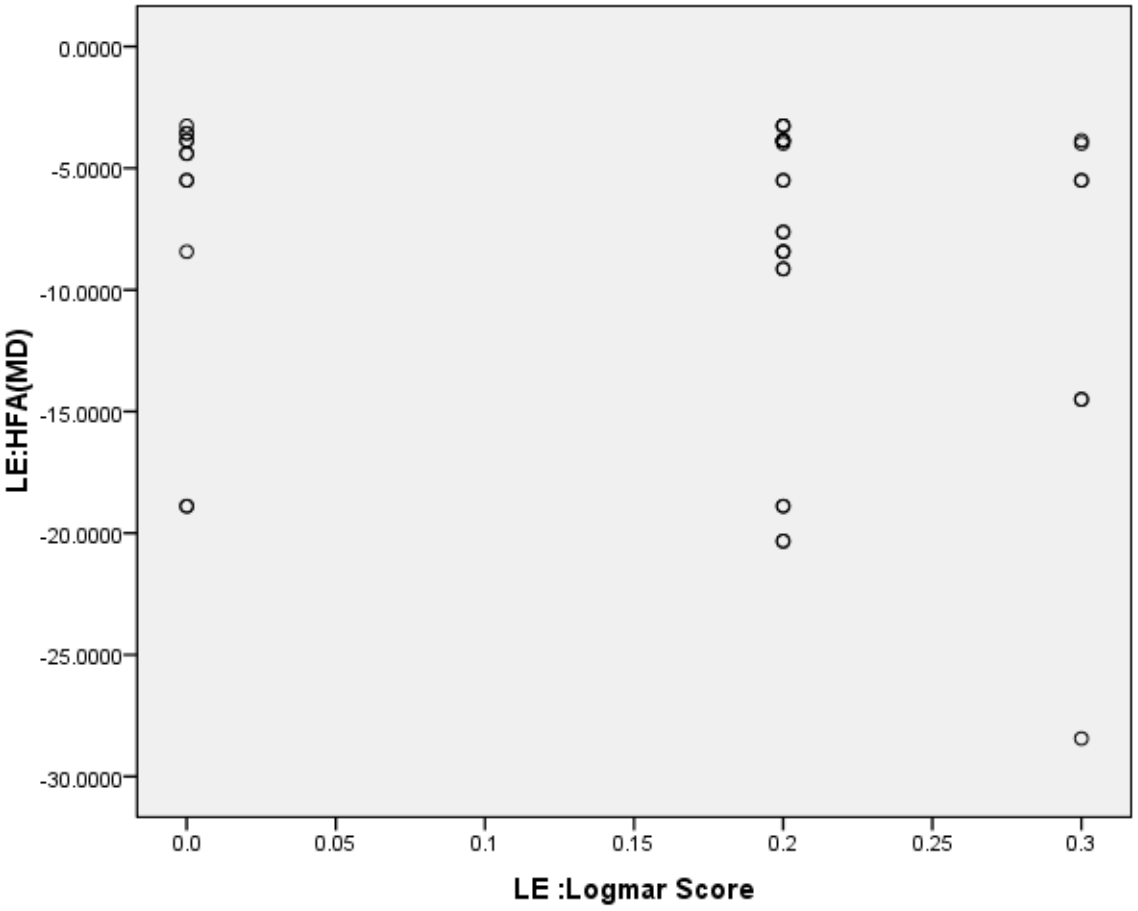


Table 17:- Correlations of Left eye HFA and LogMar SCORE

		LE:HFA(MD)
LE :Logmar Score	Pearson Correlation	-0.177
	P value	.267
	N	41

Table 17 presents the correlation analysis between Left Eye Humphrey Visual Field Analyzer (HFA) Mean Deviation (MD) and Left Eye LogMAR Score. The Pearson correlation coefficient of -0.177 suggests a weak negative correlation

DISCUSSION

DISCUSSION

In this study age distribution of subjects' shows that nearly half (48.8%) are aged 51-60 years, followed by 31.7% aged 41-50 years. Smaller groups include 14.6% under 30 years and 4.9% aged 31-40 years, totaling 41 subjects. (Table1)This distribution reflects the well-established trend of increasing glaucoma incidence with advancing age, as older individuals are more susceptible to developing this condition.

Sex distribution reveals 56.1% male and 43.9% female participants, indicating a higher male representation. Occupational diversity is notable, with clerks forming the largest group (39.0%), followed by tailors (17.1%) and others.(Table2)

Table 12 shows a positive correlation between RE (MD) and RE CS score. The correlation coefficient of 0.613, with a p-value of <0.001, suggests a strong and statistically significant positive correlation. This indicates that there is a significant relationship between visual field loss and contrast sensitivity in the right eye among the subjects studied, this is because in RE visual field loss is more which typically reduces contrast sensitivity due to diminished ability to discern light and dark areas.

Similarly Table 15 shows a moderate positive correlation between LE (MD) and LE CS score, with a Pearson correlation coefficient of 0.342 and a p-value of 0.029. This correlation aligns with findings from a study by Sohail M et al., which investigated the correlation of 10-2 and 24-2 visual field damage in Glaucomatous patients using a home based Contrast sensitivity testing Application. The test was employed in order to predict the usefulness of CS in a home based application; the test recorded logarithmic CS for a period of 8 weeks and visual field analysis also performed. The results proved a positive correlation between the two (Pearson, 0.86, P<0.0001), which was consistent with the findings of our study. [(66)]

Table 18:- Comparison of Correlations of Right eye HFA and CS SCORE

		Our study	Sohail M et al
HFA(MD)		RE: CS score	RE: CS score
RE:HFA(MD)	Pearson Correlation	0.613	0.86
	P value	<0.001	<0.001

In contrast to previous researches Ji Yong Jang & Eun Ji Lee's study on contrast sensitivity in glaucoma patients offers valuable insights into the relationship between contrast sensitivity and visual field defects. Contrary to previous assumptions, their findings suggest that contrast sensitivity does not necessarily correspond to the location of visual field damage in glaucoma patients. Instead, they observed that contrast sensitivity was more significantly affected in patients with severe visual field damage, irrespective of the location of the defects.[(69)]

Table 16 indicates a weak negative correlation between Left Eye Humphrey Visual Field Analyzer (HFA) Mean Deviation (MD) and LogMAR Score, which was not statistically significant. This finding contrasts with a study conducted by Richman et al., where contrast sensitivity was assessed using the Spaeth/Richman Contrast Sensitivity (SPARCS) test and the Pelli-Robson (PR) chart. The results were compared between eyes in three groups: MD < -12 dB and > -20 dB (group 1), MD < -20 dB and > -30 dB (group 2), or MD < -30 dB (group 3). The relationship between logMar visual acuity and SPARCS/PR scores and visual field MD was examined using multivariate regression methodology. The study found that there was no correlation between the overall SPARCS scores and PR scores in predicting the change in MD ($\beta = 0.5$, $P < 0.001$, $R2 = 61.8\%$). Severe glaucoma was predicted by total SPARCS scores of less than 45 and less than 38, respectively, with MD crossings of -20 dB (sensitivity: 70.5%; specificity:

80.9%) and -30 dB (sensitivity: 79.3%; specificity: 77.7%).”[67]

Jacob T et al's study revealed a “significant correlation between mean deviation on the Humphrey Perimeter and contrast sensitivity score on the Pelli Robson chart.”[68] This correlation indicates the interplay between visual field sensitivity and contrast sensitivity in glaucoma assessment. Conversely, the correlation between mean deviation on the Humphrey visual field and LogMAR visual acuity was weaker, aligning with the findings from our study. In Table 16, the correlation between Right Eye Humphrey Visual Field Analyzer (HFA) Mean Deviation (MD) and Right Eye LogMAR Score was examined, revealing a moderate negative correlation with a Pearson correlation coefficient of -0.428. This indicates that as the MD value increases, indicating more severe visual field loss, there is a tendency for the LogMAR Score to increase, reflecting poorer visual acuity. The correlation was found to be statistically significant with a p-value of less than 0.005.

Similarly, Table 17 also explores the correlation between Left Eye HFA MD and Left Eye LogMAR Score, showing a weak negative correlation with a Pearson correlation coefficient of -0.177. This suggests that as the MD value increases, there is a slight tendency for the LogMAR Score to decrease, indicating poorer visual acuity. However, this correlation is not statistically significant with a p-value of 0.267.

This suggests that while visual field sensitivity and contrast sensitivity are closely linked, visual acuity measured by LogMAR may not reflect changes in visual field integrity as accurately. These findings emphasize the importance of incorporating multiple visual parameters in glaucoma evaluation, with contrast sensitivity proving to be a valuable adjunct

to traditional measures like visual acuity and visual field testing.

These findings are consistent with the observations from our study, reinforcing the notion that contrast sensitivity plays a crucial role in glaucoma assessment and managementUnderstanding the impact of contrast sensitivity on glaucoma patients, particularly those with severe visual field damage, has important clinical implications. [70,71] It suggests that assessing contrast sensitivity can provide valuable information about the extent of visual impairment and disease severity in glaucoma.

Moreover, these findings contribute to our broader understanding of glaucoma pathophysiology, highlighting the complex interplay between various visual parameters in the disease process. By elucidating the relationship between contrast sensitivity and visual field defects, this study enhances our ability to comprehensively evaluate and manage glaucoma patients From a practical standpoint, these findings underscore the importance of incorporating contrast sensitivity testing into routine glaucoma evaluations. By assessing contrast sensitivity alongside traditional measures such as visual acuity and visual field testing. This personalized approach to glaucoma management has the potential to optimize patient outcomes and improve life of individuals affected by the disease.

CONCLUSION

CONCLUSION

Our study on glaucoma patients provides valuable insights into various aspects of the disease, shedding light on gender distribution, disease prevalence, age-related patterns, and correlations between key visual parameters.

Firstly, our study revealed notable gender distribution among glaucoma patients, with 23 males and 18 females indicating gender-related differences in glaucoma presentation and progression.

The age-related nature of glaucoma was also evident in our study, with the majority of cases occurring in individuals in their fifties. This highlights the importance of age-specific management strategies and regular eye screenings, particularly for individuals in this demographic. Early detection and intervention are crucial in mitigating the impact of glaucoma on vision and preventing disease progression.

Correlation analysis conducted in our study revealed important relationships between key visual parameters. We observed a negative correlation between increased logMar score (indicative of worsening visual acuity) and Mean Deviation score (reflective of visual field sensitivity) but a positive correlation was found between decreased contrast sensitivity score and Mean Deviation score. These correlations indicate the interdependence of visual acuity, contrast sensitivity, and visual field sensitivity in glaucoma assessment.

Regular assessment of these parameters allows for accurate assessment of disease severity and progression, enabling timely intervention and personalized treatment planning.

SUMMARY

SUMMARY

The current cross-sectional study was carried out in the Department of Ophthalmology, R.L.Jalappa Hospital and Research Centre, attached to Sri Devaraj Urs Medical College , Tamaka, Kolar between September 2022 till December 2023.

The study was investigated on 41 subjects, predominantly aged 51-60 (48.8%), with males slightly outnumbering females (56.1% vs. 43.9%). Clerks constituted the largest occupational group (39.0%), followed by tailors (17.1%).

Anterior segment analysis mostly revealed findings within normal limits (WNL). Primary open-angle glaucoma (POAG) was the most common type (46.3%), followed by normal tension glaucoma (NTG) (22.0%). Descriptive statistics of intraocular pressure (IOP) and Humphrey Visual Field Analyzer (HFA) mean deviation (MD) showed mean values of 24.71 mmHg (right eye) and 17.71 mmHg (left eye) for IOP, and -10.61 dB (right eye) and -8.33 dB (left eye) for HFA Mean Deviation.

These findings suggest that while there is a strong positive correlation between contrast sensitivity and visual field analysis in the right eye, the correlation is slightly weaker but still significant in the left eye.

The Pearson correlation coefficient of -0.428 indicates a moderate negative correlation between RE (MD) and LogMAR Score for the right eye. This suggests that as the HFA mean deviation for the right eye decreases (worsens), the LogMAR Score tends to increase (indicating worse visual acuity).

The p-value of 0.005 is less than 0.01, indicating a statistically significant correlation at the 1% level.

In left eye, there is a weak and not statistically significant correlation between HFA (MD)

and LogMAR Score.The relationship between visual field loss in HFA (MD) and Visual Acuity (LogMar Score) was more pronounced and significant in the Right eye compared to the left eye in the sample of 41 subjects.

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ANNEXURE

ANNEXURE 1

CASE PROFORMA	
Name:	Case No:
Age:	Date:
Sex:	1PNo:
Occupation:	DOS:
Address:	

Chief complaints:

Past history:

Diabetes Mellitus/

Hypertension/

Bronchial Asthma/Epilepsy

Family history:

Personal history:

Appetite– Sleep – Bowel–

Diet– Habits – Bladder

GPE:

Pallor/Edema/Icterus/Cyanosis /Clubbing/Lymphadenopathy

Vital signs:

a. Pulse– c) RR–

b. BP– d)Temp–

Systemic examination:

a. CVS– c.RS –

b. PA– d. CNS–

OCULAR EXAMINATION		
	RE	**LE**
1. Head Posture **2. Ocular Posture** **3. Facial Symmetry**		
5. Ocular Movements		
6. Visual Acuity a) Distant b) Near		
7.Anterior Segment		
8.IOP BY GoldmannAplannationTonometry		
9.GONIOSCOPY		
10.CS Score		
11.HFA-MD		
12.Fundus		

ANNEXURE II

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

INFORMED CONSENT FORM

Case no:

IP no:

TITLE:"A CROSS SECTIONAL STUDY TO ASSESS THE CORRELATION AMONGST VISUAL ACUITY,CONTRAST SENSITIVITY AND VISUAL FIELD ANALYSIS IN PATIENTS WITH GLAUCOMA"

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

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

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

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

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

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

ಶ್ರೀದೇವರಾಜ್‌ಅರಸ್‌ವೈದ್ಯಕೀಯಕಾಲೇಜು, ಟಮಕ, ಕೋಲಾರ

ತಿಳಿವಳಿಕೆಯಸಮ್ಮತಿನಮೂನೆ

ಕೇಸ್‌ಸಂಖ್ಯೆ:
ಐಪಿಸಂಖ್ಯೆ:

ಶೀರ್ಷಿಕೆ:"ಗ್ಲುಕೋಮಾ ರೋಗಿಗಳಲ್ಲಿ ದೃಷ್ಟಿ ತೀಕ್ಷ್ಣತೆ, ಕಾಂಟ್ರಾಸ್ಟ್ ಸೆನ್ಸಿಟಿವಿಟಿ ಮತ್ತು ವಿಷುಯಲ್ ಫೀಲ್ಡ್ ವಿಶ್ಲೇಷಣೆಯ ನಡುವಿನ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ನಿರ್ಣಯಿಸಲು ಒಂದು ಅಡ್ಡ ವಿಭಾಗದ ಅಧ್ಯಯನ"

ನಾನು, ಅಂಗೀಕರಿಸಿದ,
ಈಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳಲುಮತ್ತುಈಸಮ್ಮತಿಯರೂಪದಲ್ಲಿವಿವರಿಸಿರುವಂತೆವೈಯಕ್ತಿಕಮಾಹಿತಿಯಸಂಗ್ರಹಮತ್ತುಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನುಅನುಮೋದಿಸಲುಒಪ್ಪುತ್ತೇನೆ.

ಈಅಧ್ಯಯನದಉದ್ದೇಶ,
ತಂತ್ರದಅಪಾಯಗಳುಮತ್ತುಪ್ರಯೋಜನಗಳನ್ನುಮತ್ತುಅಧ್ಯಯನದಸಮಯದಲ್ಲಿಸಂಗ್ರಹಿಸಲ್ಪಟ್ಟಿರುವಮತ್ತುಬಹಿರಂಗಪಡಿಸುವಮಾಹಿತಿಯಗೌಪ್ಯತೆಗೆನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.ಸಂಗ್ರಹಿಸಿದಮಾಹಿತಿಯನ್ನುಸಂಶೋಧನೆಗೆಮಾತ್ರಬಳಸಲಾಗುತ್ತದೆ.

ಈಅಧ್ಯಯನದವಿವಿಧಅಂಶಗಳನ್ನುಕುರಿತುಪ್ರಶ್ನೆಗಳನ್ನುಕೇಳಲುನನಗೆಅವಕಾಶವಿದೆಮತ್ತುನನ್ನತೃಪ್ತಿಗೆನನ್ನಪ್ರಶ್ನೆಗಳಿಗೆಉತ್ತರನೀಡಲಾಗಿದೆ.

ಈಅಧ್ಯಯನದಿಂದಭಾಗವಹಿಸುವಿಕೆಯನ್ನುಯಾವುದೇಸಮಯದಲ್ಲಿನಾನುಹಿಂಪಡೆಯಲುಮುಕ್ತವಾಗಿರುತ್ತೇನೆಮತ್ತುಇದುಭವಿಷ್ಯದಕಾಳಜಿಯನ್ನುಬದಲಿಸುವುದಿಲ್ಲಎಂದುನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸುವಿಕೆನನಗೆಯಾವುದೇಹೆಚ್ಚುವರಿವೆಚ್ಚಒಳಗೊಳ್ಳುವುದಿಲ್ಲ.

ಹೆಸರು	ಸಹಿ	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿ:			
ಸಾಕ್ಷಿ:			
ಪ್ರಾಥಮಿಕ ತನಿಖಾಧಿಕಾರಿ/ವೈದ್ಯ:			

ANNEXURE III

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

PATIENT INFORMATION SHEET

TITLE:

" A CROSS SECTIONAL STUDY TO ASSESS THE CORRELATION AMONGST VISUAL ACUITY,CONTRAST SENSITIVITY AND VISUAL FIELD ANALYSIS IN PATIENT S WITH GLAUCOMA"

This information is to help you understand the purpose of the study "A CROSS SECTIONAL STUDY TO ASSESS THE CORRELATION AMONGST VISUAL ACUITY,CONTRAST SENSITIVITY AND VISUAL FIELD ANALYSIS IN PATIENT S WITH GLAUCOMA You are invited to take part voluntarily in this research study, it is important that you read and understand purpose, procedure, benefits and discomforts of the study. There are no risks associated with the various investigations done as part of the study. This study requires you to undergo vision testing, contrast sensitivity testing and visual field analysis which are non invasive tests used in routine evaluation in patients with glaucoma. Participation in this research study may not change the final outcome of your eye condition. However, patients in the future may benefit as a result of knowledge gained from this study. You will not be charged extra for any of the procedures performed during the research study. Your taking part in this study is entirely voluntary. You may refuse to take part in the study or you may stop your participation in the study at any time, without any penalty or loss of any benefits to which you were otherwise entitled before taking part in this study.

CONFIDENTIALITY Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. Your original records may be reviewed by your doctor or ethics review board. For further information,/clarification please contact Dr.SUSMITHA JOSHY, SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR – 563101.

DOCTOR's DETAILS:

DR. SUSMITHA JOSHY, MBBS

1stYEARRESIDENT

DEPARTMENT OF

OPHTHALMOLOGY,SDUMC,KOLAR–

563101

Contactno:9995068728

MailID: swzkitty@gmail.com

ಶ್ರೀದೇವರಾಜ್ಅರಸ್ಉನ್ನತಶಿಕ್ಷಣಮತ್ತುಸಂಶೋಧನಾಸಂಸ್ಥೆ,

ತಮಕ, ಕೋಲಾರ - 563101.

ರೋಗಿಯಮಾಹಿತಿಹಾಳೆ

ಶೀರ್ಷಿಕೆ:

" ಪರಸ್ಪರಸಂಬಂಧವನ್ನುನಿರ್ಣಯಿಸಲುಒಂದುಅಡ್ಡವಿಭಾಗದಅಧ್ಯಯನ

ವಿಷಯಲ್ಅಕ್ಯೂಟ್, ಕಾಂಬ್ಯಾಸ್ಟ್ರೆನ್ಸಿವಿಟಿಮತ್ತುವಿಷಯಲ್ಟೀಲ್ಡ್ಅನಾಲಿಸಿಸ್

ಗ್ಲುಕೋಮಾರೋಗಿಗಳಲ್ಲಿ"

ಈಮಾಹಿತಿಯುನಿಮಗೆಅಧ್ಯಯನದಉದ್ದೇಶವನ್ನುಅರ್ಥಮಾಡಿಕೊಳ್ಳಲುಸಹಾಯಮಾಡುತ್ತದೆ "ಎಕ್ಸಾಸ್

ದೃಶ್ಯದನಡುವಿನಪರಸ್ಪರಸಂಬಂಧವನ್ನುನಿರ್ಣಯಿಸಲುವಿಭಾಗೀಯಅಧ್ಯಯನ

ತೀಕ್ಷ್ಣತೆ, ಕಾಂಟ್ರಾಸ್ಟ್ರೆನ್ಸಿವಿಟಿಮತ್ತುವಿಷಯಲ್ಫೀಲ್ಡ್ಶ್ಲೇಷಣೆಯಲ್ಲಿ

ಗ್ಲುಕೋಮಾರೋಗಿಎಸ್

ಈಸಂಶೋಧನಾಅಧ್ಯಯನದಲ್ಲಿಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದಪಾಲ್ಗೊಳ್ಳಲುನಿಮ್ಮನ್ನುಆಹ್ವಾನಿಸಲಾಗಿದೆ, ಅದುನಿಮಗೆಮುಖ್ಯವಾಗಿದೆ

ಅಧ್ಯಯನದಉದ್ದೇಶ, ಕಾರ್ಯವಿಧಾನ, ಪ್ರಯೋಜನಗಳುಮತ್ತುಅಸ್ವಸ್ಥತೆಗಳನ್ನುಓದಿಮತ್ತುಅರ್ಥಮಾಡಿಕೊಳ್ಳಿ.

ಭಾಗವಾಗಿಮಾಡಿದವಿವಿಧತನಿಖೆಗಳೊಂದಿಗೆಯಾವುದೇಅಪಾಯಗಳಿಲ್ಲ

ಈಅಧ್ಯಯನಕ್ಕೆನೀವುದೃಷ್ಟಿಪರೀಕ್ಷೆ, ಕಾಂಟ್ರಾಸ್ಟ್ರೆನ್ಸಿವಿಟಿಪರೀಕ್ಷೆಗೆಒಳಗಾಗಬೇಕಾಗುತ್ತದೆ

ಮತ್ತುವಾಡಿಕೆಯಮೌಲ್ಯಮಾಪನದಲ್ಲಿಬಳಸಲಾಗುವಆಕ್ರಮಣಕಾರಿಅಲ್ಲದಪರೀಕ್ಷೆಗಳಾದದೃಶ್ಯಕ್ಷೇತ್ರದವಿಶ್ಲೇಷಣೆ

ಗ್ಲುಕೋಮಾಹೊಂದಿರುವರೋಗಿಗಳು.

ಈಸಂಶೋಧನಾಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸುವಿಕೆಯುನಿಮ್ಮಕಣ್ಣಿನಆಂತರಿಕಫಲಿತಾಂಶವನ್ನುಬದಲಾಯಿಸದಿರಬಹುದು

ಸ್ಥಿತಿ.ಆದಾಗ್ಯೂ, ಭವಿಷ್ಯದಲ್ಲಿರೋಗಿಗಳುಜ್ಞಾನದಪರಿಣಾಮವಾಗಿಪ್ರಯೋಜನಪಡೆಯಬಹುದು

ಈಅಧ್ಯಯನದಿಂದಪಡೆಯಲಾಗಿದೆ.ಯಾವುದೇಕಾರ್ಯವಿಧಾನಗಳಿಗೆನಿಮಗೆಹೆಚ್ಚುಪರಿಶುಲ್ಕವಿಧಿಸಲಾಗುವುದಿಲ್ಲ

ಸಂಶೋಧನಾಅಧ್ಯಯನದಸಮಯದಲ್ಲಿನಡೆಸಲಾಯಿತು.ಈಅಧ್ಯಯನದಲ್ಲಿನಿಮ್ಮಪಾಲ್ಗೊಳ್ಳುವಿಕೆಸಂಪೂರ್ಣವಾಗಿ

ಸ್ವಯಂಪ್ರೇರಿತ.

ನೀವುಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲುನಿರಾಕರಿಸಬಹುದುಅಥವಾನಿಮ್ಮಭಾಗವಹಿಸುವಿಕೆಯನ್ನುನಿಲ್ಲಿಸಬಹುದು

ಯಾವುದೇದಂಡಅಥವಾಯಾವುದೇಪ್ರಯೋಜನಗಳನಷ್ಟವಿಲ್ಲದೆ, ಯಾವುದೇಸಮಯದಲ್ಲಿಅಧ್ಯಯನಮಾಡಿ

ಇಲ್ಲದಿದ್ದರೆಈಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳುವಮೊದಲುಅರ್ಹತೆ.

ಗೌಪ್ಯತೆ

ನಿಮ್ಮವೈದ್ಯಕೀಯಮಾಹಿತಿಯನ್ನುಅಧ್ಯಯನವೈದ್ಯರುಮತ್ತುಸಿಬ್ಬಂದಿಗೌಪ್ಯವಾಗಿಡುತ್ತಾರೆಮತ್ತು

ಸಾರ್ವಜನಿಕವಾಗಿಲಭ್ಯವಾಗುವಂತೆಮಾಡುವುದಿಲ್ಲ. ನಿಮ್ಮಮೂಲದಾಖಲೆಗಳನ್ನುನಿಮ್ಮಮೂಲಕಪರಿಶೀಲಿಸಬಹುದು

ವೈದ್ಯರುಅಥವಾನೀತಿಶಾಸ್ತ್ರವಿಮರ್ಶಕಮಂಡಳಿ.ಹೆಚ್ಚಿನಮಾಹಿತಿಗಾಗಿ,/ಸ್ವಯಂಸೇವಕರಣಕ್ಕಾಗಿದಯವಿಟ್ಟುಸಂಪರ್ಕಿಸಿ

ಡಾ.ಸುಸ್ಮಿತಾಜೋಕಿ, ಶ್ರೀದೇವರಾಜ್ URS ಅಕಾಡೆಮಿಅಫ್ಲೈಯರ್

ಶಿಕ್ಷಣಮತ್ತುಸಂಶೋಧನೆ, ತಮಕ, ಕೋಲಾರ – 563101.

ವೈದ್ಯರವಿವರಗಳು:

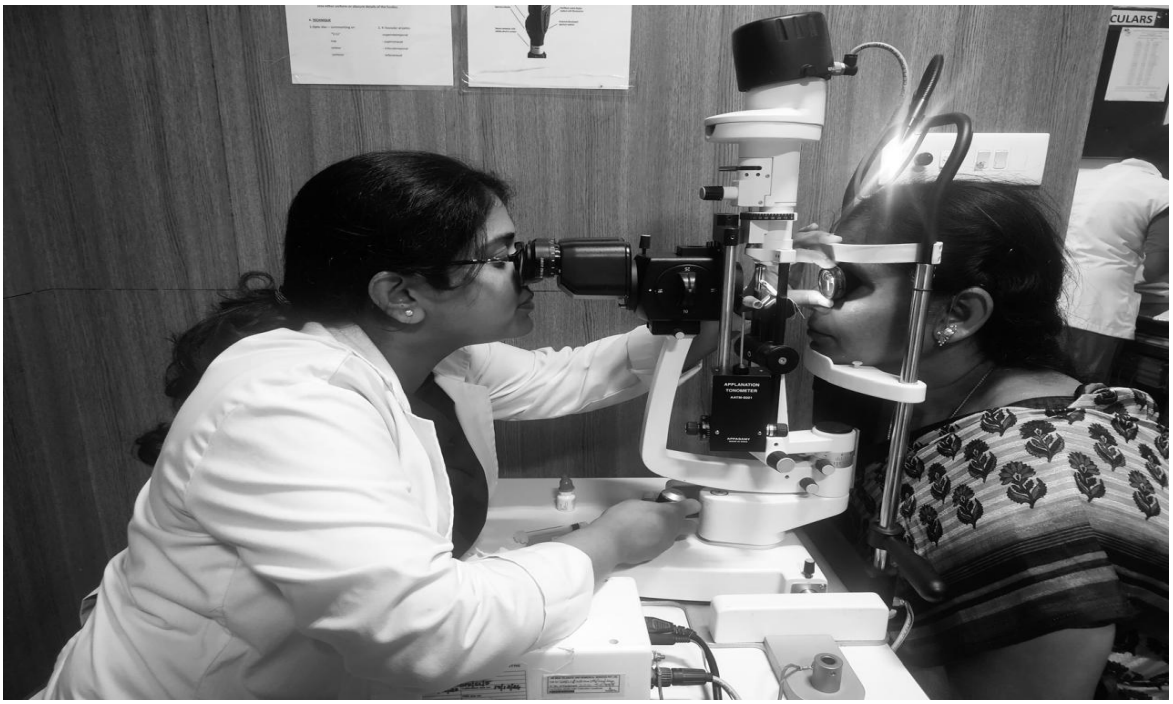
DR. ಸುಸ್ಮಿತಾಜೋಕಿ, ಎಂಬಿಬಿಎಸ್

1ನೇವರ್ಷದನಿವಾಸಿ

ನೇತ್ರವಿಜ್ಞಾನಇಲಾಖೆ, SDUMC, ಕೋಲಾರ-563101

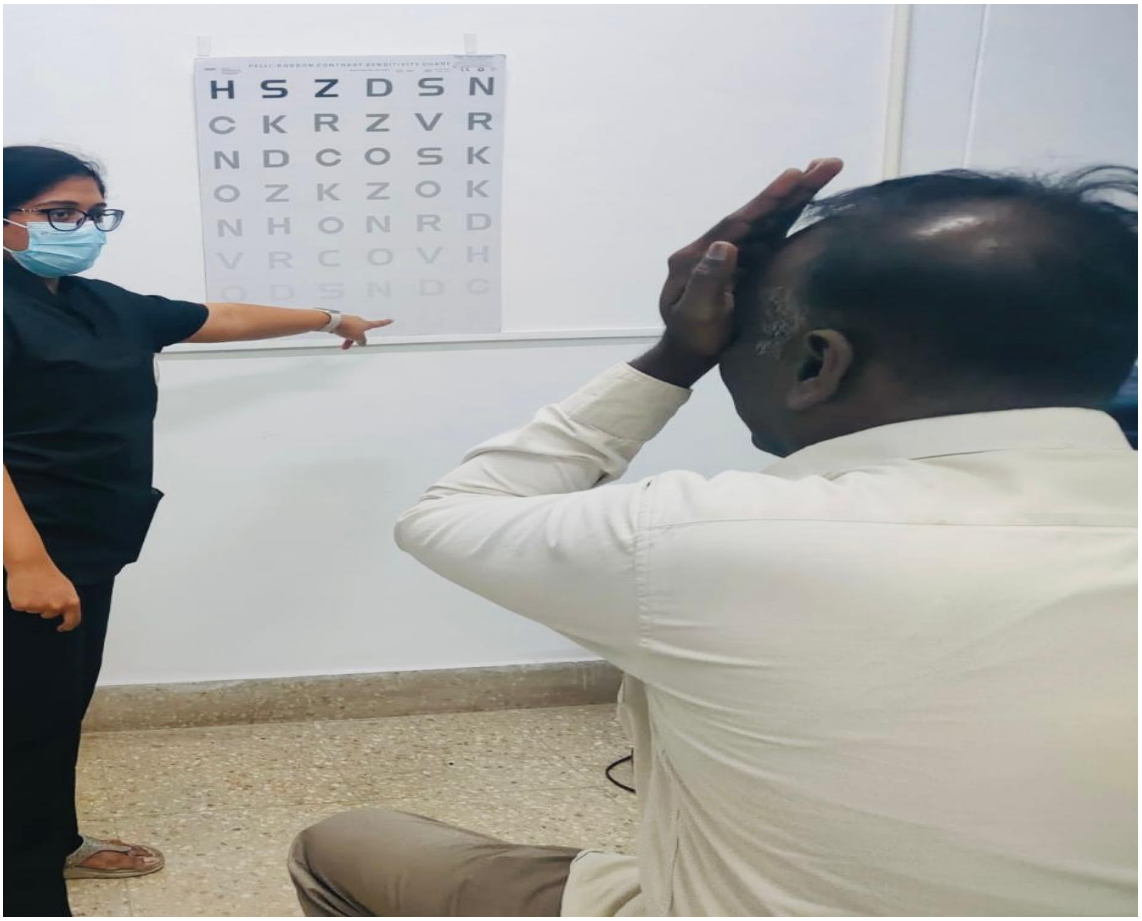
9995068728
MailID: swzkitty@gmail.com

ANNEXURE-IV



PHOTOGRAPH 1-GONIOSCOPIC EXAMINATION

PHOTOGRAPH 2-GAT EXAMINATION



PHOTOGRAPH 3-CONTRAST SENSITIVITY TEST

MASTER CHART

KEY WORDS:

GAT :Goldmann Applanation Tonometry,

Gonio:Gonioscopy,

HFA:Humphrey Visual Field Analyser,

CS:Contrast Sensitivity

SL.NO	AGE	SEX	UHID	OCCUPATION	RE :Logmar Score	LE :Logmar Score	RE :Anterior segment	LE :Anterior segment	RE :FUNDUS	LE :FUNDUS	RE:GAT(mm Hg)	LE:GAT(mm Hg)	RE:GONIO	LE:GONIO	RE:HFA(MD)	LE:HFA(MD)	PERMETRY	RE: CS score	LE:CS score	Type of glaucoma	OTHERS
1	55	M	126738	CLERK	0.2	0.3	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	17	14	Open	Open	-3.38	-3.86		2.3	2	BE:POAG	
2	30	F	121963	TEACHER	0	0.0	WNL	WNL	CD=0.6,Nasal shifting	CD=0.6,Nasal shifting	18	18	Open	Open	-3.6	-8.43		2.2	2.3	BE:NTG	
3	48	M	197870	CLERK	0.3	0	WNL	WNL	CD=0.6,Nasal shifting	CD=0.6,Nasal shifting	48	14	Open	Open	-28.8	-3.26		2.3	2.3	BE:POAG	
4	57	F	53428	TAILOR	0.3	0.2	WNL	WNL	CD=0.7,Nasal shifting, notching, laminar dot sign	CD=0.5,Nasal shifting	14	14	Open	Open	-8.4	-3.86		2.3	2.3	BE:POAG	
6	55	M	139086	CLERK	0.3	0	VH grade 2,diffuse conjunctival congestion, corneal oedema	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	44	16	Closed	Closed	—20.34	-18.9	RE:Ring scotoma	0.1	2.3	RE:PACG	
7	60	M	158648	Manager	0.3	0.2	VH grade 2	VH grade 2	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	30	20	Closed	Closed	-3.99	-3.86		2.3	2.3	BE:PACG	
8	50	F	198667	CLERK	0.2	0.2	WNL	WNL	CD=0.5 ,Nasal shifting, bayonetting,NRR thinning		20	18	Open	Open	-440	-8.43		2.3	2.3	LE:POAG	
9	55	F	175260	TAILOR	0.3	0.2	WNL	WNL	CD=0.6 ,Nasal shifting, bayonetting,NRR thinning	CD=0.6,Nasal shifting, bayonetting,NRR thinning	12	12	Open	Open	-3.86	-3.26		2.2	2.3	BE:NTG	
10	58	M	198668	DRIVER	0.3	0.2	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	20	16	Open	Open	-18.89	-18.9		2	2.3	BE:NTG	
11	50	M	189871	DRIVER	0.0	0.2	WNL	WNL	CD=0.6 ,Nasal shifting, bayonetting,NRR thinning	CD=0.6,Nasal shifting, bayonetting,NRR thinning	28	30	Open	Open	-3.86	-9.14	LE:ARCUATE SCOTOMA	2.3	2.2	BE:POAG	
12	38	F	194637	HOMEMAKER	0.3	0.3	WNL	WNL	CD=0.8,Nasal shifting, notching, laminar dot sign,RNFL defect+	CD=0.8,Nasal shifting, bayonetting,NRR thinning	16	18	Open	Open	-8.43	-5.5	RE:Ring scotoma	0.1	0.2	BE:POAG	
13	21	M	56505	Student	0.0	0.2	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	18	16	Open	Open	-3.26	-5.5		2	2	BE:POAG	
14	55	M	129024	CLERK	0.2	0.0	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	32	12	Open	Open	-30.49	-3.56	RE:BIARCUATE SCOTOMA	0.1	1.4	RE:POAG	PEX Glaucoma
15	45	M	198662	CLERK	0.2	0.2	WNL	WNL	CD=0.8,Nasal shifting, notching, laminar dot sign,RNFL defect+	CD=0.8,Nasal shifting, bayonetting,NRR thinning	28	20	Open	Open	-17.29	-7.62	RE:Arcuate scotoma	0.1	1.1	BE:POAG	
16	56	M	198664	Manager	0.0	0.3	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.6,Nasal shifting, bayonetting,NRR thinning	30	36	Open	Open	-3.5	-14.5		1.1	0.1	BE:POAG	

17	50	F	229322	TEACHER	0	0.0	WNL	WNL	CD=0.6 ,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	20	20	Open	Open	-3.8	-5.5		2.3	2.2	BE:NTG	
18	60	F	229344	TAILOR	0.3	0.2	VH grade 2	VH grade 2	CD=0.8,Nasal shifting, notching, laminar dot sign,RNFL defect+	CD=0.8,Nasal shifting, bayonetting,NRR thinning	16	12	Closed	Closed	-20.48	-20.3	BE:BIARCUATE SCOTOMA	0.1	0.1	BE:PACG	
19	30	M	198676	CLERK	0.2	0.3	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	17	14	Open	Open	-3.66	-3.99		2.3	2.3	BE:POAG	
20	48	F	198660	TEACHER	0	0.0	WNL	WNL	CD=0.6,Nasal shifting	CD=0.6,Nasal shifting	18	18	Open	Open	-6.6	-440		2	2.3	BE:NTG	
21	57	M	199497	CLERK	0.3	0	WNL	WNL	CD=0.6,Nasal shifting	CD=0.6,Nasal shifting	48	14	Open	Open	-5.5	-3.86		1.9	2.2	BE:POAG	
22	55	M	198645	TAILOR	0.3	0.2	WNL	WNL	CD=0.7,Nasal shifting, notching, laminar dot sign	CD=0.5,Nasal shifting	14	14	Open	Open	-3.88	-3.99		2	2.2	BE:POAG	
23	60	F	165838	CLERK	0.3	0	VH grade 2,diffuse conjunctival congestion, corneal oedema	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	44	16	Closed	Closed	-19.99	-440	RE:Ring scotoma	1.4	2.3	RE:PACG	
24	50	F	199492	Manager	0.3	0.2	VH grade 2	VH grade 2	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	30	20	Closed	Closed	-3.3	-3.86		1.9	2.3	BE:PACG	
25	55	M	199490	CLERK	0.2	0.2	WNL	WNL	CD=0.5 ,Nasal shifting, bayonetting,NRR thinning		20	18	Open	Open	-7.78	-8.43		2	2.3	LE:POAG	
26	58	F	190881	TAILOR	0.3	0.2	WNL	WNL	CD=0.6 ,Nasal shifting, bayonetting,NRR thinning	CD=0.6,Nasal shifting, bayonetting,NRR thinning	12	12	Open	Open	-3.3	-3.26		2	2.3	BE:NTG	
27	50	F	199762	DRIVER	0.3	0.2	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	20	16	Open	Open	-17.88	-3.86		1.1	2.3	BE:NTG	
28	38	F	199772	DRIVER	0.0	0.2	WNL	WNL	CD=0.6 ,Nasal shifting, bayonetting,NRR thinning	CD=0.6,Nasal shifting, bayonetting,NRR thinning	28	30	Open	Open	-3.86	-9.14	LE:ARCUATE SCOTOMA	2.3	1.9	BE:POAG	
29	21	M	193048	HOMEMAKER	0.3	0.3	WNL	WNL	CD=0.8,Nasal shifting, notching, laminar dot sign,RNFL defect+	CD=0.8,Nasal shifting, bayonetting,NRR thinning	16	18	Open	Open	-8.43	-5.5		0.4	0.4	BE:POAG	
30	55	F	193059	Student	0.0	0.2	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	18	16	Open	Open	-3.26	-5.5		2.3	1.9	BE:POAG	
31	45	M	193169	CLERK	0.2	0.0	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	32	12	Open	Open	-30.49	-3.56	RE:BIARCUATE SCOTOMA	0.1	1.4	RE:POAG	
32	56	M	193065	CLERK	0.2	0.2	WNL	WNL	CD=0.8,Nasal shifting, notching, laminar dot sign,RNFL defect+	CD=0.8,Nasal shifting, bayonetting,NRR thinning	28	20	Open	Open	-17.29	-7.62	RE:Arcuate scotoma	1.3	1.1	BE:POAG	

33	50	F	193094	Manager	0.0	0.3	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.6,Nasal shifting, bayonetting,NRR thinning	30	36	Open	Open	-3.5	-14.5		1.1	0.1	BE:POAG	
34	60	M	193599	TEACHER	0	0.0	WNL	WNL	CD=0.6 ,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	20	20	Open	Open	-3.8	-5.5		1.4	1.4	BE:NTG	
35	30	M	191467	TAILOR	0.3	0.2	VH grade 2	VH grade 2	CD=0.8,Nasal shifting, notching, laminar dot sign,RNFL defect+	CD=0.8,Nasal shifting, bayonetting,NRR thinning	16	12	Closed	Closed	-20.48	-20.3	BE:BIARCUATE SCOTOMA	0.1	0.2	BE:PACG	
36	48	F	194172	CLERK	0.2	0.3	WNL	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	17	14	Open	Open	-4.44	-28.4		2	2.3	BE:POAG	
37	55	F	193603	TEACHER	0	0.0	WNL	WNL	CD=0.6,Nasal shifting	CD=0.6,Nasal shifting	18	18	Open	Open	-11.09	-18.9		2.3	2.3	BE:NTG	
38	45	M	193599	CLERK	0.3	0	WNL	WNL	CD=0.6,Nasal shifting	CD=0.6,Nasal shifting	48	14	Open	Open	-20.19	-3.86		0.2	2.3	BE:POAG	
39	56	M	198674	TAILOR	0.3	0.2	WNL	WNL	CD=0.7,Nasal shifting, notching, laminar dot sign	CD=0.5,Nasal shifting	14	14	Open	Open	-15.17	-8.43		0.4	2	BE:POAG	
40	50	M	198667	CLERK	0.3	1/5	VH grade 2,diffuse conjunctival congestion, corneal oedema	WNL	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	44	16	Closed	Closed	-17.77	-3.26	RE:Ring scotoma	0.2	2.2	RE:PACG	
41	60	F	198661	Manager	0.3	0.2	VH grade 2	VH grade 2	CD=0.7,Nasal shifting, bayonetting,NRR thinning	CD=0.7,Nasal shifting, bayonetting,NRR thinning	30	20	Closed	Closed	-12.24	-18.9		0.4	0.4	BE:PACG	
42	30	M	198674	CLERK	0.2	0.2	WNL	WNL	CD=0.5 ,Nasal shifting, bayonetting,NRR thinning		20	18	Open	Open	-4.4	-3.86		1	2	LE:POAG	