"CORRELATION OF OPTIC DISC CHANGES WITH VISUAL FIELD DEFECTS IN PRIMARY OPEN ANGLE GLAUCOMA – A CROSS SECTIONAL STUDY."

REGISTRATION NO – BK0110004

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In partial fulfillment
of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

DEPARTMENT OF OPHTHALMOLOGY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM. KARNATAKA.

MAY - 2013

KLE UNIVERSITY BELGAUM, KARNATAKA

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Dr. S. B. PATIL M.S., D.O.M.S.
Professor & Head,
Department of Ophthalmology
J. N. Medical College,
Belgaum-590010
Date:
Place:

Dr. A. S. GODHI M.S., F.I.C.S.
Principal,
J. N. Medical College,
Belgaum-590010.
Date:
Place:

LIST OF ABBREVIATIONS

ACES	-	Aravind Comprehensive Eye Survey
ACG	-	Angle Closure Glaucoma
APEDS	-	Andhra Pradesh Eye Disease Study
asb	-	Apostilbs
C/D	-	Cup/Disc
CPSD	-	Corrected Pattern Standard Deviation
CNTGS	-	Collaborative Normal Tension Glaucoma Study
D	-	Diopter
dB	-	Decibels
DM	-	Diabetes Mellitus
EMGT	-	Early Manifest Glaucoma Trial
FN	-	False Negative
FP	-	False Positive
FT	-	Full Threshold
GHT	-	Glaucoma Hemifield Test
HAP	-	Hodapp Anderson Parrish
HFA	-	Humphrey Field Analyzer
HRT	-	Heidelberg Retinal Tomography
H2O2	-	Hydrogen Peroxide

IPD	-	In Patient Department
IOP	-	Intraocular Pressure
ISNT	-	Inferior Superior Nasal Temporal
MD	-	Mean Deviation
NFL	-	Nerve Fiber Layer
NTG	-	Normal Tension Glaucoma
NRR	-	Neuroretinal Rim
OAG	-	Open Angle Glaucoma
OCT	-	Optical Coherence Tomography
OHTS	-	Ocular Hypertension Treatment Study
ONH	-	Optic Nerve Head
OPD	-	Out Patient Department
PD	-	Pattern Deviation
POAG	-	Primary Open Angle Glaucoma
PPA	-	Peripapillary Atrophy
PSD	-	Pattern Standard Deviation
RCT	-	Randomized Control Trial
RGC	-	Retinal Ganglion Cells
SAP	-	Standard Achromatic Perimetry
SITA	-	Swedish Interactive Threshold Algorithm
SS	-	SITA Standard

STF	-	Short Term Fluctuation
SWAP	-	Short Wavelength Automated Perimetry
TD	-	Total Deviation
χ^2	-	Chi square

ABSTRACT

BACKGROUND

Primary open angle glaucoma (POAG) is a progressive chronic optic neuropathy in adults where intraocular pressure (IOP) and other currently unknown factors contribute to damage and in which in the absence of other identifiable causes, there is characteristic acquired atrophy of the optic nerve and loss of retinal ganglion cells and their axons with a corresponding visual field loss. This loss is usually very insidious and starts in the Bjerrum's area and leaves the patients asymptomatic in the early stages. The importance of the correlation of the optic disc changes with the visual field defects in primary open angle glaucoma cannot be stressed enough and is the need of the hour to help in detecting the glaucomatous changes early in the course of the disease and thereby help prevent blindness.

OBJECTIVES

1. To correlate the optic disc parameters with the visual field defects in primary open angle glaucoma patients using magnified photographs of the optic disc and Humphrey Field Analyzer.
2. To look for the features of the optic disc commonly associated with early visual field loss by Humphrey Visual Field Analyzer II using Swedish Interactive Threshold Algorithm program.

METHODS

This was a cross sectional study in which 96 eyes of 48 patients of primary open angle glaucoma who met the inclusion criteria were included. After obtaining written informed consent, all patients underwent a detailed optic disc evaluation by slit lamp biomicroscopy and visual field analysis with SITA Standard algorithm using Humphrey Visual Field Analyzer II - i series perimeter.

RESULTS

On correlating the type of NRR notching with the visual field defects (scotomas) using Kappa statistics, the agreement of the visual field defects with the morphometric optic disc parameter (NRR notching) was good (0.959) and statistically significant $p < 0.001$. When the increasing mean deviation (dB) was correlated with the larger cup:disc ratio using chi square test, it was found to be statistically significant with $p < 0.001$, $\chi^2 = 76.753$, DF = 6. When the increasing IOP was correlated with larger C:D ratio, p value was found to be < 0.001 which indicates statistically significant good correlation between IOP and C:D ratio.

CONCLUSION

The optic disc parameter neuroretinal rim notching correlated best with the visual field defects and is a optic disc variable superior to the cup/disc ratio in correlation with visual field defects. The earliest glaucomatous disc change is inferior neuroretinal rim notching and is a good optic disc parameter to differentiate between ocular hypertensives and POAG patients.

A large cup/disc ratio is not necessarily a sign of visual field loss. However, the correlation between increasing cup/disc ratio and increasing IOP is good, suggesting that concentric enlargement of the optic cup is largely pressure dependent. The mean deviation by HFA correlates well with the cup/disc ratio in advanced glaucoma more than in early glaucoma.

KEY WORDS: Primary open angle glaucoma; Neuroretinal rim; visual field defects; correlation.

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INTRODUCTION

Primary open angle glaucoma (POAG) also known as chronic simple glaucoma is typically characterized by an open, normal appearing anterior chamber angle and increased intraocular pressure (IOP) without any apparent ocular or systemic abnormality that might account for the elevated intraocular pressure and typical optic nerve head damage or glaucomatous visual field damage. A proposed definition of POAG, modified from the American Academy of Ophthalmology Preferred Practice Guidelines, 2005 is a multifactorial optic neuropathy in which there is characteristic atrophy of the optic nerve.

In most cases, the optic disc changes occur prior to visual field loss, [1] so the assessment of the optic disc is a very important part of the evaluation of glaucoma and ocular hypertension patients. Early glaucomatous optic disc changes are delicate, so techniques of estimation of optic discs of persons who are suspected or diagnosed as having glaucoma are requested. [2] The onset and progression of glaucoma are generally assessed by means of visual field tests and optic disc evaluation. These procedures make it possible to identify the occurrence of a diffuse or localized depression of retinal sensitivity and of structural damage to the optic disc like enlargement of cupping and focal or diffuse narrowing of the neuroretinal rim.[3]

The visual field loss in POAG is usually very insidious and starts in the Bjerrum's area and the patients are usually asymptomatic in the early stages. Having a sensitive and specific test to detect a subtle visual field loss is therefore very important in the management of glaucoma.

Sommer et al [4] observed the presence of retinal nerve fibre layer defects six years before field loss in 60% of their patients with ocular hypertension at baseline. Only a few studies have quantitatively compared the rates of disc and visual field changes among patients with glaucoma. [5] Their results indicate that measurable optic disc changes precede visual field loss in early glaucoma and in advanced stages, serial optic disc evaluation does not seem as sensitive as visual field examination in detecting further progression of glaucoma.

Visual field analysis and optic disc evaluation are the standard examination techniques used to detect the onset and progression of glaucoma. [3]

The various methods of static perimetry adopted are Goldmann perimetry, Friedmann perimetry and automated perimetry. Currently the most widely used instrument in visual field evaluation is Humphrey Visual Field Analyzer (HFA), and most common type of perimetry used is static perimetry.[6] The Swedish Interactive Threshold Algorithm (SITA) is a new computer program that has been developed for the Humphrey Visual Field Analyzer II, which reduces test taking time. Careful examination of magnified photographs of the glaucomatous discs allows prediction of the presence or absence of visual field loss. There are some errors in previous studies, such as inherent errors in visual field examination, particularly with the isolated paracentral scotoma and these may have contributed to incorrect predictions.

The purpose of the current study is to correlate the optic disc changes and the visual field defects in primary open angle glaucoma to help in detecting the glaucomatous changes early in the course of the disease and thereby help prevent blindness.

AIMS AND OBJECTIVES

1. To correlate the optic disc parameters with the visual field defects in primary open angle glaucoma patients using magnified photographs of the optic disc and Humphrey Field Analyzer.
2. To look for the features of the optic disc commonly associated with early visual field loss by Humphrey visual field analyzer II using Swedish Interactive threshold Algorithm program.

REVIEW OF LITERATURE

Primary open angle glaucoma (POAG) is defined by the American Academy of Ophthalmology's 2005 POAG Preferred Practice Pattern as 'a progressive chronic optic neuropathy in adults where intraocular pressure (IOP) and other currently unknown factors contribute to damage and in which in the absence of other identifiable causes, there is characteristic acquired atrophy of the optic nerve and loss of retinal ganglion cells and their axons'. This is associated with an anterior chamber angle that is open by gonioscopic appearance and specific visual field defects.

Early or mild glaucoma is defined as having characteristic optic nerve abnormalities with normal visual fields. The American academy of ophthalmology defines moderate glaucoma as having visual field defects in one hemifield, not within 5° of fixation.

Severe glaucoma is defined as visual field abnormalities in both hemifields or loss within 5° of fixation.

Historical background.

Glaucoma has been known in medicine since antiquity. Hippocrates described "glaucosis" as blindness which occurs in the elderly. The English ophthalmologist Dr. Banister was the first to establish the connection between increased tension of the eyeball and glaucoma.

In 1862 Donders discovered that high intraocular pressure caused blindness and called the disease "Glaucoma simplex".

The first effective surgical treatment, an iridectomy was carried out by Von Graefe in 1856.

Epidemiology

Glaucoma afflicts more than 67 million people worldwide, of whom about 10% or 6.6 million are estimated to be blind. Glaucoma is the second most frequent cause of blindness in the world, after cataract. [7]

The overall prevalence of POAG in 2010 was 1.96% of total general population, disproportionately affecting more women and Asians.[8] There were 44.7 million people with POAG of the total predicted 60.5 million people with both OAG and ACG in 2010.[8]

A significant number of glaucoma patients go undiagnosed as high as 50%-75% as reported by Baltimore Eye study and Latinos study.

Prevalence of glaucoma in India

The prevalence of glaucoma in India estimated by the population based studies is about 11.2 million persons aged 40 years and older in 2010. [9]

Among them primary open angle glaucoma is estimated to affect 6.48 million persons in 2010.[9]

Glaucoma has been declared to be the second common cause of blindness in adult population in India. [9]

The high rate of blindness in the Indian population is due to high proportion of undiagnosed glaucoma in the community.

The Aravind Comprehensive Eye Survey (ACES), a population based sample survey in rural pockets of Tamilnadu had determined prevalence of POAG to be 1.4% in adults aged 40 years and more.

The Andhra Pradesh Eye Disease Study (APEDS), a population based sample survey in urban Hyderabad had identified prevalence of POAG to be 1.62% in those aged 30 years or more.

Clinical risk factors for primary open angle glaucoma

Demographic risk factors for POAG

Age

Population based studies of prevalence and incidence of POAG consistently show a steady increase with age. As a rule of thumb, prevalence tends to roughly double for each decade over 40 years and is about 10-fold higher in the 80+ group compared to the 40 to 49 year old group.

Gender

There is a marked discordance amongst population based studies on the association between gender and POAG. The Framingham [10] study reported higher rates of POAG amongst males.

The Blue mountains [11] and St Lucia [12] studies reported higher rates in females. Others found no significant statistical association. [13, 14, 15]

Race

In general, the prevalence of POAG is highest in black populations, intermediate in whites, Hispanics and southern Asian populations and lowest in northern Asian populations.

Family history

A first degree relative with glaucoma has been consistently associated with an increased risk of POAG in prevalence surveys. [16, 17, 18]

The association between POAG and family history of POAG is stronger when the affected relative is a sibling rather than a parent or child. However, in the clinically assembled OHTS population no association between family history of POAG and POAG was found.

Genetic factors that influence POAG are complex. Although at least six genes loci have been identified with POAG, only one genetic locus GLC1A on chromosome 1q has been reported in patients with adult onset POAG. A gene that produces the protein myocilin resides within this interval, and myocilin mutations occur in upto 4.6% of patients with adult onset POAG.

Ocular risk factors

Intra ocular pressure (IOP mm Hg)

There is a strong dose response relationship between IOP and glaucoma that has consistently been shown in prevalence surveys and in longitudinal studies of incidence and progression; individuals who have pressures of 15-20 mm Hg have a low prevalence of nerve damage, whereas the prevalence of damage is higher among individuals who have pressures of 25-30 mm Hg. The most decisive new evidence to be published in recent years was demonstration by RCTs that IOP lowering decreased the incidence and progression of glaucoma compared to no treatment.

Among population based studies, the prevalence of POAG increases with increasing IOP. The causal role of IOP in optic nerve damage is evidenced by

experimental production of high pressure in primates that results in glaucomatous damage.

Corneal thickness

Corneal thickness is a risk factor for conversion from ocular hypertension to open angle glaucoma.[19] The people of African ancestry had thinner corneas and this accounted for all of the increased risk for conversion to open angle glaucoma among blacks with ocular hypertension.

Optic nerve head features

A study reported that the incidence of POAG for those with a baseline C/D ratio of more than 0.7 was 8.6 fold higher than for those with a C/D ratio of less than 0.7. [20]

The OHTS showed a 1.4 fold increase in the incidence of POAG among ocular hypertensive patients for every 0.1 unit increase in the baseline C/D ratio.[21] Also optic nerve head vertical disc diameter and the disc area are found to be associated with glaucoma.

Atrophy of neurosensory retina and retinal pigment epithelium about the optic nerve head is known as peripapillary atrophy (PPA) and has been shown to correlate with the presence of glaucoma.[22]

The prevalence of glaucoma was found to be increased 10-fold in those with disc hemorrhages but disc hemorrhages were much more common in NTG (25%) than in high tension glaucoma (8%).

Myopia

An association between myopia, particularly high myopia and open angle glaucoma has long been recognized and is supported by numerous case series and case control studies. However, the OHTS and EMGT studies have shown no association of glaucoma with myopia.

Systemic risk factors for POAG

Diabetes mellitus

The prevalence of POAG appears to be higher in the diabetic population by a factor of about 2 in most of the population based surveys. Diabetes has not yet shown to increase the incidence of glaucoma.

Blood pressure

The most meaningful blood pressure variable related to glaucoma appears to be diastolic perfusion pressure or the difference between diastolic arterial pressure and IOP. Several population based surveys have reported a seven-fold increase in the prevalence of POAG in those with lower perfusion pressures. Higher systolic and diastolic blood pressures are associated with increased IOP. [23, 24]

Migraine

There is some support for an association between migraine headaches and NTG. The CNTGS found that a history of migraine increased the risk of progression of POAG by a factor of 2.6.

Thyroid disease

In one study, thyroid disease was associated with chronic thyroid ophthalmopathy.

Smoking and alcohol

No difference in the prevalence of glaucoma were noted with mild, moderate or heavy alcohol consumption in the Beaver Dam Eye Study.[25] While a small increase in IOP was noted in smokers in the Australian Blue Mountain Eye study even after adjusting for numerous other variables,[26] the prevalence of POAG has not been observed to vary between smokers and non smokers in other studies.

Anatomy of the optic nerve head (ONH) and the retinal nerve fiber layer (RNFL)

The optic nerve head comprises of the nerve fibers that originate in the ganglion cell layer of the retina and converge upon the nerve head from all points in the fundus. At the surface of the nerve head, these retinal ganglion cell (RGC) axons bend acutely to exit the globe through a fenestrated scleral canal, called the lamina cribrosa. There is considerable variation in the size of the optic nerve head. The disc area may range from 0.68 mm^2 to 4.42 mm^2.

Divisions of the optic nerve head

- Surface nerve fiber layer
- Prelaminar region
- Lamina cribrosa region
- Retrolaminar region

Arterial supply

Posterior ciliary artery is the main source of blood supply to the optic nerve head, except for the retinal nerve fiber layer which is supplied by the retinal circulation. The blood supply in the optic nerve head has a sectoral distribution.

Capillaries

These are derived from both the retinal as well as the ciliary circulations. They resemble more closely the features of retinal capillaries than of the choriocapillaris.

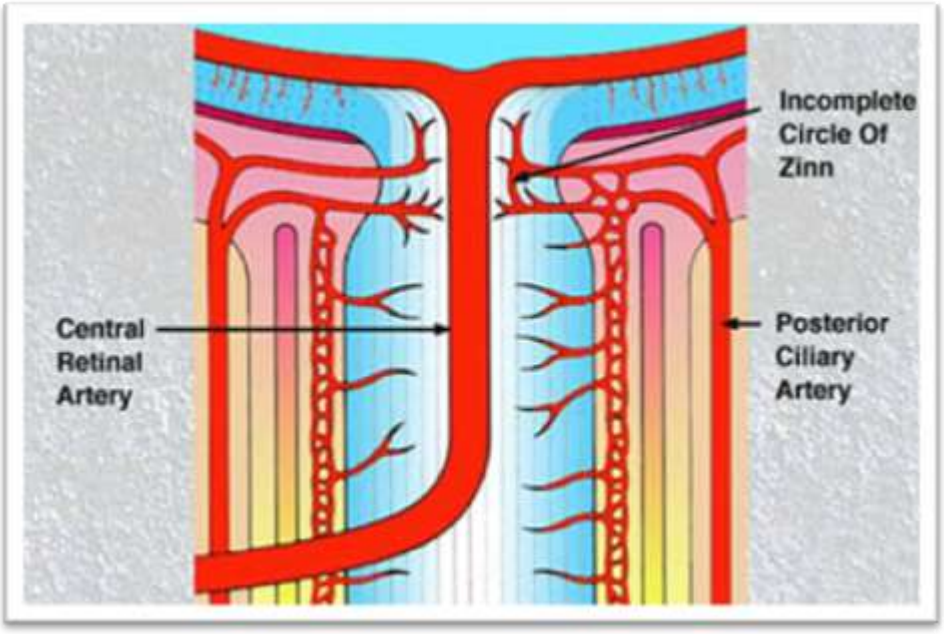


Figure 1. Arterial supply of the optic nerve head

Venous drainage

The venous drainage of the optic nerve head is almost entirely through the central retinal vein.

Astroglial support

Astrocytes provide a continuous layer between the nerve fiber and blood vessels in the optic nerve head. They provide a covering for the portions of the optic nerve head. Astrocytes also play a major role in the remodeling of the extracellular matrix of the optic nerve head and synthesizing growth factors and other cellular mediators that may affect the axons of the RGC's and contribute to the health or susceptibility to the disease.

Retinal nerve fiber layer

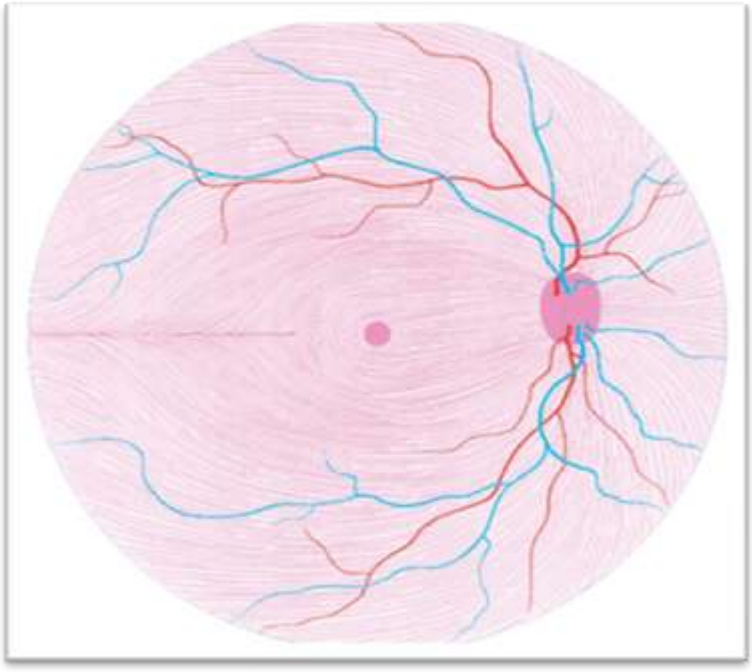


Figure 2. Arrangement of retinal nerve fiber layer

As the axons traverse the nerve fiber layer from the ganglion cell bodies to the optic nerve head , they are distributed in a characteristic pattern.

Fibers from the temporal periphery originate on either side of a horizontal dividing line, the median raphe, and arch above or below the fovea as the arcuate nerve fibers, while those from the central retina, the papillomacular fibers and the nasal fibers take a more direct path to the nerve head. The arcuate nerve fibers occupy the superior and inferior temporal portions of the optic nerve head, with axons from the peripheral retina taking a more peripheral portion in the nerve head. The arcuate nerve fibers are the most susceptible to early glaucomatous damage. The papillomacular fibers spread over approximately one third of the distal optic nerve, primarily inferotemporally, where the axonal density is the higher. They intermingle with the extramacular fibers, which may explain the retention of central vision in early glaucomatous optic atrophy.

Pathogenesis

Raised IOP

A raised IOP results from resistance to the aqueous humor outflow. This obstruction to the aqueous humor outflow in POAG is associated with alterations in conventional outflow pathway. In the trabecular meshwork there is a decrease in the endothelial cell number, alteration in endothelial cell function, decrease in inter trabecular spaces, loss of the trabecular beams and thickening of basement membrane which are all associated with an increased resistance to aqueous outflow resulting in raised IOP. In POAG, within the trabecular meshwork, collagen abnormalities like fragmentation, orientation changes and abnormal spacing are noted.

Diminshed aqueous outflow

1) An obstruction of the trabecular meshwork by foreign material. Several investigators have noted the accumulation of foreign material in the trabecular meshwork and juxtacanalicular tissue, including pigment, red blood cells, glycosaminoglycans, amorphous material[27], extracellular lysosomes, plaquelike material, [28] and protein. [29]
2) A loss of trabecular endothelial cells.
3) A reduction in pore density and size in the inner wall endothelium of Schlemm's canal.
4) A loss of giant vacuoles in the inner wall endothelium of Schlemm's canal. Giant vacuoles may play a crucial role in moving fluid from the meshwork into the lumen of Schlemm's canal.
5) A loss of normal phagocytic activity.
6) Disturbance of neurologic feedback mechanisms.

Altered corticosteroid metabolism

The corticosteroid hypothesis has been extended to include a generalized sensitivity to the effects of glucocorticoids in patients with POAG.

Dysfunctional adrenergic control

In analogous fashion to the corticosteroid theory, others have proposed that the diminished outflow facility in patients with POAG could be explained by an increased sensitivity to adrenergic agonists.

Oxidative damage

The meshwork contains glutathione, which may protect the endothelial cells from the effects of hydrogen peroxide (H2O2) and other oxidants. This interesting hypothesis is still the subject of active research.

Optic nerve cupping and atrophy

Cupping consists of backward bowing of the lamina cribrosa, elongation of the laminar beams, and loss of the ganglion cell axons in the rim of neural tissue. Histologic studies indicate that optic nerve cupping includes the loss of all three elements of the disc – axons, blood vessels, and glial cells. Most authorities believe that the lamina cribrosa is the site of glaucomatous optic nerve damage

The ischaemic theory postulates that compromise of the microvasculature with resultant ischaemia in the optic nerve head is responsible. Loss of capillaries or alteration in capillary blood flow causes interference with the delivery of nutrients or removal of metabolic products from axons, failure of blood flow regulation and delivery of hostile vasoactive substances to the blood vessels of the optic nerve head.

The direct mechanical theory suggests that raised IOP directly damages the retinal nerve fibers as they pass through the lamina cribrosa.

Diagnosis and clinical features

POAG is diagnosed by assessing a combination of clinical factors including the level of intraocular pressure (IOP), optic disc appearance and visual field damage.

History

A comprehensive evaluation of an individual with glaucoma should begin with eliciting detailed history which includes a review of the family, ocular and systemic history, use of systemic and ocular medications, past ocular surgery and known local and systemic intolerance to the use of glaucoma medications.

Clinical evaluation

Vision and refraction

Best corrected visual acuity is to be determined. Correction of refractive error is essential for accurate perimetry.

Pupils

Relative afferent pupil defect detects asymmetric optic nerve damage and is an important finding in glaucoma.

Slit lamp biomicroscopy

Anterior segment examination is usually normal in POAG.

Intraocular pressure (IOP mm Hg)

The mean value of IOP in a large normal population is 16 mmHg, with a standard deviation of 3 mmHg.

IOP in general population is not Gaussian in distribution but skewed toward higher pressures and an IOP > 22 mm Hg does not necessarily indicate abnormality.

IOP is preferably measured with Goldmann type of applanation tonometry or its equivalent before performing gonioscopy or dilatation of the pupil. In case

Goldmann applanation tonometer is not available, Schiotz tonometer offers a viable alternative. Time of IOP measurement is to be noted.

Gonioscopy

Gonioscopic evaluation of the anterior chamber angle is an essential diagnostic tool in glaucoma. Goldmann 3 mirror goniscopic lens is the prototype instrument employed in viewing the anterior chamber angle.

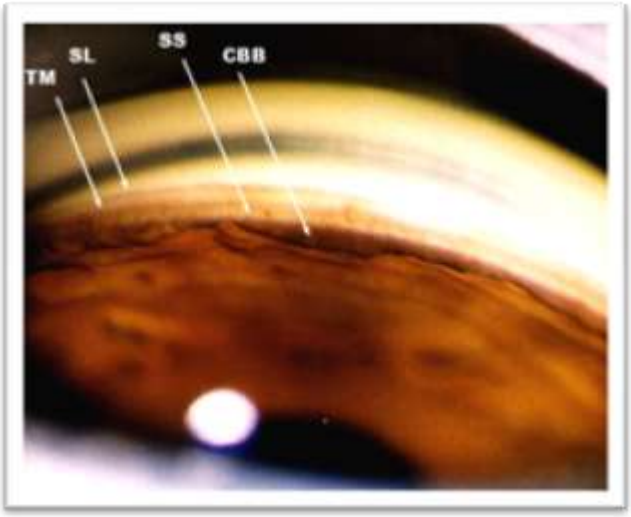


Figure 3. Anterior chamber angle structures visible on gonioscopy

Shaffer's system of grading anterior chamber angle

Grade Number	Angle width	Description	Risk of closure
4	45-35	Wide open	Impossible
3	35-25	Wide open	Impossible
2	20	Narrow	Possible
1	10	Extremely narrow	Probable
Slit	Slit	Narrow to slit	Probable
0	0	Closed	Closed

Optic disc evaluation

A magnified, preferably stereoscopic, examination of the optic disc using a 60-90 diopter (D) lens or a contact lens with the slit lamp is the ideal method of examining the ONH. Retinal examination also requires an indirect ophthalmoscope. The indirect ophthalmoscope alone is not good enough to comment on the optic disc. In experienced hands, the direct ophthalmoscope can provide valuable information too.

Stereo photographs are the current gold standard but the optic disc findings should at least be documented, preferably with a drawing or imaging for comparison with future examinations. The size of the disc can be easily estimated on the slit lamp with a 60 D lens. The magnification factor for the 60 D lens is 1, for 78 D is 1.13 and for 90 D is 1.41. A narrow slit-beam height is adjusted vertically till it just encompasses the margins of the optic disc.

The optic disc is slightly oval in shape and it contains a central area of pallor, the optic cup. The tissue between the cup and disc margin is called the neuroretinal rim. The neuroretinal rim is composed of axons of the retinal ganglion cells that exit the eye through optic nerve. Death or destruction of the retinal ganglion cells, as in glaucoma, is reflected by loss or thinning of neuroretinal rim and enlargement of the cup.

Optic disc signs in POAG

Generalized

- Large optic cup
- Asymmetry of cups between the two eyes
- Progressive enlargement of the cup

Focal

- Narrowing or notching of the neuroretinal rim
- Vertical elongation of cup
- Cupping up to the rim margin
- Regional pallor
- Splinter hemorrhage
- Nerve fiber layer defects
- Bayoneting of retinal vessels
- Overpass vessel phenomenon

Non specific signs of glaucomatous damage

- Exposed lamina cribrosa (laminar dot sign)
- Nasal displacement of retinal vessels
- Baring of circumlinear vessels
- Peripapillary crescent

Generalized enlargement of cup may be the earliest change detected in glaucoma. Focal enlargement of the cup may appear as localized narrowing or notching of neuroretinal rim. Deep or localized notching, where lamina cribrosa becomes visible at the disc margin is referred to as acquired optic disc pit. The cup becomes vertically oval if notching occurs at one or both of the inferior and superior aspect of the optic disc. Notching or thinning and loss of neuroretinal rim is associated with bayoneting of retinal vessels.

Disc hemorrhages appear as linear red streaks at or near the disc margin. Individuals with normal tension glaucoma are more likely to have such changes than

those with POAG.[30] Most eyes with disc hemorrhages tend to a have progressive visual field loss and hence it is a reliable prognostic sign.

Neuroretinal rim evaluation

Loss of axons in glaucoma is reflected as abnormalities of the neuroretinal rim. NRR is usually thickest inferiorly, followed by superior, nasal and temporal quadrants. This is the "ISNT" rule, seen in 80% of the normals. While that is not specific enough, and there may be normal variations, any change in this pattern is suspicious. If the inferior rim is thinner than the superior, that could suggest pathology. Certainly, an inferior or superior rim that is equal to or thinner than the temporal rim is highly suspicious. The temporal rim should be the thinnest. Localized narrowing of the inferior or superior rim that does not extend to the rim is also suspicious. If the rim extends to the edge of the disc for a clock hour it is called a notch. A notch is characteristic of glaucoma and usually produces a functional field defect.

Advanced glaucomatous cupping

If the progressive changes of glaucomatous optic atrophy are not arrested by appropriate measures to reduce the IOP, the typical course is eventual loss of all neural rim tissue. The ultimate result is total cupping, which is clinically seen as white disc with loss of all neural rim tissue and bending of all vessels at the margin of the disc. This has also been called “bean pot” cupping.

Retinal nerve fiber layer loss

Glaucomatous optic atrophy is associated with loss of axons in nerve fiber layer. The gold standard for the examination of the NFL is red free photography, but

the NFL can be examined clinically using the green filter on the slit lamp or ophthalmoscope. It is sometimes clearly seen on indirect ophthalmoscopy too, both with and without the green filter. The normal arcuate NFL is seen as fine bright striations. When viewed from the superior to the inferior arcuate area, the NFL has a bright, dark, bright pattern, the "dark" being the region between the disc and the macula. The inferior arcuate NFL has a larger area and is more clearly seen than the superior arcuate NFL, consistent with the NRR thickness.

Nerve fiber layer defects appear as dark stripes or wedge shaped defects of varying width in peripapillary area or as diffuse loss of striations and these correlate well with the visual field changes.[31] Focal abnormalities consist of slit like grooves or wedge defects. Diffuse nerve fiber loss is more common in glaucoma but is also difficult to appreciate. The nerve fiber layer defect may be diffuse or localized and may be the initial sign of glaucomatous damage. [32]

The optic nerve and/or nerve fiber layer imaging techniques include the Heidelberg Retinal Tomograph (HRT III), OCT and the NFL Analyzer (GDx VCC). The World Glaucoma Association consensus on imaging states that these instruments lack the sensitivity and specificity for routine clinical use. In the hands of experts, however, they may provide valuable clinical information. These instruments can help corroborate our suspicions and help support our diagnosis, but are not required for routine clinical diagnosis.

Visual field analysis

Visual field evaluation is a complex task that has become an integral part of glaucoma evaluation.

Visual field testing is used in three distinct ways in glaucoma evaluation and management: diagnosis, assessment of severity and determination of progression.

In 1939, Traquair cleverly described a normal visual field as an island of vision surrounded by a sea of blindness.

The 'top of island ' corresponds to fovea with highest light sensitivity (fixation point); it declines towards periphery, and the bottom corresponds to the peripheral visual field with the lowest light sensitivity.

The normal visual field has an oval shape: temporal field extends to 100-110 degrees, inferior to 70-75 degrees, and superior and nasal to 60 degrees.

A blind spot is an absolute scotoma corresponding to optic nerve head and is located 15 degrees temporal to fovea.

Historical considerations

The concept of visual field dates to the fifth century BC, when Hippocrates described hemianopic defects. As early as 150 BC, Ptolemy attempted to measure the visual field.

In 1801, Thomas Young made the first genuine measurements of the visual field.

In the mid nineteenth century, Foster developed the first arc perimeter, which predominated until 1889 when Bjerrum discovered the tangent screen. In the 1950s, Goldmann discovered hemispheric projection perimeter which became the clinical standard for the next 30 years.

Lynn and Tate demonstrated the first static automated perimeter in 1969. Fankhauser, Heijl and Krakau are credited for the rapid development of automated static perimetry.

Glaucomatous visual field defects

Glaucomatous field damage results from damage to the intraocular portion of the optic nerve extending from the retinal ganglion cells to just posterior to the lamina cribrosa.

Peripheral loss

Defects along the peripheral boundaries of the visual field (peripheral nasal steps, vertical steps and temporal sector defects) are most often found in association with scotoma in the more central arcuate area, although in some patients with early glaucomatous visual field loss, peripheral defects may be the only detectable abnormality.

Localized visual field defects

The glaucomatous process typically causes initial damage to one or more axon bundles, creating a localized visual field defect which constitute the most definitive early evidence of visual field loss from glaucoma.

Arcuate defects

Bjerrum described an arcuate visual defect, which he showed is strongly suggestive of glaucoma. This arcuate scotoma starts from the blind spot and arches above or below fixation, or both, to the horizontal median raphe, corresponding to the arcuate retinal nerve fibers.

Early visual loss in glaucoma commonly occurs within this arcuate area, especially in the superior half which correlates with predilection of the inferior and superior temporal poles of the optic nerve head for early glaucomatous damage.[33] They most often appear first as one or more localized defects, or paracentral scotomas.

Occasionally, the early arcuate defect may connect with the blind spot and taper to a point in a slightly curved course, which has been referred to as a seidel scotoma.

As the isolated defects enlarge and coalesce, they form an arching scotoma that eventually fills the entire arcuate area from the blind spot to the median raphe, which is called an arcuate or Bjerrum scotoma.

With further progression, a double arcuate (or ring) scotoma develops. Although the arcuate defect is probably the most reliable early form of glaucomatous field loss it is not pathognomonic.

Nasal steps

The loss of retinal nerve fibers rarely proceeds at the same rate in the upper and lower portions of an eye. Therefore, a step like defect is frequently created where the nerve fibers meet along the median raphe. Because the superior field is involved somewhat more frequently than the inferior portion in the early stages of glaucoma, the nasal step more often results from a greater defect above the horizontal midline, which is referred to as a superior nasal step. However, inferior nasal steps are not uncommon.

Unequal contraction on the peripheral side of the defect due to loss of corresponding bundles of peripheral arcuate nerve fibers, produces a defect that has been called the peripheral nasal step of Ronne. Nasal step often begins as an isolated scotoma in nasal periphery.[34]

Vertical step

A stepwise defect along the vertical midline, referred to as a vertical step or hemianopic offset, is a less common feature of glaucomatous field loss than the nasal step, but has been reported to occur in approximately 20% of cases.[35]

Generalized and central depression of the visual field

Central vision is typically one of the last regions to be totally lost, but studies have shown mild central and diffuse reduction in the visual field even in the early stages of glaucoma.[36,.37, 38] The mechanism for this is uncertain, although it appears to represent pressure induced damage, with diffuse nerve fiber loss.

Concentric contraction

Generalized reduction in the visual field may become manifest as a concentric constriction of the visual field which has been noted to precede other detectable glaucomatous field defects in many patients.[39]

Isopter contraction, as an early field defect of glaucoma, is often more marked in the nasal field, which has been called "crowding of the peripheral nasal isopters".[40]

Enlargement of the blind spot

Enlargement of the blind spot, due to depression of peripapillary retinal sensitivity, is also considered to be an early glaucomatous field change.

Enlargement of the blind spot can also be produced in normal individuals with threshold targets so that it is not a pathognomonic sign of glaucoma. [41]

Angioscotomata

Angioscotomata are long, branching scotomas above and below the blind spot which are presumed to result from shadows created by the large retinal vessels. Angioscotomata may represent an early glaucomatous visual field defect, [42] although it is technically difficult to demonstrate and not highly diagnostic.

Temporal sector defect

Because the retinal nerve fibers nasal to the optic nerve head converge on the disc by a direct route, a lesion involving these fiber bundles produces a sector defect temporal to the blind spot.[43] This defect usually appears later in the course of glaucomatous field loss.

Advanced glaucomatous field defects

The natural history of progressive glaucomatous field loss is the eventual development of a complete double arcuate scotoma, which coalesce nasally at the horizontal meridian and may extend to the peripheral limits in all areas except temporally. This results in a central island and a temporal island of vision in advanced glaucoma.

With continued damage, these islands of vision progressively diminish in size until the tiny central island is totally extinguished, which may occur abruptly. The temporal island of vision is more resistant and may persist long after central vision is lost. However, it too will eventually be destroyed if the glaucoma is not controlled, leaving the patient with no light perception.

Visual field testing and glaucoma diagnosis

Visual field testing is critical in diagnosis of glaucoma Changes in the optic disc appearance in the form of focal or diffuse loss of neuroretinal rim resulting in an increase in size of cup, while diagnostic for glaucoma, generally take years to develop and it is difficult to document this change.

A guideline for minimal abnormality on a visual field required to make the diagnosis of glaucoma by SAP are

1. Abnormal glaucoma hemifield test
2. Pattern standard deviation abnormal at P<5% level
3. Cluster of 3 or more points on pattern deviation plot abnormal at P<5 %, at least 1 at the P<1% level in an expected area of the visual field.

The first two criteria, the glaucoma hemifield test and pattern standard deviation, were used in the Ocular Hypertension Treatment Study (OHTS) as visual field endpoints for the development of primary open angle glaucoma. [44]

The latter criterion, pointwise analysis may be more sensitive to very early defects.

If any of the three criteria listed are present in the absence of other causes, such as nonglaucomatous optic neuropathies or chorioretinal pathology, then diagnosis of glaucoma should be considered.

False positive testing in glaucoma suspects and ocular hypertensives has been found to be fairly common, so repeat testing of a suspected early visual field defect is important before concluding that glaucoma is present. In the OHTS, for instance 86% of suspected new visual field defects disappeared on repeat testing. [45]

Most defects are localized taking the form of a paracentral scotoma or nasal step. As glaucoma progresses, these defects typically enlarge and coalesce into arcuate defects, corresponding to thinning of the retinal nerve fiber layer.

The best place to look for early glaucomatous visual field defects is the pattern deviation plot rather than the gray tone printout or total deviation plot.

Visual field and assessing the severity of glaucoma

The severity of the visual field defects can be used to judge the severity of functional damage to the visual system from glaucoma.

This is important to determine the aggressiveness of initial therapy and to assess the success of ongoing treatment. There are several visual field severity grading scales including those used in the Advanced Glaucoma Intervention Study, the Collaborative Initial Glaucoma Treatment Study, the Glaucoma Staging System developed by Brusini and colleagues and the Hodapp Anderson Parrish system. [46]

The preferred one to use is an expanded version of visual severity scale proposed by Hodapp Anderson and Parrish for the Humphrey Field Analyzer.

This scale is easy to use in clinical practice, captures the full range of stages in glaucoma progression and is based on three distinct features of the visual field that should be assessed when deciding on the severity of the functional loss, which include the size of the defect, the depth of the defect, and the proximity of the defect to fixation.

The pattern of visual field loss progression in glaucoma, from early to severe, is predictable in light of the pattern of thinning of the retinal nerve fiber layer which

usually begins with the superior and inferior arcuate fibers, then the papillomacular bundle, and finally the nasal fibers.

When the arcuate fibers are lost, the visual field shows only the central and temporal islands remaining which correspond to the remaining papillomacular and nasal fibers, respectively.

The next fibers to be lost are typically the papillomacular fibers which results in central visual acuity loss and eccentric fixation.

Visual field severity grading system for the Humphrey visual field analyzer (Stage 0-5): [47]

Stage O: No or minimal defect/Ocular Hypertension.

Does not meet any criteria for stage 1

Stage 1: Early defect

Mean deviation (MD) ≤-6.00 dB and at least one of the following

A On pattern deviation plot, there exists a cluster of 3 or more points in an expected location of the visual field depressed below the 5% level at least 1 of which is depressed below the 1% level.

B Corrected pattern standard deviation/pattern standard deviation significant at P<0.05.

C Glaucoma hemifield test "outside normal limits".

Stage 2: Moderate defect

MD of -6.01 to 12.00 dB and at least one of the following

A On pattern deviation plot, greater than or equal to 25% but fewer than 50% of points depressed below the 5% level, and greater than or equal to 15% but fewer than 25% of points depressed below 1% level.

B At least 1 point within central 5^0 with sensitivity of <15 dB but no point within central 5^0 with sensitivity of <0 dB.

C Only 1 hemifield containing a point with sensitivity <15 dB within central 5^0 of fixation .

Stage 3 : Severe defect

MD of -12.01 dB to – 20.00 dB and at least one of the following :

A On pattern deviation plot, greater than or equal to 50% but fewer than 75% of points depressed below the 5% level and greater than or equal to 25% but fewer than 50% of points depressed below 1% level .

B Any point within central 5^0 with sensitivity of < 0 db.

C Both hemifields containing a point(s) with sensitivity <15dB within 5^0 of fixation.

Stage 4: Advanced defect

MD of -20.00dB and at least one of the following :

A On pattern deviation plot, greater than or equal to 75% of points depressed below the 5% level and greater than or equal to 50% of points depressed below 1% level.

B At least 50% of points within central 5^0 with sensitivity of <0 dB.

C Both hemifields containing greater than 50% of points with sensitivity <15 dB within 5^0 of fixation.

Stage 5: End stage disease

Unable to perform Humphrey visual fields in "worst eye" due to central scotoma or "worst eye" visual acuity of 20/200 or worse due to primary open angle glaucoma.

Visual field and assessing progression in glaucoma

Glaucoma progression is due to the death of retinal ganglion cells and their axons, which results in peripheral, followed by central vision loss.

Assessment of changes in the visual field, optic disc and retinal nerve fiber layer are the primary ways one diagnoses progression in glaucoma. It is generally believed that structural changes in the optic nerve and retinal nerve fiber layer can be detected before functional changes with SAP, although the Early Manifest Glaucoma Trial found all of their progressive cases except one with SAP rather than optic disc photographs evaluated with flicker chronoscopy. [48]

In clinical practice following SAP for signs of change is critical in the management of glaucoma and is typically performed every 1-2 years or more frequently if glaucoma is poorly controlled or rapidly progressive. [49]

There are several ways in which a visual field defect can progress in glaucoma. Localized defects can enlarge or deepen, and finally a new distinct localized defect can appear. When looking for enlarging or deepening of existing defects, one can apply numerical pointwise criteria such as that used in the Collaborative Normal Tension Glaucoma Study[50] or one of several statistical

programs that analyze individual points such as the Glaucoma Progression Analysis program for the Humphrey Visual Field Analyzer. [51]

Using the global indices such as worsening of the mean deviation (MD) and pattern standard deviation (PSD), can be difficult. The MD is a single measure of the depression of the patient's visual field compared to age matched controls. It may be getting worse from localized or diffuse changes, the latter most commonly a manifestation of cataract worsening.

The pattern deviation value is not the best measure of a worsening visual field in glaucoma, however, because it gets higher initially in glaucoma but then lower as more and more of the visual field becomes affected.

If the MD and PSD are both worsening, this is better evidence of worsening of a localized defect such as commonly occurs in glaucoma.

Standard achromatic perimetry

Perimetry is used to describe the measurement of the visual field (Gr. Peri: around, Gr. Metrein: to measure).

The visual field is all that space or area that is seen by a steadily fixating eye. It is described as an island or hill of vision surrounded by a sea of darkness (Traquair). It is a three- dimensional structure and extends 60° nasally, 90° temporally, 50° superiorly and 70° inferiorly. The acuity of vision is sharpest at the top of the hill (corresponds to the fovea) and then decreases progressively to the periphery with the nasal slope steeper than temporal.

Perimetry aims to draw the map of the island of vision, such that it is a true representation for each eye and also aims to present it in a way which is clinically useful.

Standard visual field testing involves measuring the contrast sensitivity or the ability of an observer to just distinguish the target from the background.

Conventional visual field testing is performed with a white target against a more dimly illuminated white background [white on white or Standard Achromatic Perimetry (SAP)]

Two main types of SAP currently used in clinical practice are kinetic and static.

Kinetic perimetry

Kinetic perimetry defines threshold by moving the test object from a non seeing (subthreshold) to a seeing (suprathreshold) area and recording the point at which it is first seen in relation to fixation. The boundaries, or contour lines, are called isopters.

Kinetic perimetry is typically performed manually by confrontation, on a tangent screen or with a Goldmann perimeter.

In kinetic perimetry, the stimulus usually is presented approximately 2^{o} per second toward fixation until the patient first perceives it. The stimulus is subsequently moved to another meridian in an periphery out of view and advanced toward fixation again until the patient sees it.

By repeating these manoeuvres at approximately 15^0 intervals around 360^0 of the visual field, the examiner defines a series of points that can be connected to describe an isopter corresponding to the stimulus used.

The careful examination of a visual field using Goldmann perimeter is time consuming and requires a highly trained technician. However it allows for a careful peripheral examination, and some patients especially elderly or those with advanced field loss, perform better on manual perimeters compared to automated devices.

Static perimetry

Static perimetry involves the presentation of stationary test objects, using either suprathreshold or threshold presentations.

Basic principles of visual field testing

1. Technician

To accurately interpret visual fields, the interpreter must be familiar with the skills and variations of the technician performing the tests. The technician can improve patient performance by monitoring the patient consistently during the examination.

2. Stimuli

The typical stimuli used in clinical perimetry are spots of light of various predefined combinations of diameter and intensity projected on the background. The visibility of the stimulus also depends on how far the eye is positioned from the screen and the brightness of the background. The other factors affecting perception of the stimulus include the length of time the stimulus is presented, colour of the stimulus and of the background and the condition of the eye and the patient.

The Humphrey perimeter uses projected stimuli. The standard white stimuli can be varied in intensity over a range of 5.1 log units (51 decibels) between 0.08 and 10,000 apostilbs (asb). The decibel (dB) value refers to retinal sensitivity, rather than to stimulus intensity with 0dB corresponding to the maximum brightness that the perimeter can produce (10,000 asb) and 51dB to 0.08 asb.

In standardized testing with a size III white stimulus, the dimmest stimulus that can be seen foveally by a young, well- trained observer is at most about 38 to 40dB. Thus the upper 10 decibels of stimulus range from 41 dB to 51 dB really fall outside the range of human vision.

3. Stimulus size

The standard target for both kinetic and static perimetry is a white disc, the stimulus value of which can be adjusted by varying the target size or luminosity relative to that of the background. In normal subjects, the mean retinal sensitivity has been shown to increase with the increasing size of the test object. [52]

The Humphrey perimeter is capable of testing with the five Goldmann stimulus sizes but the 0.43 degree Goldmann size III stimulus is used most of the time.

In practice, size V which is occasionally employed in advanced field loss, is the only other commonly used stimulus size. Size V is also the standard stimulus size in blue yellow perimetry (SWAP).

4. Stimulus duration/exposure time

The exposure time will also affect the stimulus visibility. The stimulus presented over a larger period of time may become more visible, the phenomenon called temporal summation.

However, after the temporal summation is complete, which happens typically after 0.1 second, the image is not seen any better.

The Humphrey perimeter uses a stimulus duration of 200 milliseconds (ms), which is long enough for visibility to be little affected by small variations in duration, but still shorter than the latency for voluntary eye movements (about 250ms).

5. Background illumination

The level of background illumination affects the contour of the hill of vision and thus appearance of the visual field. Brighter background illumination increases the slope of the central field and may influence the appearance of field defects.

Standard Humphrey perimetry projects stimuli against a background with a brightness of 31.5 apostilbs. This background illumination was originally used by the Goldmann perimeter and has been adopted as a standard by the International Perimetric Society.

5. Area tested

To compare visual field charts, the same region of the visual field must be tested during serial examinations. For most purposes, tests examine alongside vertical and horizontal meridia.

Physiologic factors that influence visual field

The following factors should be compensated for, if possible or otherwise should be considered when interpreting the fields.

Patient variables

Age

Increasing age is associated with a reduction in retinal threshold sensitivity.[53]

This effects starts as early as 20 years of age, progresses linearly throughout life and involves the peripheral and superior areas more than the pericentric and inferior portions of the field.

Standard automated perimetry protocols compensate for age influence by using age bracketed databases.

The increase in fluctuation that occurs as the test moves toward the periphery is also greater with age. Mean sensitivity of the visual field decreases approximately 0.58-1.0 dB per decade. Increased age may be associated with increased variability in repeated test results over time.

Ocular variables

1. Pupil size

Pupillary diameter of less than 3mm can cause generalized depression of the visual field. It is usually best to test the field with a pupil that is at least 3mm in diameter.

Significant miosis may depress central and peripheral threshold sensitivities and exaggerate field defects.

Mydriasis has less influence on the visual field, although pupillary dilatation with tropicamide 1% in healthy subjects was shown in one study to significantly reduce threshold sensitivity with automated perimetry.[54]

2. Clarity of ocular media

Cataracts produce glare and change the intensity of the stimulus. Therefore, a cataract can cause or exaggerate central or peripheral field defects, which could be mistaken for the development or progression of glaucomatous field loss.

Reduced clarity of the ocular media from other causes such as corneal disturbance, a cloudy posterior lens capsule after cataract surgery or vitreous opacities may also affect visual fields.

Applanation tonometry before automated static threshold perimetry was found in a study to have no detrimental effect on the visual field results.[55]

3. Refractive error and retinal blur

When the projected stimulus is not focused on the retina, the edge of the stimulus is blurred, contrast is decreased and the stimulus may not be detected by the patient. The larger the stimulus, the less it is to be affected by the blur.

Refractive errors primarily influence the central field. [56] When a size III stimulus is used refractive errors of 1 diopter or less may not need to be corrected, because it usually will cause only slightly more than 1dB of general reduction of sensitivity.[57]

Mild myopia does not need correction, unless the refractive error exceeds 3 diopters.

Hyperopia has a greater influence on perimetric results, especially for the central field, and even small hyperopic refractive errors can significantly alter threshold sensitivity.[56,57,58]

Age tables are available to aid in determining the appropriate correction for presbyopia.

Astigmatism should be corrected unless the cylinder is less than 1 diopter in which case it can be included as the spherical equivalent.

Psychological factors that influence visual fields

The patient's understanding of the test and his or her alertness, concentration, fixation and co-operation all influence the results of visual field testing.

Fatigue effects

Full Threshold protocols take a long time to complete, and patients usually find visual field testing exhausting.

Fatigue causes artificially decreased sensitivity in the areas of existent glaucomatous defect.[59] Fatigue may also cause decreased performance in patients with glaucoma within central 10 degrees, as well as increased deterioration of the mean defect and localized loss in the periphery.[60]

Automated static perimetry

Automated perimetry is accepted as the standard way of measuring the visual field.

The major limitations of tangent screens and arc perimeters were lack of standardization of the test objects and the background, as well as patient fixation.

These needs were addressed in the era of standardization, which began in the middle of the 20th century with the contributions of Goldmann. The main problem that remained however was the subjectivity of both the patient and perimetrist.

Although the subjectivity of the patient has not been eliminated, the influence of the perimetrist was eliminated to variable degrees with the advent of automated perimetry in the 1970s.

In the last half of the twentieth century, several automated perimeters became available. Currently, the most widely used one is the Humphrey Field Analyzer (HFA) and the most common type is static perimetry.

Advantages of automated static perimetry are

- Shorter test duration
- Reproducible standardized testing conditions.
- No examiner bias.
- Can compare visual fields obtained in different centers.
- Availability of normative data.
- Sophisticated statistical analysis and more accurate monitoring.
- Better data storage capability.
- Higher sensitivity.
- Stimuli presentation at random locations and therefore improved patient fixation and absence of local retinal adaptation .
- No need for highly trained perimetrists.

The Humphrey field analyzer consists of four basic elements : the bowl or projection surface, the optical system, the central processor and the patient interface.

The bowl

The bowl of the HFA II is a patented, aspherical or bullet shaped surface upon which stimuli are projected. It was adopted because it improves patient ergonomics and reduces instrument size.

The distance from the eye to the centre of the bowl is 30 centimeters.

The bowl surface is textured to provide an almost perfectly matte finish known as a Lambertian surface which provides no direct or specular reflections.

The optic system

The Humphrey perimeter's optical system provides stimuli of known brightness for a known amount of time in a known location, and against a background of known brightness.

All five standard Goldmann stimulus sizes (I through V) are available, although most testing is done with the size III.

Stimuli are presented by aiming the projection system at the particular location to be tested, adjusting a set of neutral density filters to obtain the correct stimulus brightness and then opening the mechanical shutter for a fixed time, usually 200 milliseconds. Background brightness and stimulus brightness are checked at the beginning of each test and constantly during testing.

The central processor

The Humphrey's perimeter's central processor not only fulfills many of the functions commonly seen in a standard desktop computer, it also must control the optical system as well as make complex, split second strategy adjustments based upon each patient response.

The system has a hard disk for program and data storage, a disk drive, and a video screen. All clinical data must be safeguarded by frequent backing up.

A printer is also available so that visual field test results may be printed for future reference.

The patient interface

The patient interface consist of a chin rest, a forehead rest, a trial lens holder, the patient response button and the instrument table and chair.

Test patterns/test programs

The standard programs on the Humphrey are the 30-2, 24-2, 10-2 and the macular grid program. The most commonly used are limited to the central 24 to 30 degrees with a 6 degrees separation between test locations.

In the 30-2, the central 30 degrees of the visual field are tested. It consists of 76 points 6 degrees apart on either side of the vertical and horizontal axis, such that the innermost points are three degrees from fixation.

In the 24-2 program, 54 points are examined. It is near similar to the 30-2 except that two peripheral nasal points at 30 degrees on either side of the horizontal axis are included while testing the central 24 degrees.

The 10-2 program tests 68 points 2 degrees apart in the central 10 degrees. This program helps to assess and follow up fixation characteristics in patients with an advanced disease along with the macular test which examines 16 points in the central 5 degrees each being 2 degrees apart.

Programs are also available to study the peripheral field beyond 30 degrees either in the nasal quadrant or for 360 degrees. The peripheral studies can be performed alone or in conjunction with a central field program and usually have wider target separation. Static testing of the peripheral nasal fields has been shown to provide valuable additional information in detecting glaucomatous field defects.

Testing strategies

Fully automated perimeters provide suprathreshold and full threshold measurements.

1. Suprathreshold static perimetry
2. Threshold related screening
3. Full threshold perimetry

Suprathreshold static perimetry

Suprathreshold static perimeters present a stimulus brighter than the anticipated normal value for the corresponding retinal location.

Threshold related screening

Herein, the intensity of the light presented is 5dB brighter than the actual threshold at the test point in question. This allows the entire field to be screened quickly. It can used as a screening test for detection and follow up known pathologies.

Full threshold perimetry

In automated perimetry, threshold values are defined as stimuli intensity detected 50% of the time. The most commonly used programs measure the retinal threshold at 70 to 80 points within the central 24 to 30 degrees.

In the Full Threshold strategy, stimuli are presented at predetermined locations using a 4-2 algorithm where the threshold is crossed twice, initially in a 4dB increments followed by 2dB increments.

Examination of the central 24^0 visual field using this technique takes 15-20 min.

Fastpac

Another thresholding strategy to reduce testing time is the FASTPAC program of the Humphrey field analyzer which estimates threshold from a single threshold crossing in 3dB increments, in contrast to the standard double threshold crossing with 4dB and 2dB. This strategy has been evaluated by several investigative teams most of whom agree that it provides time reduction at some expense of accuracy and reliability.

Swedish Interactive Threshold Algorithm (SITA)

In recent years, a new threshold strategy known as SITA has become increasingly popular. This algorithm uses standard 24-2 or 30-2 patterns to assess the visual field based on the probability analysis of the patterns of glaucomatous damage and is more time efficient than standard threshold strategies.

Two versions of SITA are currently available

SITA Standard and SITA Fast

SITA standard takes approximately half the time to complete as compared to the Standard Full Threshold program and SITA Fast takes about half the time of the FASTPAC algorithm.

During the SITA test, the computer also produces an information index, which stops the test at the location being examined when threshold reaches a preselected level.

The goal of any test of the visual function is to improve test sensitivity and specificity. In the world of perimetry, one way to achieve this is by improving test reliability and variability by decreasing test duration without sacrificing the quality of the results. This has been accomplished using a Swedish Interactive Threshold Algorithm (SITA). The SITA strategy uses more efficient mathematical methods for estimation of threshold values based on normative data, patient age and patient responses during the test.

The threshold values are adjusted in real time during the test and pace is altered depending on the patient response speed.

The test time is also reduced by eliminating retest trials for short term fluctuation determination and redundant questioning for assessing false positive (FP) and false negative (FN) responses. By using this smart strategy of dynamic testing adjustment, SITA standard reduced the test duration by 50% compared to the Full Threshold strategy, importantly this reduction in the test duration is not done by sacrificing quality. SITA matches and even surpasses the accuracy of the Full Threshold strategy. [61,62]

To further decrease the test duration by 30-50% SITA fast strategy was designed. However test retest reliability for this strategy is worse compared to the SITA standard.

Humphrey single field printout

There are eight parts to the single field printout. Each has to be examined serially before drawing a conclusion.

Reproducibility (Zone – 1)

First assess the reproducibility of the concerned fields at the onset, check the printed information at the top of the page, to ensure listing of the correct patient, the type of test done (30-2, 24-2, 10-2), eye in question and date of birth (the software package statistically compares the patients response with age corrected normal population).

The recorded visual acuity, refraction and pupil size are important parameters as they all can affect the data.

Reliability (Zone-2)

Interpretation of any visual field should start with the analysis of reliability indices.

These indices are located in the upper-left corner of the single field analysis printout.

Following are the reliability indices available on a Full Threshold and SITA printouts.

Fixation losses

Fixation losses are noted as the ratio of the number of times the patient responded when he saw a target placed in the blind spot against the total number of times fixation was tested.

Several methods of fixation monitoring are available on the HFA II

1. Fixation monitoring on the screen – A technician can monitor the patients eye on a screen to ensure a steady fixation.
2. Heijl Krakau technique (blind spot monitoring) - When a fixation monitor is set at blind spot, a Heijl Krakau technique [63] is used, where stimuli are projected several times during the test into an area of a previously mapped blind spot. If a patient responds to such stimuli it is assumed to be a fixation loss.
3. Gaze tracker – HFA II also has an option of a gaze tracker where the alignment of corneal reflexes and pupil is used to assess subjects fixation throughout the test.

Fixation losses exceeding 20% of the total trials are flagged, it is bracketed (XX) and is indicative of questionable reliability. However, not all fixation losses are due to unsteady gaze.

A "pseudo loss" of fixation is seen when there is a improper location of the blind spot, or when the initial blind spot is present near the edge of a scotoma, so even though it is presented throughout the test, it is occasionally visible.

Another source of error in this method is patient head tilt during the test. Even a small tilt can move the blind spot and result in artificially high fixation losses.

False positive (FP) errors

It is expressed as a ratio of the number of times the patient responds to a pause in the testing sequence without presentation of the target against the total number of pauses. It is the single most significant reliability indicator.

Bracketing occurs when FP's are 33% but often 15-20% rate can also destroy credibility of a field.

A high FP ratio, will be accompanied by a high positive mean defect, white areas on the gray scale which is an indication of very high threshold levels (white scotomas), a high number of fixation losses and a message of abnormal high sensitivity on GHT.

In a Full Threshold or FASTPAC strategy, false positive (FP) errors are registered when a patient responds to a catch trial in which an auditory stimulus is given in the absence of a visual stimulus.

SITA does not use 'catch trials' to register FP, instead it calculates them as responses that occurs outside the normal response time window. Patients with high FP errors may be overly anxious and reassuring them that it is normal to miss 50% of the stimuli can usually cure "trigger happy" patients.

False negative (FN) errors

False negative errors are expressed as a ratio and occur when the patient does not respond when a point previously thresholded is retested with a brighter stimulus.A 33% FN ratio is considered excessive and makes the test unreliable.

However the presence of a scotoma and a high number of FN errors, with all other reliability measures being normal is indicative of a reliable field.

In a Full Threshold or FASTPAC strategy, false negative errors are recorded when a patient does not respond to stimuli 9dB brighter than previously registered at that location.

Two common causes of increased FN are:

1. Patient fatigue (can produce a 'cloverleaf field')

2. End stage glaucoma.

SITA does not spend extra time during a test on catch trial; instead, FN errors are estimated at the end of the test as stimuli when, during threshold testing, a patient denied seeing a stimulus that was later found to be brighter. Only test points from the relatively normal parts of the visual field are considered in this analysis.

Visual field plots

Threshold sensitivity values (dB) are plotted from stimuli presentations at pre determined test locations.

Gray scale (Zone-3)

The gray scale is a schematic representation of threshold values. It helps to appreciate a general pattern of the visual field and draws attention to abnormal areas which require further careful studying.

It is a rough indicator of the extent of field damage but can be misleading.

Each point on the grayscale is represented by a symbol of varying darkness which corresponds to the threshold level at that point.

Total deviation plots (Zone-4)

Total deviation plots are calculated as a difference between the patient's threshold values and those of the age- matched normals, at each point.

This depending on whether the patient did better or worse than expected is expressed as a positive or negative number.

TD plots are center-weighted such that an abnormal central point is assigned more significance than a peripheral point. These are presented as dB plot (top left plot on the single field analysis) and as a probability plot (bottom left plot on the single field analysis).

These probability symbols increase in significance from a set of 4 dots to a black box, P<5%, <2%, <1% and 0.5%. The presence of black box indicates that a few normal subjects will have that score and it does not necessarily correspond to an absolute defect. Many points with P<0.5% are relative defects and their actual threshold is available from the raw data.

Pattern deviation plot (Zone-5)

Pattern deviation (PD) dB and probability plots are located to the right of the TD plots on the single field analysis printout.

PD is calculated by adjusting the overall sensitivity (TD values) of the visual field by the seventh most sensitive non-edge point, to differentiate focal defects from generalized changes.

Therefore, PD plots highlight focal visual field defects and ignore generalized changes.

Raw data/numeric data (Zone-6)

It is the actual threshold score for each threshold point. Areas flagged in the pattern and total deviation plot should be inspected carefully for confirmatory signs like double threshold points of abnormal or foci of high local fluctuation. This should be followed by a geographic survey of the entire numeric data.

Global indices (Zone-7)

The global indices are presented in the lower right hand corner of the printout and include.

Mean deviation (MD)- It represents an average deviation of the patients visual field from the age matched controls.

Its calculation is centre weighted .A negative MD indicates a depressed field while a positive value represents a higher than normal sensitivity.

MD does not help in differentiating diffuse from focal loss.

Pattern standard deviation (PSD)

It represents the degree of irrregularity in the field. The higher the value the more uneven is the field indicating a focal visual field defect.

Usually an increase in PSD in a patient with glaucoma suggest progression.

Short term fluctuation (STF)

STF is available in the Full Threshold, but not in the SITA strategy

It represents the consistency of patient responses during a single test and determined by retesting the same ten points.

Corrected pattern standard deviation (CPSD)

CPSD represents a PSD corrected for STF. This is done in the attempt to eliminate irregularities in the visual field secondary to unreliable patient responses.

It is available in the full threshold but not in the SITA strategy.

Glaucoma Hemifield test (Zone-8)

The glaucoma Hemifield test (GHT) is used to identify localized visual field typical of glaucoma. [64]

In the test, both inferior and superior hemifield are divided into five zones and the average threshold values of the superior and inferior mirror images are compared.

Five different outcomes of the GHT are possible

1. GHT within normal limits.
2. GHT borderline.
3. GHT outside normal limits.
4. General reduction of sensitivity.
5. Abnormally high sensitivity.

Correlation between optic nerve head and visual field defects

In most glaucoma patients, clinically recognizable disc changes precede detectable field loss, and the absence or presence of glaucomatous visual field loss can usually, but not always, be predicted from the appearance of the optic nerve head. [65]

Quigley and coworkers [66] attempted to correlate axon loss in the optic nerve head with the visual field defects. Their work suggested that not only does nerve fiber loss occur before reproducible field defects in some patients

with elevated IOP, but the extent of axonal loss maybe much greater than the corresponding visual field change. With standard perimetric techniques, 25% to 35% of the retinal ganglion cells may be lost in an eye with a normal field by the time reproducible early defects are found and 10% or fewer axons may remain by the stage of severe field loss.

When correlating retinal ganglion cell atrophy with automated perimetry in patients with glaucoma, a 20% loss of cells, especially large ganglion cells in the central 30 ° of the retina, correlated with a 5 dB sensitivity loss, whereas a 40% loss corresponded with a 10 dB decrease, and some ganglion cells remained in areas with 0 dB sensitivity.[66]

The nature of the optic nerve head cupping can also be used to predict the type (in addition to the presence) of field loss. Extensive or focal absence of the neural rim tissue, especially at the superior and the inferior poles, is the most reliable indicator of visual field disturbance and is usually associated with a field defect in the corresponding area. In some eyes, field loss may occur before the pallor reaches the disc margin.

Unusual cases have been reported with field damage despite round, symmetric cups .[67]

The ability to predict impending glaucomatous visual field loss by the appearance of the optic nerve head is less accurate than correlating disc damage with established field loss. No single parameter or combination of parameters in glaucomatous optic atrophy is totally satisfactory for this purpose. The parameters that correlate best with visual field loss are magnification corrected measurements of the neuroretinal rim area and defects

in the retinal nerve fiber layer. Diffuse structural changes in the ONH are associated with diffuse depression of the visual function, and localized changes correlate more with localized visual field changes. In a study, examination of stereoscopic optic disc photographs allowed accurate prediction of glaucomatous and normal fields to be made in 82% and 95% eyes, respectively and for visual field loss to be correctly located in upper and lower half in 83% and 91% of cases respectively. The high correlation between the state of the visual field and the optic disc, means that, in evaluation of the glaucoma patient, the appearance of the optic disc and the visual fields should be in agreement. [65]

In another study, Balaszi G et al, studied the neuroretinal rim area in suspected glaucoma and early open angle glaucoma patients and concluded that there is a statistically significant relation between the neuroretinal rim area and the visual function and that the neuroretinal rim area is a variable that is superior to the cup-disc ratio in its correlation with visual function. [68] In a study to assess the glaucomatous disc for features of the optic disc commonly associated with early visual field loss, it was observed that the parameter with the highest probability value to differentiate between the optic disc of early visual field loss and the optic disc of normal subjects was " ratio of the thinnest NRR width in the vertical sectors to the NRR width of the temporal sector ≤ 85 % [2]

Glaucomatous enlargement of the optic cup follows a pattern of vertical extension of the orifice of the cup and focal notching in the neuroretinal rim is always associated with visual field loss. [65]

MATERIALS AND METHODS

This was a cross sectional study conducted in the Department of Ophthalmology from January 2011 to December 2011 to correlate the optic disc changes with the visual defects by observing magnified fundus photographs and using Humphrey's visual field analyzer SITA standard 30-2 program.

SOURCE OF DATA

All diagnosed primary open angle glaucoma patients and glaucoma suspects attending ophthalmic OPD and IPD in KLE's Dr Prabhakar Kore Hospital and MRC Belgaum were included in the study. The procedure and investigations were explained to the patients and informed written consent was taken.

Method of Collection of Data

Study Design: cross sectional study

Sample Size: 48

Based on previous statistics of patients input.

Duration: One year (01st Jan 2011 to 31st Dec 2011).

A total of 48 patients who fulfilled the following inclusion criteria were included in the study.

Inclusion criteria:

1. A known diagnosis of primary open angle glaucoma or glaucoma suspect
2. Patients older than 40 yrs of age.
3. An applanation tonometer intraocular pressure of $\geq$ 21 mm of Hg.
4. Gonioscopically open angle.

5. A combination of optic disc changes like cupping, notching, thinning, or pallor of the neuroretinal rim suggestive of POAG.

The following patients were excluded from the study

1. Participants not willing to give informed consent.
2. Refractive error of > 5 dioptres of sphere or 2.5 dioptres of cylinder.
3. History of ambylopia.
4. History of any disease or trauma to the eye being tested.
5. Abnormal pupillary reaction.
6. History of use of any medication that may affect the visual field.
7. History of cerebrovascular accident.
8. Patients with dense cataracts.
9. Eyes with any retinal pathologic condition that may affect visual field.
10. Suspicious appearing optic nerves or visual field defect associated with identifiable cause such as chorioretinal scar.

After obtaining informed written consent, a comprehensive evaluation of an individual with glaucoma was begin with eliciting detailed history which includes a review of the family and ocular and systemic history, use of systemic and ocular medications, past ocular surgery and known local and systemic intolerance to the use of glaucoma medications.

Clinical evaluation

Vision and Refraction

Best corrected visual acuity was determined and correction of refractive error was essential for accurate perimetry. The patients were classified into three groups : emmetropic, myopic and hypermetropic.

Pupils

Pupil size was noted and both direct and consensual light reflex was noted. The relative afferent pupillary defect was tested with swinging flash light test.

Biomicroscopy

Biomicroscopy of the anterior segment was performed with the help of slit lamp to detect signs of underlying disease.

Conjunctiva was examined for vasodilation and presence of papillae and follicles. Cornea was examined for punctate epithelial defects, microcystic epithelial edema and endothelial abnormalities.

The anterior chamber depth was estimated with a narrow slit beam directed at an angle of 60 degrees onto the cornea at limbus (Van Herick method).

Evaluation of iris was performed before dilatation to know the presence of heterochromia, atrophy, transillumination defects and ectropion uvea.

The lens was examined after dilatation and exfoliative deposits, phacodenesis, subluxation, and dislocation was noted along with lens size, shape and clarity.

Intraocular pressure (IOP mm Hg)

IOP was measured with Goldmann type of applanation tonometer before performing gonioscopy or dilatation of the pupil.

Technique of IOP estimation by applanation tonometer was as follows:

A drop of topical anaesthetic was placed in each eye and the tip of moistened fluorescein strip was touched to the tear film on the inner aspect of the lower lid. The tonometer and prism were set in correct position on slit lamp. The tension knob was set at 1 gm and the 0 graduation mark of the prism was set at white line on the prism holder, the cobalt filter was used with the slit beam opened maximally. The angle between the illumination and microscope was 60 degrees. The patient was seated comfortably on the slit lamp and the tonometer prism was advanced until the tip of the prism touches the cornea. A monocular view of the central applanated zone and the fluorescein stained tear film was obtained. The tip of the applanation prism was adjusted until the two equal semicircles were seen in view. The fluorescein rings were approximately 0.25-0.30 mm in thickness. If fluorescein rings were thinner IOP was underestimated and additional fluorescein may be added. A wide fluorescein ring may over estimate IOP and excessive dye was dried with a tissue and IOP was estimated. The tension knob was rotated until the inner border of fluorescein rings approximate. The reading obtained was multiplied by 10 to give the IOP in millimetres of mercury. IOP was measured in each eye until three consecutive readings were found within 1 mm Hg.

GONIOSCOPY

Gonioscopic evaluation of the anterior chamber angle was done with help of Goldmann 3 mirror gonioscopic lens.

Technique

The patient sits with the head firmly against the head rest of the slit lamp and a local anaesthetic (4% lignocaine) was instilled. 1% methylcellulose solution was placed on the corneal aspect of the gonioscopic mirror. With the patient looking up , one edge of the lens was placed in the lower fornix, the upper lid was elevated, the patient was instructed to gaze straight and the lens was rotated against the eye. An inverted image of the opposite angle was viewed in the mirror. The scleral spur and the Schwalbe's line were most consistent angle landmarks which were used for identification of angle structures and their grading. Schwalbe's line was identified as the termination of the corneal light wedge; using a narrow slit beam sharply focussed, one could observe two linear reflections, one from the external surface of the cornea and its junction with sclera, and other, the internal surface of cornea. These two reflections meet at the Schwalbe's line. The scleral spur was identified by the site of insertion of the iris on the ciliary face, convexity of iris and the prominence of the peripheral iris roll. The Shaffer's system was used to grade the anterior chamber angle as follows :

Shaffer's system of grading anterior chamber angle

Grade Number	Angle width	Description	Risk of closure
4	45-35	Wide open	Impossible
3	35-25	Wide open	Impossible
2	20	Narrow	Possible
1	10	Extremely narrow	Probable
Slit	Slit	Narrow to slit	Probable
0	0	Closed	Closed

OPTIC DISC EVALUATION

All patients had pupillary dilatation with 0.8% tropicamide and 5% phenylephrine. The optic nerve was evaluated using a slit lamp biomicroscope and a posterior pole lens (90 D lens) which offers a stereoscopic and a magnified view of the optic nerve head and retinal nerve fiber layer. The slit beam rather than diffuse illumination was used in detecting changes in the contour of the optic disc. These techniques require patient cooperation and pupillary dilatation for an adequate view of the optic disc details. The cup: disc ratio was measured and recorded. The presence of peripapillary atrophy, notching and optic disc hemorrhage was noted.

The neuroretinal rim was examined carefully for notching , thinning or pallor. Position of the blood vessels was recorded. The disc was also examined for non specific signs like Bayoneting sign and laminar dot sign. Magnified photographs were taken with a canon retinal digital camera to correlate with the visual field defects.

VISUAL FIELD EVALUATION

All visual field examinations were carried out using the same Humphrey Visual Field Analyzer II- i series (Zeiss company) and by the same perimetrist.

Each patient's distance correction was entered into the perimeter's software. The software calculates the trial lens power needed according to the date of birth of the patient entered.

Using the Humphrey Visual Field Analyzer II- i series (Zeiss), SITA standard test using program 30-2 and a size III stimulus on a white background was performed. Calculations of the total and pattern deviation plots and global indices (mean deviation, MD and pattern standard deviation, PSD) were derived using STATPAC for SITA, version A10.1.

Visual fields with any abnormal reliability parameter (fixation losses > 33 %, false positive responses >33%, or false negative responses >33 %) were excluded and repeat testing was performed on a subsequent date.

SINGLE FIELD ANALYSIS PRINT OUT OF 30-2 SITA STANDARD ALGORITHM WAS ANALYSED AS FOLLOWS:

All the 8 zones of the single field analysis print out were examined serially for detecting the glaucomatous visual field defect.

Minimal criteria for glaucomatous visual field defect were as follows:

1. Glaucoma hemifield test (GHT) outside normal limits.
2. PSD (for SITA standard) with P values <5%.
3. A cluster of three or more points on the pattern deviation plot in a single hemifield (superior or inferior) with p values < 5% , one of which must have a P value <1%.

Any one of the preceding criteria was considered sufficient evidence of a glaucomatous visual field defect.

SITA (SS) Standard fields were used to classify patients into mild, moderate, or severe glaucomatous defects using the Hodapp Anderson Parrish (HAP) system.

Hodapp Anderson Parrish Visual Field Severity Score

Criteria for Early Defect

1. Mean deviation no worse than - 6 dB.
2. On pattern deviation plot, fewer than 25% of points depressed below the 5% level, and fewer than 15% of points depressed below the 1% level.
3. No point within central 5^0 with sensitivity < 15 dB.

Criteria for Moderate Defect

1. Mean deviation worse than - 6dB but no worse than -12 dB.
2. On pattern deviation plot, fewer than 50% of points depressed below the 5% level, and fewer than 25% of points depressed below the 1% level.
3. No point within central 5^0 with sensitivity of < 0 dB.
4. Only one hemifield containing a point with sensitivity <15 dB within 5^0 of fixation.

Criteria for Severe Defect

1. Mean deviation worse th an -12 dB.
2. On pattern deviation plot, more than 50% of points depressed below the 5% level and more than 25% of points depressed below the 1% level.
3. Any point within central 5^0 with sensitivity of < 0 dB.

4. Both hemifields containing a point or points with sensitivity < 15 dB within 5^0 of fixation.

STATISTICAL METHODS

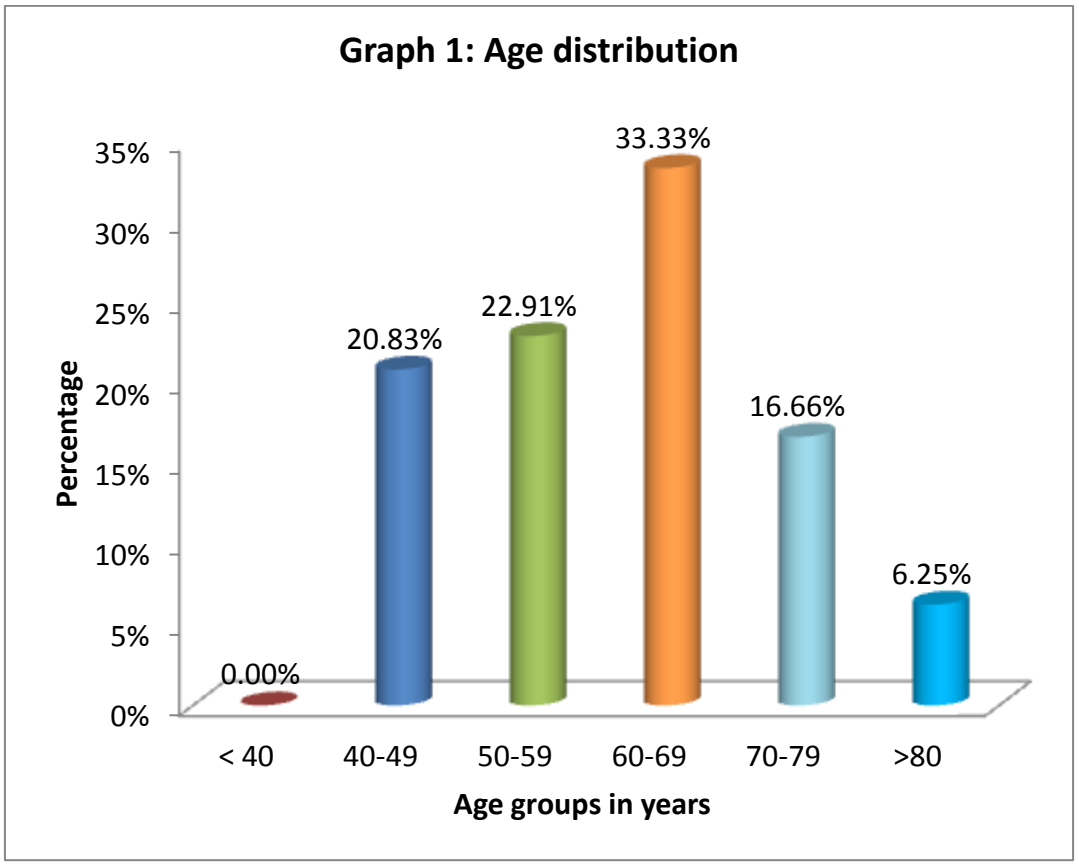
Distribution of demographic and clinical characteristics were noted. Correlation between IOP and C:D ratio was calculated using chi square test. Correlation of visual field defects with the optic disc changes was calculated using Kappa statistics. Correlation between mean deviation and C:D ratio was calculated using chi square test.

OBSERVATIONS AND RESULTS

The present study was conducted at KLES Dr Prabhakar Kore Hospital and MRC, Belgaum and J N Medical College during the period of 01st January to 31st December 2011. During this period 48 patients who fulfilled the selection criteria were included in the study. Ninety six eyes of 48 patients were examined for optic nerve head changes and visual field defects included in all the analysis of the results.

TABLE 1 - AGE DISTRIBUTION

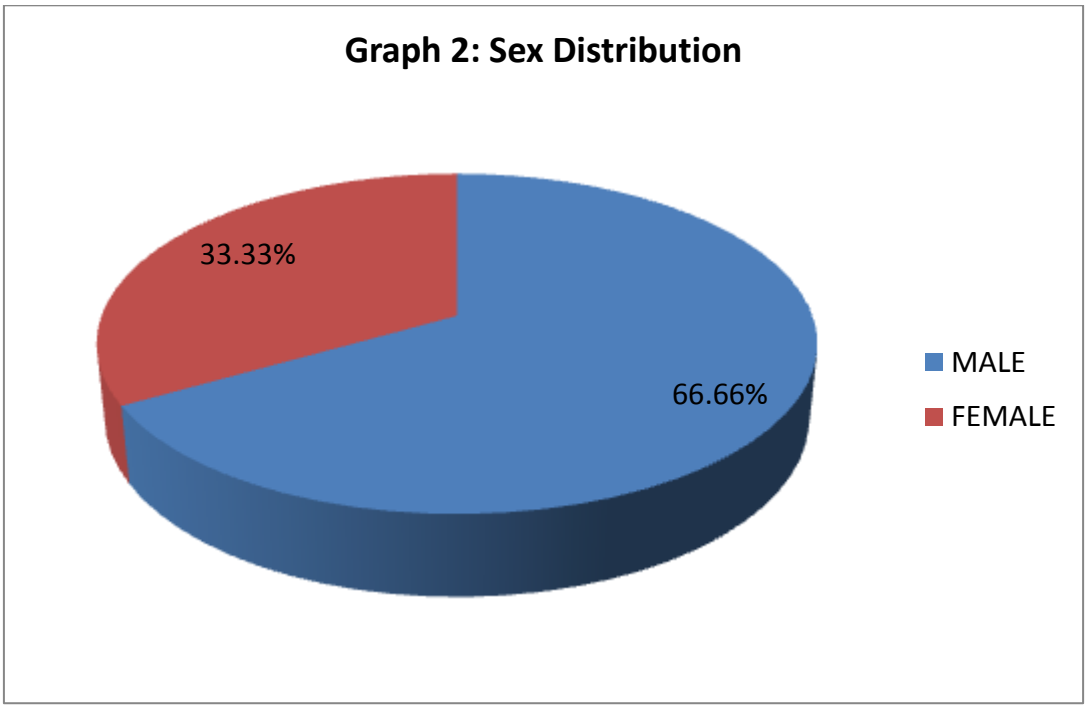
AGE GROUP	NUMBER OF PATIENTS	PERCENTAGE
< 40	0	0.00%
40-49	10	20.83%
50-59	11	22.91%
60-69	16	33.33%
70-79	8	16.66%
>80	3	6.25%
TOTAL	48	100%



Out of 48 patients, 16 patients (33.33%) belonged to the age groups of 60 to 69 years and 8 patients (16.66%) belonged to the age group of 70 to 79 years of age. 11 patients (22.91%) belonged to 50 to 59 years of age group, 10 patients (20.83%) belonged to the age group of 40 to 49 years and 3 patients (6.25%) belonged to the age group of > 80 years. No patients were found below 40 years of age. Maximum number of patients belonged to 6^{th} and 7^{th} decade of age. We had less number of patients above 80 years (6.25%). Mean age of patients was 60.04 ± 11.76.

TABLE 2- SEX DISTRIBUTION

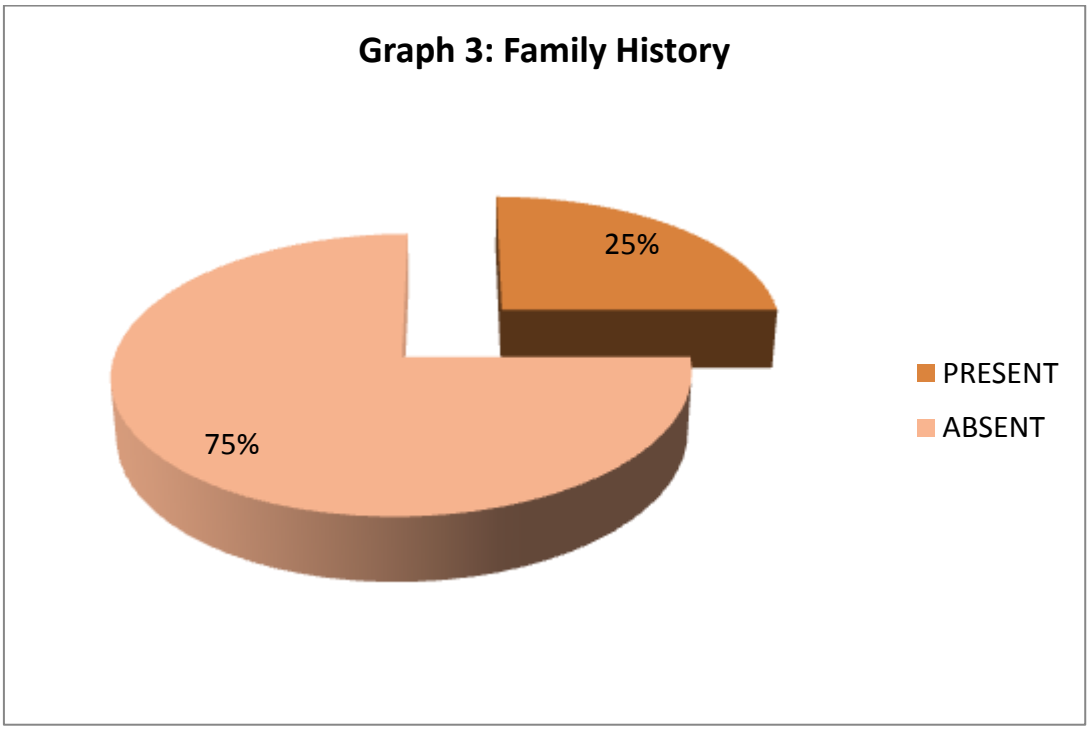
SEX	NUMBER OF PATIENTS	PERCENTAGE
MALE	32	66.66%
FEMALE	16	33.33%
TOTAL	48	100%



In the present study out of 48 patients, 32 (66.66%) were males and 16 (33.33%) were female patients.

TABLE – 3 FAMILY HISTORY

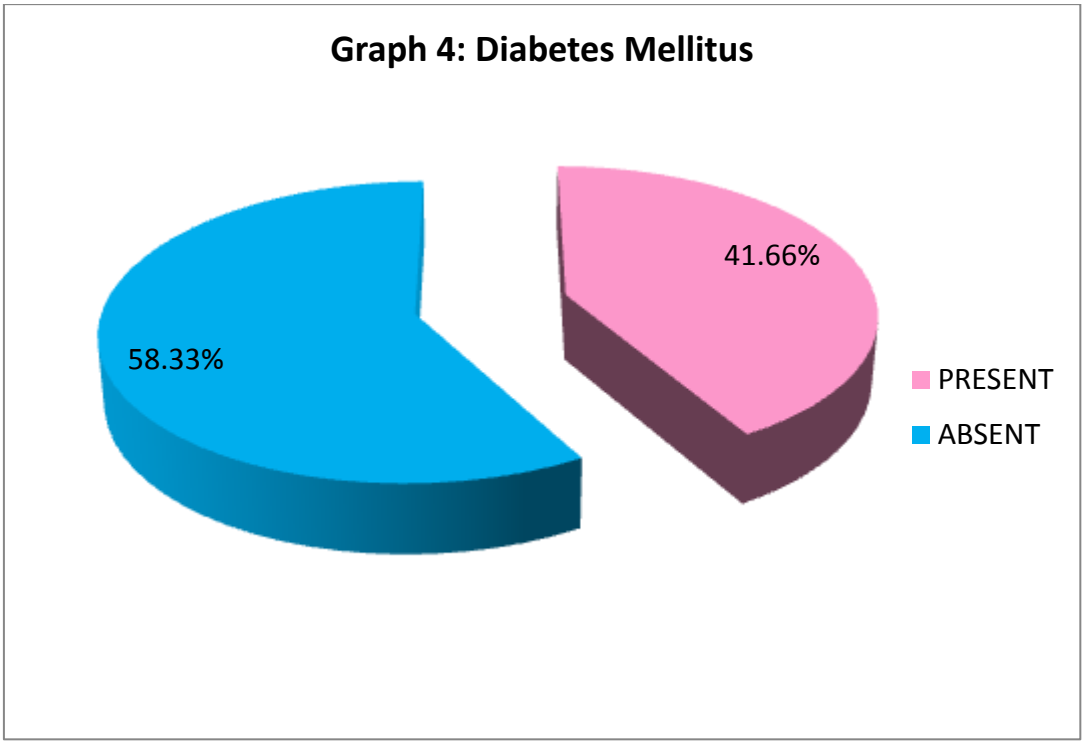
FAMILY HISTORY	NUMBER OF PATIENTS	PERCENTAGE
PRESENT	12	25%
ABSENT	36	75%
TOTAL	48	100%



In the present study, 12 patients (25%) had a positive family history of POAG in first degree relative (sibling, parent, or child) and 36 patients (75%) had negative family history.

TABLE - 4 DIABETES MELLITUS

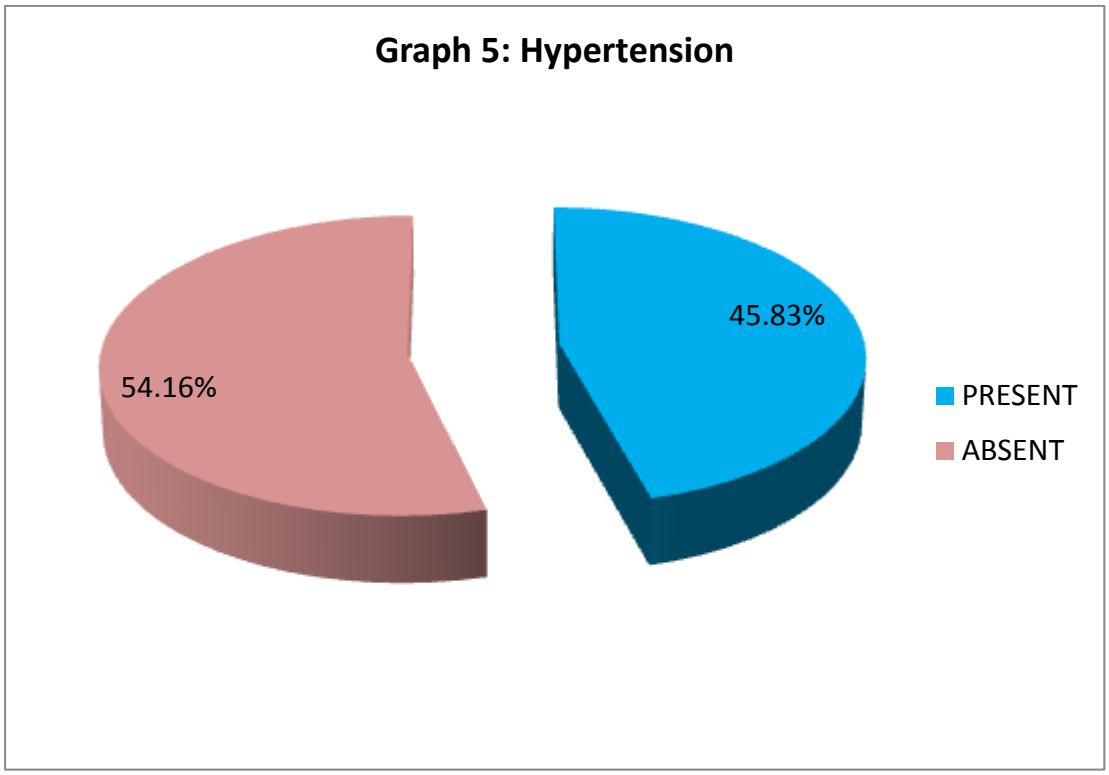
DIABETES MELLITUS	NUMBER OF PATIENTS	PERCENTAGE
PRESENT	20	41.66%
ABSENT	28	58.33%
TOTAL	48	100%



Out of 48 patients in the present study, 20 patients (41.66%) had diabetes mellitus and 28 patients (58.33%) did not have diabetes mellitus.

TABLE 5 - HYPERTENSION

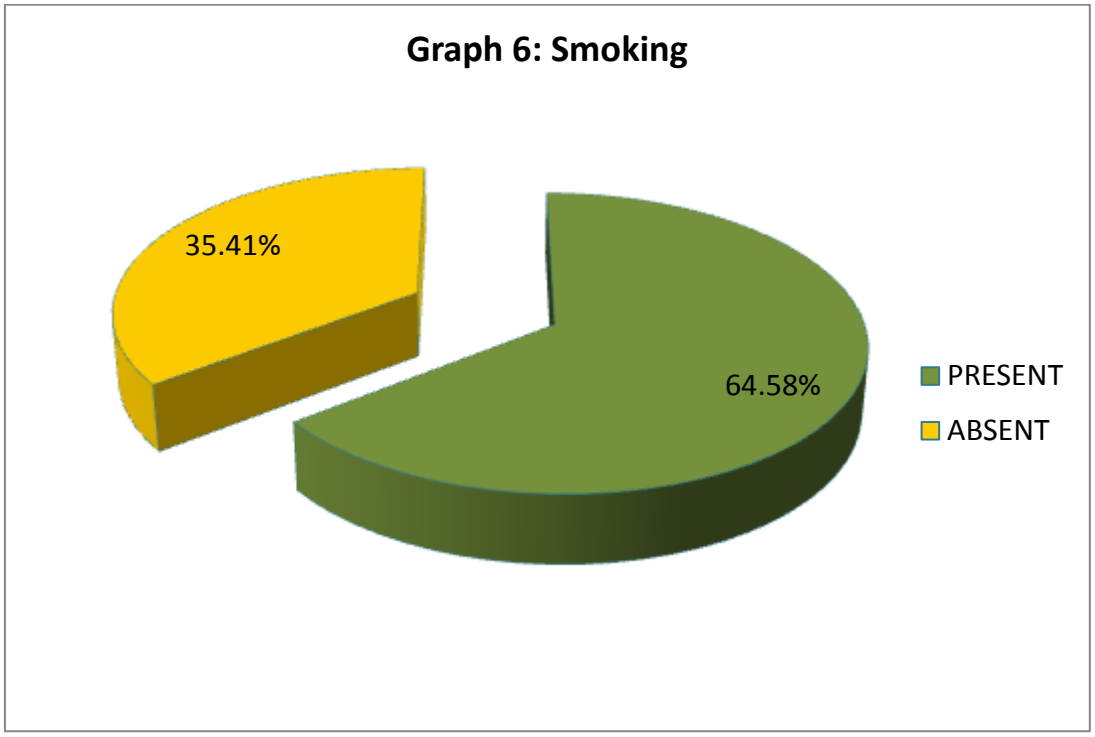
HYPERTENSION	NUMBER OF PATIENTS	PERCENTAGE
PRESENT	22	45.83%
ABSENT	26	54.16%
TOTAL	48	100%



Out of 48 patients in present study, 22 patients (45.83%) gave history of hypertension and 26 patients (54.16%) did not give history of hypertension.

TABLE 6 - SMOKING

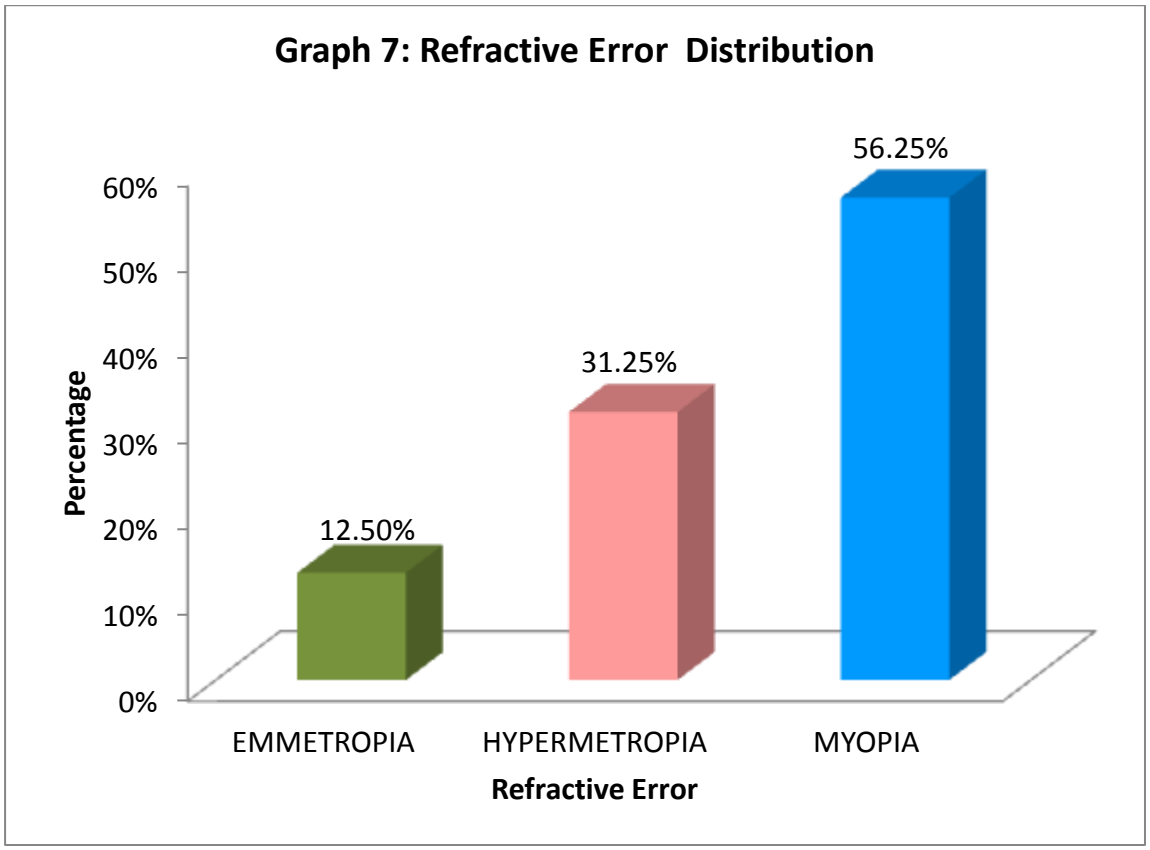
SMOKING	NUMBER OF PATIENTS	PERCENTAGE
PRESENT	31	64.58%
ABSENT	17	35.41%
TOTAL	48	100%



Out of 48 patients in the present study, 31 patients (64.58%) were smokers and 17 patients (35.41%) were non smokers.

TABLE 7 - REFRACTIVE ERROR DISTRIBUTION

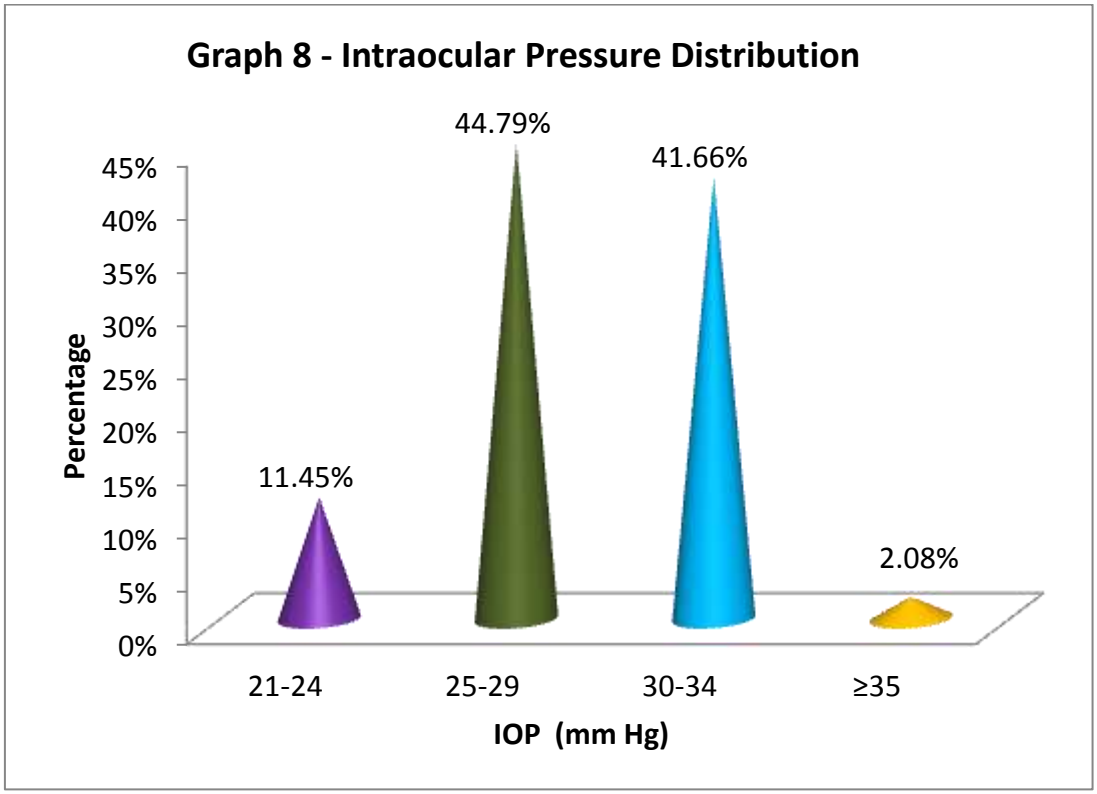
REFRACTIVE ERROR	NUMBER OF PATIENTS	PERCENTAGE
EMMETROPIA	6	12.50%
HYPERMETROPIA	15	31.25%
MYOPIA	27	56.25%
TOTAL	48	100%



Out of 48 patients in the present study, 27 patients (56.25%) were myopic, 15 patients (31.25%) were hypermetropic and 6 patients (12.50%) were emmetropic.

TABLE 8 - INTRAOCULAR PRESSURE (IOP mm Hg) DISTRIBUTION

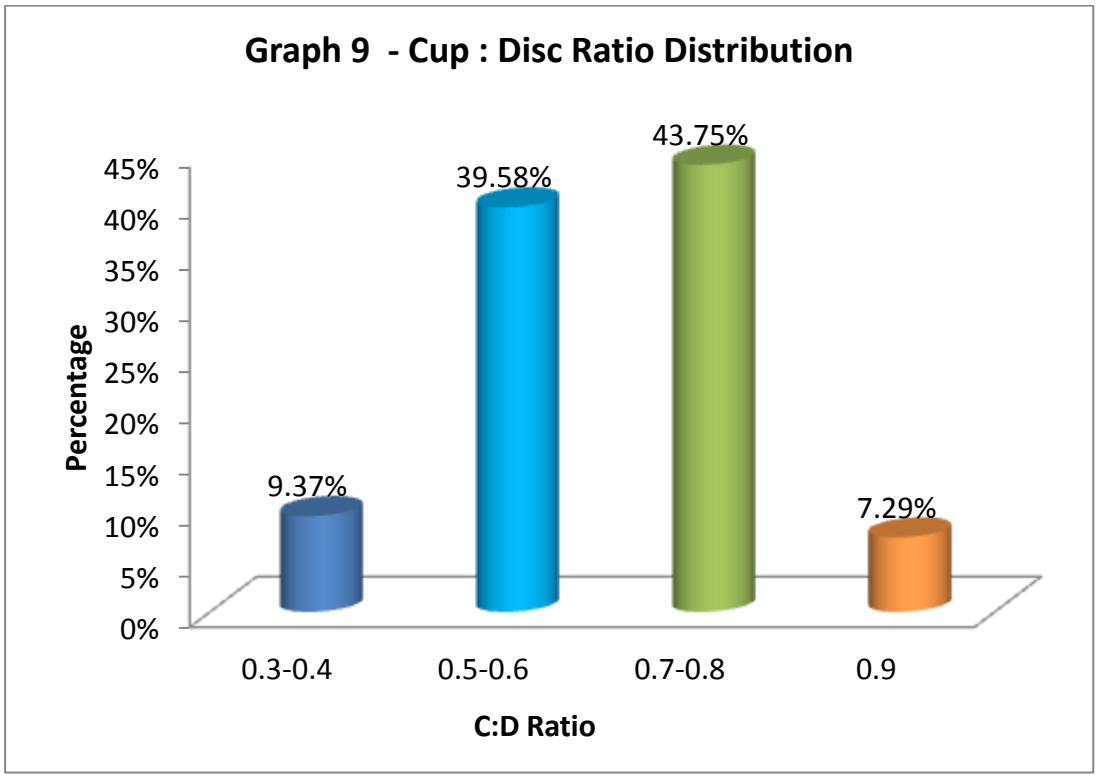
IOP (mm Hg)	NUMBER OF EYES	PERCENTAGE
21-24	11	11.45%
25-29	43	44.79%
30-34	40	41.66%
≥35	2	2.08%
TOTAL	96	100%



In the present study, 43 eyes (44.79%) had IOP in the range of 25 to 29 mm Hg, 11 eyes (11.45%) had IOP in the range of 21 to 24 mm Hg and 40 eyes (41.66%) had IOP in the range of 30 to 34 mm Hg. More number of eyes (44.7%) had IOP in the range of 25 to 29 mm Hg and 2 eyes (2.08%) had IOP ≥ 35 mm Hg. Mean IOP of 96 eyes was 27.66 ± 3.23 mm Hg.

TABLE 9 - CUP : DISC RATIO DISTRIBUTION

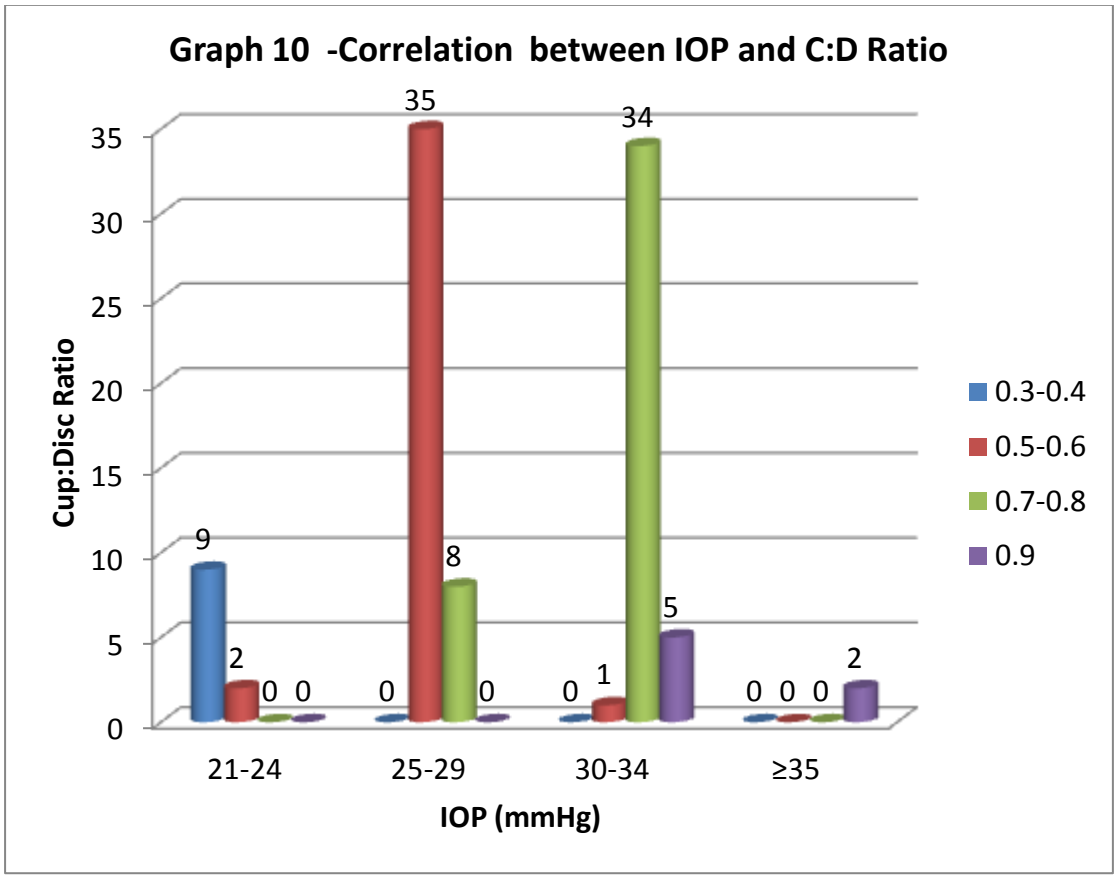
C : D RATIO	NUMBER OF EYES	PERCENTAGE
0.3-0.4	09	9.37%
0.5-0.6	38	39.58%
0.7-0.8	42	43.75%
0.9	07	7.29%
TOTAL	96	100%



Out of 96 eyes of 48 patients included in the present study, 42 eyes (43.75%) had C:D ratio in the range of 0.7 to 0.8 . 38 eyes (39.58%) had C:D ratio in the range of 0.5 to 0.6. Around 7 eyes (7.29%) had C:D ratio of 0.9, 9 eyes (9.37%) had C:D ratio in the range of 0.3 to 0.4. More number of eyes (43.75%) had C:D ratio in the range of 0.7 to 0.8 . Mean C:D ratio of 96 POAG eyes was 0.64 ± 0.15.

TABLE 10 - CORRELATION BETWEEN IOP (mm Hg) AND C:D RATIO

C : D RATIO	IOP (mm Hg)			
	21-24	25-29	30-34	≥35
0.3-0.4	9	-	-	-
0.5-0.6	2	35	1	-
0.7-0.8		8	34	-
0.9	-	-	5	2
TOTAL	11	43	40	2

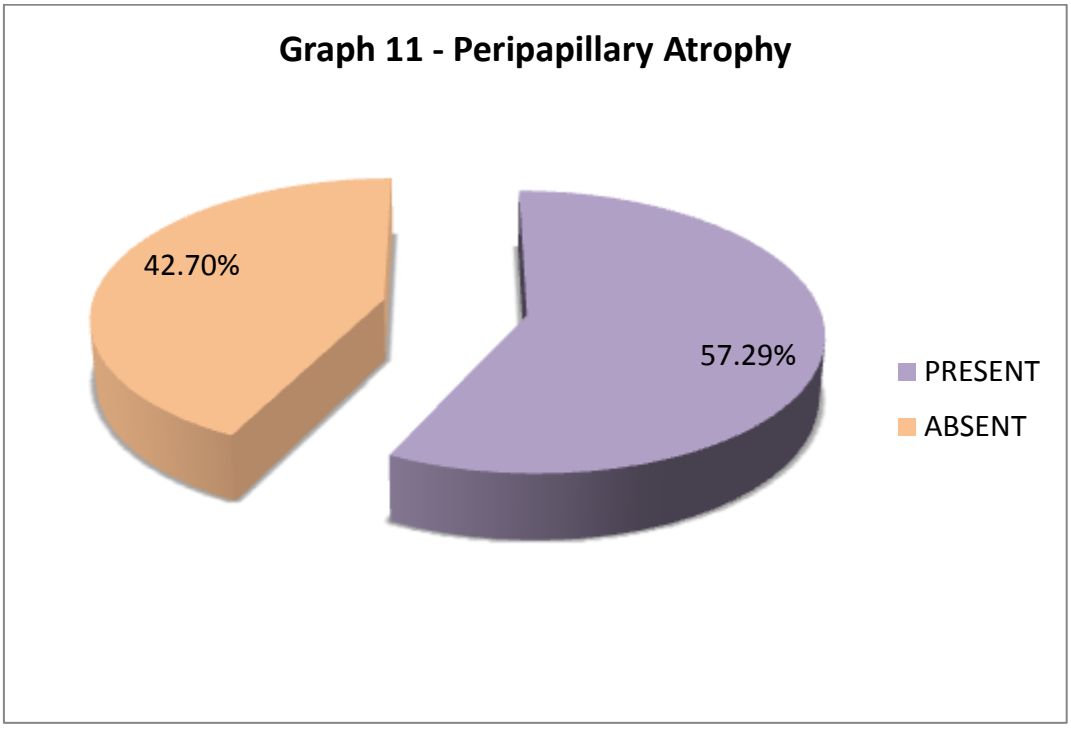


In the present study, among 11 eyes with IOP in the range of 21 to 24 mm Hg, 9 eyes had C:D ratio of 0.3 to 0.4 and 2 eyes had C:D ratio of 0.5 to 0.6. In eyes with IOP in the range of 25 to 29 mm Hg, no eyes had C:D ratio of 0.3 to 0.4, 35 eyes had C:D ratio of 0.5 to 0.6, 8 eyes had C:D ratio of 0.7 to 0.8 and no eyes had C:D ratio of 0.9 and in eyes with IOP in the range of 30 mm Hg to 34 mm Hg, 1 eye had a C:D ratio of 0.5 to 0.6, 34 eyes had a C:D ratio of 0.7 to 0.8 and 5 eyes had C:D ratio of 0.9. In eyes with IOP more than 35 mm Hg, only 2 eyes had a C:D ratio of 0.9.

When the increasing IOP was correlated with larger C:D ratio, *p* value was found to be < 0.001 which indicates statistically significant good correlation between IOP and C:D ratio.

TABLE 11 - PERIPAPILLARY ATROPHY

PERIPAPILLARY ATROPHY	NUMBER OF EYES	PERCENTAGE
PRESENT	55	57.29%
ABSENT	41	42.70%
TOTAL	96	100%

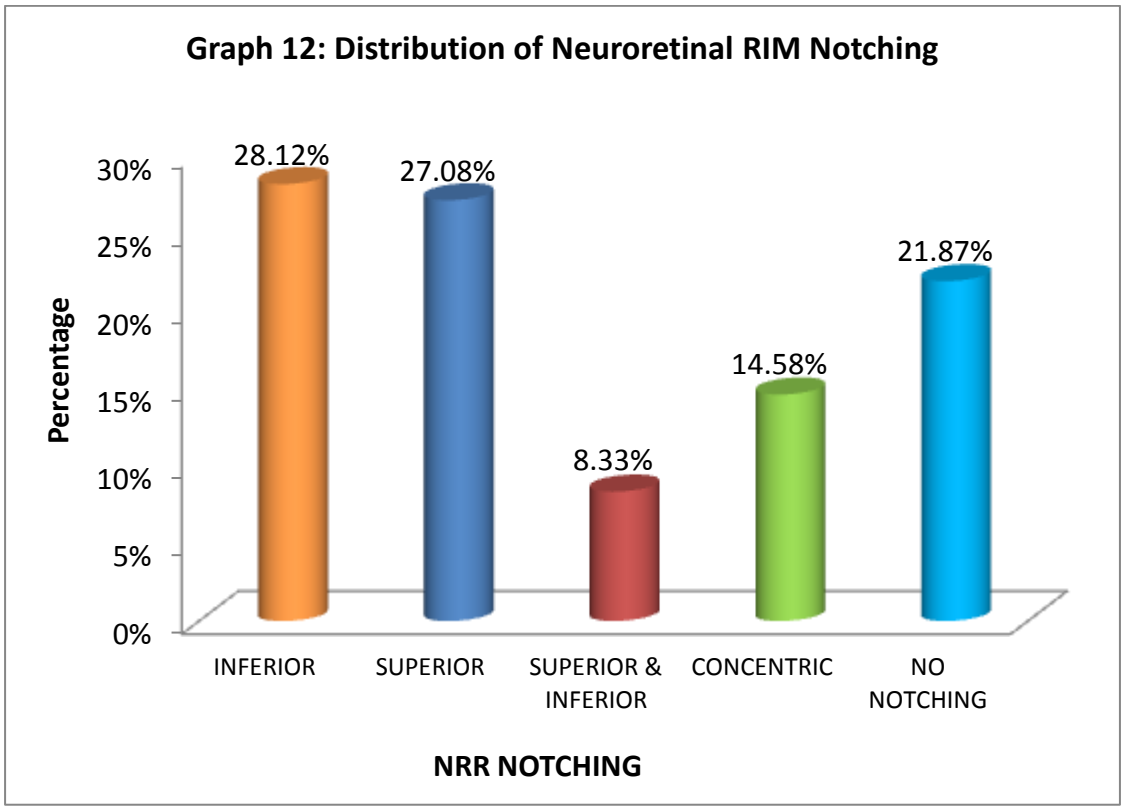


Out of 96 eyes of 48 patients of POAG, peripapillary atrophy was present in 55 eyes (57.29%) and was absent in 41 eyes (42.70%).

In the 96 eyes of 48 patients of POAG, no patient showed optic disc splinter hemorrhages.

TABLE – 12 DISTRIBUTION OF NEURORETINAL RIM NOTCHING

TYPE OF NRR NOTCHING	NO. OF EYES	PERCENTAGE
INFERIOR NOTCHING	27	28.12%
SUPERIOR NOTCHING	26	27.08%
SUPERIOR & INFERIOR NOTCHING	8	8.33%
CONCENTRIC NOTCHING	14	14.58%
NO NOTCHING	21	21.87%
TOTAL	96	100%

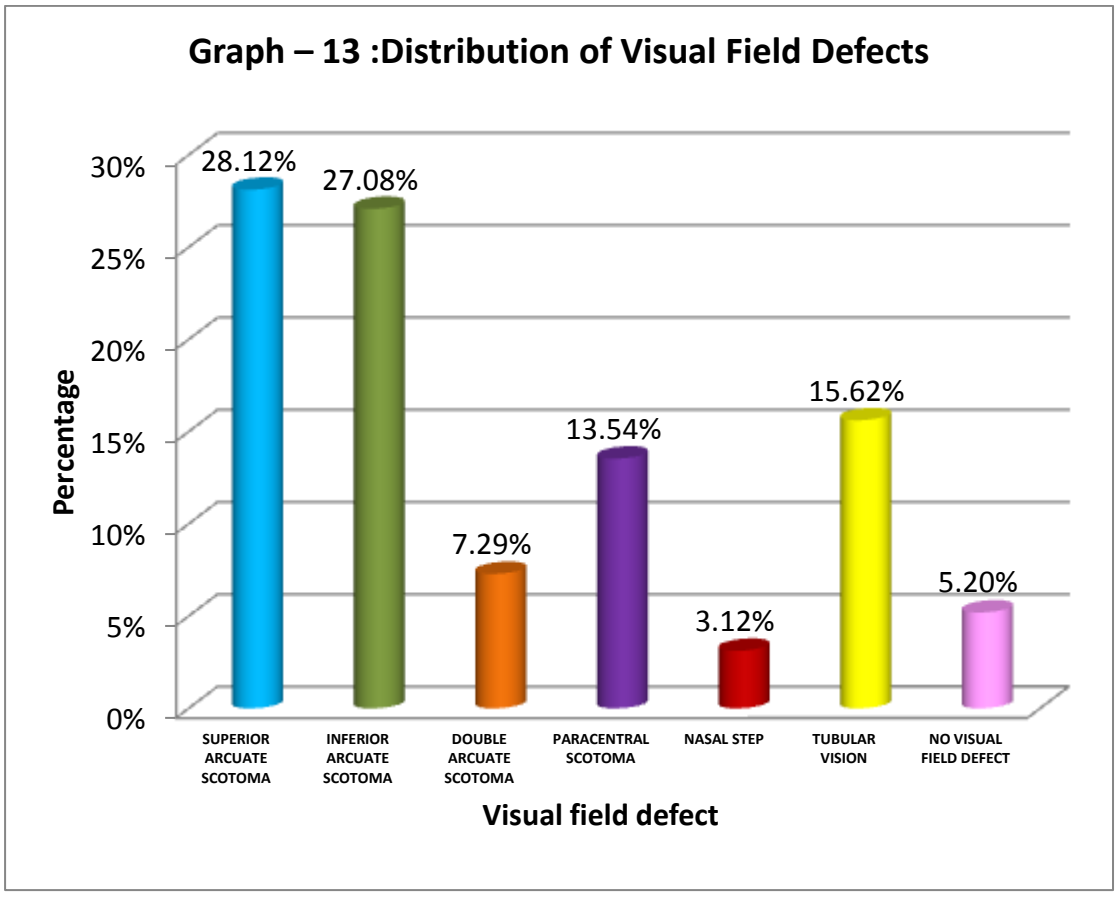


In the present study, out of the 96 eyes of 48 patients of POAG, 75 eyes (78.12%) showed neuroretinal rim notching and 21 eyes (21.87%) showed no notching. Out of the 75 eyes which had neuroretinal rim notching, 27 eyes (28.12%) had inferior notching, 26 eyes (27.08%) had superior notching, 14 eyes (14.58%) had concentric notching and 8 eyes (8.33%) had both superior and inferior notching.

More number of eyes (28.12%) had inferior rim notching.

TABLE – 13 DISTRIBUTION OF VISUAL FIELD DEFECTS

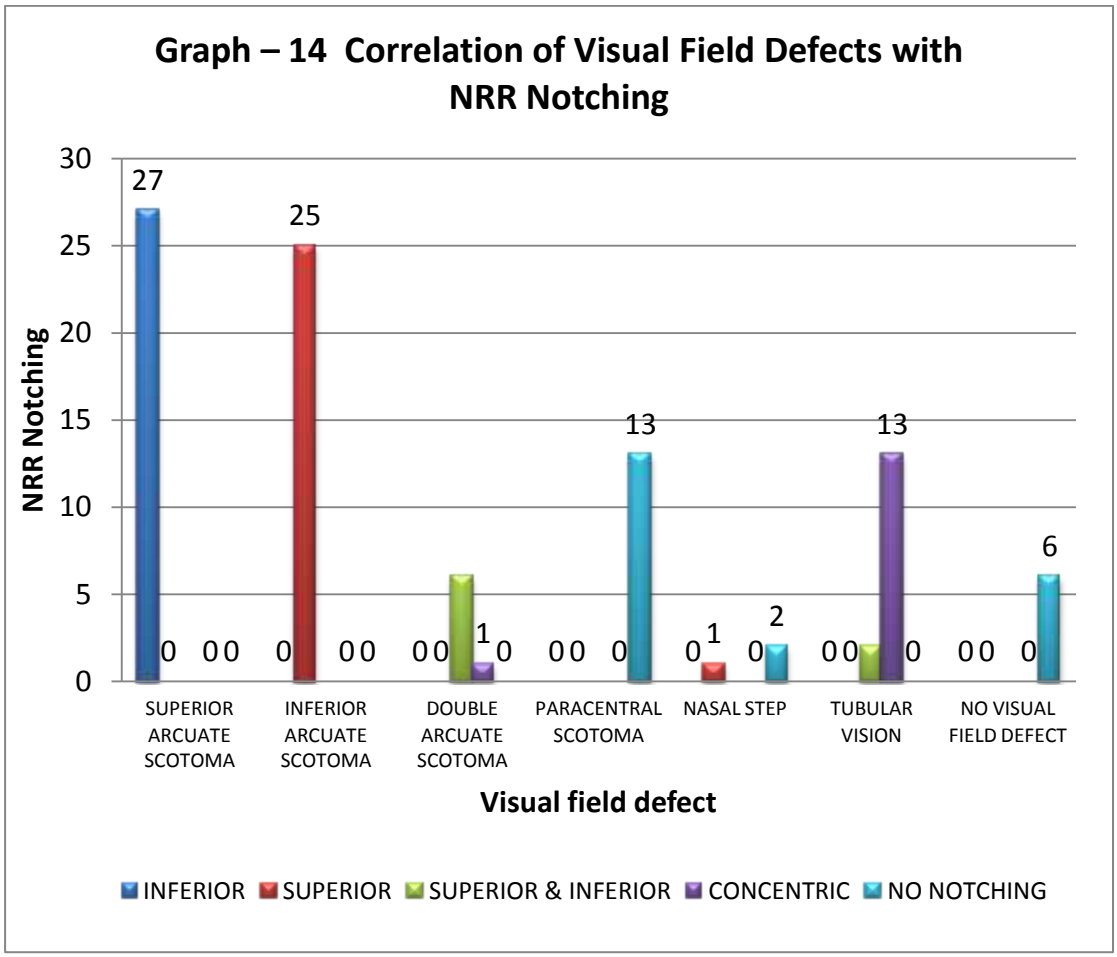
TYPE OF VISUAL FIELD DEFECT	NO. OF EYES	PERCENTAGE
SUPERIOR ARCUATE SCOTOMA	27	28.12%
INFERIOR ARCUATE SCOTOMA	26	27.08%
DOUBLE ARCUATE SCOTOMA	7	7.29%
PARACENTRAL SCOTOMA	13	13.54%
NASAL STEP	03	3.12%
TUBULAR VISION	15	15.62%
NO VISUAL FIELD DEFECT	05	5.20%
TOTAL	96	100%



In the present study, out of the 96 eyes of 48 patients with POAG, 27 eyes (28.12%) had superior arcuate scotoma, 26 eyes (27.08%) had inferior arcuate scotoma, 15 eyes (15.62%) had tubular vision, 13 eyes (13.54%) had paracentral scotoma, 7 eyes (7.29%) had double arcuate scotoma and 3 eyes (3.12%) had nasal step. Only 5 eyes (5.20%) had no visual field defects.

TABLE – 14 CORRELATION OF VISUAL FIELD DEFECTS WITH NRR NOTCHING

VISUAL FIELD DEFECT	TYPE OF NRR NOTCHING				
	INFERIOR	**SUPERIOR**	**SUPERIOR & INFERIOR**	**CONCENTRIC**	**NO NOTCHING**
SUPERIOR ARCUATE SCOTOMA	27	-	-	-	-
INFERIOR ARCUATE SCOTOMA	-	26	-	-	-
DOUBLE ARCUATE SCOTOMA	-	-	6	1	-
TUBULAR VISION	-	-	2	13	-
PARACENTRAL SCOTOMA (13), NASAL STEP (3), NO VISUAL FIELD DEFECT(5)	-	-	-	-	21



In the present study, out of the 96 eyes of 48 POAG patients, all 27 eyes with inferior neuroretinal rim notching had superior arcuate scotoma and all 26 eyes with superior neuroretinal rim notching had inferior arcuate scotoma. Six eyes with vertical elongation of the cup due to superior and inferior NRR notching had double arcuate scotoma and 1 eye with concentric NRR notching had double arcuate scotoma. Two eyes with superior and inferior NRR notching had tubular vision and 13 eyes with concentric NRR notching had tubular vision. Out of the 15 eyes with tubular vision 7 eyes had central 5 degrees of tubular vision and 8 eyes had central 10 degrees of tubular vision. Twenty one eyes had no NRR notching, out of which 13 eyes showed an isolated paracentral scotoma, 3 eyes had a nasal step and 5 eyes showed no visual field defect.

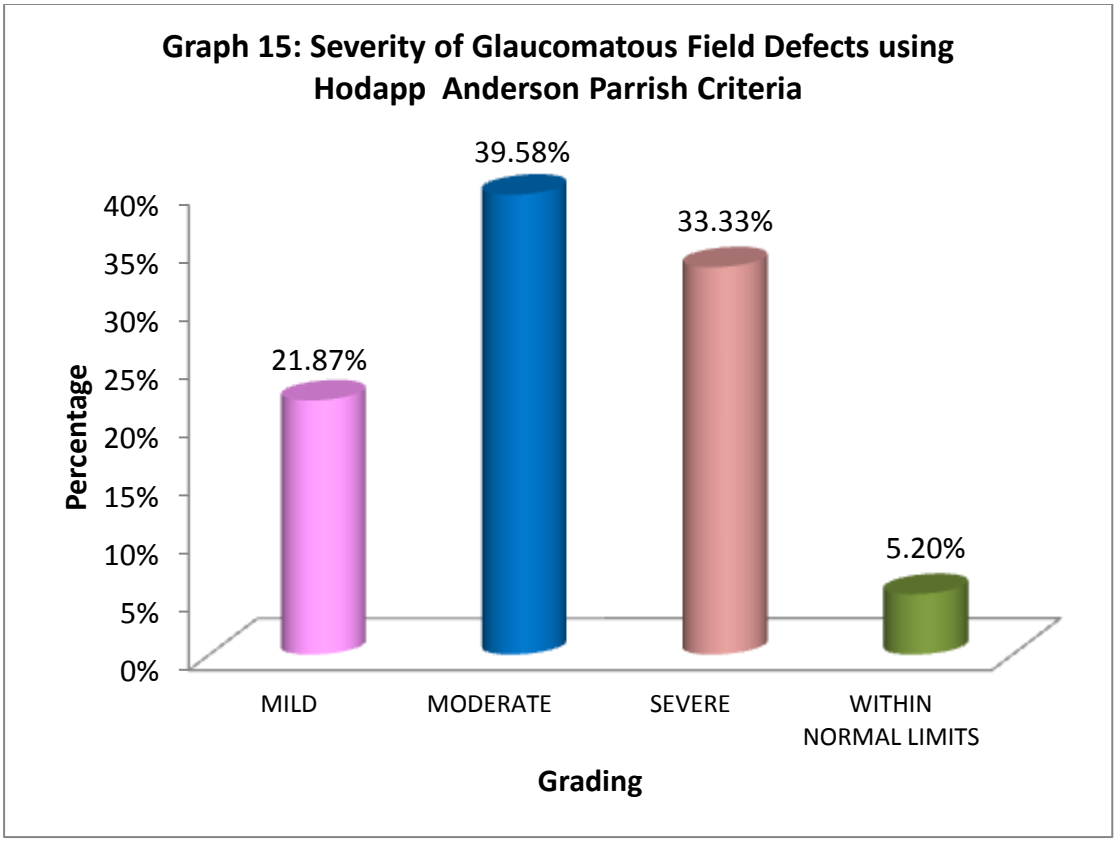
Kappa statistics is a measure of the degree of nonrandom agreement between observers or measurements of the same categorical variable .

In the present study, on correlating the type of NRR notching with the visual field defects (scotomas) using Kappa statistics, the agreement of the visual field defects with the morphometric optic disc parameter (NRR notching) was good (0.959) and statistically significant (*p<0.001).*

It was observed that neuroretinal rim loss was always associated with visual field defects except in the 13 eyes (13.14%) with isolated paracentral scotoma and in 3 eyes (3.12%) with a nasal step.

TABLE-15 DISTRIBUTION OF THE SEVERITY OF GLAUCOMATOUS FIELD DEFECTS USING HODAPP ANDERSON PARRISH CRITERIA

GRADING	NUMBER OF EYES	PERCENTAGE
MILD	21	21.87%
MODERATE	38	39.58%
SEVERE	32	33.33%
WITHIN NORMAL LIMITS	5	5.20%
TOTAL	96	100%



The visual field defects were classified as mild, moderate and severe visual field defects according to Hodapp Anderson Parrish (HAP) criteria.

Hodapp Anderson Parrish Visual Field Severity Score

Criteria for Early Defect

4. Mean deviation no worse than - 6 dB.
5. On pattern deviation plot, fewer than 25% of points depressed below the 5% level, and fewer than 15% of points depressed below the 1% level.
6. No point within central 5^0 with sensitivity < 15 dB.

Criteria for Moderate Defect

5. Mean deviation worse than - 6dB but no worse than -12 dB.
6. On pattern deviation plot, fewer than 50% of points depressed below the 5% level, and fewer than 25% of points depressed below the 1% level.
7. No point within central 5^0 with sensitivity of < 0 dB.
8. Only one hemifield containing a point with sensitivity < 15 dB within 5^0 of fixation.

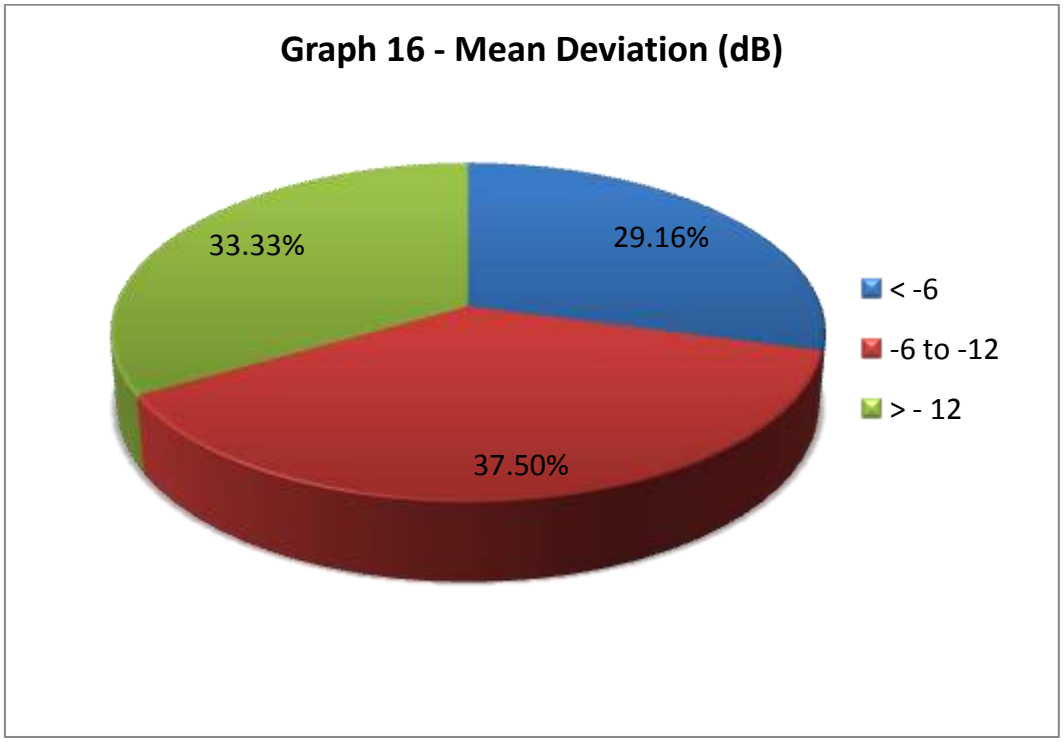
Criteria for Severe Defect

5. Mean deviation worse than -12 dB.
6. On pattern deviation plot, more than 50% of points depressed below the 5% level and more than 25% of points depressed below the 1% level.
7. Any point within central 5^0 with sensitivity of < 0 dB.
8. Both hemifields containing a point or points with sensitivity < 15 dB within 5^0 of fixation.

In the present study, 38 eyes (39.58%) had moderate visual field defects, 32 eyes (33.33%) had severe visual field defects, 21 eyes (21.87%) had mild visual field defects. Out of the 96 eyes, 5 eyes had no visual field defects.

TABLE – 16 MEAN DEVIATION (dB)

MEAN DEVIATION (dB)	NUMBER OF EYES	PERCENTAGE
< -6	28	29.16%
-6 to -12	36	37.5%
> - 12	32	33.33%
TOTAL	96	100%



In the present study, among the 96 eyes of 48 patients with POAG, 36 eyes (37.5%) had a mean deviation between -6 dB to -12 dB, 32 eyes (33.33%) had a mean deviation of more than -12 dB and only 28 eyes (29.16%) had a mean deviation of less than -6 dB.

The highest mean deviation in the present study was – 30.95 dB with a C:D ratio of 0.9 , concentric neuroretinal rim notching and tubular vision and the lowest mean deviation was 0.76 dB with a C:D ratio of 0.3 with no neuroretinal rim notching and a normal visual field.

TABLE – 17 CORRELATION BETWEEN MEAN DEVIATION dB AND C:D RATIO

C : D RATIO	MEAN DEVIATION (dB)			
	> 0	**0 to – 6**	**- 6 to – 12**	**> - 12**
0.3-0.4	5	4	-	-
0.5-0.6	-	19	19	-
0.7-0.8	-	-	17	25
0.9	-	-	-	7
TOTAL	5	23	36	32

In the present study, among the 96 eyes of 48 POAG patients, 9 eyes with a C:D ratio in the range of 0.3 to 0.4 had a mean deviation less than – 6 dB , 19 eyes with a C:D ratio in the range of 0.5 to 0.6 had a mean deviation less than – 6 dB and 19 eyes had a mean deviation between -6 dB and – 12 dB. 17 eyes with a C:D ratio of 0.7 to 0.8 had a mean deviation between – 6 dB to – 12 dB and 25 eyes had a mean deviation more than – 12 dB. 7 eyes with 0.9 C:D ratio had mean deviation more than – 12 dB. None of the eyes with a C:D ratio of 0.7 to 0.9 had mean deviation less than – 6 dB. When the increasing mean deviation (dB) was correlated with the larger cup:disc ratio using chi square test, it was found to be statistically significant with $p<0.001$, $\chi^2 = 76.753$, DF = 6.

DISCUSSION

The present cross sectional study included 96 eyes of 48 known POAG patients and glaucoma suspects who met the inclusion criteria.

Visual field analysis and optic disc evaluation are critical features used in the diagnosis and management of glaucoma. SITA standard white-on-white automated perimetry is currently the gold standard for the diagnosis, grading and detection of progression of glaucomatous visual field defects in primary open angle glaucoma. These procedures make it possible to identify the occurrence of a diffuse or localized depression of retinal sensitivity and of structural damage to the optic disc.

In glaucoma patients, it is important to detect glaucomatous visual field loss and begin adequate treatment as early as possible. In most cases, the optic disc changes occur prior to the visual field loss, so the optic disc assessment is a very important part of the evaluation of glaucoma and ocular hypertension patients. Glaucomatous optic atrophy leads to morphologic changes in the disc such as loss of neuroretinal rim, deepening of the optic cup, baring of the lamina cribrosa pores, enlargement of the peripapillary atrophy and localized and diffuse loss of the retinal nerve fibre layer and to psychophysical defects such as visual field loss.

Only a few studies have correlated the visual field defects with the morphometric disc changes among patients with glaucoma and their results indicate that measurable optic disc changes precede visual field loss in early glaucoma and in advanced stages, serial optic disc evaluation seems not as sensitive as visual field examination. Due to some inherent errors in visual field examination in previous studies, it may have contributed to incorrect predictions.

Therefore, the current study was done to correlate the optic disc parameters with the visual field defects by assessing the optic disc and to look for the features of the optic disc commonly associated with visual field loss by Humphrey visual field analyzer II using Swedish Interactive threshold Algorithm program.

When the age distribution was studied in our 48 POAG patients, the youngest patient was of 40 years and the oldest was of 82 years .The mean age of the studied population was 60.04 years and a standard deviation of 11.76 years. Suzuki et al [69] in his study found the mean age of 119 POAG patients was 63.8 ± 12.0 years which correlates with our study. Gyasi et. al [70] in his study found that the mean age of the studied population was 53.2 ±16.3 years which was slightly lower because the mean age of onset of POAG is usually lower in African population than Asian population.

Lin [71] in his study found that 80.97 % of POAG patients were over 40 years old . In a study by Song[72] he showed that in the POAG patients, age range was between 18 and 78 years and peak was seen in the population over 40 years old. From the results of above studies it is seen that POAG is uncommon before 40 years of age.

The number of POAG patients significantly increased from 20.83% in 40 to 49 years of age group to 33.33% in 60 to 69 years age group indicating that there is an increase in the number of patients with POAG with each subsequent decade of life. Increased age may reflect the cumulative effects of many factors that cause the ageing optic nerve head to be more vulnerable to IOP, even of normal range leading to POAG.

The influence of gender on glaucoma has not been as straight forward as may be expected from the generally skewed elevation in IOP among women after 40 years

of age. Results from different prevalence studies have not been conclusive in showing gender preponderance as some studies report male prevalence of POAG to be twice as high as females or vice versa while others report no such association at all. In the present study, there were double number of male patients compared to the female patients. Our findings are similar to the hospital based study done by Gyasi et al [70] , where among the total of 446 POAG patients, there were nearly twice as many males (n=292 [65.5%]) compared to females (n=154[34.5%]).

Lin et al [73] in his investigation showed that the number of male POAG patients was 2.55 times that of female POAG patients which also correlates with our study.

C. Hong et al [74], in his study, found that POAG showed a slightly higher frequency of occurrence in males (54.2 %).

These findings probably reflect the socio-cultural aspects of male dominance in our rural and sub-urban population where men control the family wealth and are more likely to have the upper hand in accessing ' pay- for- health ' care services.

A family history of POAG is generally considered to be an important risk factor for POAG and having a first degree relative with glaucoma has been consistently associated with an increased risk of POAG. In the present study only 25 % of patients had a positive family history of POAG in first degree relative (sibling, parent or child). Kellerman et al [75] in his study found that a family history of glaucoma was found in 13 to 25 % of glaucoma patients which correlates with the results of our study. In the Tajimi Study, the information obtained in the interview with participants about the family history of glaucoma was very less (5/119). [69]

As with any cross sectional study, this study had important limitations of high degree of illiteracy in our rural and suburban population leading to the decreased awareness of glaucoma in our study population and the use of oral reports of glaucoma history among relatives and also recall problems in patients about family history of POAG because of which less number of patients gave a positive family history.

There is an association between diabetes mellitus and POAG, as people with DM are more prone to POAG and diabetics tend to have higher IOP compared to non diabetics. In the present study of 48 POAG patients, 41.66% of patients were having diabetes mellitus. Lin et al [76] found that in his study group of 76,673 POAG patients, more than 30% (30.2%) had diabetes mellitus which is similar to the results obtained in the present study. Similar results were found by Jau- Der Ho et al [77] in their study of 4032 patients with POAG and found that 1043 patients (25.9%) gave a positive history of diabetes mellitus. In our study quite a number patients of POAG were found to be diabetic indicating that diabetes mellitus may be commonly seen in POAG patients.

Systemic hypertension may be associated with primary open angle glaucoma as the capillary circulation at the disc may be more precarious in a patient with systemic hypertension. In the present study 45.83 % of POAG patients gave a positive history of hypertension. Lin et al [76] in the study of 76,673 POAG patients found that more than half (50.5%) of patients had hypertension which correlates with present study. Jau- Der et al [77] in his study of 4032 POAG patients found that 1968 patients (48.8%) had a positive history of hypertension which is similar to the results of present study. Leighton et al [78] found that systolic and diastolic blood pressure

readings were significantly greater in open- angle glaucoma than either normal controls or in low tension glaucoma.

Smoking is one of the risk factors for POAG. So when the history of smoking was evaluated in the present study, 64.58 % of patients were found to be smokers. Hasnain et al [79] in his study found that out of 66 patients with POAG, 41 were smokers (62.12%) which was similar to the results obtained in the present study. Suzuki et al [69] found that among 119 POAG patients in his study 51 patients (42.85%) were smokers.

Myopia is a one of the risk factors for POAG and patients with POAG were more likely to have myopia than hypermetropia which is commonly associated with angle closure glaucoma. In the present study more number of the patients were found to be myopic (56.25%) and hypermetropia was less common (31.25%). Suzuki et al [69] in his study found that among 119 patients with POAG, 40.18% of patients were myopic. However individuals with myopia were not found to have a higher incidence or progression of glaucoma in the ocular hypertension treatment study or the early manifest glaucoma trial.

Intraocular pressure remains the most significant risk factor for POAG and indeed the only one that can be currently modulated. The mean IOP of 48 patients' 96 eyes in the present study was found to be 27.66 ± 3.23 mm Hg. Chul Hong et al [74] in his clinical study of 206 Korean glaucomatous patients found that mean IOP of POAG patients was 33.4 ± 15.5 mm Hg. In the present study the mean IOP of 96 eyes of 48 POAG patients was slightly lower may be due to the fact that maximum number of patients included in the study were known POAG patients already on antiglaucoma medications.

When the distribution of IOP across the present study was studied , more number of eyes (44.79%) were found to have IOP in the range of 25 to 29 mm Hg compared to the 21 to 24 mm Hg (11.45%), 30 to 34mm Hg (41.66%) and ≥ 35mm Hg (2.08 %) which indicated that there are more number of patients were with higher IOPs. In the present study, 86.45 % of patients had IOP in the range of 25 to 34 mm Hg which indicated that delay in seeking help and poor glaucoma status in our rural and suburban population and non compliance to medications.

The cup disc ratio is the parameter that is used to define glaucoma and is also treated as a risk factor as many studies have reported a higher CD ratio in POAG patients. Mean C:D ratio of 96 eyes of 48 POAG patients was 0.64 ± 0.15. Wensor et al [80] in the study of prevalence of glaucoma in the Melbourne Visual Impairment Project found that in 56 diagnosed POAG patients the mean C : D ratio was 0.74 with standard deviation of 0.28 which was similar to the results of present study.

In the present study 51.04 % of patients had cup : disc ratio more than 0.7. In the study by Gyasi et al [70] on presentation patterns of primary open angle glaucomas in North Eastern Ghana more than seventy percent (70.2 %) eyes had cup to disc ratio greater than 0.8 which correlates with our study.

Sommer et al [81] found that 70 % of glaucomatous eyes showed vertical contour cup : disc ratios greater than, or equal to, a value of 0.6 at the time glaucomatous visual field loss first became evident. Armaly [82] reported that cup : disc ratio was increased when visual field loss was first detected in patients with glaucoma. In the present study, the number of eyes with C:D ratio more than 0.7 is lesser compared to other studies as we have included glaucoma suspects.

It is found that higher the IOP, the larger the cup-disc ratio. In the present study correlation between IOP > 21 mm Hg and cup disc ratio of > 0.7 was found to be statistically significant with *p* value < 0.001 which indicates that higher C:D ratio was found in patients with high IOP. Varma and colleagues [83] found that the higher the IOP, the larger the cup-disc ratio. Gyasi et al [70] in his study found that the relationship between high intraocular pressure (IOP > 30 mm Hg) and vertical cup disc ratio = 1.0 was found to be statistically significant ($p = 0.0001$). It is possible that these findings derive from simple back-ward forces associated with the IOP, causing the disc surface to be positioned in a way that it enlarges the cup at higher IOP.

The diffuse loss of visual sensitivity from glaucoma is largely pressure dependent and maybe secondary to diffuse axonal dysfunction, leading to progressive concentric enlargement of the optic nerve cup. However, in the localized loss group, visual field loss seems less pressure dependent Here, loss may occur at low pressures as well as high pressures.

In the present study among 96 eyes of 48 patients of POAG, peripapillary atrophy was present in 57.29 % of eyes. Primrose et al [84] in a survey of new glaucoma patients over 40 years of age found that in 52 POAG patients the typical peripapillary halo was observed in 28 patients (53.8 %).

In the present study of 48 POAG patients, no patient showed optic disc hemorrhage . Optic disc hemorrhages usually occur more commonly in patients with normal-tension glaucoma than in patients with POAG.

In the present study other disc changes of POAG like nasalization of vessels, laminar dot sign and baring of circumlinear vessels were observed but were not evaluated as they may not be specific for POAG.

In the present study, patients with branch retinal vein occlusion with POAG were not included as one of the exclusion criterias in this study was patients with any retinal pathology that may affect the visual field analysis.

In POAG, it is difficult to detect the early changes of glaucoma with the standard achromatic perimetry (SAP) because nerve fiber degeneration and loss of visual fields do not progress in parallel in the early stages of glaucoma. The early changes in POAG are those in the thickness of the nerve fiber layer and in the morphology of the optic disc.

In the present study, out of the 96 eyes of 48 patients of POAG, 75 eyes (78.12%) showed neuroretinal rim notching and 21 eyes (21.87%) showed no notching.

More number of eyes (28.12%) had inferior rim notching followed by superior rim notching (27.08%) which conforms to the "ISNT" rule. Therefore , in our study, the earliest glaucomatous disc change was inferior neuroretinal rim notching and was a good optic disc parameter to differentiate between glaucoma suspects and early glaucoma.

In normals, the temporal rim should be the thinnest. Localized narrowing of the inferior or superior rim is characteristic of glaucoma and is usually predicted to produce a visual field defect.

A B Cullinane et al [85] studied topographic parameters of the disc in ocular hypertensives and in early glaucomatous eyes and concluded that early glaucomatous damage more commonly may occur in the inferotemporal region of the optic disc and is an effective measurement parameter in the early detection of optic disc change in glaucoma.

A normal NRR configuration is inferiorly broader than superiorly and the smallest temporally. Quigley et al [86] concluded that in glaucoma, the rate of loss of NRR on superior and inferior poles is rapid to an hour glass shaped atrophy.

It is the inferotemporal slope of the neuroretinal rim which significantly separates the ocular hypertensives from the normals. [85]

Sung Ming Hyung et al[87], in their study of forty three eyes of 28 patients reported that the parameter with the highest probability to differentiate between the optic disc of early visual field loss and normal subjects was "ratio of the thinnest NRR width in the vertical sectors to the NRR width of the temporal sector $\leq$ 85% suggesting that the neuroretinal rim is a good predictive parameter for detection of early visual field loss.

Balazsi et al [68] reported that neuroretinal rim thinning is an optic disc variable that is superior to the C:D ratio in its correlation with visual function and also in its ability to differentiate among normal eyes, eyes with suspected glaucoma and early glaucoma.

Jonas BJ and Grundler EA[88] reported that the most useful optic disc parameter for the early detection of glaucomatous optic nerve damage was the shape of the neuroretinal rim. An abnormal rim shape strongly indicates glaucomatous nerve damage.

Therefore, in the current study, early inferotemporal NRR change is consistent with the above studies. However, these findings are slightly different from Dong et al[89] who reported that sloping of the nasal inferior sector of the NRR is the earliest indicator of glaucomatous nerve damage.

The pattern of visual field loss in eyes with early damage is of utmost importance because it can facilitate our understanding of the pathophysiology of glaucomatous optic nerve damage. In the present study, in the distribution of the visual field defects. out of the 96 eyes of 48 patients with POAG more number of eyes had arcuate scotomas with superior arcuate scotomas being more common than inferior arcuate scotomas followed by tubular vision and double arcuate scotomas. The glaucoma suspects either had isolated paracentral scotomas, nasal step or normal visual fields. The patients with normal visual fields had C:D ratios in the range of 0.3 to 0.7 without neuroretinal rim notching indicating that a large optic cup is not necessarily a sign of visual field loss. However, the 13 eyes with paracentral scotomas without NRR notching showed a discrepancy between abnormal visual field and normal neuroretinal rim.

Hitchings RA, Spaeth LG [65] reported that in an individual case a large optic cup is not necessarily a sign of visual field loss. The neurons comprising the NRR are relatively constant in number, they have a constant cross sectional area. In one eye, there was a normal visual field despite 'bean pot' cupping. The undermining of the NRR is not an indicator of visual field loss. This correlates with our study.

The visual field scotoma distribution of open angle eyes in Tabriz and graz [90] showed that the most common visual field defect was nasal and peripheral scotoma beyond 20 degrees of fixation in Tabriz and arcuate visual field defect in Graz patients which is consistent with our study.

The existence of a good disc-field correlation means that the evaluation of the glaucoma patient is incomplete without ensuring agreement between the appearance of the optic disc and the state of the visual function. Therefore, in the present study,

we have correlated the optic disc parameters with the visual field indices. On correlating the NRR changes with the patterns of visual field defects we found that all eyes with inferior and superior NRR notching had superior and inferior arcuate scotomas, respectively. Eyes with both superior and inferior NRR notching with vertical elongation of the cup and one eye with concentric notching had double arcuate scotomas.

Most of the eyes with concentric NRR notching had tubular vision with a C:D ratio of 0.8 to 0.9. Thirteen eyes with no NRR notching had isolated paracentral scotomas and 3 eyes had nasal step. Five eyes with no NRR notching but cupping in the range of 0.4 to 0.7 had normal visual fields. On applying Kappa statistics, there was almost perfect correlation between optic disc parameter (NRR) and visual field defects indicating that examination of the optic disc in glaucoma allows accurate prediction of visual field defects. The C:D ratio correlated weakly with the pattern of visual field defects in our study. Both the visual fields and the optic disc must be given equal importance as either may show the first evidence of glaucomatous damage.

Hitchings RA[65] concluded that examination of optic disc photographs allowed accurate prediction of glaucomatous and normal fields to be made in 82% and 95% of eyes respectively and for visual field loss to be correctly located in the upper and lower half in 83% and 91% of cases, respectively. This correlates with our study.

Jonas BJ[88] reported that correlation between mean visual field defect and neuroretinal rim area was similar to a logarithmic function. A discrepancy between visual fields and optic disc was seen in younger individuals than in old patients. In eyes with normal visual fields, rim shape was the most important variable for

detecting glaucomatous nerve damage. In the early stage of glaucoma, the NRR area decreased more than the mean visual field defect increased. It explains why some eyes with increased IOP, abnormal optic discs, have normal visual fields.

Macri A et al [91] reported that the agreement of visual field analysis with all the optic disc parameters was good (kappa = 0.690). This correlates with our study.

Hyung et al [87] concluded that the ratio of the thinnest vertical NRR width to the temporal NRR width ≤ 85% has a high diagnosability for early field loss detection.

Cullinane BA [85] concluded that the C:D ratio and NRR area significantly separated the OHT from the POAG patients.

Miglior et al [3] reported occurrence of measurable optic disc damage before the onset of visual field defects OHT and progressive visual field loss before the development of optic disc deterioration in well established POAG. However, as our study did not address progression of the disease it is not possible to add any definitive conclusions.

Keltner LJ et al[92] concluded that both the visual fields and optic disc must be monitored with equal diligence. Changes in the ONH based on photographs and visual field changes have an increased risk of developing glaucoma. Some studies have reported that methods evaluating structural changes in glaucoma are more reliable than visual field analysis in early glaucoma. However, these studies have been conducted with scanning laser polarimetry using GDx – VCC and Heidelberg retinal tomography which can calculate more number of optic disc parameters for correlation.

So from the above studies it is seen that the NRR is the most superior optic disc variable to detect field loss and C:D ratio correlates weakly compared to NRR. Clinically, an individual with increased intraocular pressure, typical optic disc changes and normal visual fields is not only an ocular hypertensive but also a glaucomatous patient in need of treatment. The high correlation between the state of visual field and optic disc means that, in the evaluation of visual functions of a glaucoma patient, the appearance of the optic disc and the visual field should be in agreement.

The visual field defects were classified as mild, moderate and severe visual field defects according to Hodapp Anderson Parrish (HAP) criteria. In the present study, maximum eyes had moderate visual field defects and only 5 eyes had no visual field defects indicating the insidious onset of the disease and diagnosis of patients only after the disease has progressed to an extent to which glaucomatous optic nerve damage has already occurred.

The mean deviation (MD) values were obtained by the SITA standard strategy of the 30-2 program of HFA. The MDs of only reliable fields were used in the analysis.

The highest mean deviation was -30.95 dB with 0.9 C:D ratio and concentric NRR notching with central 5 degrees of tubular vision, graded as severe field defect according to HAP. On correlating the mean deviation (MD) with the C:D ratio it was found to be statistically significant ($p < 0.001$). However, there were a few eyes with 0.5 to 0.6 C:D ratio with a mean deviation less than – 6 dB.

Omodaka K et al[93] reported that the vertical C:D ratio had the highest correlation coefficient to the mean deviation among all the optic disc parameters. This correlates with our study.

Cullinane BA [85] found a significant difference in visual field mean deviation (MD) between normal and POAG patients. Among the highest individual correlations were those found between visual field MD and C:D ratio ($r = -0.40$, $p < 0.001$) which correlates with our study.

Lan WY et al [94] reported that in the early stage of glaucoma, a small change in the MD was accompanied by a large amount of rim loss and in later stages, a large change in MD was accompanied by a small change in rim loss.

For MD between 0 dB to – 6dB, the relative rim loss can range from 0 to 70% and for MD between – 6 dB and -12 dB, the relative rim loss ranged only from 40 % to 80 %. When the patients with MD worse than – 10 dB were excluded from the study, the correlation weakened considerably.

Therefore, in our study, this explains why a few eyes with C:D ratio of 0.5 to 0.6 had MDs less than -6 dB with only a mild visual field defect. However, in our study, 33.33 % eyes had MD more than – 12 dB with advanced glaucoma making the correlation with the C:D ratio statistically significant.

CONCLUSION

The conclusions drawn from the present study were as follows:

- In the present study, the visual field defects correlated with the optic disc parameter neuroretinal rim notching with $p < 0.001$, Kappa statistics = 0.959.
- The neuroretinal rim shape is a optic disc parameter superior to the C:D ratio in detecting early glaucomatous nerve damage and to predict visual field loss.
- The inferior neuroretinal rim notching is the earliest glaucomatous nerve damage detected in POAG patients in this study.
- Isolated paracentral scotomas and the arcuate scotomas are the commonest early visual field defects in the present study.
- The correlation between the increasing C:D ratio and the intraocular pressure was statistically significant $p < 0.001$. The diffuse loss of visual sensitivity from glaucoma is largely pressure dependent leading to progressive concentric enlargement of the optic nerve cup.
- However, a large C:D ratio is not necessarily a sign of visual field loss as the neurons comprising the NRR remain constant.
- Hodapp Anderson Parrish (HAP) scale can be helpful in classifying glaucoma severity to set target intraocular pressure goals.
- The mean deviation by HFA correlates with the C:D ratio in advanced glaucoma more than in early glaucoma $p < 0.001$, DF = 6, $\chi^2 = 76.753$.

- The high correlation between the state of visual field and optic disc means that, in the evaluation of visual functions of a glaucoma patient, the appearance of the optic disc and the visual field should be in agreement and both the visual fields and optic disc must be monitored with equal diligence as either of the two may show the first evidence of glaucomatous optic nerve damage.

SUMMARY

A one year cross sectional study was done to correlate the optic disc changes with the visual field defects by optic disc evaluation and by using Humphrey Visual Field Analyzer at KLES Dr Prabhakar Kore Hospital and MRC, Belgaum.

A total of 96 eyes of 48 primary open angle glaucoma patients fulfilling the inclusion criteria were included in the study. The intraocular pressure was measured by applanation tonometry and gonioscopy was done with Goldmann 3 mirror gonioscopic lens. The optic disc was evaluated by slit lamp biomicroscopy using 90D lens and magnified photographs were taken with a canon retinal digital camera.

Central field testing was performed with the 30-2 program SITA standard strategies. Correlation between the optic disc parameters and the visual field defects was calculated using Kappa statistics and Chi square test.

Mean age of patients was 60.04 ± 11.76. Thirty two of 48 glaucoma patients were male (66.66%) 16 were female (33.33%). There were more number of males in the present study.

The mean IOP of 96 eyes of 48 patients was 27.66 ± 3.23 mm Hg. and the mean C:D ratio was 0.64 ± 0.15.

In present study, on correlating the neuroretinal rim notching with the visual field defects using kappa statistics, it was found to be statistically significant with $p < 0.001$. We correlated the increasing C:D ratio with the IOP using chi square test and it was found to be statistically significant $p < 0.001$. The mean deviation (dB) was correlated with the C:D ratio and was statistically significant $p < 0.001$ in advanced glaucoma more than in early stages of glaucoma.

The neuroretinal rim is an optic disc variable that is the most superior optic disc variable to detect field loss and C:D ratio correlates weakly compared to the NRR.

In our study, the earliest glaucomatous disc change was inferior neuroretinal rim notching and was a good optic disc parameter to differentiate between glaucoma suspects and early glaucoma.

A large C:D ratio is not necessarily a sign of visual field loss as the neurons comprising the NRR remain constant.

Hodapp Anderson Parrish (HAP) scale can be helpful in classifying glaucoma severity to set target intraocular pressure goals.

Thus the current study shows that an individual with increased intraocular pressure, typical optic disc changes and normal visual fields is not only an ocular hypertensive but also a glaucomatous patient in need of treatment.

The high correlation between the state of visual field and optic disc means that, in the evaluation of visual functions of a glaucoma patient , the appearance of the optic disc and the visual field should be in agreement and both the visual fields and optic disc must be monitored with equal diligence as either of the two may show the first evidence of glaucomatous optic nerve damage.

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ANNEXURE – I:

PROFORMA FOR DATA COLLECTION

Patient ID No | | | |

Name of the Patient:

Age: | | | Years

Sex: | | (1-Male, 2-Female)

OP No: | | | | | | | |

IP No: | | | | | | |

Date: | | | | | | | | |

Is the patient eligible for study | | 1- Yes, 2-No

Has informed consent been given | | 1- Yes, 2-No

Chief complaints and history of present illness

1. Diminution of vision (Tick whichever is applicable)

Gradual		Distant		Painless	
Sudden		Near		Painful	

Duration | | (1 –Right eye, 2 –Left eye, 3-Both)

2. Coloured haloes | | (1-Yes, 2-No) if yes specify

3. Photophobia | | (1-Yes, 2-No) if yes specify

4. Lacrimation | | (1-Yes, 2-No) if yes specify

5. Redness of eye | | (1-Yes, 2-No) if yes specify

6. H/o wearing spectacles ☐ (1-Distance, 2- near, 3-Both)

7. H/o Frequent change of presbyopic glasses ☐ (1- Yes, 2- No)

Past History (1-Yes, 2- No) if yes , specify the details

1. Any disease, surgery, trauma to the eye being tested ☐

2. Diabetes ☐ Duration

3. Hypertension ☐ Duration

4. Thyroid disease ☐

5. Cerebrovascular event ☐

6. Any retinal pathologic condition affecting the visual field ☐

Family History –of Glaucoma ☐ (1-Yes, 2-No) if yes, specify the details

Treatment history/Drug intake (1-yes, 2-No) if yes, specify the drug used, duration, frequency.

1. Miotics ☐

2. Systemic medication ☐

3. Steroids ☐

Personal History

1. Smoking ☐ (1-Yes, 2 –No)
2. Alcoholism ☐ (1-Yes, 2 –No)
3. Diet ☐ (1-Veg,2-Non Veg,3-Mixed)
4. Appetite ☐ (1-Good, 2- Reduced)
5. Bowel and bladder ☐ (1-Regular, 2-Not regular)

GENERAL PHYSICAL EXAMINATION

Pulse /minute

BP mm of Hg

RR /minute

CVS ☐ 1-Normal, 2-Abnormal, if abnormal specify

RS ☐ 1-Normal, 2-Abnormal, if abnormal specify

CNS ☐ 1-Normal, 2-Abnormal, if abnormal specify

P/A ☐ 1-Normal, 2-Abnormal, if abnormal specify

OCULAR EXAMINATION

	Right Eye	Left Eye
1) Visual Acuity	☐	☐

1-6/6to6/9,2-6/12to6/18,3-6/24to6/36,4-6/60 ,5-cf 3 mt to cf mt, 6-cfcf,7-PLPR

1. With pinhole	☐	☐
2. With spectacles	☐	☐
3. Near vision	☐	☐

(1-n6,2-n8,3-n10,4-n12,5-n18,6-n36,7-n<36)

2) Adnexa	1-Normal 2-Abnormal If abnormal specify	☐	☐
3) Conjunctiva	1- Normal 2 -Congested	☐	☐

4) Sclera 1-Normal ☐ ☐

2-Abnormal (If abnormal ,then specify)

5) Cornea

a) Corneal sensation 1-Normal ☐ ☐

2-Diminished

3-Absent

b) Corneal oedema 1-present, 2-absent ☐ ☐

c)Vascularization 1-present, 2-absent ☐ ☐

d)Bullous keratopathy 1-present, 2-absent ☐ ☐

e)Keratic Precipitates 1-present, 2-absent ☐ ☐

6) Anterior chamber Depth 1-Normal ☐ ☐

2-Shallow

3-Deep

7) Iris 1-Normal ☐ ☐

2-Abnormal (if abnormal, then)

2a) Loss of pattern (1-present,2 –absent) ☐ ☐

2b) Atrophic patches (1-present,2-absent) ☐ ☐

2c) Posterior synechiae (1-present,2-absent) ☐ ☐

2d) Peripheral anterior synechiae ☐ ☐

(1-present,2-absent)

2e)Neovascularization (1-present,2-absent) ☐ ☐

2f) Coloboma (1-present,2-absent) ☐ ☐

2g) Holes (1-present,2-absent) ☐ ☐

2h) Glaucoma flaken (1-present,2-absent) ☐ ☐

8) Pupil

1-Size (mm) ☐ ☐

2-Shape ☐ ☐

3 Reaction i-Brisk

ii-Sluggishly reactive

iii-non-reactive

A direct light reflex ☐ ☐

B Consensual light reflex ☐ ☐

4 Afferent pupillary defect ☐ ☐

(1-Present, 2-Absent) ☐ ☐

9) Lens 1. Normal

2-Cataract A-Immature, B-Mature, C-Hypermature

3. Pseudophakia

Right eye Left eye

10.Refraction Correction

INVESTIGATIONS

Intraocular pressure (IOP mm Hg) measurement

Applanation Tonometer

RE mm Hg

LE mm Hg

2) GONIOSCOPY

According to Shaffer's system of grading the angle width

RE

	Superior	Inferior	Temporal	Nasal
Grade				

LE

	Superior	Inferior	Temporal	Nasal
Grade				

3) FUNDUS EXAMINATION by 90 D Slit Lamp Biomicroscopy

OD OS

1. Glow 1-Good,2 –faint,3-absent

2. Media 1-clear,2-hazy

3. Disc 1- normal,2-abnormal

4. C:D 1- 0.3 to 0.4

2- 0.5 to 0.6

3- 0.7 to 0.8

4- 0.9

5. Vessels A Arteries 1-Normal

2-Narrowing

B Veins 1-Normal

2-Dilated

3-Tortous

4-Sheathing

C. AV ratio

6. Background

7. Macula

Disc examination in glaucoma by 90 D slit lamp biomicroscopy

OD OS

a) Cup :disc ratio

1- 0.3 to 0.4

2- 0.5 to 0.6

3- 0.7 to 0.8

4- 0.9

b) Neurorentinal rim 1-normal,2-thining

c) Notching 1-present,2-absent

d) Pallor areas on the disc 1–present,2-absent

e) Position of blood vessels 1-normal,2-nasalisation

f) Splinter hemorrhages 1–present,2-absent

g) Peripapillary atrophy 1–present,2-absent

h) Pulsations of retinal arterioles 1–present ,2-absent

i) Bayoneting sign 1–present,2-absent

j) Laminar dot sign 1–present,2-absent

k) Atrophy of retinal nerve fiber layer 1–present,2-absent

l) Barring of circumlinear vessels 1- present,2-absent.

4) Visual field evaluations

It is done with 30-2 program Swedish interactive threshold algorithm (SITA standard) using Humphrey's field analyzer.

A) Glaucoma hemifield test OD OS

1-within normal limits

2-borderline

3-outside normal limits

B) Mean deviation (MD)

1 < -6dB

2 > -6 dB but < - 12 dB

3 > -12dB

C) Pattern deviation plot

1- <25% points depressed below 5% level

<15% points depressed below 1% level

2- >25% points depressed below 5% level

<25% points depressed below 1% level

3- >50% points depressed below 5% level

>25% points depressed below 1% level

D) PSD

1-Depressed to the P<5%

2-Not depressed to the P<5%

E) Sensitivity in central 5^0 field

I- No point within the central 5^0 with sensitivity less than 15dB

II- No point within the central 5^0 with sensitivity less than 0dB

III-Any point within the central 5^0 with sensitivity less than 0dB

F) Superior and inferior hemifield

i-No hemifield containing a point(s) with sensitivity less than 15dB within 5^0 fixation.

ii- Only one hemifield containing a point(s) with sensitivity less than 15dB within 5^0 fixation

iii-Both hemifield containing a point(s) with sensitivity less than 15dB within 5^0 fixation

G) Reliability indices

a) Fixation losses 1<20%

2>20%

b) False positive errors 1<33%

2>33%

c) False negative errors 1<33%

2>33%

5) Magnified fundus photographs taken after dilatation of the pupil with 0.8% tropicamide and 5% phenlyephrine eye drops.

ANNEXURE - II

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr/Mrs/Miss

You are invited to participate in our research study titled **"CORRELATION OF OPTIC DISC CHANGES WITH VISUAL FIELD DEFECTS IN PRIMARY OPEN ANGLE GLAUCOMA – A ONE YEAR CROSS SECTIONAL STUDY"** conducted by **Dr. _____,** Post Graduate in MS Ophthalmology under guidance of **Dr. _____,** Professor in the Department of Ophthalmology, J.N. Medical College, Belgaum.

Respected Sir/Madam, we request you to participate in our study as you are eligible to do so.

Your participation in the research is voluntary. Your decision whether to or not to participate in the study will not affect your relationship with J. N. Medical College. If you decide to participate you are free to withdraw at any time. You will be told all the new information available about the study and you will be given free will to decide about participation and continuation in this study.

PURPOSE OF THE STUDY

The purpose of the current study is to correlate the optic disc changes with the visual field defects in primary open angle glaucoma using the Humphrey's field analyzer with 30-2 program , Swedish interactive threshold algorithm.

Procedure involved

If you agree to enroll yourself in this study, I will ask your present, past, family and personal history. Then you will be clinically examined in detail by slit lamp examination, refraction done for best corrected visual acuity, IOP measured by applanation tonometer, gonioscopy done by Goldmann three mirror lens to look for the angles, fundoscopy by 90D SLE for detailed examination of the optic disc. Magnified fundus photographs will be taken with canon retinal camera.

Then visual field evaluation will be done with SITA Standard using Humphrey field analyzer.

Risks and benefits

With all the necessary precautions taken there is a minimal risk involved with the tests.

Your participation may benefit you and others suffering from the same disease in future by helping us to learn more about the disease process and the efficiency of these tests in detecting glaucomatous field defects.

COSTS FOR PARTICIPATING IN THIS RESEARCH

There will be no extra cost incurred by the participant. The participant will however have to pay for the investigations and tests which are part of the existing management protocol for this ailment.

There is no commitment for any reimbursement or any other compensation for the participant.

PRIVACY AND CONFIDENTIALITY

The only people to know that you are a research subject are members of the research team. No information provided by you during the research will be disclosed to others without your written permission.

AUTHORIZATION TO PUBLISH RESULTS

When the results of this study/research are published in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

CONTACT DETAILS

If you have any questions and clarifications or if you need help at any time during the study period about this research/study. You may please contact.

1) Chief Investigator Dr. _____, Dept of Ophthalmology, JNMC, Belgaum, Ph.No : _____.
2) Dr. _____, Professor, Guide, Department of Ophthalmology. JNMC, Belgaum. Ph : _____
3) Dr. _____, Principal, JNMC, Belgaum and Chairman of Institutional Ethics Committee. Ph . _____.

CONSENT FOR PARTICIPATION IN RESEARCH TRIAL

I , Mr/ Mrs / Ms _____voluntarily agree for the participation as a subject for this study. By signing this consent form, I am not giving up any of my legal rights , I may withdraw from the study anytime . I am signing the consent form after having read or been read for me in my vernacular language about the study in detail including the risks and benefits and having all my questions answered .

Signature or the Left Thumb Print of participant :

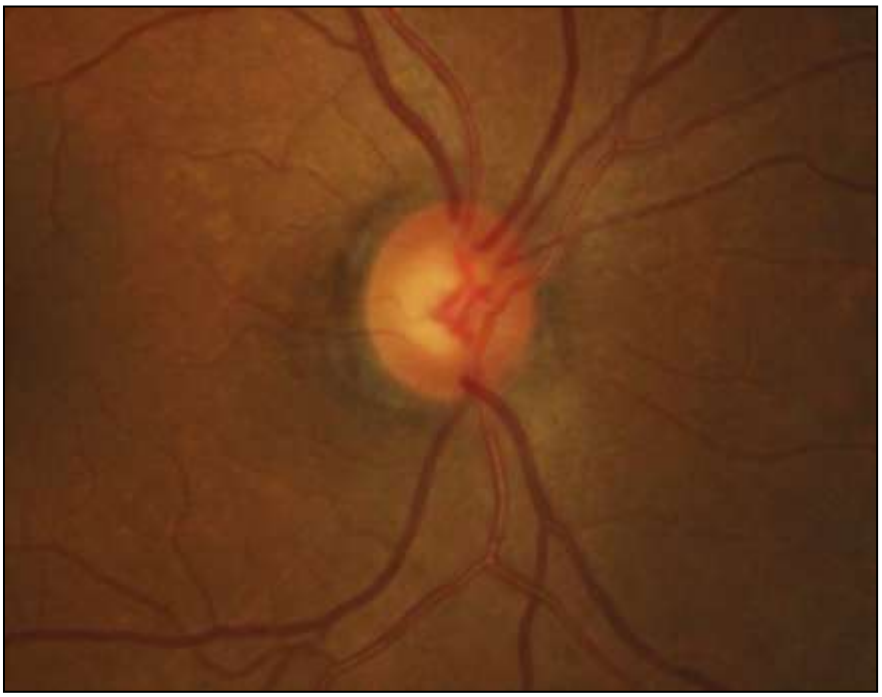
Investigators Name :

Investigators Signature :

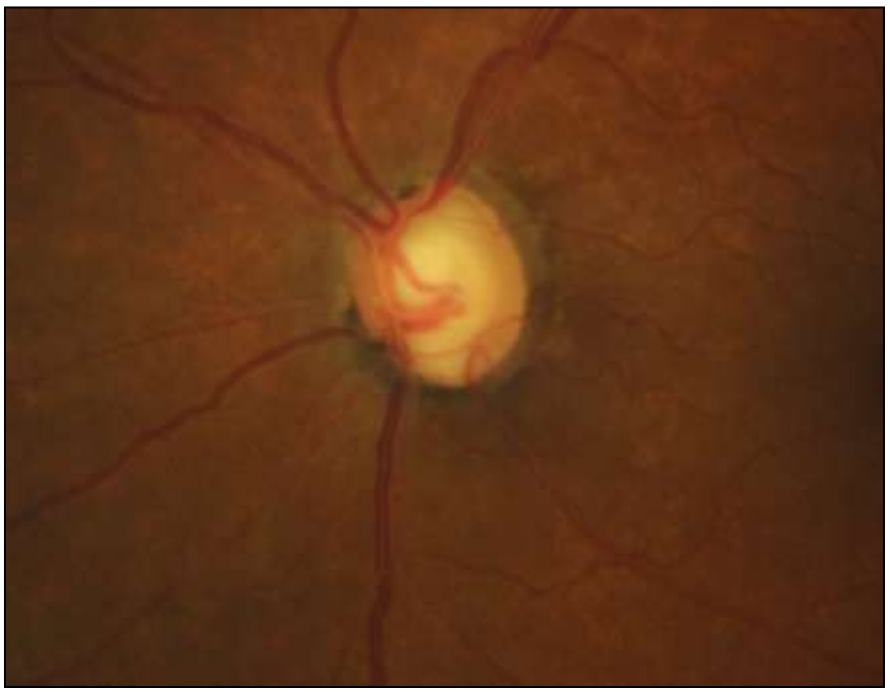
Date :

Place :

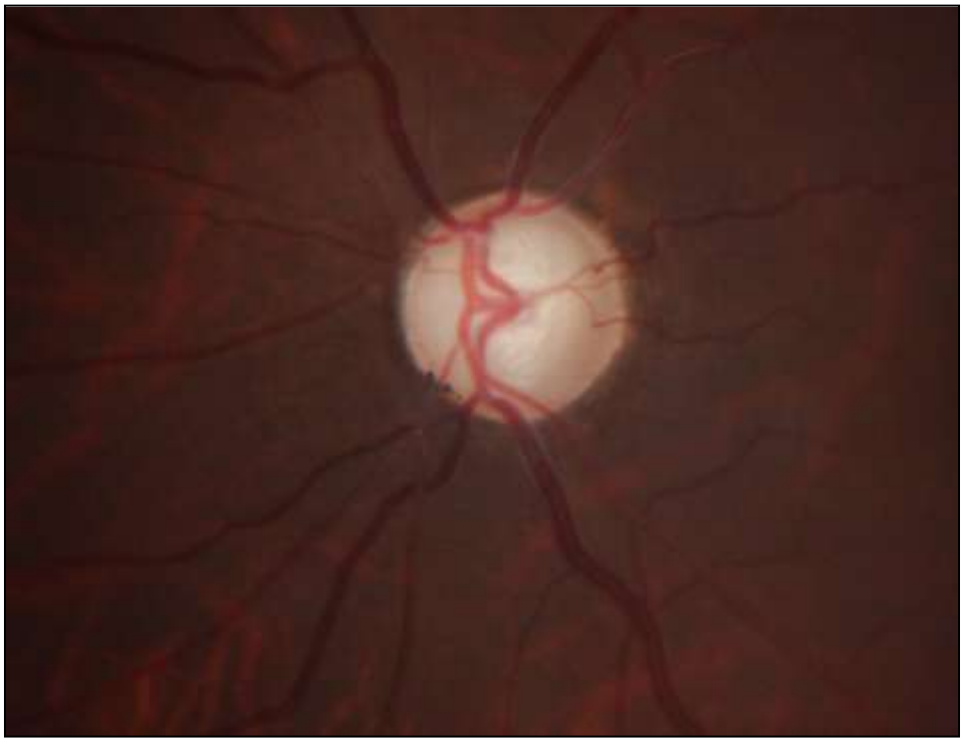
ANNEXURE – III
PHOTOGRAPHS



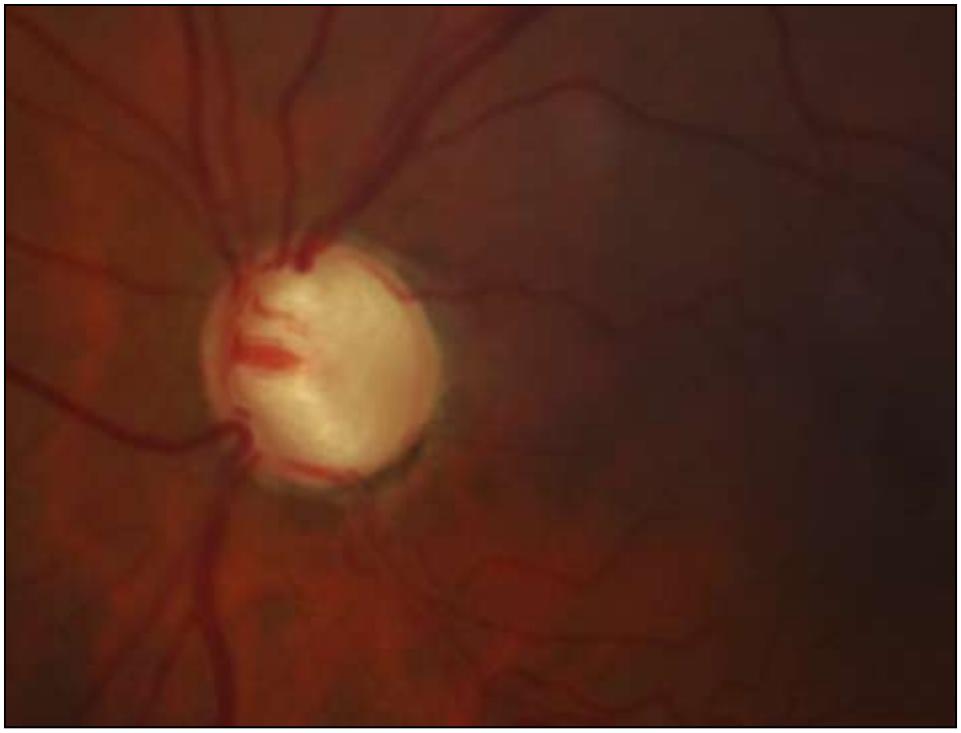
Photograph No.1: Fundus photograph of optic disc showing normal C:D ratio of 0.3 to 0.4 with healthy neuroretinal rim



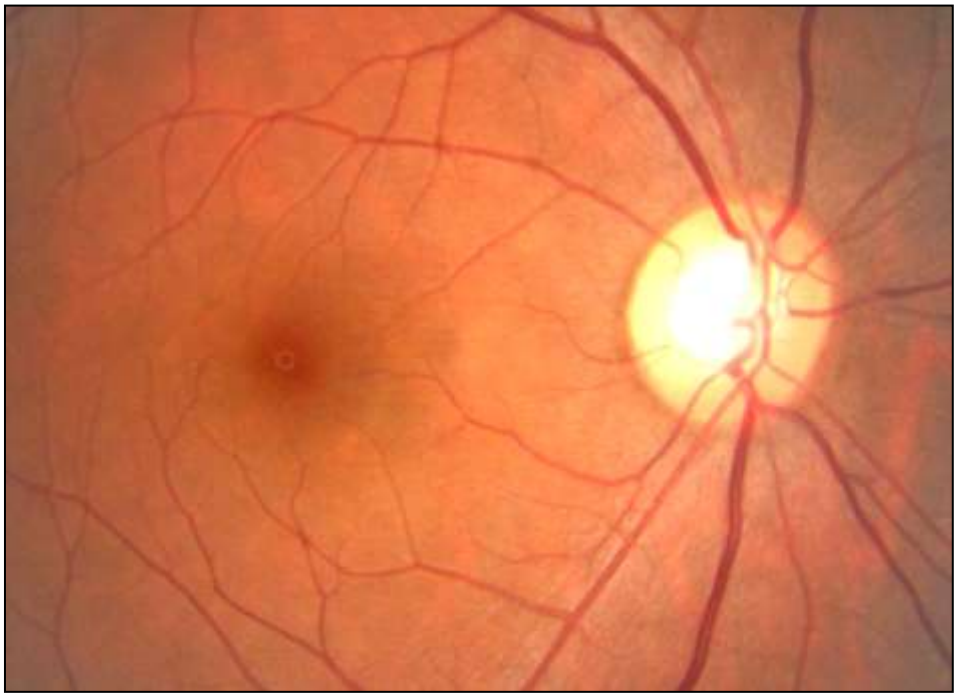
Photograph No.2: Fundus photograph of optic disc showing inferior notching with peripapillary atrophy



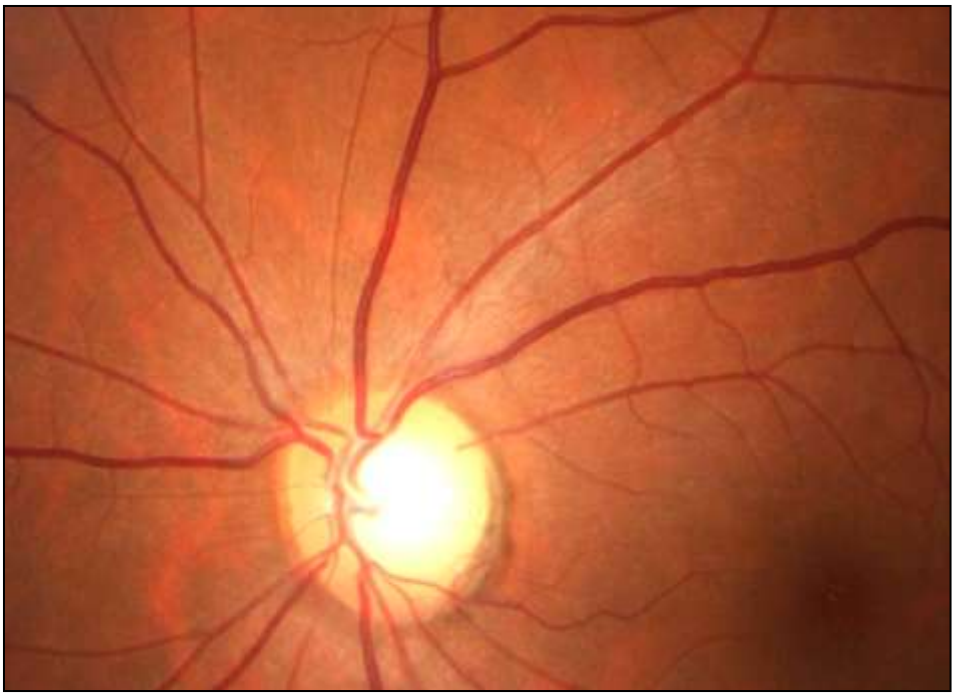
Photograph No.3: Fundus photograph of optic disc showing diffuse thinning of NRR, 0.7 cupping with laminar dot sign



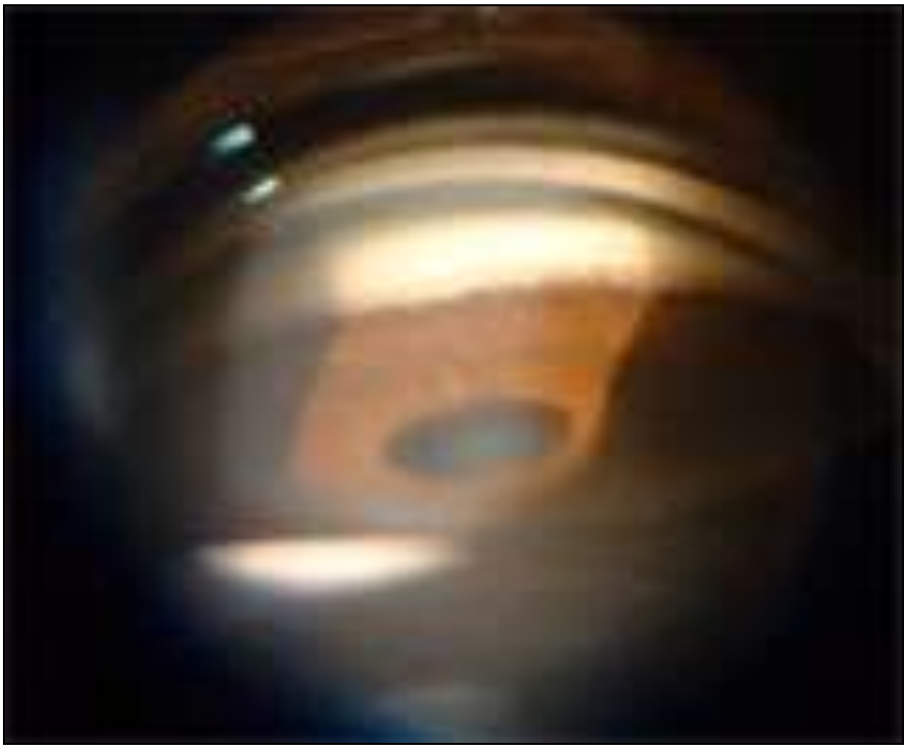
Photograph No.4: Fundus photograph showing 0.9 cupping , 'bean pot' cupping nasalization of vessels and peripapillary atrophy



Photograph No. 5 : Fundus photograph showing 0.6 cupping with nasalization of vessels and superior neuroretinal rim notching.



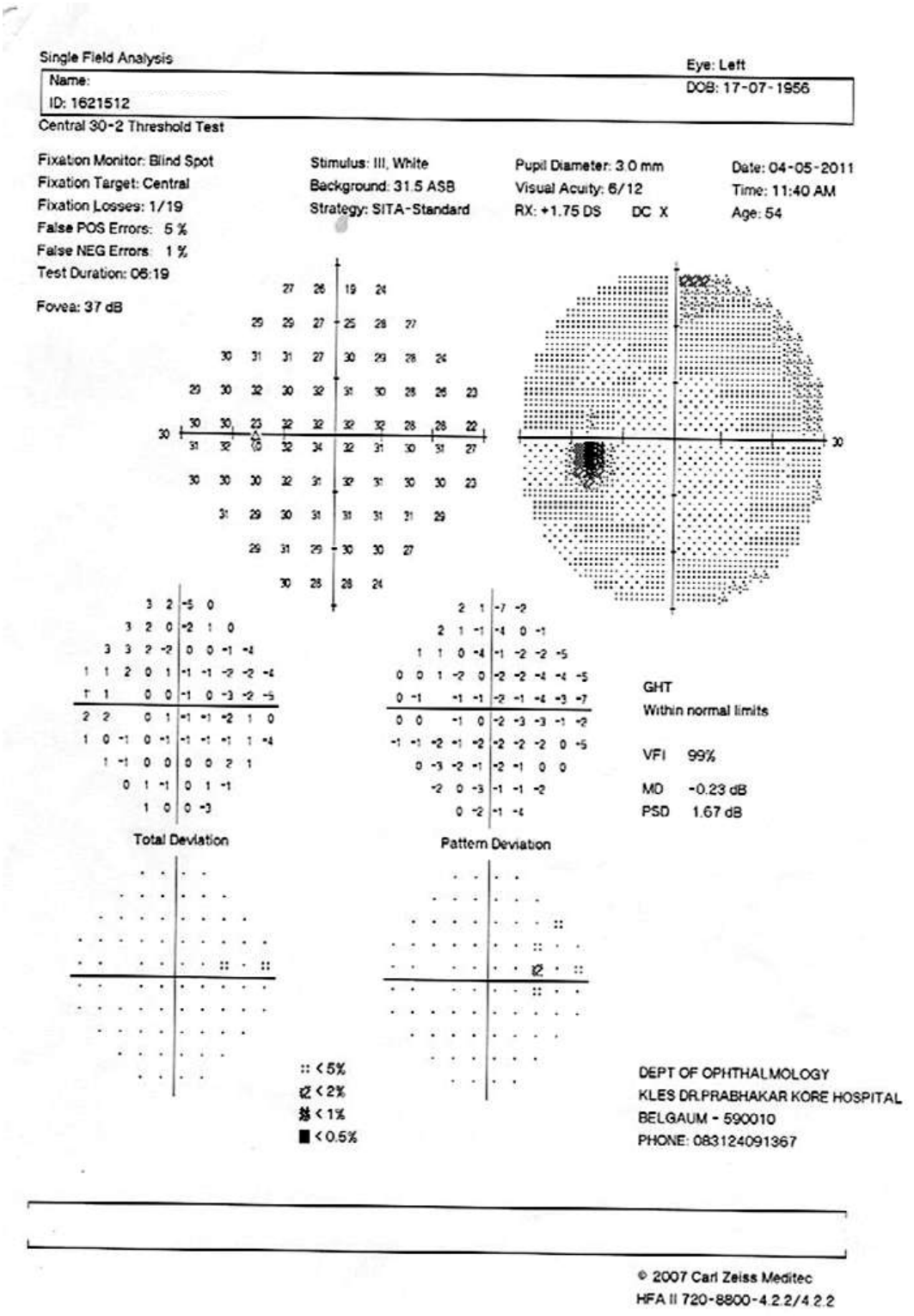
Photograph No. 6: Fundus photograph showing 0.6 cupping with superior and inferior neuroretinal rim notching



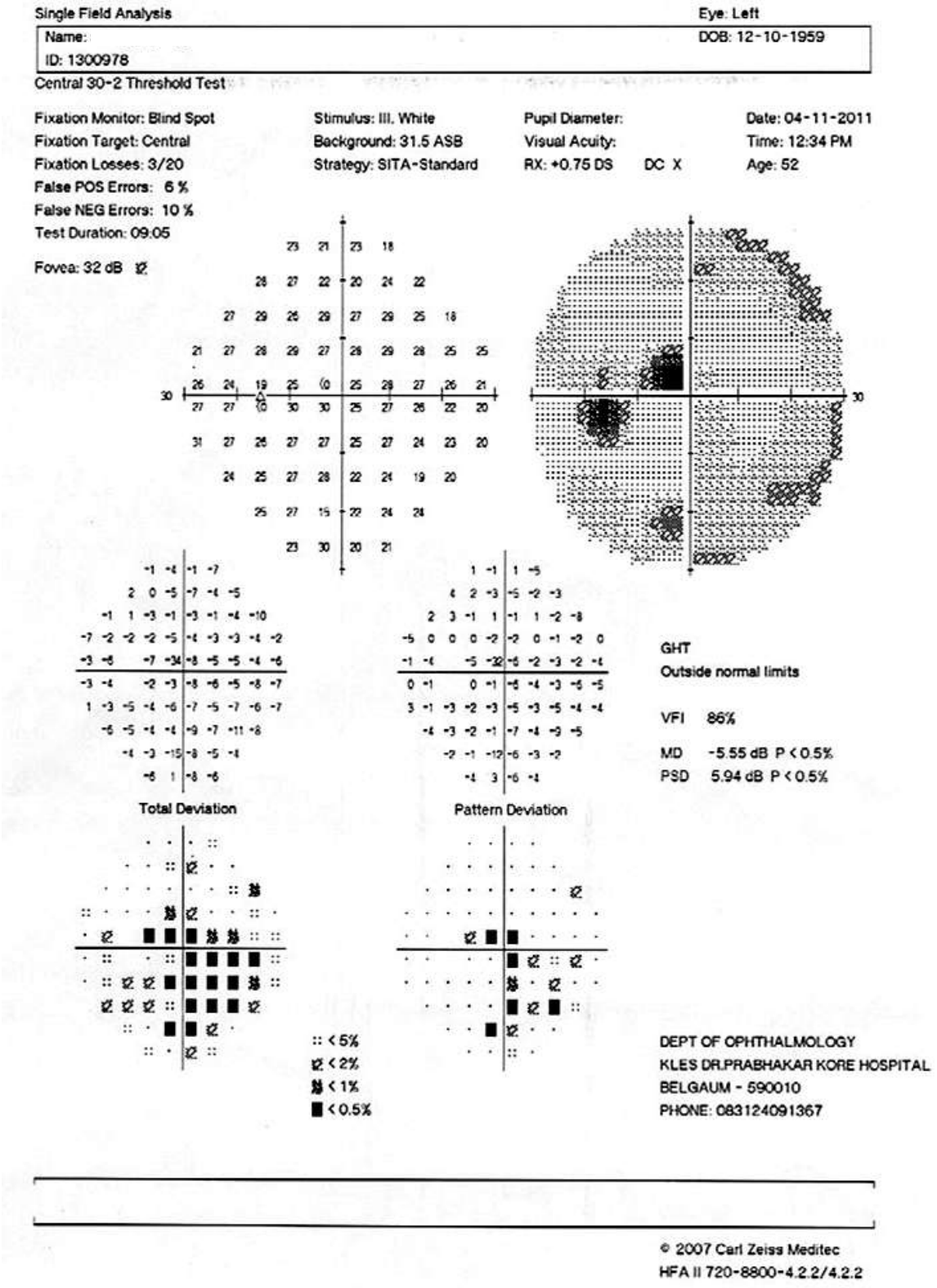
Photograph No. 7 : Open angle structures visible on Gonioscopy with Goldmann 3 mirror Gonioscope

ANNEXURE - IV

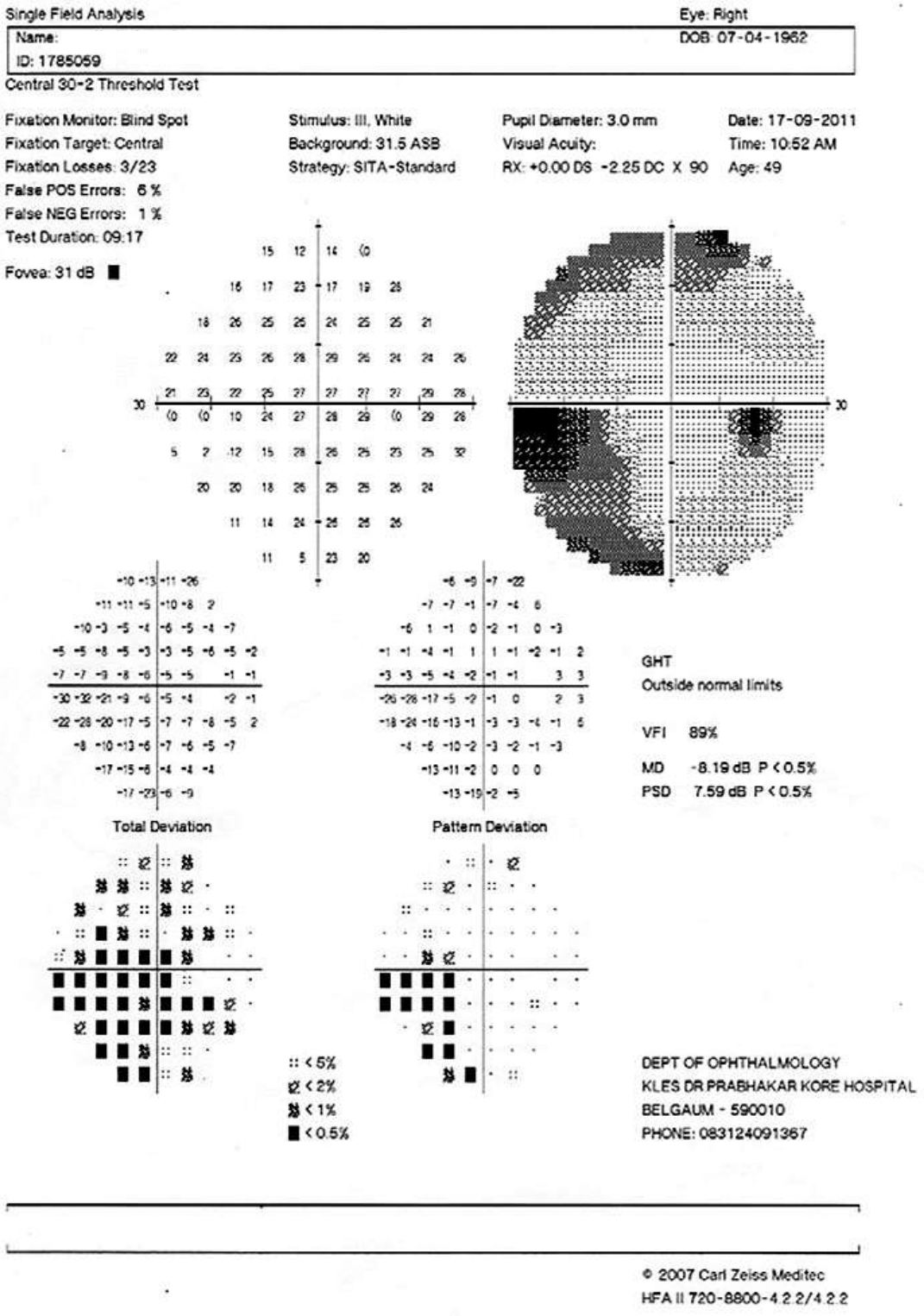
LIST OF SINGLE FIELD ANALYSIS PRINTOUTS



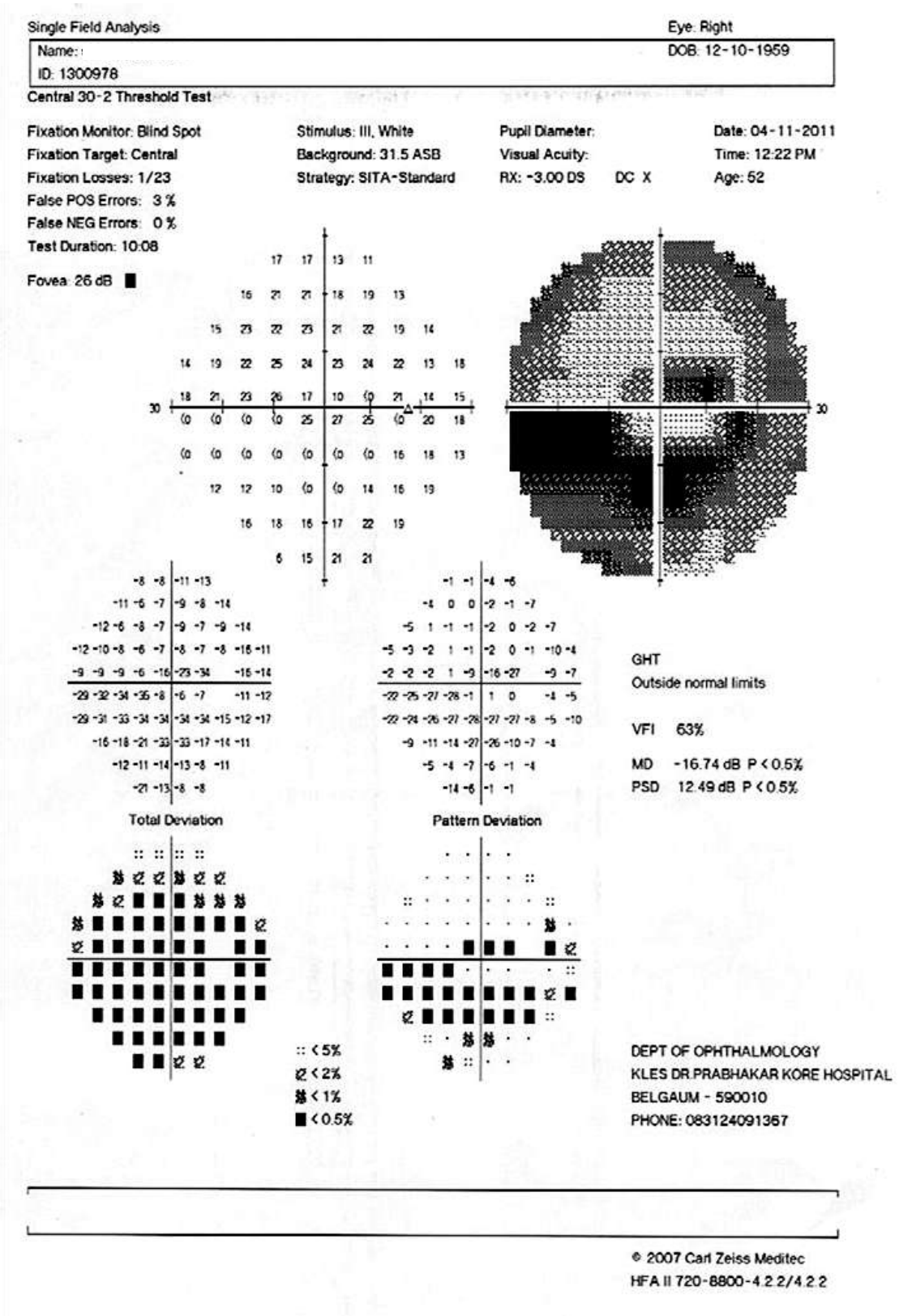
1) Single field analysis print out of 30-2 SITA standard showing normal visual field



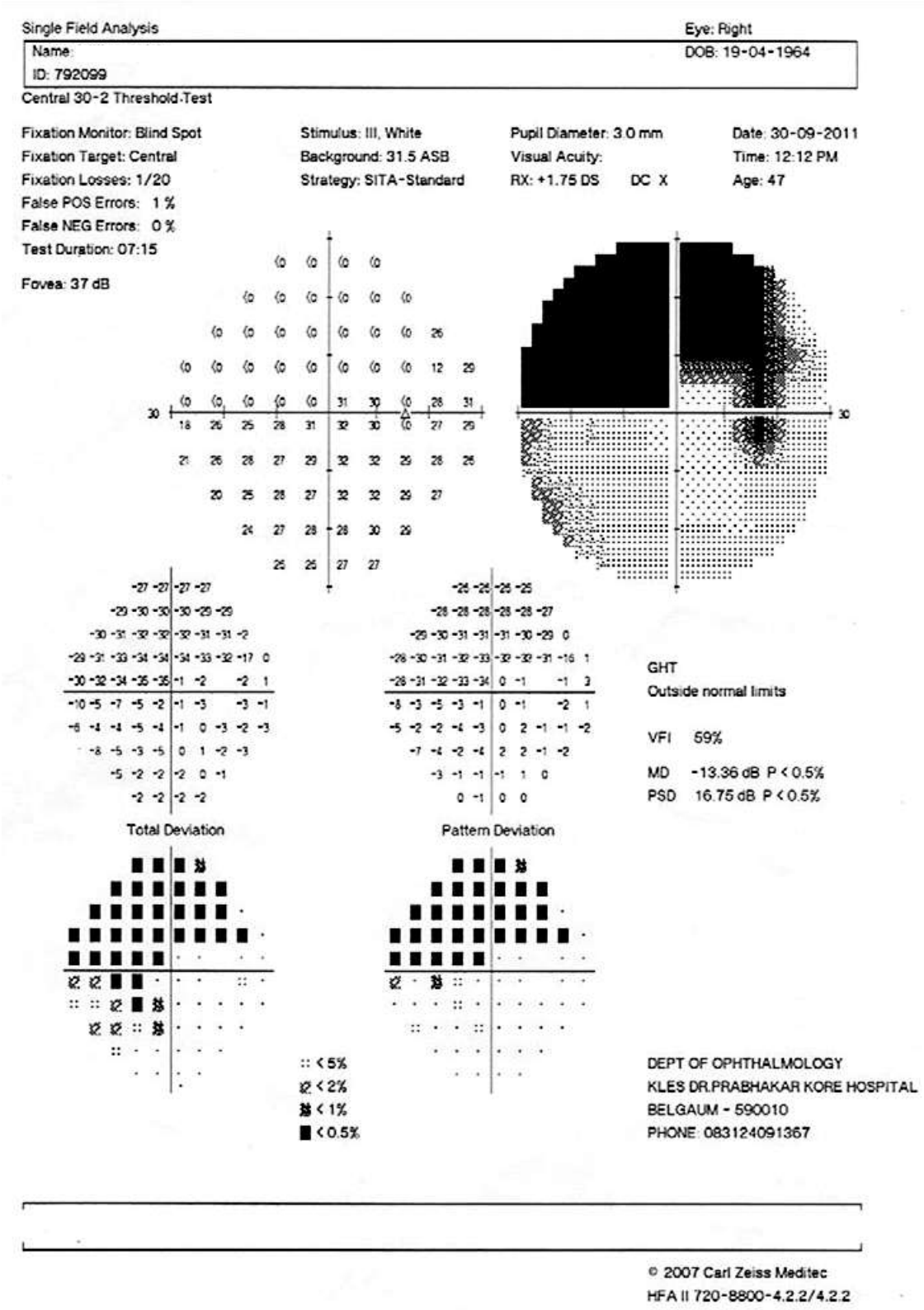
2) Single field analysis print out of 30-2 SITA standard showing mild visual field defect with early paracentral scotoma



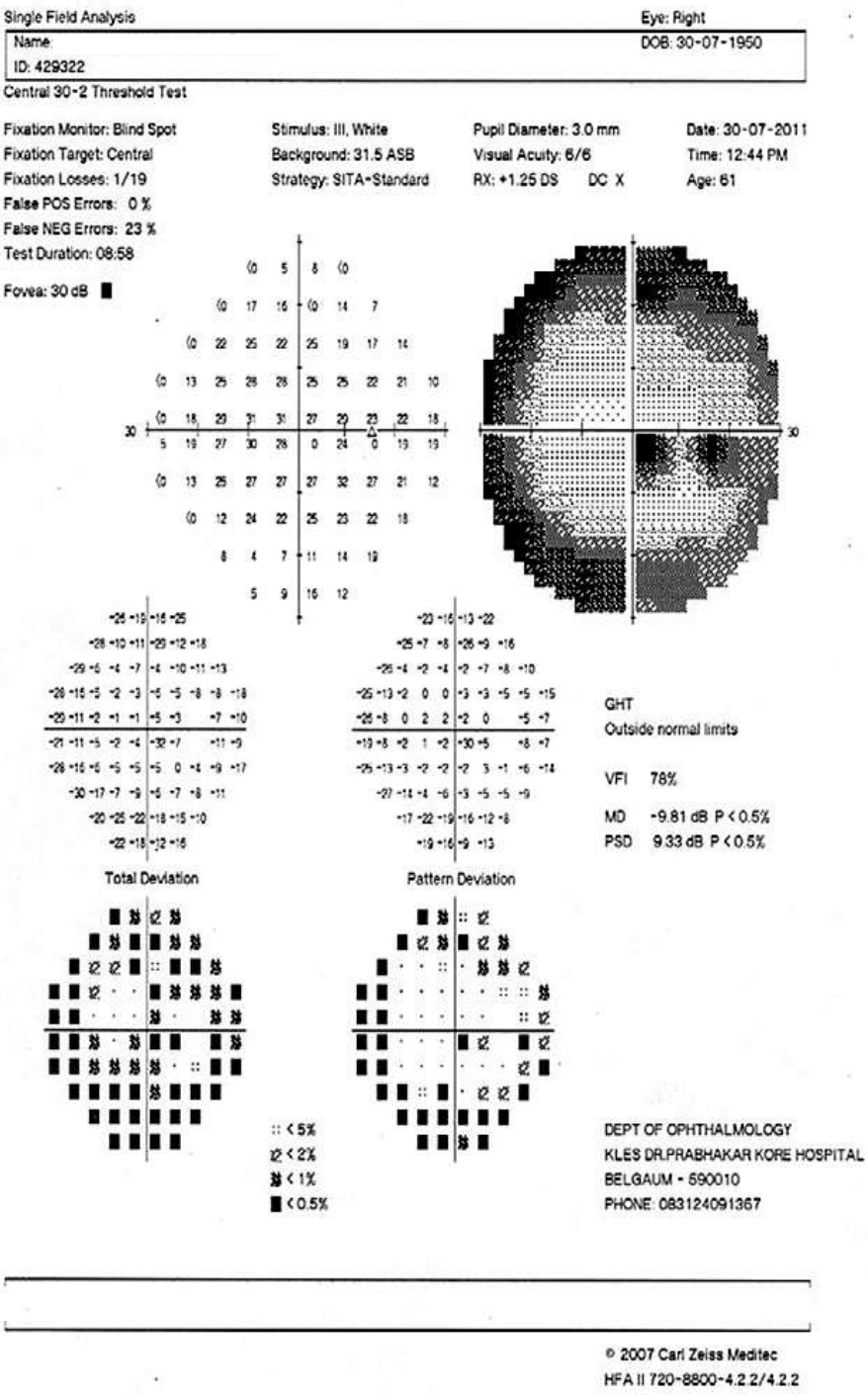
3) Single field analysis print out of 30-2 SITA standard showing moderate visual field defect (inferior nasal step)



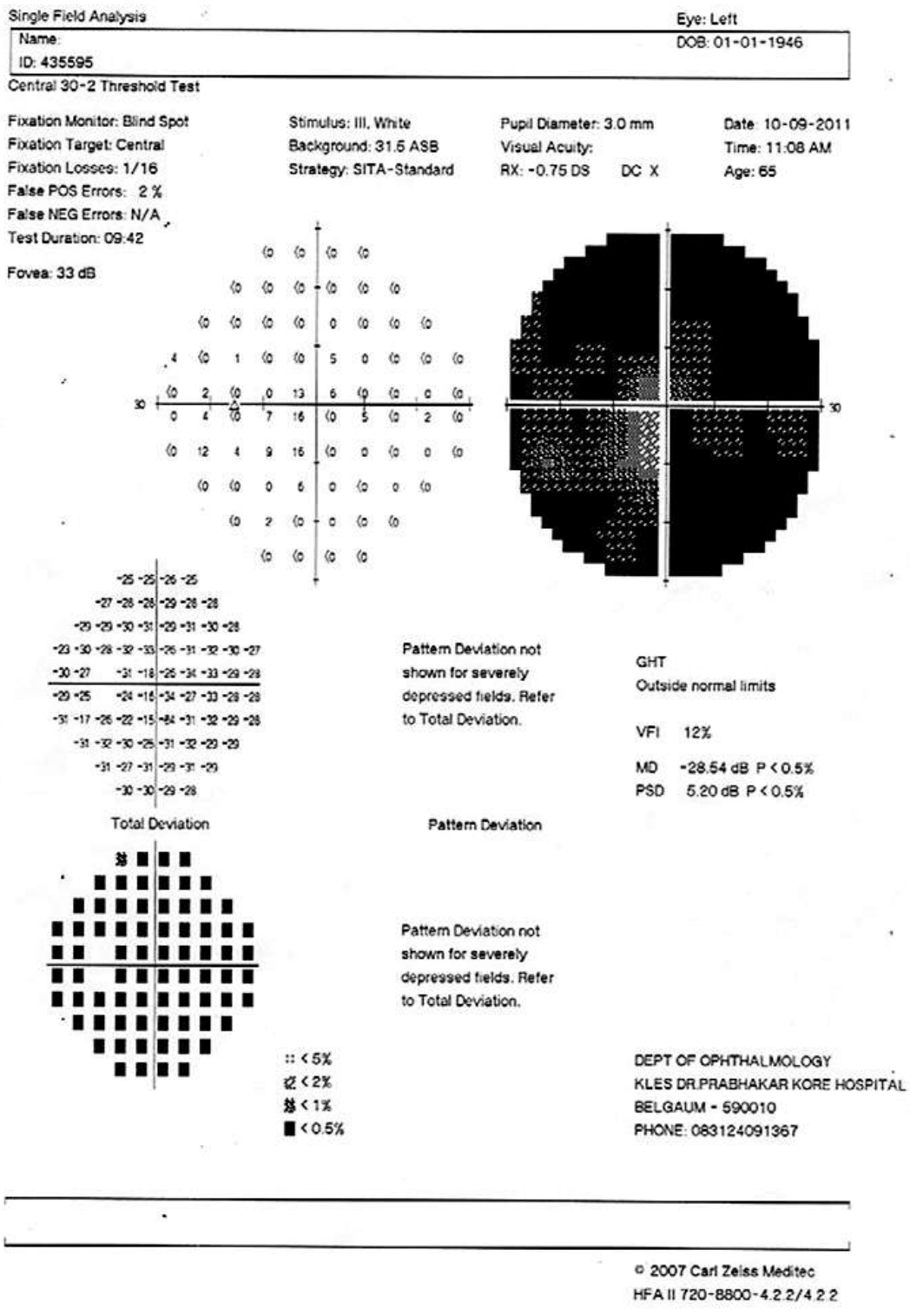
4) Single field analysis print out of 30-2 SITA standard showing severe visual field defect (inferior arcuate scotoma)



5) Single field analysis print out of 30-2 SITA standard showing severe visual field defect (superior arcuate scotoma)



6) Single field analysis print out of 30-2 SITA standard showing moderate visual field defect (double arcuate scotoma)



7) Single field analysis print out of 30-2 SITA standard showing severe visual field defect (tubular vision)

ANNEXURE – V: MASTER CHART

SI No	IP/OP No.	Name	Age	sex	DM	HTN	F/Ho	H/o treatment	H/o smoking	VA		refractive error		IOP mmHg		Gonioscopy		Optic nerve head changes RE			Optic nerve head changes LE			visual field defect		Mean deviation dB		GHT		Sensitivity in central 5 deg		HAP grading	
										RE	LE	RE	LE	RE	LE	RE	LE	C:D	PPA	NRR thinning	C:D	PPA	NRR thinning	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
1	792099	Lalit Jain	42	M	Y	Y	N	Y	Y	6/9	6/9	m	m	30	30	IV	IV	0.7	P	inf	0.7	P	sup & inf	SAS	DAS	-13.36	-27.37	ONL	ONL	III	III	SEV	SEV
2	354656	Manik	64	M	N	Y	N	N	Y	6/24	CF 5mt	m	m	25	33	IV	IV	0.5	P	inf	0.8	P	C	SAS	TV	-5.22	-25.26	ONL	ONL	II	III	MOD	SEV
3	605415	Appasab	76	M	N	N	N	N	Y	6/12	6/36	h	h	35	30	IV	IV	0.9	P	C	0.8	P	C	TV	TV	-27.26	-28.69	ONL	ONL	III	III	SEV	SEV
4	1992716	savithree	82	F	N	Y	Y	N	N	6/36	6/36	m	m	26	25	IV	IV	0.7	P	A	0.6	P	A	NS	IPS	-9.82	-5.43	ONL	ONL	I	I	MOD	mild
5	1474454	shanta	45	F	N	N	N	N	N	6/12	6/12	h	h	25	30	IV	IV	0.5	A	inf	0.7	A	inf	SAS	SAS	-5.75	-6.25	ONL	ONL	I	I	mild	mild
6	908925	sunita	61	F	N	N	N	N	N	6/12	6/12	m	m	25	25	IV	IV	0.5	A	A	0.5	A	A	NS	IPS	-5.08	-5.73	ONL	ONL	I	I	mild	mild
7	784161	machandra	68	M	Y	N	N	N	Y	6/9	6/9	h	h	22	31	IV	IV	0.4	P	A	0.6	A	inf	IPS	SAS	0.75	-11.25	WNL	B	I	I	WNL	MOD
8	1723570	mohammed	60	M	Y	Y	N	N	Y	6/9	6/18	m	m	25	25	IV	IV	0.5	P	sup & inf	0.5	P	sup & inf	DAS	DAS	-4.81	-11.35	ONL	ONL	II	II	mild	MOD
9	608724	saroja	52	F	N	N	N	Y	N	6/9	6/6	m	m	27	25	IV	IV	0.6	P	inf	0.6	P	inf	SAS	SAS	-5.93	-5.97	ONL	ONL	I	I	mild	Mild
10	586962	anand	40	M	N	N	Y	N	Y	6/6	6/6	e	e	30	30	IV	IV	0.8	A	sup	0.7	A	sup	IAS	IAS	-11.97	-10.09	ONL	ONL	I	I	MOD	MOD
11	425384	ramappa	67	M	Y	N	N	Y	Y	6/6	6/6	e	e	25	25	IV	IV	0.5	P	A	0.5	P	sup	WNL	IAS	-4.31	-4.6	ONL	ONL	I	I	mild	Mild
12	1300978	shivayogi	52	M	N	Y	Y	Y	Y	6/6	6/6	m	m	25	30	IV	IV	0.6	P	sup	0.7	P	sup	IAS	IAS	-11.74	-6.55	ONL	ONL	I	II	MOD	Mild
13	2032125	vahida	47	F	N	N	N	Y	N	6/6	6/6	m	m	30	30	IV	IV	0.7	A	sup	0.7	A	sup	IAS	IAS	-10.55	-7.34	ONL	ONL	I	I	MOD	MOD
14	2017250	srinivas	76	M	Y	Y	N	Y	Y	6/9	6/9	h	h	22	22	IV	IV	0.4	P	A	0.4	P	sup	IPS	IAS	0.63	0.65	WNL	WNL	I	I	WNL	WNL
15	785703	shankar	84	M	N	Y	N	N	Y	6/6	6/6	m	m	31	30	IV	IV	0..9	P	C	0.8	P	inf	TV	SAS	-16.75	-10.1	ONL	ONL	III	I	SEV	MOD
16	1621512	Carmalita	54	F	N	N	Y	N	N	6/9	6/6	m	m	30	30	IV	IV	0.7	P	inf	0.7	P	inf	SAS	SAS	- 7.25 -8.34	-8.34	B	ONL	I	I	MOD	MOD
17	1604911	shankar	52	M	Y	Y	N	N	Y	6/12	6/12	m	m	23	22	IV	IV	0.4	P	A	0.3	P	sup	IPS	IAS	0.76	0.65	WNL	WNL	I	I	WNL	WNL
18	1551425	somnath	58	M	Y	Y	N	N	Y	6/9	6/9	m	m	25	26	IV	IV	0.5	A	A	0.5	A	A	WNL	WNL	-3.24	-4.74	B	ONL	I	I	Mild	Mild
19	1421721	maruti	62	M	N	N	N	Y	Y	6/36	6/18	m	m	24	31	IV	IV	0.6	P	sup	0.8	P	sup	IAS	IAS	-11.11	-11.39	ONL	ONL	I	I	MOD	MOD
20	1317075	shashikala	65	F	Y	Y	N	Y	N	6/6	6/6	e	e	31	32	IV	IV	0.8	P	inf	0.8	P	sup & inf	SAS	DAS	-8.91	-14.03	ONL	ONL	I	I	MOD	SEV
21	1325772	sridevi	47	F	N	N	Y	Y	N	6/9	6/9	m	m	33	30	IV	IV	0.8	P	inf	0.8	P	inf	SAS	SAS	-10.91	-10.82	ONL	ONL	III	I	MOD	MOD
22	1584855	sudha	73	F	N	Y	N	N	N	6/9	6/18	m	m	25	25	IV	IV	0.6	A	sup	0.6	A	sup	IAS	IAS	-5.79	-4.89	ONL	ONL	I	I	Mild	Mild
23	2004713	shivaji	68	M	Y	Y	N	Y	Y	6/6	6/6	e	e	25	32	IV	IV	0.5	A	sup	0.8	A	sup	IAS	IAS	-7.7	-11.35	ONL	ONL	I	I	MOD	MOD

24	1676706	sripal	76	M	N	N	N	Y	Y	6/18	6/9	m	m	35	26	IV	IV	0.9	P	C	0.6	P	sup	TV	IAS	-25.96	-4.56	ONL	ONL	III	I	SEV	Mild
25	2005623	vivekanand	65	M	Y	N	Y	N	Y	6/12	6/12	h	h	27	31	IV	IV	0.6	A	inf	0.7	A	sup	SAS	IAS	-3.88	-7.24	ONL	ONL	I	I	Mild	MOD
26	588713	nijaguni	73	M	N	Y	N	N	N	6/6	6/6	m	m	22	30	IV	IV	0.3	A	A	0.7	A	inf	WNL	SAS	-1.43	-10.68	ONL	ONL	I	I	Mild	MOD
27	1784258	mahadev	65	M	Y	N	N	Y	Y	6/24	6/36	m	m	31	34	IV	IV	0.8	A	Sup & inf	0.9	A	sup & inf	TV	TV	-19.22	-22.73	ONL	ONL	III	III	SEV	SEV
28	435595	drupada	65	F	N	Y	N	Y	Y	6/18	6/60	m	m	30	31	IV	IV	0.8	P	C	0.7	P	C	TV	TV	-24.66	-28.54	ONL	ONL	III	III	SEV	SEV
29	1786686	prakash	61	M	Y	N	N	N	N	6/6	6/18	h	h	32	34	IV	IV	0.7	A	inf	0.9	A	C	SAS	TV	-27.39	-30.95	ONL	ONL	I	III	SEV	SEV
30	1694160	vijaykumar	62	M	N	Y	Y	N	Y	6/9	6/6	e	e	26	25	IV	IV	0.6	P	sup	0.5	P	sup & inf	IAS	DAS	-11.95	-10.4	ONL	ONL	I	III	MOD	MOD
31	1785059	ananth	51	M	N	Y	N	Y	Y	6/6	6/6	m	m	29	25	IV	IV	0.7	P	sup	0.6	P	C	NS	TV	-18.91	-11.89	ONL	ONL	I	III	SEV	MOD
32	1005732	shankar	71	M	Y	N	N	Y	Y	6/9	6/9	m	m	23	23	IV	IV	0.4	A	A	0.4	A	A	IPS	IPS	-5.75	-4.57	ONL	ONL	I	I	Mild	Mild
33	1903425	neeta	55	F	Y	N	N	Y	N	6/6	6/6	h	h	28	25	IV	IV	0.7	A	inf	0.5	A	A	SAS	IPS	-17.88	-12.88	B	ONL	I	I	SEV	MOD
34	1915797	laxmi	70	F	N	N	Y	Y	N	6/9	6/24	m	m	30	28	IV	IV	0.8	P	C	0.7	P	C	DAS	TV	-16.92	-23.02	ONL	ONL	III	II	SEV	SEV
35	373427	amina	56	F	Y	Y	N	N	N	6/9	6/9	h	h	26	27	IV	IV	0.5	A	A	0.6	A	sup	IPS	IAS	-11.11	-11.92	ONL	ONL	I	I	MOD	MOD
36	1698215	yashwant	72	M	N	N	N	Y	Y	6/6	6/6	e	e	22	30	IV	IV	0.4	P	sup	0.8	P	C	IAS	TV	-5.2	-28.4	ONL	ONL	I	III	Mild	SEV
37	438931	akkamahadevi	40	F	Y	N	Y	N	N	6/6	6/6	m	m	30	30	IV	IV	0.7	A	inf	0.7	A	inf	SAS	SAS	-12.5	-12.77	ONL	ONL	I	I	SEV	MOD
38	1720156	irappa	55	M	N	Y	N	Y	Y	6/6	6/6	m	m	30	30	IV	IV	0.7	P	sup	0.7	P	sup	IAS	WNL	-13.73	-15.5	ONL	WNL	I	I	SEV	SEV
39	1683781	kumar	40	M	Y	N	N	N	Y	6/6	6/6	h	h	29	28	IV	IV	0.7	P	sup	0.7	P	inf	IAS	SAS	-18.5	19.31	ONL	ONL	I	I	SEV	SEV
40	1851127	prakash	48	M	N	N	Y	N	Y	6/6	6/6	m	m	25	25	IV	IV	0.6	A	A	0.6	A	A	IPS	SAS	-11.77	-10.52	ONL	ONL	I	I	MOD	MOD
41	1988882	gangappa	47	M	Y	N	N	N	Y	6/60	6/60	h	h	25	26	IV	IV	0.5	P	A	0.5	P	A	IPS	IPS	-10.71	-9.56	ONL	ONL	I	I	SEV	SEV
42	101389	abraham	67	M	N	N	N	Y	Y	6/12	6/12	m	m	26	25	IV	IV	0.5	P	sup	0.5	P	sup	IAS	IAS	-11.44	-11.61	ONL	ONL	I	I	MOD	MOD
43	1892122	parvati	80	F	N	Y	N	Y	N	CF CF	6/36	h	h	30	32	IV	IV	0.9	P	C	0.9	P	C	TV	TV	-24.44	-24.28	ONL	ONL	II	III	SEV	SEV
44	1898707	anthony	51	M	Y	Y	Y	Y	Y	6/9	6/9	h	h	25	25	IV	IV	0.6	A	inf	0.6	A	inf	SAS	SAS	-10.42	-9.42	ONL	ONL	I	I	MOD	MOD
45	443412	somanna	52	M	N	Y	Y	N	Y	6/6	6/6	m	m	24	30	IV	IV	0.5	P	sup	0.7	P	inf	IAS	SAS	-10.94	-12.22	ONL	B	I	I	MOD	MOD
46	1852733	gunavanthi	60	F	N	Y	N	Y	N	6/12	6/12	h	h	28	28	IV	IV	0.7	A	A	0.7	A	A	IPS	IPS	-18.93	-16.13	ONL	B	I	I	SEV	SEV
47	1817109	jaswant	43	M	N	N	Y	N	Y	6/9	6/9	h	h	25	25	IV	IV	0.5	A	inf	0.5	A	inf	SAS	SAS	-11.92	-8.52	B	ONL	I	I	MOD	MOD
48	593596	basavaraj	62	M	Y	N	N	Y	Y	6/9	6/9	h	h	31	32	IV	IV	0.8	A	Sup & inf	0.8	A	inf	DAS	SAS	-17.23	-15.06	ONL	ONL	I	I	SEV	SEV

ABBREVIATIONS AND KEY FOR MASTER CHART

A	-	Absent
B	-	Borderline
C	-	Concentric
C : D	-	Cup : Disc ratio
DAS	-	double arcuate scotoma
Deg	-	degrees
DM	-	Diabetes Mellitus
e	-	emmetropia
F/ HO	-	Family history of
GHT	-	Glaucoma Hemifield Test
HAP	-	Hodapp Anderson Parrish
h	-	hypermetropia
HTN	-	Hypertension
Inf	-	Inferior
IAS	-	inferior arcuate scotoma
IPS	-	isolated paracentral scotoma
IOP	-	Intraocular Pressure
LE	-	Left Eye
M	-	Male

m - myopia

MD - Mean Deviation

MOD - Moderate

NS - nasal step

NRR - neuroretinal rim

N - No

ONL - Outside normal limits

P - Present

PPA - Peripapillary Atrophy

RE - Right Eye

SAS - superior arcuate scotoma

SEV - Severe

Sup - Superior

Sup & inf - superior and inferior

I - No point within the central 5^0 with sensitivity less than 15dB

II - No point within the central 5^0 with sensitivity less than 0dB

III - Any point within the central 5^0 with sensitivity less than 0dB

IV - Open angles

WNL - Within normal limits

Y - Yes