“THE ASSESSMENT OF MACULAR THICKNESS VARIABILITY IN PRIMARY OPEN ANGLE GLAUCOMA USING OPTICAL COHERENCE TOMOGRAPHY”

By

DR.RAVEENA J,

MBBS

Dissertation submitted to the

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

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

Under the guidance of

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2025

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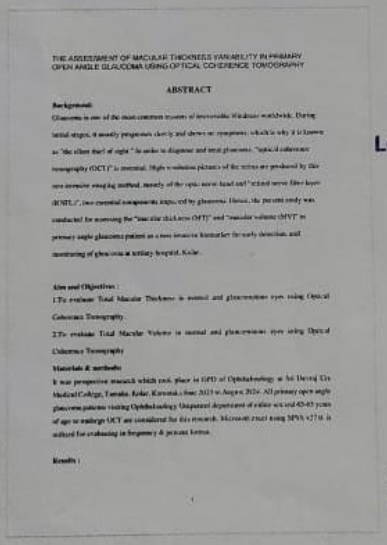

THE ASSESSMENT OF MACULAR THICKNESS VARIABILITY IN PRIMARY OPEN ANGLE GLAUCOMA USING OPTICAL COHERENCE TOMOGRAPHY

ABSTRACT

Background:

Glaucoma is one of the most common causes of irreversible blindness worldwide. During initial stages, it usually progresses slowly and shows no symptoms, which is why it is known as "the silent thief of sight." In order to diagnose and treat glaucoma, "optical coherence tomography (OCT)" is essential. High-resolution pictures of the retina are produced by this non-invasive imaging method, namely of the optic nerve head and "retinal nerve fiber layer (RNFL)", two essential components impacted by glaucoma. Hence, the present study was conducted for assessing the "macular thickness (MT)" and "macular volume (MV)" in primary angle glaucoma patient as a non-invasive biomarker for early detection and monitoring of glaucoma at tertiary hospital, Kolar.

Aim and Objectives :

1.To evaluate Total Macular Thickness in normal and glaucomatous eyes using Optical Coherence Tomography.

2.To evaluate Total Macular Volume in normal and glaucomatous eyes using Optical Coherence Tomography

Materials & methods:

It was prospective research which took place in OPD of Ophthalmology at Sri Devraj Urs Medical College, Tamaka, Kolar, Karnataka from 2023 to August 2024. All primary open angle glaucoma patients visiting Ophthalmology Outpatient department of either sex and 45-65 years of age to undergo OCT are considered for this research. Microsoft excel using SPSS v27.0 is utilized for evaluating in frequency & percent format.

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Last, but not the least, I would like to express my gratitude to the Almighty for all his blessings.

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LIST OF ABBREVIATIONS

SL NO.	ABBREVIATION	FULL FORM
1.	OCT	Optical Coherence Tomography
2.	RNFL	Retinal nerve fibre layer
3.	NFL	Nerve fibre layer
4.	IOP	Intraocular pressure
5.	POAG	Primary open angle glaucoma
6.	VF	Visual field
7.	RGC	Retinal ganglion cell
8.	ONH	Optic nerve head
9.	FDT	Frequency doubling technology
10.	SWAP	Short wave automated perimetry
11.	SITA	Swedish interactive thresholding algorithm
12.	ALT	Argon laser trabeculoplasty
13.	SLT	Selective laser trabeculoplasty
14.	MRI	Magnetic resonance imaging
15.	HFA	Humphrey field analysis
16.	IPL	Inner plexiform layer
17.	GCIPL	Ganglion cell inner plexiform layer
18.	HVF	Humphrey visual field
19.	MT	Macular thickness
20.	OMT	Outer macular thickness
21.	IMT	Inner macular thickness
22.	CMT	Central macular thickness
23.	VD	Vascular perfusion density
24.	GS	Glaucoma suspect

ABSTRACT

PURPOSE

Glaucoma is a group of eye diseases that damage the optic nerve, often due to elevated intraocular pressure, and is a leading cause of irreversible blindness worldwide. It typically develops slowly and without noticeable symptoms in its early stages, earning it the nickname "the silent thief of sight." Optical Coherence Tomography (OCT) plays a vital role in the diagnosis and management of glaucoma. This non-invasive imaging technique provides high-resolution cross-sectional images of the retina, particularly the retinal nerve fiber layer (RNFL) and the optic nerve head, which are key structures affected by glaucoma. Hence, the present study was conducted for assessing the macular thickness in primary angle glaucoma patient as a non-invasive biomarker for early detection, diagnosis, and monitoring of glaucoma at tertiary hospital, Kolar.

AIMS AND OBJECTIVES

1. To evaluate the Total Macular Thickness in normal and glaucomatous eyes using Optical Coherence Tomography.
2. To evaluate the Total Macular Volume in normal and glaucomatous eyes using Optical Coherence Tomography

MATERIAL AND METHODS

It was a prospective research which took place in OPD of Ophthalmology at Sri Devraj Urs Medical College, Tamaka, Kolar, Karnataka June 2023 to August 2024. All primary open angle glaucoma patients visiting Ophthalmology Outpatient department of either sex and 48-65 years

of age to undergo OCT are considered for this research. After obtaining consent, demographic details were noted & then subjected for detailed ophthalmic examination of both eyes including Visual acuity, Slit lamp bio microscopy, IOP by Goldman Applanation Tonometry, Fundus examination, macular thickness and Total Macular Volume by OCT.

RESULTS

The mean age was 57.1 years in glaucoma patients and 58.0 years in controls, and both groups had an identical gender distribution. Macular thickness in all quadrants—superior, inferior, nasal, and temporal was significantly reduced in glaucomatous eyes compared to normal controls, with the most marked thinning observed in the nasal quadrant (244.90 µm in glaucoma vs. 269.35 µm in normals). In the outer macular ring, our study found that glaucomatous eyes exhibited significantly reduced thickness across all quadrants compared to normal eyes. The most pronounced reductions were again noted in the superior and nasal quadrants, with mean values of 229.65 µm versus 248.75 µm (superior), and 223.65 µm versus 249.10 µm (nasal) in glaucoma and control groups, respectively. total macular thickness and volume were significantly reduced in glaucoma patients compared to normal controls, with mean macular thickness measuring 237.4 µm in glaucomatous eyes versus 245.0 µm in normal eyes. The mean intraocular pressure (IOP) in our study was significantly higher in glaucoma patients (20.4 mmHg) compared to normal subjects (12.5 mmHg).

CONCLUSION

This study identified a significant reduction in macular thickness and volume in glaucomatous eyes compared to normal controls, with the most pronounced thinning in the nasal and superior quadrants across both inner and outer macular regions. A strong negative correlation between intraocular pressure and macular parameters was observed, suggesting a role for pressure-

induced and vascular mechanisms in glaucomatous damage. These findings support the utility of OCT-derived macular metrics as sensitive, non-invasive biomarkers for early glaucoma detection and monitoring

Key words: Macular volume, Macular thickness, POAG, OCT

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INTRODUCTION

THE ASSESSMENT OF MACULAR THICKNESS VARIABILITY IN PRIMARY

OPEN ANGLE GLAUCOMA USING OPTICAL COHERENCE TOMOGRAPHY

Glaucoma is a chronic, degenerative visual neuropathy. The distinctive appearance of the optic nerve sets glaucoma apart from the majority of other types of acquired optic neuropathy. The optic-nerve cup enlarges in glaucoma as the neuroretinal rim of the optic nerve gradually thins. Optic-nerve cupping is the term used to describe this condition. It is brought on by the loss of retinal ganglion cell axons, as well as the vasculature and supporting glia. The typical pink hue of the residual neuroretinal rim is still there. Cupping does not form and the optic-nerve tissue loses its pink hue in other ocular neuropathies. Arteritic anterior ischemic optic neuropathy is an uncommon exception, where cupping may take place.[1]

If left untreated, glaucoma patients may lose total eyesight following the loss of peripheral vision. Glaucoma is categorized based on anterior-segment abnormalities that potentially raise intraocular pressure, even though the condition often manifests without an increase in intraocular pressure. The cornea and crystalline lens, which are both devoid of a blood supply, are fed by the circulatory system of the anterior region of the eye. Aqueous humor, which is produced by the ciliary body, circulates throughout the anterior chamber and drains via the trabecular meshwork in the iridocorneal angle, which is the angle created between the iris and the cornea.[2] Reduced aqueous outflow, increased aqueous humor production, is the cause of elevated intraocular pressure.[3]

The iridocorneal angle's appearance is used to categorize glaucomas. Primary and secondary kinds are further subdivided into open-angle, closed-angle, and developmental types. Normal-tension glaucoma, may coexist with “primary open-angle glaucoma (POAG)”. Both adult-

onset (occurring after the age of forty) and juvenile-onset (occurring between the ages of three and forty) forms of primary open-angle glaucoma are included.[3]

A certain pattern of anatomical and functional abnormalities (visual field (VF) exams shows loss of vision) is used to diagnose glaucoma, a progressive optic neuropathy. Early glaucomatous damage to the macula has long been known to occur.[4–6] However, until recently, early macular degeneration was often disregarded. For instance, there are six test points in the most used VF test for glaucoma. These test spots are outside of the densest zone of RGCs, as we will see. In any event, the ability to assess the thickness of the RGC layer in the human macula in vivo using "optical coherence tomography (OCT)" has rekindled interest in macular injury.[7]

To help diagnose glaucoma, imaging techniques have evaluated the thickness of the "retinal nerve fiber layer (RNFL)" (scanning laser polarimetry and OCT) and examined the shape of the optic nerve head (scanning laser ophthalmoscopy). Ganglion cells are grouped in four to six layers in the macular region, and they account for thirty to thirty-five percent of the thickness of the retinal macular. A large reduction in the thickness of the retinal or retinal nerve fiber layer is brought about as a consequence of the loss of macular ganglion cells. Due to the high cell density of the macula, which comprises more than fifty percent of all retinal ganglion cells, it is an excellent region for identifying early cell loss and changes over the course of time.[8] According to a number of studies, the loss of RGCs causes a reduction in macular thickness and volume in glaucomatous eyes; this result is correlated with RNFL thickness and visual field abnormalities.[9] In the present work, we have investigated the role of macular thickness and macular volume in glaucoma.

AIMS AND

OBJECTIVES

1. To evaluate Total Macular thickness in normal and glaucomatous eyes using Optical Coherence Tomography .

2. To evaluate Total Macular Volume in normal and glaucomatous eyes using Optical Coherence Tomography

REVIEW OF LITERATURE

ANATOMY OF EYE

The human eye is a spherical that is filled with fluid and is surrounded by three layers of tissue. Sclera is a strong white fibrous tissue that makes up the majority of the outermost layer of the structure. On the other hand, this opaque outer layer gets changed into the cornea in the front of the eye. The cornea is a specialized transparent tissue that allows light rays to enter the eye. The iris, the ciliary body, and the choroid are the three unique structures that are found in the intermediate layer of tissue. These structures are connected with one another. The colorful part of the eye that is seen through the cornea is referred to as the iris since it is visible. Under the control of the nervous system, it is possible to modify the size of the pupil, which is the opening in the middle of the eye, owing to the presence of two sets of muscles that perform opposing activities. The lens is surrounded by a ring of tissue known as the ciliary body. This ring of tissue has a muscle component that plays a significant role in regulating the lens's refractive power, as well as a vascular component known as the ciliary processes, which are responsible for producing the fluid that fills the front of the eye. The choroid is made up of a dense capillary bed, which is the primary source of blood supply for the photoreceptors that are located in the retina.[10]

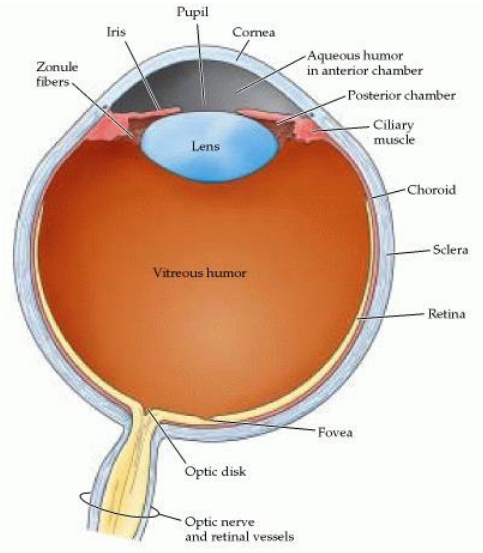


Figure 1: Anatomy of the human eye[10]

The cavity of the eyeball, which is placed behind the pupil and the iris, is where the lens is found. The zonule fibers are responsible for its suspension from the ciliary body muscles. Accommodation is provided by the ciliary body muscles while near vision is being performed. The anterior cavity and the vitreous cavity are the two cavities that are created when the lens acts as a partition between the interior of the eyeball. The region that is located anterior to the lens is known as the anterior cavity, and it is subdivided into two chambers: the anterior chamber and the posterior chamber. The space between the cornea and the iris that is known as the anterior chamber. It is placed behind the iris and in front of the zonular fibers and lens. Both of the chambers that make up the anterior cavity are filled with aqueous humor, which is a fluid that is watery and provides nourishment to the cornea and lens. Every ninety minutes, the epithelium of the ciliary body is responsible for the production of the aqueous humor, which is then refilled. This fluid that is found within the eye will eventually make its way to Schlemm's canal, which is responsible for draining the fluid into the circulation of the body. The vitreous cavity, which is situated between the lens and the retina, is the region of the eyeball that is the most extensive cavity.[11] The vitreous body is a jelly-like material that is translucent and is located inside the vitreous chamber. It is responsible for compressing the retina against the choroid. The vitreous body, in contrast to the aqueous humor, does not experience fast replenishment via circulation. Phagocytic cells are present in it, and they are responsible for removing debris in order to keep the vision clear. [12]

ANATOMY OF RETINA

It is the retina that makes up the third and most innermost layer of the eyeball. There is a neuronal layer and a pigmented layer that make up this structure. Between the choroid and the neural portion of the retina is where you'll find the pigmented layer, which is a sheet of epithelial cells that contain melanin. The neural layer, also known as the sensory layer, is an extension of the brain that is responsible for the intensive processing of visual input prior to the transmission of electrical impulses across the axons of the optic nerve. The ganglion cell layer, the bipolar cell layer, and the photoreceptor layer are the three separate layers that make up the neurons that make up the retina respectively. Before reaching the photoreceptor layer, light must first travel through the ganglion cell layer as well as the bipolar cell layer simultaneously. Photoreceptors are specialized cells that are responsible for the conversion of nerve impulses into light rays. Rod cells and cone cells are the two cell types that make up photoreceptors. More than 100 million rods and around 6 million cones are found in each retina. Color vision is produced by cones, whereas rods are responsible for our ability to see in low light. When diverse combinations of blue, green, and red cones are stimulated, color vision is produced as a consequence. Eventually, the information that is seen reaches the optic disk, which is sometimes referred to as the blind spot due to the fact that it does not contain any rod or cone cells. In the precise center of the posterior region of the retina is a flat area known as the macula lutea. This place is very important for vision. Cones are the only cells that can be found in the fovea centralis, which is situated in the geographical center of the macula lutea. Specifically, it is the region that has the finest visual acuity or resolution, with the maximum density of cone photoreceptors and the absence of rod receptors. In order to concentrate on anything that we are looking at, this is the area of the eye that we employ. [12]

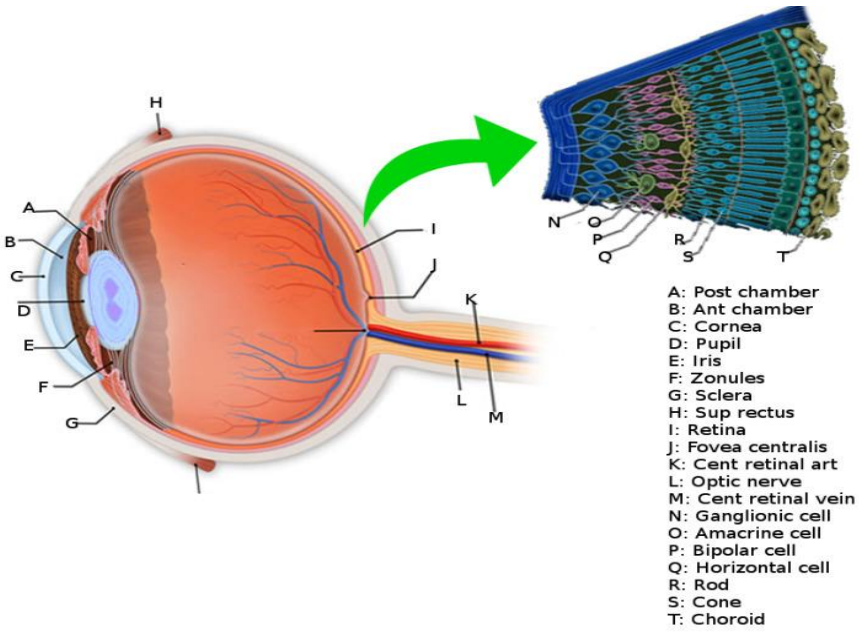


Figure 2:The Tunics of the Eye

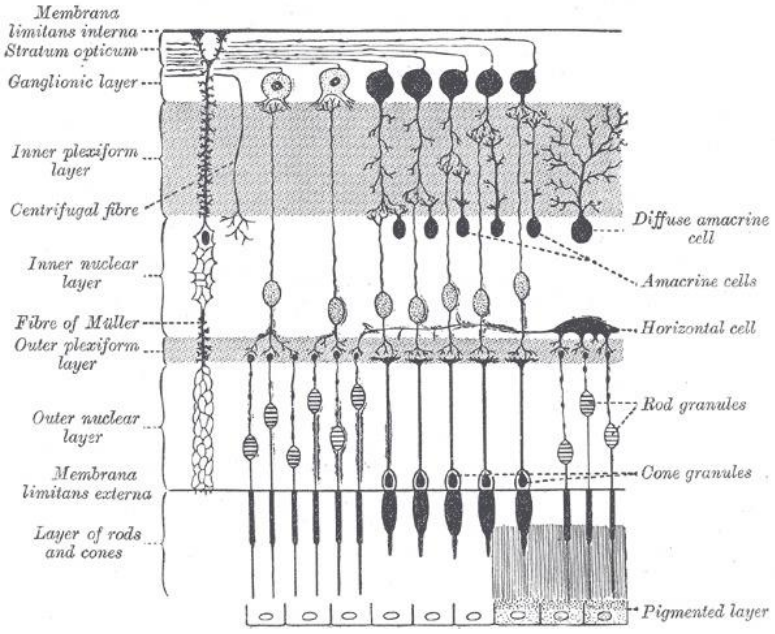


Figure 3: Anatomy of retina[13]

Anatomy of Macula

The macula, generally known as the macula lutea due to its yellowish pigmentation, is the highly delicate region of the retina and provides the maximum level of visual clarity. In a fundoscopic investigation, it is discovered temporally from the optic disc. The carotenoids which constitute the macular pigments as well as impart them their yellowish shade are lutein as well as zeaxanthin. Such pigments are well known for their ability to screen blue light as well as possess anti-inflammatory effects. Lutein as well as zeaxanthin supplementation have been demonstrated to improve pigment concentration as well as is linked to a lower incidence of diabetic retinopathy in grownups and preterm birth retinopathy in newborns. The fovea, an avascular dip in the macula's center that houses a significant number of cones, is located there.

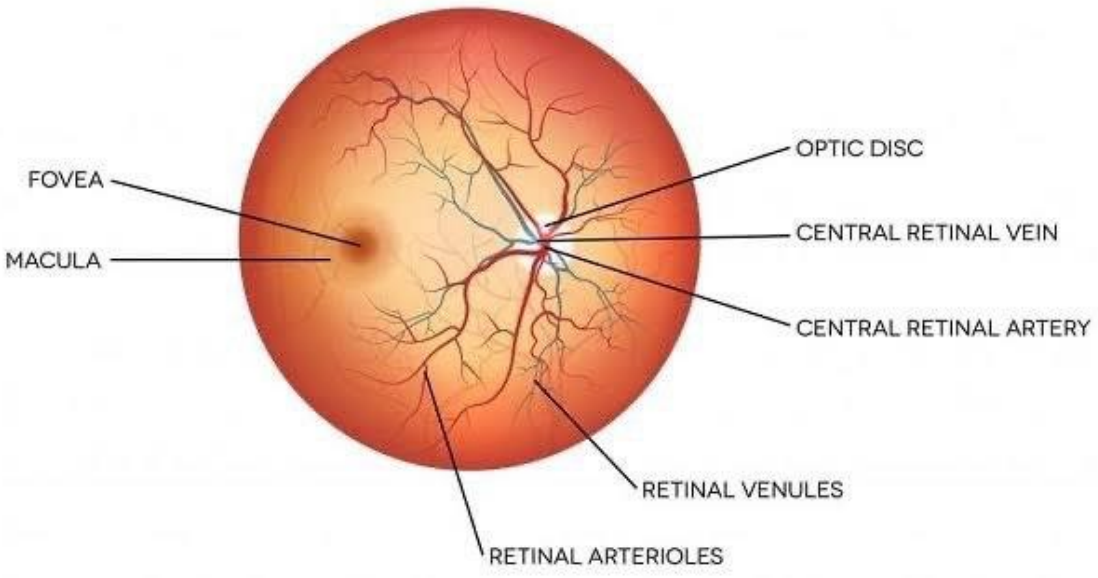


Figure 4: Normal Fundus picture

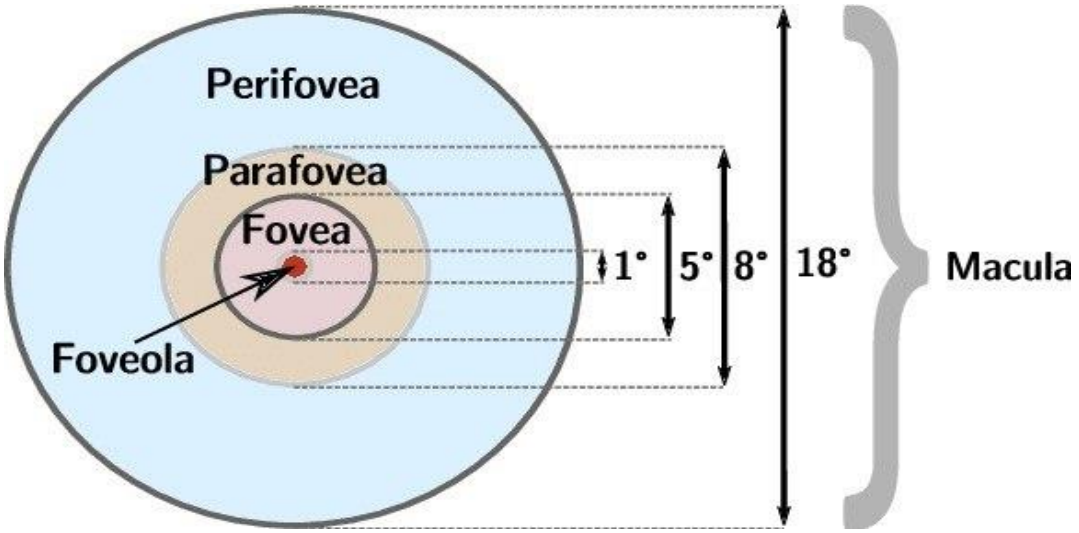


Figure 5: Anatomy of Macula

The Foveola:

Even though it covers a little portion of the visual field i.e., 1°, the foveola, that has dimensions of 0.35 millimeters in diameter as well as 0.13 millimeters in width, is the region of the retina with the maximum degree of visual clarity. This is explained by the fact that only cone photoreceptors are present, as well as by the fact that it is not vascularized. Because the choriocapillaris, that illuminates via the foveola, has strong choroidal circulation, it generally seems deeper red as compared to the surrounding retina.

BLOOD SUPPLY AND LYMPHATICS

The retina is vascularized by blood vessels as well as the choroid. The choroid provides the retina's external layers, and its internal layers are supplied by branches of large blood vessels. Following are the details of each vessel which contributes to retina’s vasculature.

Central Retinal Artery – Principal blood artery which feeds the retina's internal layers; it passes through optic nerve sheath. The superior as well as inferior arcades of central retinal

artery will eventually create the blood-retina barrier. A significant branch of the ocular artery is where it starts.

Central Retinal Vein – the primary drainage system for the retina, which passes inside the optic nerve's sheath together with the major retinal artery.

Long Posterior Ciliary Arteries – Near the optic nerve entrance region, these 2 arteries emerge from the ophthalmic artery and then penetrate the sclera.

Short Posterior Ciliary Arteries – These vasculatures arise from ophthalmic artery as a couple branches, then divide into ten to twenty tiny blood vessels which pierce the posterior sclera as well as surround optic nerve. Optic cup is nourished by the circle of Zinn. The Bruch membrane as well as external retina are additionally supplied by posterior ciliary arteries.

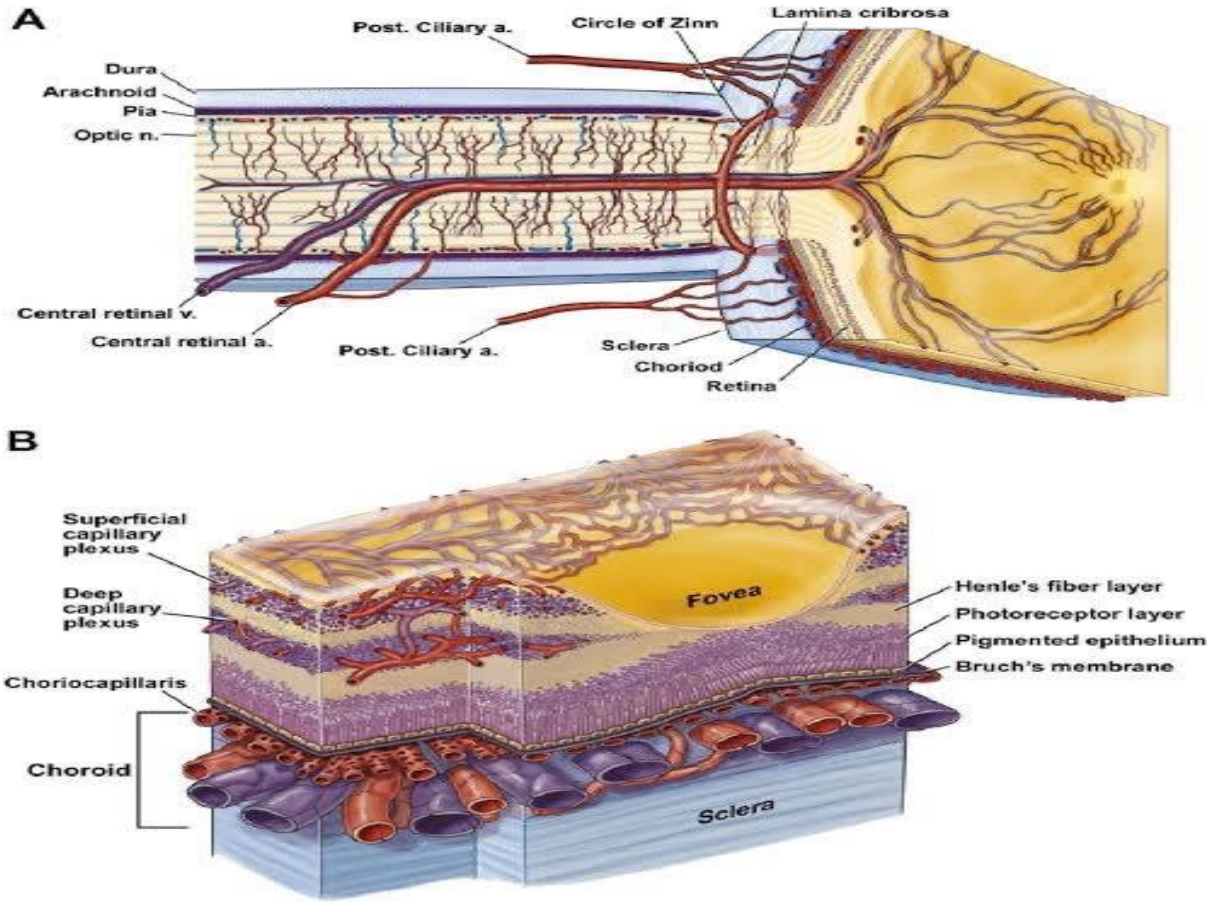


Figure 6: Vasculature of Retina

Choroid –2nd principal layer, also known as the tunic of eye. Choriocapillaris is thickest behind the fovea as well as gets thinner as it moves outward. No lymphatic vessels in the retina are observed.[21]

GLAUCOMA

The gradual progressive degradation of retinal ganglion cells and their axons is a frequent feature of glaucoma, a group of progressive optic neuropathies that cause a distinctive look of the optic disc and a pattern of vision loss. Both the basic underpinnings of the illness and the mechanisms influencing its course are still poorly understood. However, the only risk factor that has been shown to be treated is intraocular pressure. Glaucoma may develop into vision impairment and eventually blindness if treatment is not received. Primary open-angle glaucoma is an age-related and sneaky kind of the condition.[17]

EPIDEMIOLOGY

The age-standardized prevalence rate of glaucoma fell from 111.92 [95% UI: 94.76 to 130.28 per 100,000] in 1990 to 94.68 (95% UI: 80.42 to 110.87 per 100,000) in 2019, while the number of prevalent glaucoma cases worldwide increased from 3,881,624 [95% UI: 3,301,963 to 4,535,045] in 1990 to 7,473,400 (95% UI: 6,347,183 to 8,769,520) in 2019. From 1990 to 2019, the DALY number for glaucoma rose from 442,182 (95% UI: 301,827 to 626,486) to 748,308 (95% UI: 515,636 to 1,044,667). Age-standardized DALY rates and the sociodemographic index (SDI) were substantially inversely correlated.[18]

The largest regional burden of worldwide blindness (23.5%) due to glaucoma is attributed to India.[19] After cataract and refractive error, glaucoma is the third most common cause of blindness in India.[20] In India, 8.9 million people are blind and 11.9 million suffer from glaucoma.[19] In India, glaucoma accounts for 12.8% of blindness. Because there are fewer community-based studies and a relatively poor awareness and detection rate (more than 90% of glaucoma goes undiagnosed), it is challenging to assess the true burden of the illness and

the many subtypes of glaucoma in the Indian population.[21] The pattern of glaucoma differs in various parts of India because of the country's great geographic and ethnic diversity.[22]

Classification/Types[23]

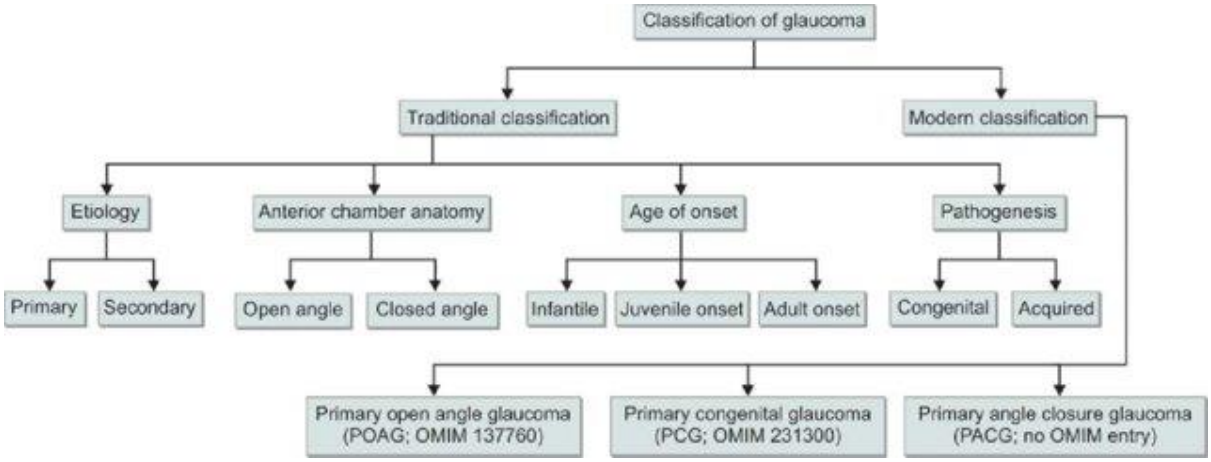


Figure 7: Classification Of Glaucoma

Glaucoma comes in a variety of forms (Figure). Optic disc excavation, sometimes referred to as cupping, is a distinctive deformation and remodelling of the ONH as a reaction to biomechanical stress and strain associated with "intraocular pressure (IOP)", which may happen at any IOP level. It is the distinguishing hallmark of this group of diseases. As per the anatomical structure of the aqueous humour outflow channel, the two primary kinds of glaucoma are open-angle glaucoma and angle-closure glaucoma.[24]

Angle-closure glaucoma is quite common in people of Chinese and other Asian races, whereas open-angle glaucoma makes up the bulk of glaucoma cases in people of European and African ancestry.[25]

Primary open-angle glaucoma

POAG is the most common kind of open-angle glaucoma. The majority of POAG patients will not go blind, although they often have significant impairments in their "quality of life (QOL)"

and visual function. Around 6 million incidents of blindness are predicted to result from POAG globally in 2020.[24]

Prevalence and Burden in India and the World

In 2015, it was predicted that between 35 and 58 million individuals globally have POAG. As the world's population ages, it is anticipated that the prevalence of POAG would rise to between 53 and 65.5 million afflicted people by 2020.[26] There are significant differences in POAG prevalence amongst populations. Ethnicity also affects prevalence.[26,27] POAG is most common among Black people (>5% of people 60 years of age), followed by Hispanic or Latino people (2.7%), Asian people (about 2%), and white people (roughly 1.5%)5. Furthermore, depending on the individual's place of birth, the prevalence might differ among each ethnic group. The greatest incidence of POAG is seen in elderly Hispanic or Latino people (18%), then among black people (15%), white people (7%), and Asian people (5%). These persons are all 85 years of age or older, and the frequency is considerably greater in those over 90 years of age. POAG is more common among Black people than in those of other races.[24]

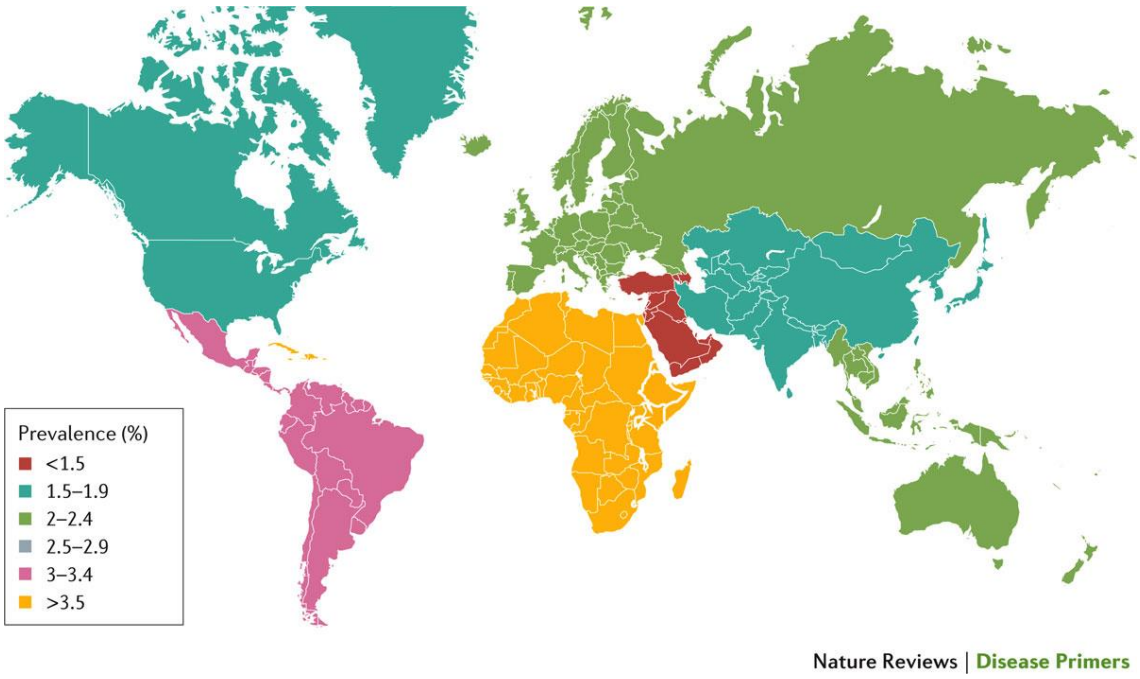


Figure 8: Primary open-angle glaucoma Prevalent- worldwide.[26]

Causes and risk factors

Glaucoma is a serious public health issue because it causes irreparable injury to the optic nerve and retinal ganglion cells, as well as a progressive loss of peripheral vision. Anatomical, genetic, vascular, and immunological variables are all part of its complex etiology. In all forms of glaucoma, age is a major risk factor for the gradual loss of retinal ganglion cells. Medical diseases including diabetes, high blood pressure, and heart disease, as well as a family history of the illness in a direct related (mother, father, brother, sister, or children), are additional risk factors for developing glaucoma.[28]

Key Risk Elements for Primary Open-Angle Glaucoma[3]

Risk Factor	Prevalence of Glaucoma	Relative Risk of Glaucoma*
	%	
Race		
Black	4.2	
White†	2.1	
Asian	1.4	
Older age (odds ratio per decade increase)		
Black		1.6
White†		2.1
Asian		1.6
Elevated intraocular pressure		
<15 mm Hg		1.0
16–18 mm Hg		2.0
19–21 mm Hg		2.8
22–29 mm Hg		12.8
30–34 mm Hg		39.0
Diastolic perfusion pressure (adjusted odds ratio)‡		
≥50 mm Hg		1.0
40–49 mm Hg		1.7
30–39 mm Hg		2.1
<30 mm Hg		6.2
Family history in first-degree relative (adjusted odds ratio)		2.9
Myopia (adjusted odds ratio)		1.6–3.3
Thin central cornea (hazard ratio per 40-μm decrease)		1.7

Table 1: Risk Factor And Prevalence Of Glaucoma

The juvenile or early adult type of POAG, which is often featured by very high “intraocular pressure (IOP)”, is typically where disease-associated mutations in myocilin arise. The prevalence of myocilin mutations in adult populations with primary open-angle glaucoma varies between 3% and 5%. In around 90% of cases, individuals with these mutations exhibit the glaucoma phenotype. Myocilin-related glaucoma's exact mechanism is yet unknown. It seems that mutations change the myocilin protein in a manner that interferes with the IOP's regular control. Forms of myocilin linked to disease disrupt protein transport, which causes misfolded proteins to accumulate intracellularly. It is believed that the intraocular pressure rises as a result of insufficient protein secretion.[3,29]

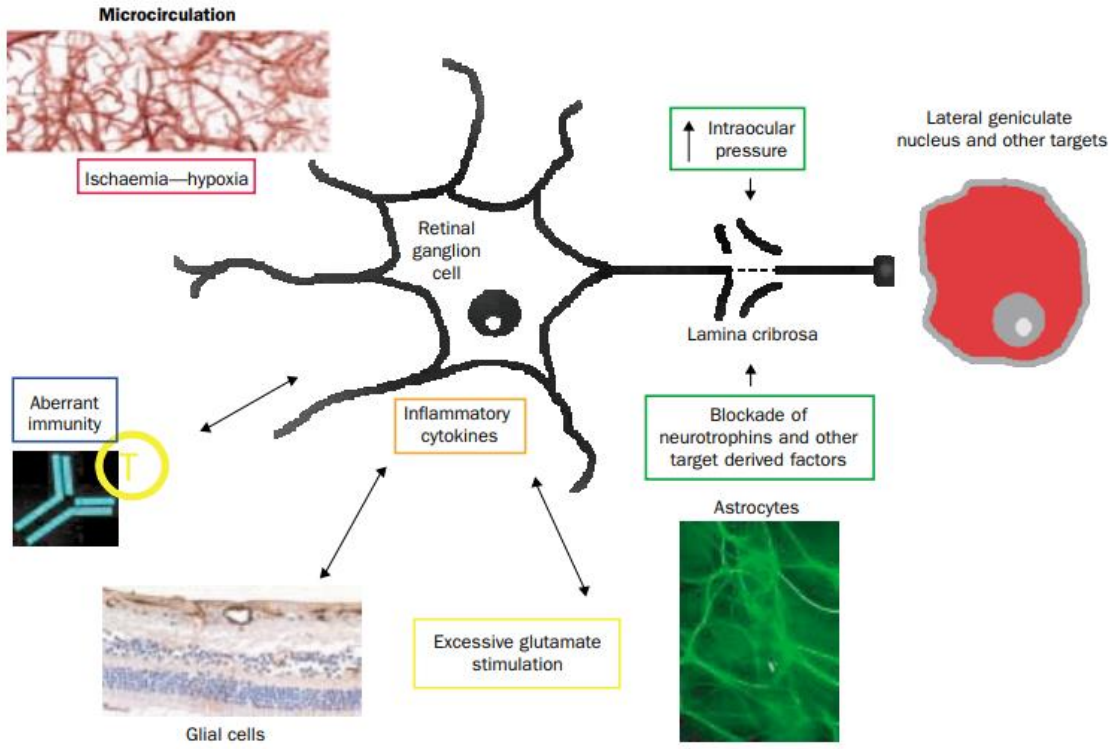


Figure 9: Pathogenesis of glaucomatous neurodegeneration: Influencing factors[17]

Pathophysiology

Despite the fact that the precise cause of glaucoma is unknown, there is a correlation between the loss of retinal ganglion cells and IOP. IOP is estimated by the equilibrium between the

ciliary body's production of aqueous humor and its drainage through two distinct channels, the trabecular meshwork and the uveoscleral outflow pathway. In individuals who have open-angle glaucoma, achieving aqueous outflow through the trabecular meshwork is more challenging. In contrast, people with angle-closure glaucoma usually have blocked access to the drainage routes.[29]

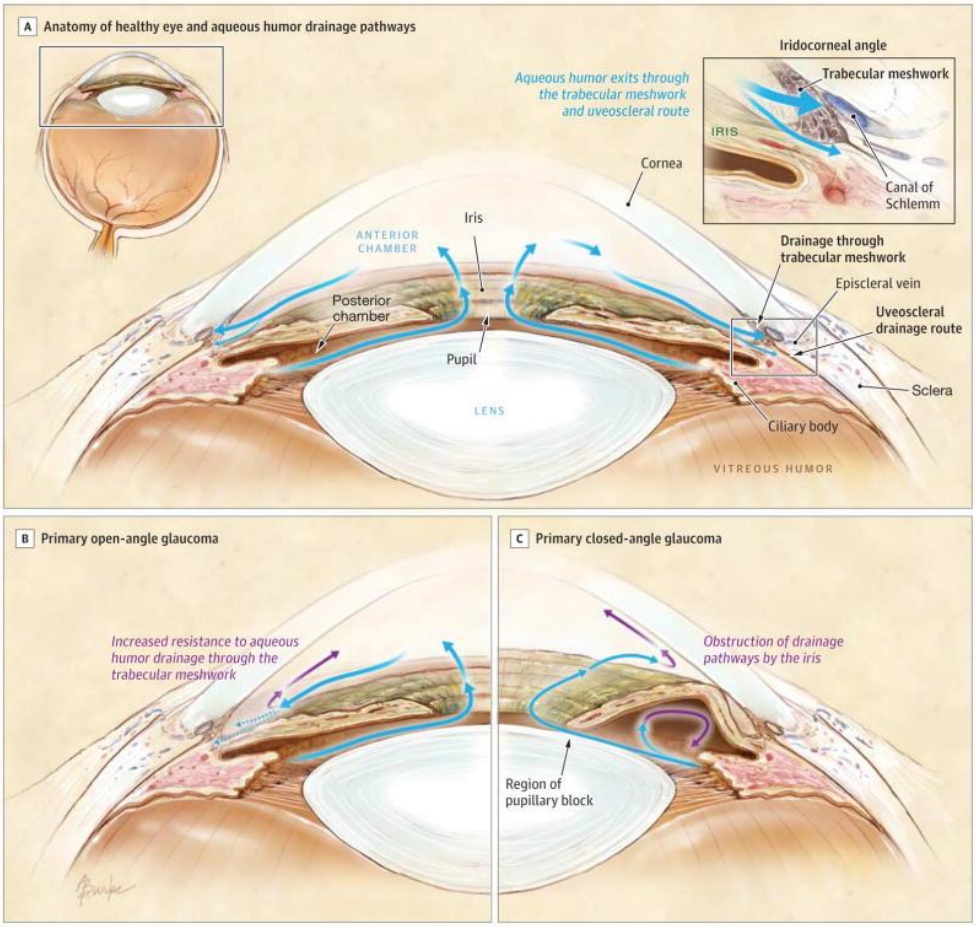


Figure 10:Healthy and Glaucomatous Eyes' Aqueous Humor Drainage Pathways[29]

The posterior components of the eye, in particular the lamina cribrosa and the tissues that surround it, are susceptible to experiencing mechanical stress and strain as a consequence of the pressure that is present inside the eye. The lamina where the optic nerve fibers (retinal ganglion cell axons) leave the eye is where the sclera is punctured.[30] The weakest part of the pressurized eye's wall is the lamina. Within the eye, the lamina cribrosa may undergo

compression, deformation, and remodelling as a result of the stress and strain that is caused by intraocular pressure. It is possible for this to result in mechanical axonal injury as well as interruption of axonal transport, which inhibits the brainstem target (relay neurons of the lateral geniculate nucleus) from retrogradely supplying critical trophic nutrients to retinal ganglion cells. During the course of research conducted on cats and monkeys with experimentally induced ocular hypertension, it was shown that both orthograde and retrograde axonal transport at the level of the lamina cribrosa were inhibited.[31,32] Early in the pathophysiology of glaucoma in experimental systems, disrupted axonal transport leads to vesicle collections and microtubule and neurofilament disarray in the prelaminar and postlaminar areas.[33] Similar ultrastructural alterations in optic nerve fibers are seen in glaucoma-affected postmortem human eyes. In times of intraocular pressure-induced metabolic stress, it may be challenging to fulfill high energy demands since retinal ganglion cells and astrocytes may also exhibit mitochondrial malfunction.[30,34]

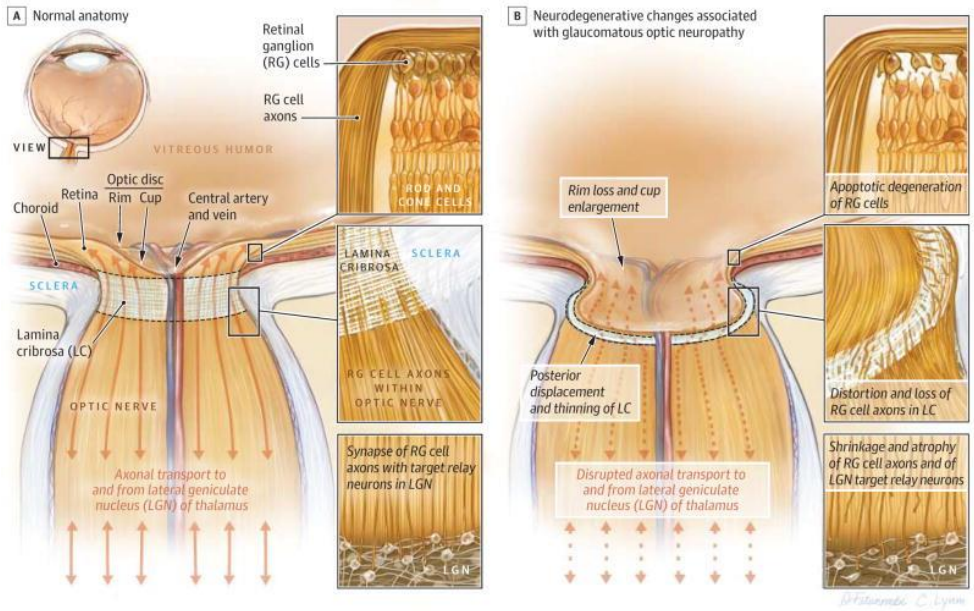


Figure 11: An illustration of the neurodegenerative alterations and normal anatomy linked to glaucomatous optic neuropathy.[29]

Clinical features

Despite the fact that glaucoma is consistently associated with higher intraocular pressure, a number of population-based investigations revealed that 25% to 50% of glaucoma patients had IOP less than 22 mm Hg. Even after years of follow-up, a significant portion of individuals with increased intraocular pressure never acquire glaucoma, despite the close correlation between the two conditions.[17] Until the illness is severe and there is significant brain damage, glaucoma advances without exhibiting any symptoms. When symptoms do appear, the condition causes vision loss, which lowers quality of life and makes it harder to carry out regular tasks like driving. To reduce the disease's course, early intervention is crucial. Patients who are at risk of developing glaucoma should be referred to an eye care professional.[29]

Characteristic alterations in the appearance of the optic nerve head and retinal nerve fiber layer arise with retinal ganglion cell death and optic nerve fiber loss in glaucoma. When an ophthalmoscopic examination of the optic nerve head is performed, these abnormalities, which constitute the most important component of a glaucoma diagnosis, may be identified. It is hard to overestimate the relevance of doing an appropriate ophthalmologic examination of the eye in order to identify glaucoma at an early stage. Visual fields gradually deteriorate due to retinal ganglion cell loss; this generally starts in the midperiphery and might proceed centripetally until only a central or peripheral island of vision is left.[29]

Diagnosis

A fundoscopic examination, gonioscopy, tonometry, optical coherence tomography (OCT), and visual field testing are all part of the evaluation process. Given that IOP is the biggest risk factor among them, tonometry is crucial.[35] Although there are a number of different kinds of tonometers, Goldmann applanation tonometry is the gold standard for individuals with glaucoma, increased IOP, and risk factors.[36,37] When Goldmann applanation tonometry is not

feasible, such as for bedridden patients, noncollaborative people, children, or those allergic to anesthetic drops, other tonometers may be taken into consideration.[38,39]

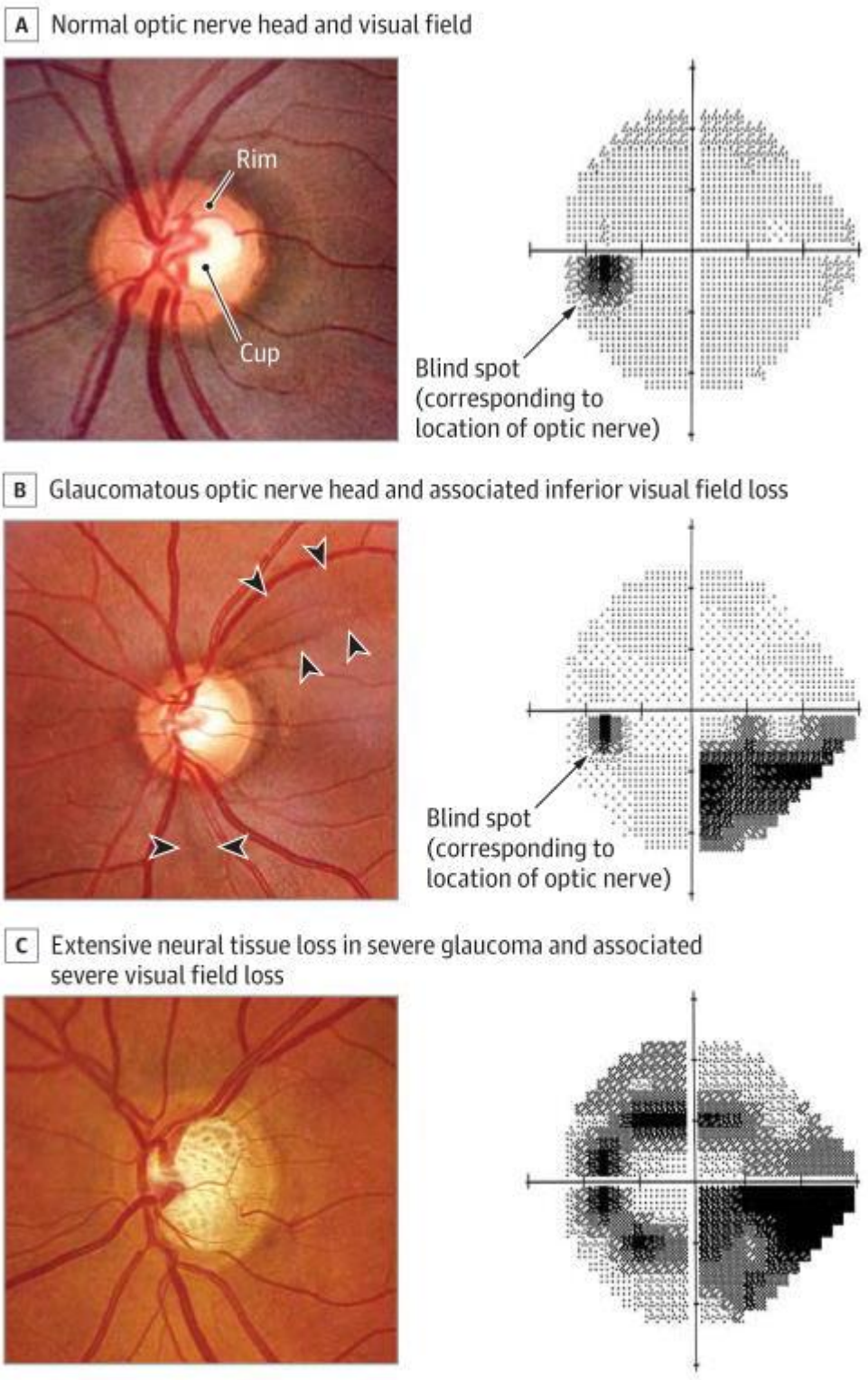


Figure 12:Visual Field Test Results with Normal, Glaucomatous Optic Nerve Heads.[29]

Fundoscopic examination: During a normal eye exam, POAG is often identified when the patient is still asymptomatic and before a discernible loss of vision. The primary method for assessing the ONH and RNFL in order to diagnose glaucoma is slit-lamp biomicroscopy. For ONH examination, both direct and indirect fundoscopy are inadequate; direct fundoscopy does not provide a binocular view of the ONH, while indirect fundoscopy's magnification is insufficient to enable a trustworthy evaluation of the ONH.[24]

Slit-lamp biomicroscopy: To distinguish glaucomatous from non-glaucomatous optic neuropathies, slit-lamp biomicroscopy is used to carefully examine the color of the neuroretinal rim and the arrangement of the optic disc.[24]

Digital imaging: While optic disc photography is still helpful for recording the ONH and RNFL configuration, digital imaging technologies have given doctors a more efficient, objective, and quantitative way to help them diagnose glaucoma. The three commercially accessible imaging systems for assessing the ONH and RNFL structures are optical coherence tomography (OCT), confocal scanning laser ophthalmoscopy, and scanning laser polarimetry.[40–42]

Perimetry: Since RNFL and ONH abnormalities found by clinical examination or digital imaging tools often occur before observable visual field loss, perimetry is not necessary for the diagnosis of glaucoma. In individuals with glaucoma, a significant decrease in RNFL thickness as determined by OCT may be seen up to eight years before visual field abnormalities appear.[43] The most effective method for diagnosing and tracking POAG is automated static threshold perimetry, such as Humphrey field analysis. Patients may spend less time testing thanks to a variety of algorithms that have been verified for POAG (e.g. Swedish Interactive Threshold Algorithm, SITA). According to estimates, 50% of ganglion cells must be destroyed before a field defect manifests itself, and patients with early POAG may test with normal visual fields. Because of this justification, new perimetric tests like frequency doubling technology (FDT) and short-wavelength automated perimetry (SWAP) have been developed. By focusing on a

particular fraction of ganglion cells with sparse distribution, these tests may be able to improve the early diagnosis of functional loss.[44]

IOP measurement Although elevated IOP is a risk factor for the onset and progression of glaucoma, it is not a diagnostic necessity for glaucoma. Goldmann applanation tonometry is the most often used method for measuring intraocular pressure (IOP), despite the fact that there are other contact and non-contact tonometers available in clinical practice. However, the cornea's shape and biomechanical characteristics affect IOP readings obtained by Goldmann applanation tonometry. When the cornea is thin or soft, IOP is often underestimated; when the cornea is thick or stiff, IOP is typically overstated. The assessment of central corneal thickness, which is often done using corneal pachymetry (ultrasonography), is another component of the clinical examination of glaucoma that takes into consideration the variations in the cornea's characteristics. There are currently no validated tools for measuring corneal flexibility.[24]

Management

By maintaining eyesight, POAG therapy aims to improve the patient's quality of life. (AAO) At the moment, this means bringing the IOP down to a level that should stop more optic nerve injury.[45]

Despite several trials demonstrating comparable or better IOP control with first laser therapy or surgery, topical medicines are often considered the first-line treatment for POAG.[46] Due to the inherent risks of complications, surgery has not been routinely used as a primary therapy in most settings, despite the possibility for good IOP control.

Topical drugs: By encouraging aqueous humor outflow or decreasing aqueous humor generation, topical drugs administered directly to the ocular surface may reduce intraocular pressure.[47] Digital punctal occlusion or eyelid closure for at least one minute after application may prevent these medications from entering the systemic circulation via the nasolacrimal duct.

Class	Drugs	Mechanisms of action	Notes
Aqueous humour outflow (uveoscleral pathway)			
Prostaglandin analogues	Latanoprost, travoprost bimatoprost and tafluprost	Increase in outflow of aqueous humour, primarily through the uveoscleral pathway by matrix metalloproteinase expression, and remodelling of the uveoscleral outflow tract	• Normally used as a first-line therapy • Highly effective and well tolerated
Aqueous humour outflow (conventional pathway)			
Cholinergic agonists	Pilocarpine and carbachol	Increase in aqueous humour outflow through the trabecular meshwork due to ciliary muscle contraction	Very effective, but the use is compromised by dim vision (owing to pupillary constriction), discomfort and myopia
Aqueous humour formation			
β-Adrenergic receptor blockers	Timolol, betaxol, carteolol and levobunolol	Reduce the secretion of aqueous humour secretion from the ciliary body	Few ocular adverse effects but might cause systemic adverse effects, such as fatigue and bradycardia
α-Adrenergic receptor agonists	Apraclonidine and brimonidine	• Decrease aqueous humour inflow by inactivating adenylyl cyclase in ciliary processes • Mediates the release of noradrenaline via activation of α2-adrenergic receptors and might increase uveoscleral outflow	• Sedation can be minimized by occluding nasolacrimal tear duct (for up to 2 minutes) • These drugs might have neuroprotective effects, but can induce allergy, particularly with the 0.2% formulation
Carbonic anhydrase inhibitors	Dorzolamide, brinzolamide, acetazolamide and methazolamide	Decrease in aqueous humour formation by inhibiting the production of carbonic anhydrase and bicarbonate in the ciliary epithelium	Application using eye drops is better tolerated than oral administration

Table 2: Pharmacotherapy drugs used in POAG therapy[24,47]

Regarding the treatment of POAG, laser trabeculoplasty is quite significant. By stimulating trabecular meshwork cells, which results in the remodeling of the local extracellular matrix and an increase in aqueous humor outflow, laser treatments to the trabecular meshwork reduce the intraocular pressure. But we still don't completely understand the exact mechanics behind IOP decreases. For more than 30 years, argon laser trabeculoplasty, or ALT, has been used in clinical practice. However, SLT, which uses a neodymium-doped yttrium aluminum garnet laser, is becoming more and more popular. SLT lowers IOP just as well as ALT, but it has fewer side effects and causes less mechanical harm to the trabecular meshwork. In one St. Lucian research, those with POAG of West African descent who had first SLT therapy saw a long-lasting reduction in their intraocular pressure.[48] Additionally, a meta-analysis's results revealed a 6.9–35.9% decrease in IOP one year after SLT.[24,49]

Surgery: A partial-thickness scleral flap covering a sclerectomy (a tiny hole in the sclera) from the anterior chamber of the eye into the subconjunctival region is used in trabeculectomy. For

more than 40 years, trabeculectomy has been the most common glaucoma operation. Because cataract extraction surgery, which includes replacing a cloudy natural lens with a clear artificial intraocular lens, has the potential to bring about an increase in the anterior chamber angle and a drop in IOP, lens extraction is not recommended for the treatment of POAG. This impact is often minor and short-lived. New options to close the gap between topical treatments and surgery for IOP management have been made available by minimally invasive glaucoma procedures.

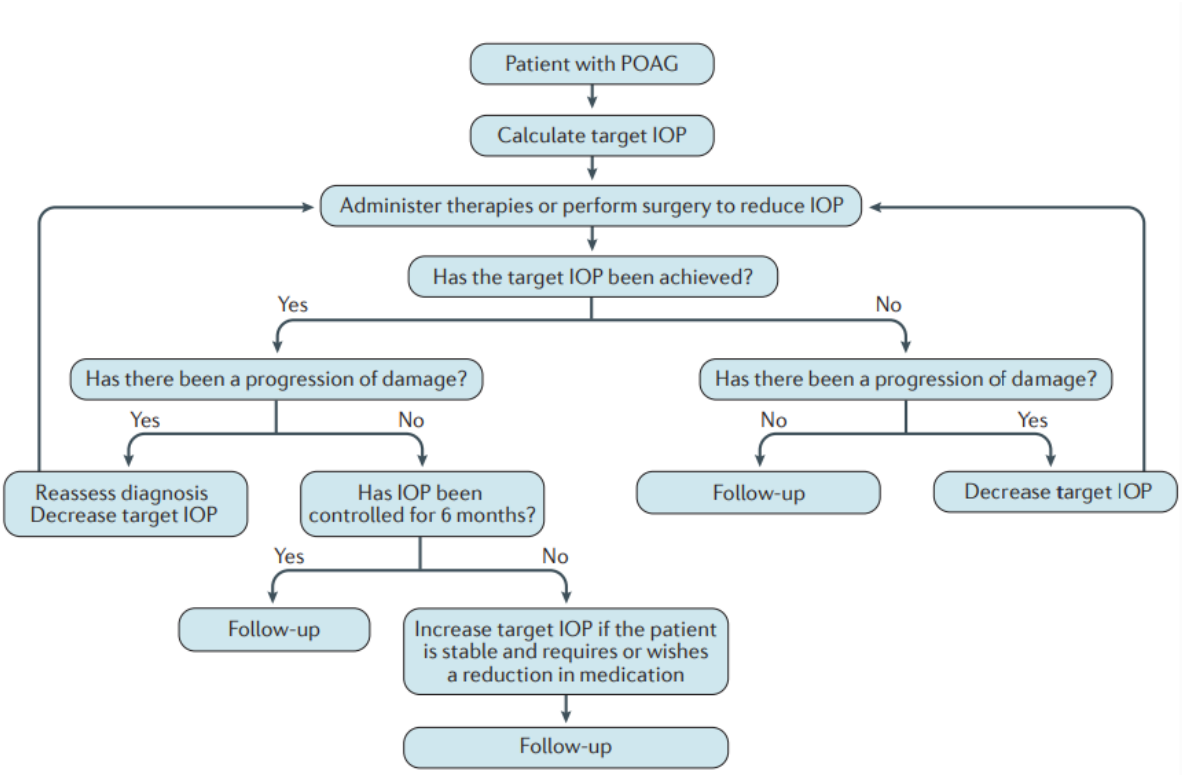


Figure 13: Management strategy for newly diagnosed primary open-angle glaucoma.[24]

Prognosis & Complications

The majority of OAG patients will not lose their vision in their lifetime, but advanced POAG may result in optic atrophy and loss of light sensitivity. OAG progression risk factors include:[50,51]

- Aging
- High IOP
- A smaller optic rim area or a higher cup-to-disc ratio
- Atrophy of beta peripapillaries
- Hemorrhage on the disc
- thin central corneal thickness
- Diminished hysteresis of the cornea
- Low pressure of ocular perfusion
- Poor adherence to treatment
- The process of pseudoexfoliation

According to research findings, the cumulative likelihood of untreated instances of end-stage glaucoma in at least one eye within ten years was 35%.

Glaucoma is a leading cause of visual impairment that may impact many facets of quality of life and hinder performance in a wide range of everyday activities, including reading, walking, and driving.[52] Individuals who have glaucoma may also be more vulnerable to falls and car accidents.[53] Understanding when and how glaucoma causes disability is crucial to adjusting the dose of treatment to the pace of functional degradation and the likelihood of impairment, as the existing medications for glaucoma may have negative side effects.

OPTICAL COHERENCE TOMOGRAPHY (OCT)

Optical coherence tomography has become a valuable imaging method by offering new high-resolution cross-sectional data on several macula pathological characteristics. It gives medical professionals the ability to accurately and consistently assess retinal thickness quantitatively.[54] OCT produces in vivo pictures without affecting the tissue being examined since it is entirely noninvasive. Real-time and video-rate image viewing is made possible by rapid scanning rates and signal processing. Compared to other medical imaging techniques like ultrasound or MRI, OCT has a much greater resolution. It combines a lateral resolution similar to confocal scanning laser ophthalmoscopy with an axial resolution that may approach that of confocal microscopy.[55]

Principle & Technique

Due to their similar operating principles, OCT and medical ultrasonography are often compared. Waves are sent to the tissue being examined by both medical imaging modalities, and the tissue structure reflects the waves. The depth of the reflection is determined by analyzing the back-reflected waves and measuring their delay. Near-infrared light, which is used in OCT, travels much more quickly than ultrasound. Due to the impossibility of directly measuring the delays of back-reflected waves, a reference measurement is employed. A portion of the light is supplied to a reference arm of known length, while another component is directed to the sample via an interferometer.

The light from the light source is split into two beams: the center beam and the reference beam. The detector then combines the back-reflected light from both arms once more and records it. In order to obtain an A-scan, the reference arm needs to be scanned. This allows the detector to capture one depth profile of the sample simultaneously. This must be done again for every point of the lateral scan.[56]

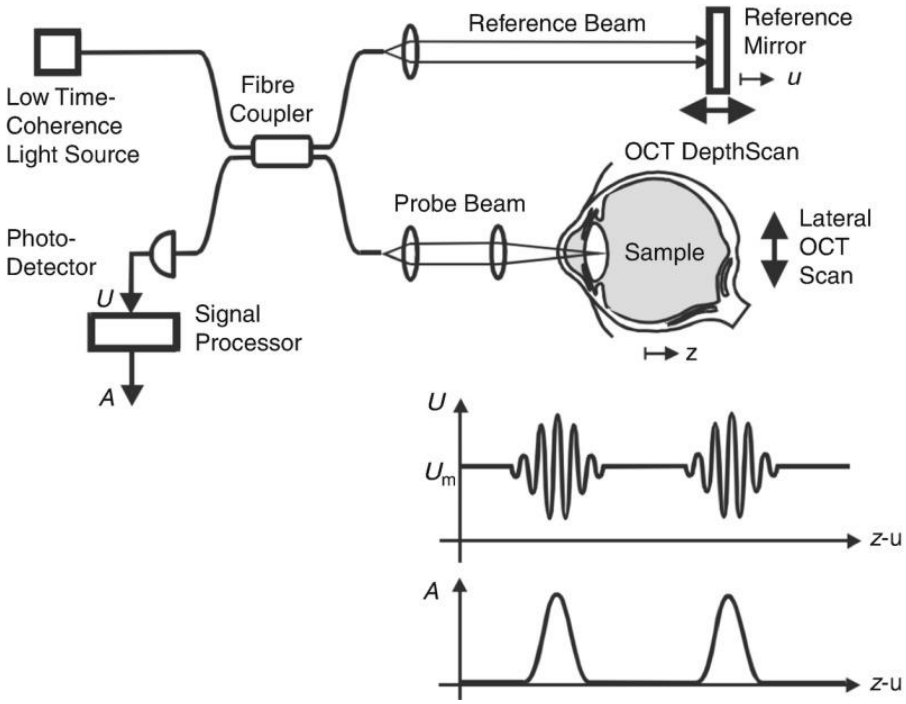


Figure 14: Principle of OCT

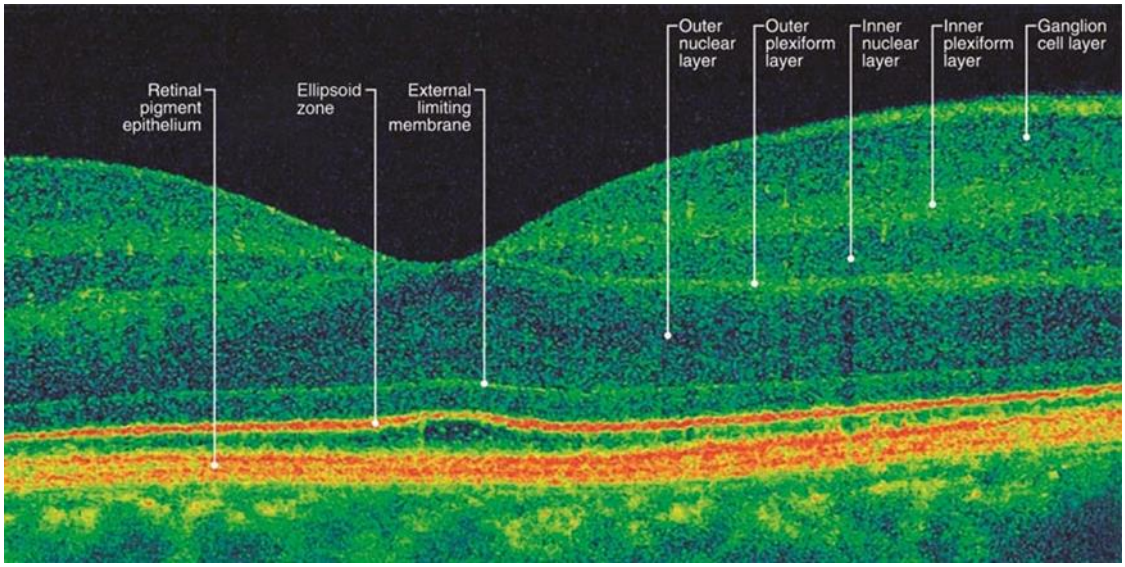


Figure 15 : Normal OCT picture with Retinal layers

Figure 16 : OCT Machine

HUMPHREY FIELD ANALYSER (HFA)

An important diagnostic tool for assessing the visual field and providing important information about neurological and ocular disorders is the Humphrey Visual Field (HVF) test. While patterns like 10-2 provide in-depth central vision examination, often utilized patterns like 24-2 and 30-2 help identify field abnormalities like scotomas or glaucomatous damage.[57]

Principle & Technique

The visual field printout displays the retina's light sensitivity measurement at a specific region. In a healthy person, the fovea has the most sensitivity in the retina, which decreases as one moves toward the periphery. Apostilb is used to quantify the light stimulus's intensity.[58] On the other hand, retinal sensitivity and apostilb have an inverse relationship. Additionally, the human eye reacts to a variety of apostilbs. Thus, decibels (0–50 in typical automated perimetry) are used to quantify light sensitivity. The intensity of the light stimulus is expressed logarithmically in decibels. The Weber-Fechner law, which states that sensation rises with the stimulus's logarithm, is adapted in this way.[59] Decibel and retinal sensitivity are directly

correlated. The brightest light stimulus, 10,000 apostilbs in a Humphrey field analyzer, is represented by a decibel (dB) of zero, while the dimmest stimulus, 0.1 apostilbs, is represented by a dB of fifty. Therefore, a location on the retina with the lowest sensitivity will be able to see a stimulus at zero decibels (0 dB), and vice versa. An absolute scotoma is indicated if the 0 dB stimulus is not seen. Because it is relative, the decibel level varies from machine to machine. On the Humphrey visual field analyzer, a 0 dB value corresponds to 10,000 apostilbs, however on the Octopus perimetry, it corresponds to 4000 apostilbs. One apostilb on the Humphrey visual analyzer and 0.4 apostilb on the Octopus perimetry are represented by a 40 dB value.[57]

Figure 17 : HFA machine

Macular Volume and Thickness in Glaucoma

In glaucoma, the cells that die are called “retinal ganglion cells (RGCs)”. The RNFL is composed of RGC axons, which leave the eye via the optic nerve. Peripapillary rNFL losses on SD-OCT or increased optic nerve cupping are indicators of the progression of glaucomatous optic neuropathy. More than half of RGC corpses are found within ~5 mm or 16° of the fovea.

Cell bodies are arranged in stacks of up to six layers.[60] Therefore, by examining the overall retinal thickness, minor losses of ganglion cell bodies—which, along with the rNFL, account for more than 30% of the retinal thickness—can be identified. When there is no additional macular disease present, total macular thickness serves as a proxy for tissue thickness loss brought on by glaucoma.

The loss of the "inner plexiform layer (IPL)", ganglion cell bodies, and RNFL—tissues that are lost in glaucoma—is consequently reflected in changes in the overall macular thickness loss in glaucoma. Because of the high degree of reflectivity from these two border areas of the retina, optical reflecting technologies like the OCT can readily and reliably assess total macular thickness. Retinal Thickness Analyzer was the first device to measure retinal thickness. Time domain OCT was used later. The center 6 mm × 6 mm perifoveal region was assessed by the Stratus OCT using data from six radial line scans that intersected at the fovea. The data in between the lines was interpolated to construct a map of macular thickness.[61,62] When compared to peripapillary RNFL measurements, this method of interpolating data with sparse measurements in a wide retinal area did not seem to be helpful in the diagnosis and treatment of glaucoma.[61] SD-OCT subsequently made it possible to assess bigger retinal regions at faster acquisition speeds. As a result, a more accurate retinal (macular) thickness map may be produced by measuring retinal thickness with a higher concentration of data points and far less interpolation of data. For this, many software techniques from various instrument manufacturers are used, such as a grid of lines or a raster of lines throughout the macular area.

It has been shown that in glaucoma, macular thickness correlates with both peripapillary RNFL thickness and optic nerve cupping.[63] "Humphrey Visual Field (HVF)" characteristics and estimated RGC count have been linked to macula thickness reductions in glaucomatous and normal eyes.[64,65] While imaging the macula enables the measurement of the overall thickness

of the macula, peripapillary RNFL scans show the ring of tissue around the optic nerve. This makes it possible to map RGCs and macula thickness to the visual field for comparison.

Compared to the more peripheral retina included in the visual field, the macular region occupies comparatively few locations in the standard 24-2 or 30-2 visual field, thus visual field deficiencies in this area need a higher number of ganglion cell losses. Thus, visual field testing cannot identify small decreases of macular thickness, increasing the possibility of early glaucoma detection using this modality.[45]

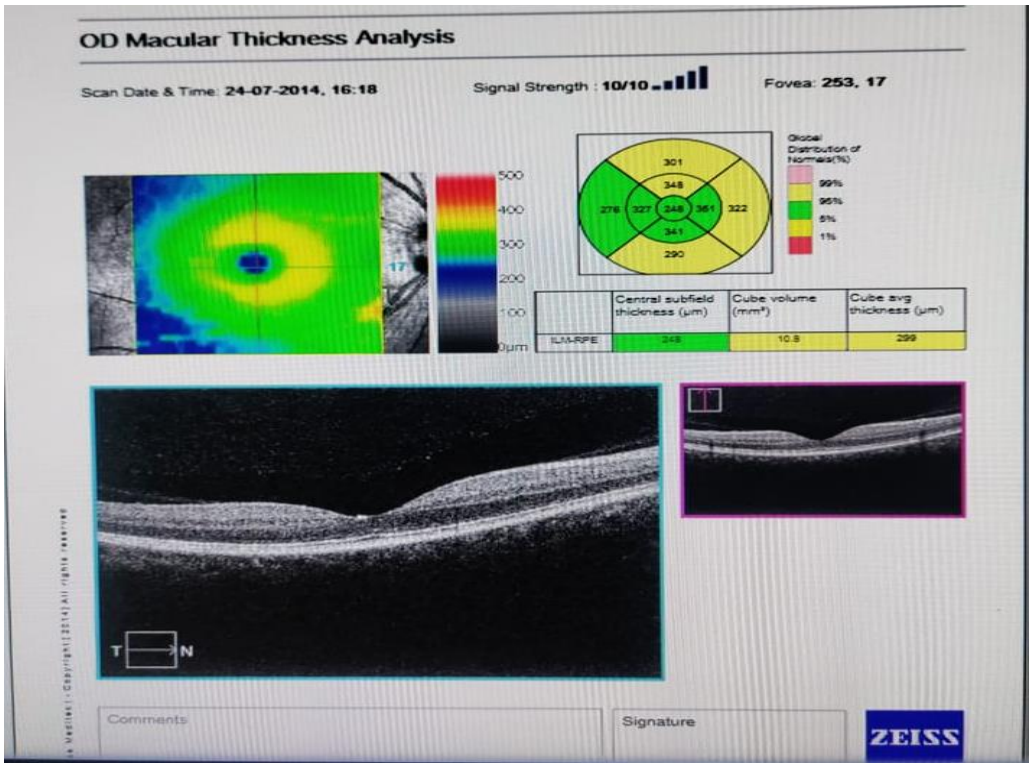


Figure 18: OCT report

Studies in the past on Total Macular Thickness and volume in glaucomatous eyes using OCT

Sharma et al. used OCT to evaluate the retinal macular thickness and volume in patients with POAG to normal people. TMV, OMT, and IMT values were considerably lower in the POAG group than in the control group, but CMT did not vary, most likely because there were no

ganglion cells in the central region. Macular thickness and volume measures may thus be utilized to diagnose glaucoma, particularly in individuals who have disc abnormalities. Measures of macular thickness showed a strong correlation with glaucoma diagnosis.[9]

In patients with glaucoma and ocular hypertension (OH), **Mota et al**. found a correlation between macular thickness (MT) and VF characteristics, as well as with variations in the thickness of the retinal nerve fiber layer (RNFL). VF characteristics and the decline in MT were shown to be significantly correlated ($p<0.05$). When we compared the LV in the group with average MT 270 to 300 μm, the findings were more significant ($p<0.05$). In the group with MT < 270 μm, asymmetry between the superior and inferior macula linked with LV ($p<0.05$). Additionally, there was a strong association ($p < 0.05$) between the decline of the superior and inferior MT and the thinning of the superior-temporal and inferior-temporal RNFL. In individuals with glaucoma, measures of retinal thickness in the macula using spectral domain OCT correlate with VF and RNFL characteristics. Static computerized perimetry created using Octopus 101® was used to first illustrate this link. By showing a link between structure and function, these findings may be a useful tool for glaucoma patients' evaluation and follow-up.[66]

Giovanni et al. applied OCT to measure macular volume in both glaucomatous and normal eyes. They noticed notable variations between the groups. Subjects with advanced glaucoma (mean±SD=6.678 +/- 0.455 mm3) showed much less volume than those with normals (mean±SD=7.35 +/- 0.455 mm3) and early glaucoma (mean±SD=7.09 +/- 0.475 mm3). OCT tomograms combined with volumetric examination of macular thickness might be a helpful tool for tracking and recording individuals with both advanced and early glaucoma. According to our data and other authors' findings, there is a substantial correlation between OCT macular volumes and glaucoma state.[67]

Using OCT, **Chaturvedi et al.** evaluated myopes' variations in macular volume and RNFL thickness and their importance for the early detection of POAG. 4 quadrants and an inner circle were used to measure the RNFL thickness. Both groups' mean inner circle thicknesses were statistically significant, measuring 101.48 μm (SD±13.34 μm) and 92.38 μm (SD±11.99 μm), respectively. The superior, nasal, and inferior quadrants showed a significant difference as well. The difference in the temporal quadrant was not statistically significant. A 6mm diameter was used to compute the macular volume. Group A's mean value was 7.82 mm±0.54 mm^3. In Group B, the mean value was 7.44mm3±0.98mm3. According to the statistical analysis, there is a statistically significant difference between the two groups. An accepted method for identifying open angle glaucoma at the preperimetric stage is RNFL thickness. Additionally, it is discovered that the risk and macular volume are connected. POAG is thought to be more likely to develop in myopes. Therefore, it should be obligatory to test RNFL thickness using OCT in High Myopes.[68]

Ojima et al. assessed the macular volume and RNFL thickness in glaucoma patients' eyes and normal eyes, comparing the diagnostic utility of these parameters in glaucoma patients' eyes. When comparing eyes with early glaucoma to normal eyes, there was a substantial reduction in macular volume in six of the nine macular sectors and RNFL thickness in eight of the twelve peripapillary sectors. In the eyes of patients with advanced glaucoma, the RNFL and macular volume were both decreased, with the exception of the thickness of the RNFL in the foveal region and the thickness of the retina in the papillomacular region. The average RNFL (0.963) had a greater “area under the receiver-operating characteristic curve (AUROC)” than the macular volume (0.919).[69]

The idea that retinal thickness and glaucoma diagnosis are related was assessed by Guedes et al. When comparing normal participants with either early or advanced glaucoma, all NFL

parameters in both prototype and commercial OCT machines were statistically substantially different ($P < 0.05$). There were substantial differences between normal participants and advanced glaucomatous eyes in terms of inner ring, outer ring, and mean macular thickness in both prototype and commercial OCT devices ($P < 0.05$). OCT measurements of macular and NFL thickness revealed statistically significant associations with glaucoma, however the link was greater for NFL thickness than macular thickness. The results obtained from the prototype and commercial OCT machines showed excellent agreement. Clinical evaluation of glaucoma may benefit from OCT measures of macular and NFL thickness.[61]

Ganglion cell-inner plexiform layer (GCIPL) and RNFL thicknesses were linked with macular and peripapillary "vascular perfusion density (VD)" in glaucoma patients and "glaucoma suspects (GS)" by **Triolo et al.** The glaucoma group's peripheral RNFL, GCIPL, and macular RNFL thicknesses were considerably lower than those of the controls and GS ($P < 0.05$). In the glaucoma group, peripheral VD reduced on average and in the superior and inferior quadrants ($P < 0.05$), whereas macular VD did not change significantly across groups ($P > 0.05$). There was an association between RNFL thickness and VD at the peripapillary region, but no statistically significant relationship between GCIPL thicknesses and macular VD was seen across all groups (all $P > 0.05$).[70]

Greenfield et al. used OCT to correlate the thickness of the RNFL with the macular thickness among both glaucomatous and normal eyes. The mean RNFL thickness, pattern standard deviation, and visual field mean defect were all substantially correlated with mean macular thickness ($p<0.05$). The mean +/- SD macular thickness in the field defect quadrant (277 +/- 28 micro m) was considerably lower ($P =.005$) than in the unaffected quadrant (286 +/- 27 micro m) in glaucomatous eyes with visual field loss confined to 1 hemifield ($n = 11$). Compared to the unaffected quadrant (121 +/- 39 micro m), the mean RNFL thickness in the

damaged quadrant (89 +/- 53 micro m) was substantially lower (P =.009). Changes in macular thickness may be a proxy for retinal ganglion cell loss in glaucoma, since they are closely linked to alterations in visual function and RNFL structure.[71]

MATERIALS AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA:

Patients visiting Ophthalmology outpatient department at R.L. JALAPPA HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR attached to SRI DEVARAJ URS MEDICAL COLLEGE for routine check-up or refraction.

SAMPLE SIZE: 40

STUDY DESIGN: Cross sectional study

INCLUSION CRITERIA:

1.Patients with primary open angle glaucoma

2. Healthy Control group with no history of glaucoma or retinal pathology IOP of <21mm Hg, normal optic nerve head appearance

EXCLUSION CRITERIA:

1. Angle closure glaucoma
2. Diabetic retinopathy
3. Macular degeneration
4. Macular edema
5. Epiretinal membrane
6. Retinal detachment
7. Cataract
8. High myopia
9. Presence of non glaucomatous optic nerve disease
10.Previous ocular surgery or trauma

METHODOLOGY

Each patient will be assessed by detailed history and clinical examination of both the eyes will be done by various methods as follows-

Setting- Ophthalmology Out Patient Department.

All primary open angle glaucoma patients visiting Ophthalmology Outpatient department of either sex and 45-65 years of age to undergo OCT are considered for this research.

Each patient will be assessed by detailed history and clinical examination of

both the eyes will be done by various methods as follows

1. Best corrected visual acuity by snellens chart.
2. Slit lamp bio microscopy for evaluation of anterior segment.
3. Posterior segment evaluation done by indirect ophthalmoscopy

and or +90D bio-microscopy.

4. Assessment of Intraocular pressure by Goldmann Applanation

Tonometer

5. Gonioscopy- Shaffer's grading system will be used.
6. Macular thickness and volume by Optical coherence

tomography(time domain Zeiss primus-200)

7. Field analysis done by white on white Humphrey Field Analyzer (Carl Zeiss)

SAMPLE SIZE ESTIMATION

Sample size variants estimate for total macular thickness as reported in the study by Anjali sharma etal. Macular Thickness variability in primary open angle glaucoma patients using optical coherence tomography.[72] with 95% confidence interval and 80% power the estimated total sample size will be 40 with 20 patients in each group

Sample size = 4 Pq/ (L^2)

P = Expected proportion in population based on previous studies

q = 100- P

L = allowable error

STATISTICAL METHODS USED FOR STUDY

Data was entered in MS Excel and analysed by SPSS v27.0. Categorical variables are expressed in frequency and percent. Continuous variables were expressed in mean (SD) and median (IQR). Chi-square test was applied to test association between categorical variables. Association between glaucoma and the continuous variables was tested by Mann-Whitney test. P value below 0.05 is taken as significant.

RESULTS

Normal and glaucoma patients equally included.

Table 3: Frequency among Groups

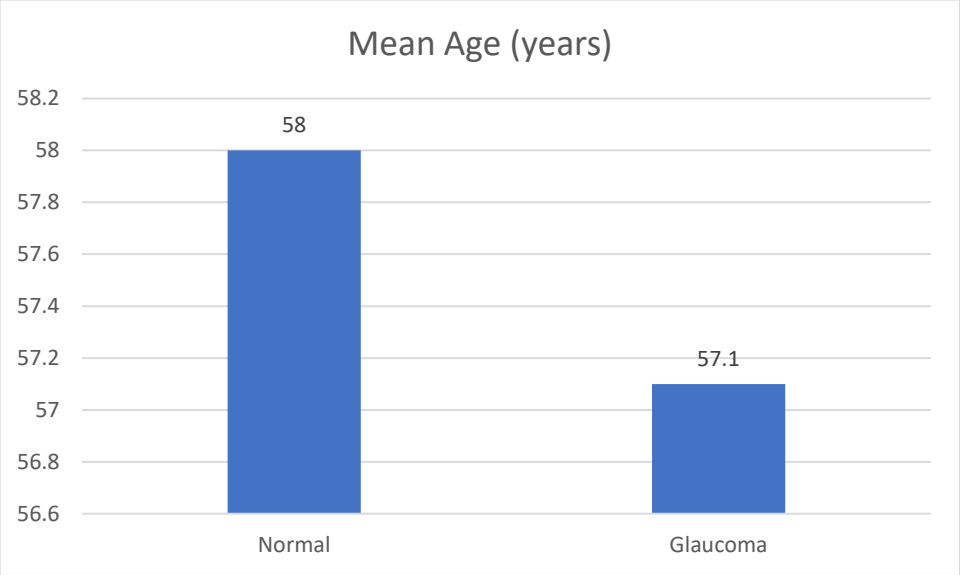
Group

	Frequency	Percent
Normal	20	50.0
Glaucoma	20	50.0
Total	40	100.0

Table 4: Age distribution

	Normal				Glaucoma			
	Mean	Median	SD	IQR	Mean	Median	SD	IQR
AGE	58.00	59.00	4.15	56,60.75	57.10	56.00	2.29	55.25,58

Mean age of normal and glaucoma patients were 58 and 57.1 years, respectively.



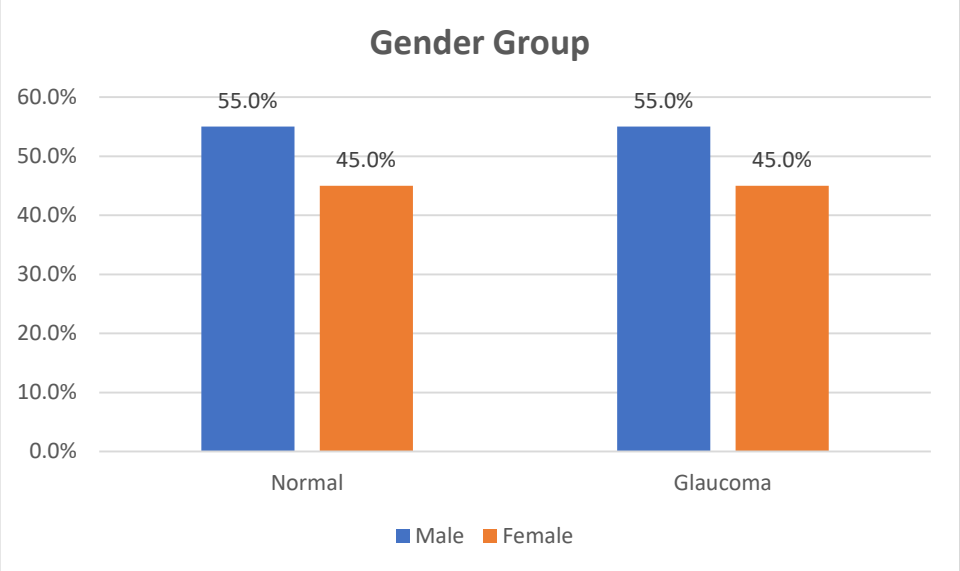
Graph 1: Mean age

GENDER Group

Table 5: Gender wise distribution

			Normal	Glaucoma	P value
GENDER	Male	Frequency	11	11	1.000
		Percent	55.0%	55.0%	
	Female	Frequency	9	9	
		Percent	45.0%	45.0%	
Total		Frequency	20	20	
		Percent	100.0%	100.0%	

Most of the participants in normal and glaucoma groups were males (55%).



Graph 2: Gender wise distribution

Table 6: Mean macular thickness (1-3mm) among groups in microns

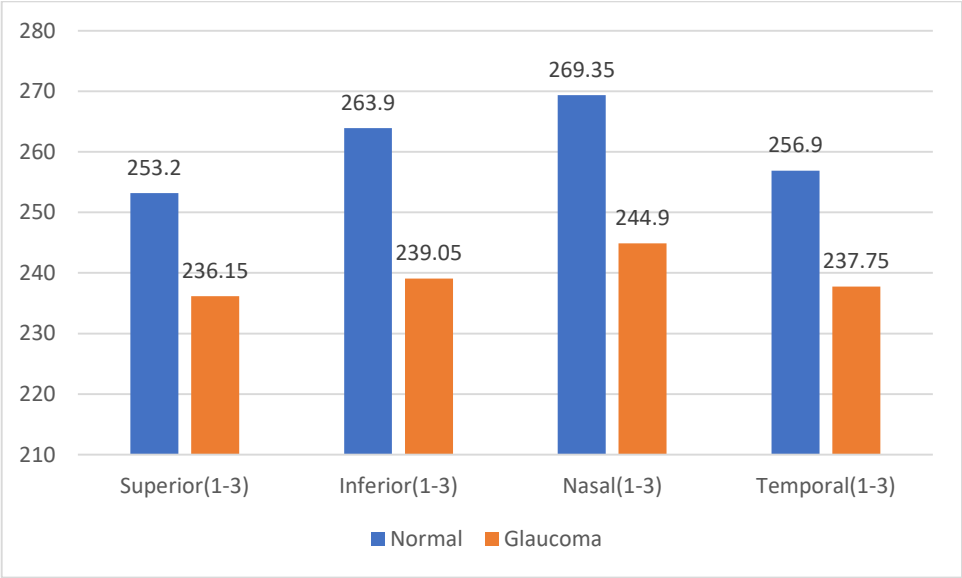
	Normal				Glaucoma			
	Mean	Median	SD	IQR	Mean	Median	SD	IQR
Superior(1-3)	253.20	260.00	14.91	234,266	236.15	233.00	14.36	230,240
Inferior(1-3)	263.90	270.00	14.16	250,274	239.05	237.50	20.22	222.5,247.75
Nasal(1-3)	269.35	272.00	9.07	270,275	244.90	250.00	13.40	236,252
Temporal(1-3)	256.90	260.00	8.98	249,264	237.75	239.00	16.16	223.5,245.5

The mean Superior macular thickness (1-3) in normal and glaucoma patients were 253.20 µm and 236.15 µm

The mean Inferior macular thickness (1-3) in normal and glaucoma patients were 263.90 µm and 239.05 µm

The mean Nasal macular thickness (1-3) in normal and glaucoma patients were 269.35 µm and 244.90 µm

The mean temporal macular thickness (1-3) in normal and glaucoma patients were 256.90 µm and 237.75 µm



Graph 3: Mean macular thickness (1-3mm)

Table 7: Mean macular thickness (1-6mm) among groups in microns

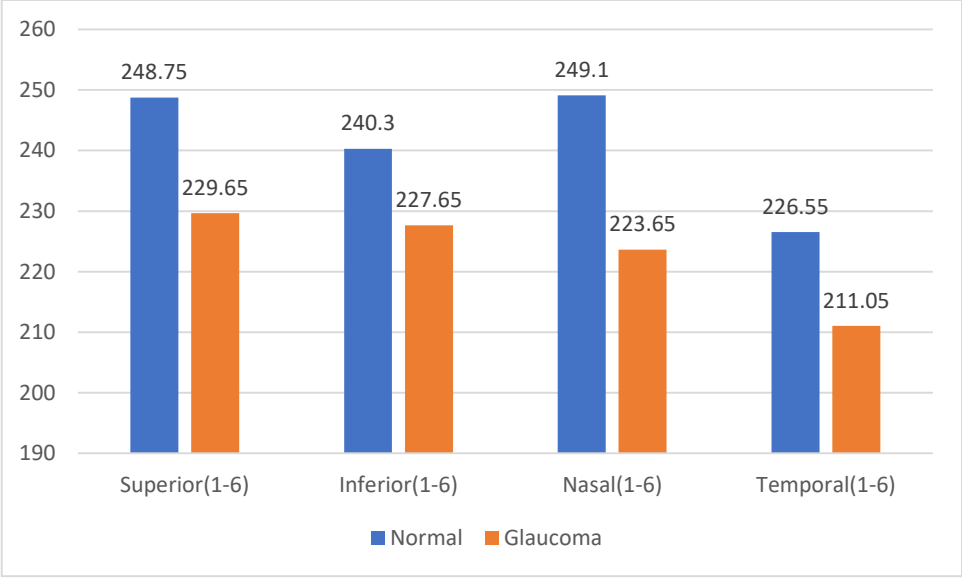
	Normal				Glaucoma			
	Mean	Median	SD	IQR	Mean	Median	SD	IQR
Superior(1-6)	248.75	254.00	10.13	237.75,255	229.65	230.00	14.80	216.25,234
Inferior(1-6)	240.30	242.00	7.34	240,246	227.65	226.00	8.82	224,230
Nasal(1-6)	249.10	253.00	12.14	253,256	223.65	220.00	15.10	216.5,228
Temporal(1-6)	226.55	230.00	9.86	228,232	211.05	208.00	11.17	203.25,215

The mean Superior macular thickness (1-6) in normal and glaucoma patients were 248.75 µm and 229.65 µm

The mean Inferior macular thickness (1-6) in normal and glaucoma patients were 240.30 µm and 227.65 µm

The mean Nasal macular thickness (1-6) in normal and glaucoma patients were 249.10 µm and 223.65 µm

The mean temporal macular thickness (1-6) in normal and glaucoma patients were 226.55 µm and 211.05 µm



Graph 4: Mean macular thickness (1-6mm)

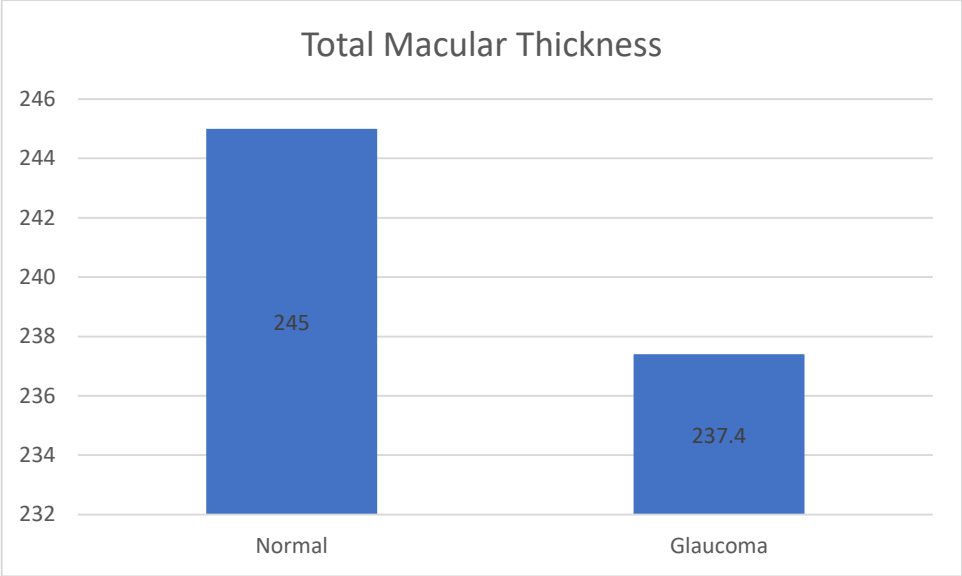
Table 8: TMT TMV IOP among groups

	Normal				Glaucoma			
	Mean	Median	SD	IQR	Mean	Median	SD	IQR
Total Macular Thickness	245.00	247.00	12.80	233,257.75	237.40	235.50	15.28	222.25,253.75
Total Macular Volume	6.65	6.70	0.17	6.6,6.7525	6.26	6.20	0.23	6.1,6.3
IOP	12.50	12.00	1.82	12,14	20.40	20.00	2.56	18,22

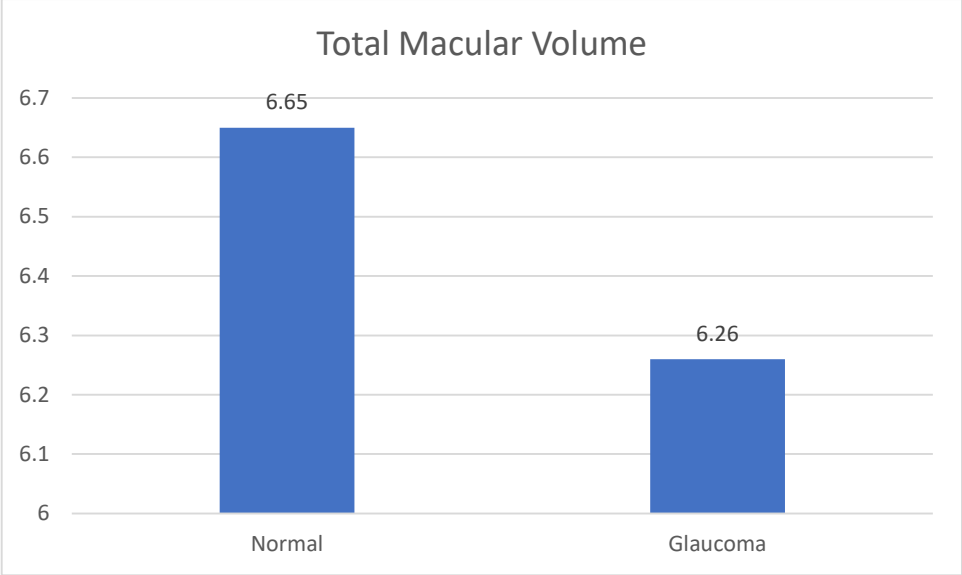
The mean Total Macular Thickness in normal and glaucoma patients were 245.00 µm and 237.40 µm.

The mean Total Macular Volume in normal and glaucoma patients were 6.6 mm^3 and 6.26 $mm^{3.}$

The mean IOP in normal and glaucoma patients were 12.50 mmHg and 20.40 mmHg.

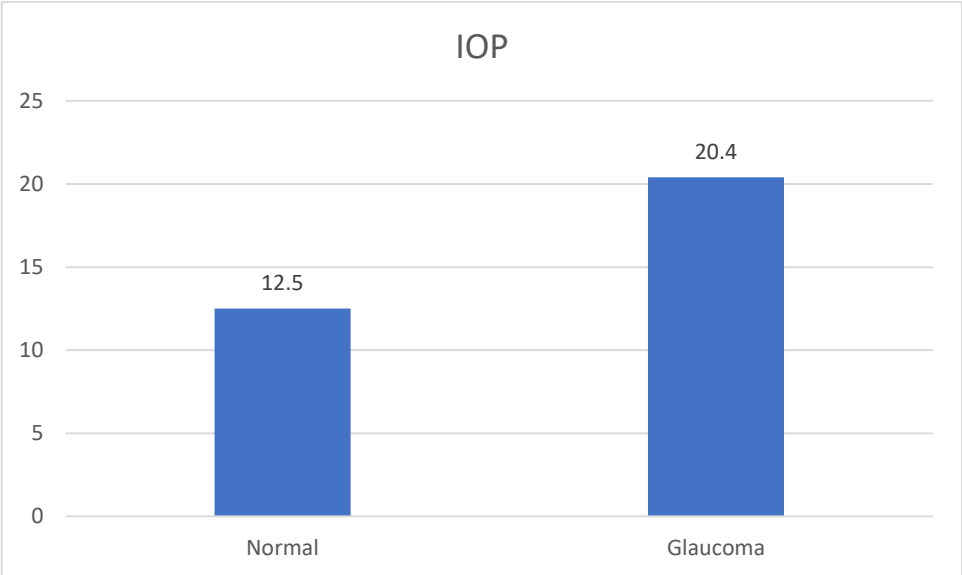


Graph 5: Total macular thickness among groups



Graph 6: Total macular volume among groups

Macular thickness and volume was significantly lower in glaucomatous eyes than normal eyes in all quadrants



Graph 7: Mean IOP among groups

Table 9: Mean ranks

Ranks

	normal/glaucoma	N	Mean Rank	P value
superior(1-3)	Normal	20	25.90	0.003
	Glaucoma	20	15.10	
	Total	40		
inferior(1-3)	Normal	20	27.25	<0.001
	Glaucoma	20	13.75	
	Total	40		
nasal(1-3)	Normal	20	29.10	<0.001
	Glaucoma	20	11.90	
	Total	40		
temporal(1-3)	Normal	20	27.03	<0.001
	Glaucoma	20	13.98	
	Total	40		
superior(1-6)	Normal	20	27.85	<0.001
	Glaucoma	20	13.15	
	Total	40		
inferior(1-6)	Normal	20	27.13	<0.001
	Glaucoma	20	13.88	
	Total	40		
nasal(1-6)	Normal	20	27.68	<0.001
	Glaucoma	20	13.33	
	Total	40		
temporal(1-6)	Normal	20	27.03	<0.001
	Glaucoma	20	13.98	
	Total	40		
Total Macular Thickness	Normal	20	24.85	0.018
	Glaucoma	20	16.15	
	Total	40		
total volume	Normal	20	27.60	<0.001
	Glaucoma	20	13.40	
	Total	40		
IOP	Normal	20	10.60	<0.001
	Glaucoma	20	30.40	
	Total	40		

DISCUSSION

DISCUSSION

Glaucoma is a chronic, progressive optic neuropathy that results in irreversible visual impairment, primarily due to the degeneration of RGCs. OCT has become an essential diagnostic tool for detecting early structural changes, particularly within the macula, which houses over 50% of RGCs. In this study, we analyzed a total of 40 eyes—20 from glaucomatous patients and 20 from age- and sex-matched normal controls—to evaluate differences in macular thickness, macular volume, and intraocular pressure (IOP).

In our study, both the glaucomatous and normal groups were age- and sex-matched, with 20 subjects in each group. The mean age was 57.1 years in glaucoma patients and 58.0 years in controls, and both groups had an identical gender distribution (55% male, 45% female), minimizing demographic bias. Anjali Sharma et al[72]. similarly found no significant differences in age (50.48 vs. 50.52 years; $p = 0.962$) or gender distribution (male:female ratio 52:24 vs. 44:24; $p > 0.05$) between their glaucoma and control cohorts. Mona Abdelkader et al.[73] also reported no significant differences in age, gender, or refractive error among their study participants, with comparable age means (40 ± 15 vs. 38 ± 13 years) and nearly equal sex distribution (50% male and female in normals; 53% female and 47% male in glaucoma patients). Complementing these findings, Viviane Guedes et al.[74] observed an age-related trend across glaucoma stages, with mean age increasing from 50.1 years in normals to 65.9 years in advanced glaucoma, along with modest gender variation. Collectively, these studies—including ours—highlight the importance of matching demographic variables to ensure valid comparisons and emphasize the increasing age-related risk of glaucoma progression.

In our study, macular thickness in all quadrants—superior, inferior, nasal, and temporal—was significantly reduced in glaucomatous eyes compared to normal controls, with the most marked

thinning observed in the nasal quadrant (244.90 µm in glaucoma vs. 269.35 µm in normals). Inferior and temporal regions also showed substantial reductions. These differences were statistically highly significant ($p < 0.001$ for all quadrants), indicating the diffuse nature of glaucomatous damage in the macula. Our findings align with those of Anjali Sharma et al.[72], who reported a mean inner macular thickness of 243.96 µm in glaucoma patients versus 263.56 µm in normals, with significant thinning across all quadrants—temporal (233.09 ± 24.47 µm vs. 256.23 ± 9.6 µm), superior (247.34 ± 18.07 µm vs. 265.10 ± 10.04 µm), nasal (249.50 ± 17.61 µm vs. 265.90 ± 11.59 µm), and inferior (245.92 ± 17.32 µm vs. 267.03 ± 9.28 µm), all $p < 0.0001$. Similarly, Mona Abdelkader et al. [73]observed a significant reduction in mean inner ring macular thickness in glaucomatous eyes (230 ± 15 µm) compared to normals (269 ± 20 µm), with $p = 0.001$

Similarly, in the outer macular ring, our study found that glaucomatous eyes exhibited significantly reduced thickness across all quadrants compared to normal eyes. The most pronounced reductions were again noted in the superior and nasal quadrants, with mean values of 229.65 µm versus 248.75 µm (superior), and 223.65 µm versus 249.10 µm (nasal) in glaucoma and control groups, respectively. These differences were statistically significant ($p < 0.001$) in all quadrants, reaffirming the presence of diffuse macular thinning in glaucomatous eyes. Supporting our results, Anjali Sharma et al.[72] reported consistent findings in the outer 6 mm macular region, where glaucoma patients (Group A) had significantly lower thickness than controls (Group B): temporal (199.18 ± 12.63 µm vs. 223.84 ± 14.58 µm), superior (216.85 ± 13.99 µm vs. 235.52 ± 13.18 µm), nasal (231.38 ± 17.79 µm vs. 259.90 ± 37.04 µm), and inferior (203.06 ± 23.79 µm vs. 230.68 ± 14.32 µm), all with $p < 0.0001$. Complementing these findings, Mona Abdelkader et al.[73] also observed a statistically significant reduction in outer ring macular thickness in glaucoma patients (230 ± 12 µm) compared to normals (245 ± 15 µm; $p = 0.002$). Collectively, these consistent observations highlight the diagnostic value of

evaluating outer macular thickness—particularly in the superior and nasal regions—for early detection and progression monitoring in glaucoma.

In our study, total macular thickness and volume were significantly reduced in glaucoma patients compared to normal controls, with mean macular thickness measuring 237.4 µm in glaucomatous eyes versus 245.0 µm in normal eyes.. Greenfield et al[75]. similarly demonstrated that macular thickness was significantly thinner in glaucomatous eyes. Leung et al.[76] reported notable reductions in both mean macular thickness (1–6 mm) and quadrant-specific thicknesses (1–3 mm) among glaucoma patients. Furthermore, Mona Abdelkader et al.[73] observed not only a significant reduction in total macular thickness but also found that macular nerve fiber layer (NFL) thickness had a stronger correlation with visual function ($R = 0.7$, $p = 0.02$) than total macular thickness ($R = 0.45$, $p = 0.05$). These combined findings emphasize the clinical utility of macular structural metrics—particularly macular thickness and NFL measurements—in assessing glaucomatous damage and its functional impact.

Our study demonstrated a significant reduction in total macular volume among glaucoma patients (mean: 6.26 mm^3) compared to normal subjects (6.65 mm^3), with a highly significant p-value (< 0.001), consistent with the growing body of evidence linking macular thinning to glaucomatous damage. Anjali Sharma et al.[72] similarly reported lower macular volumes in glaucoma patients (6.18 ± 0.39 mm^3) versus controls (6.64 ± 0.17 mm^3), while David E. Lederer et al[77] showed a stepwise decline in volume from normal (2.37 ± 0.11 mm^3) and early glaucoma eyes (2.27 ± 0.13 mm^3) to advanced glaucoma eyes (2.12 ± 0.23 mm^3), all with statistically significant differences. A. Giovannini et al[78] also observed significantly higher macular volumes in normal (7.35 ± 0.455 mm^3) and early glaucoma (7.09 ± 0.475 mm^3) eyes compared to advanced glaucoma cases (6.678 ± 0.455 mm^3). Pravda Chaturvedi et al.[79] found a statistically significant difference in macular volume between glaucomatous (7.44 ± 0.98

mm^3) and normal eyes (7.82 ± 0.54 mm^3) in a 6 mm scan diameter. Collectively, these findings—including ours—reinforce the utility of macular volume not only as a structural biomarker of disease severity but also as a potential predictor of glaucoma risk.

As expected, the mean intraocular pressure (IOP) in our study was significantly higher in glaucoma patients (20.4 mmHg) compared to normal subjects (12.5 mmHg), with a highly significant p-value (< 0.001). This elevated IOP was associated with reduced macular thickness, reinforcing the hypothesis that increased IOP contributes to retinal ganglion cell loss via both mechanical compression and vascular dysregulation. These results are consistent with those of Anjali Sharma et al.,[72] who reported a significantly higher mean IOP in glaucoma patients (23.22 mmHg) versus controls (14.45 mmHg), with a p-value of < 0.0001, supporting the central role of IOP in glaucomatous structural damage measurable through OCT. In contrast, Huiyuan Hou et al., [80]in a cohort of 213 subjects (287 eyes), found no significant differences in IOP or other systemic and ocular parameters among healthy, preperimetric, and early glaucoma subjects (all p > 0.1), suggesting that in earlier stages or more heterogeneous populations, factors other than IOP alone may contribute to glaucomatous changes. This underscores the importance of integrating structural imaging like OCT with IOP monitoring for comprehensive glaucoma assessment.

CONCLUSION

CONCLUSION

Our study demonstrates that macular thickness and volume are significantly reduced in glaucomatous eyes compared to normal controls, with the most pronounced thinning observed in the nasal and superior quadrants. These findings were consistent across both the inner and outer macular rings. Additionally, a strong association between elevated intraocular pressure and reduced macular parameters was observed, underscoring the role of mechanical and vascular stress in glaucomatous neurodegeneration. These results, supported by previous literature, affirm the value of OCT-derived macular measurements as sensitive and non-invasive biomarkers for early detection, diagnosis, and monitoring of glaucoma.

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SUMMARY

SUMMARY

Glaucoma is a chronic, degenerative visual neuropathy. With the help of optical coherence tomography (OCT), assessment of macula's structures can be carried out more thoroughly. A cross-sectional study was done to assess and compare the Total Macular Thickness (MT) and volume (MV) between normal and glaucomatous using OCT. The study was done among 20 POAG patients and 20 normal eyes, attending a tertiary eye care institute outpatient department of Ophthalmology at R.L.J Hospital and Research Centre, attached to Sri Devaraj Urs Medical College between June 2023 to August 2024, after obtaining the approval from Institutional Ethics Committee.

Mean age of normal and glaucoma patients was 58 and 57.1 years. Most of the participants in normal and glaucoma groups were males (55%). The mean Superior macular thickness (1-3) in normal and glaucoma patients were 253.20 µm and 236.15 µm.The mean Inferior macular thickness (1-3) in normal and glaucoma patients were 263.90 µm and 239.05 µm. The mean Nasal macular thickness (1-3) in normal and glaucoma patients were 269.35 µm and 244.90 µm.The mean temporal macular thickness (1-3) in normal and glaucoma patients were 256.90 µm and 237.75 µm. The mean Superior macular thickness (1-6) in normal and glaucoma patients were 248.75 µm and 229.65 µm.The mean Inferior macular thickness (1-6) in normal and glaucoma patients were 240.30 µm and 227.65 µm.The mean Nasal macular thickness (1-6) in normal and glaucoma patients were 249.10 µm and 223.65 µm. The mean temporal macular thickness (1-6) in normal and glaucoma patients were 226.55 µm and 211.05 µm. The mean Total Macular Thickness in normal and glaucoma patients were 245.00 µm and 237.40 µm.The mean Total Macular Volume in normal and glaucoma patients were 6.6 mm^3 and 6.26 mm^3

Macular thickness and volume were significantly lower in glaucomatous eyes in comparison with normal eyes in all quadrants. A strong association between elevated intraocular pressure and reduced macular parameters was observed, underscoring the role of mechanical and vascular stress in glaucomatous neurodegeneration.

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ANNEXURE- I

CASE PROFORMA

CASE PROFORMA	
Name:	Case No:
Age:	Date:
Sex:	OP No:
Occupation:	DOE:
Address:	

Chief complaints:

H/O presenting illness:

Past history:

DM / HTN / BA / Epilepsy

Family history:

Personal history:

Appetite – Sleep – Bowel –

Diet – Habits – Bladder –

GPE:

Pallor / Edema /Icterus / Cyanosis / Clubbing / Lymphadenopathy

Vital signs:

a. Pulse – c) RR –

b. BP – d) Temp –

Systemic examination:

a. CVS – c. RS –

b. PA – d. CNS –

OCULAR EXAMINATION		
TESTS	**RE**	**LE**
HEAD POSTURE OCULAR POSTURE FACIAL SYMMETRY		
EXTRAOCULAR MOVEMENTS Ductions Versions		
VISUAL ACUITY: Distant Near		
ANTERIOR SEGMENT		
INTRAOCULAR PRESSURE		
GONIOSCOPY		
FUNDUS Distant direct ophthalmoscopy Direct ophthalmoscopy Indirect ophthalmoscopy		
OCT 1.OMT 2.IMT 3.TMT 4.TMV		
HFA		

ANNEXURE- II

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

PATIENT INFORMATION SHEET

PATIENT INFORMATION SHEET

This information is to help you understand the purpose of the study THE ASSESSMENT OF MACULAR THICKNESS VARIABILITY IN PRIMARY OPEN ANGLE GLAUCOMA USING OPTICAL COHERENCE TOMOGRAPHY.

You will not be charged for any of the tests. All the tests are routine tests and absolutely no risks are associated with various investigations. We would like to get your consent to take part voluntarily in this research study, it is important that you read and understand the purpose, procedure, benefits and discomforts of the study.

1.What is the purpose of this study?

Is to study and compare macular thickness variability in glaucomatous eyes and normal eyes

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

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

- Slit lamp bio microscopy
- Posterior segment evaluation done by indirect ophthalmoscopy and or +90D bio-microscopy.
- Assessment of Intraocular pressure by Goldmann Applanation Tonometer
- Gonioscopy
- Macular thickness by Optical coherence tomography
- Field analysis done by white on white Humphrey Field Analyzer

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

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

CONFIDENTIALITY

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

You may refuse to take part in the study or you may stop your participation in the study at any time, without a penalty or loss of care in the future.

Extra monetary benefits or money will not be paid for taking part in the study.

For further information/ clarification please contact

For further information/ clarification please contact

Dr RAVEENA J (PH NO: 8921602069)

JUNIOR RESIDENT

DEPT OF OPHTHALMOLOGY,

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

ಶ್ರೀ ದೇವರಾಜ್ ಯುಆರ್ಎಸ್ ಅಕಾಡೆಮಿ ಆಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ ಅಂಡ್ ರಿಸರ್ಚ್, ತಮಕಾ, ಕೋಲಾರ್ - 563101

ರೋಗಿಯ ಮಾಹಿತಿ ಪತ್ರ

ಆಪ್ಟಿಕಲ್ ಕೊಹೆರೆನ್ಸ್ ಟೊಮೊಗ್ರಫಿಯನ್ನು ಬಳಸಿಕೊಂಡು ಪ್ರಾಥಮಿಕ ತೆರೆದ ಕೋನ ಗ್ಲುಕೋಮಾದಲ್ಲಿ ಮ್ಯಾಕ್ಯುಲರ್ ದಪ್ಪದ ವ್ಯತ್ಯಾಸದ ಮೌಲ್ಯಮಾಪನದ ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳಲು ಈ ಮಾಹಿತಿಯು ನಿಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ

ಯಾವುದೇ ಪರೀಕ್ಷೆಗಳಿಗೆ ನಿಮಗೆ ಶುಲ್ಕ ವಿಧಿಸಲಾಗುವುದಿಲ್ಲ. ಎಲ್ಲಾ ಪರೀಕ್ಷೆಗಳು ವಾಡಿಕೆಯ ಪರೀಕ್ಷೆಗಳು ಮತ್ತು ಸಂಪೂರ್ಣವಾಗಿ ಯಾವುದೇ ಅಪಾಯಗಳು ವಿವಿಧ ತನಿಖೆಗಳೊಂದಿಗೆ ಸಂಬಂಧ ಹೊಂದಿಲ್ಲ. ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಪಾಲ್ಗೊಳ್ಳಲು ನಿಮ್ಮ ಒಪ್ಪಿಗೆಯನ್ನು ಪಡೆಯಲು ನಾವು ಬಯಸುತ್ತೇವೆ, ನೀವು ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಕಾರ್ಯವಿಧಾನ, ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಅನಾನುಕೂಲಗಳನ್ನು ಓದುವುದು ಮತ್ತು ಅರ್ಥಮಾಡಿಕೊಳ್ಳುವುದು ಮುಖ್ಯವಾಗಿದೆ.

1. ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶವೇನು?

ಗ್ಲುಕೋಮಾಟಸ್ ಕಣ್ಣುಗಳು ಮತ್ತು ಸಾಮಾನ್ಯ ಕಣ್ಣುಗಳಲ್ಲಿ ಮ್ಯಾಕ್ಯುಲರ್ ದಪ್ಪದ ವ್ಯತ್ಯಾಸವನ್ನು ಅಧ್ಯಯನ ಮಾಡುವುದು ಮತ್ತು ಹೋಲಿಸುವುದು

2. ಯಾವ ವಿವಿಧ ತನಿಖೆಗಳನ್ನು ಬಳಸಲಾಗುತ್ತಿದೆ? ಯಾವುದೇ ಸಂಬಂಧಿತ ಅಪಾಯಗಳಿವೆಯೇ?

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಒಳಗೊಂಡಿರುವ ವಿವಿಧ ತನಿಖೆಗಳೊಂದಿಗೆ ಸಂಪೂರ್ಣವಾಗಿ ಯಾವುದೇ ಅಪಾಯಗಳು ಸಂಬಂಧಿಸಿಲ್ಲ

• ಸ್ಲಿಟ್ ಲ್ಯಾಂಪ್ ಬಯೋ ಮೈಕ್ರೋಸ್ಕೋಪಿ

• ಪರೋಕ್ಷ ನೇತ್ರದರ್ಶಕ ಮತ್ತು ಅಥವಾ +90D ಜೈವಿಕ ಸೂಕ್ಷ್ಮದರ್ಶಕದಿಂದ ಹಿಂಭಾಗದ ವಿಭಾಗದ ಮೌಲ್ಯಮಾಪನವನ್ನು ಮಾಡಲಾಗುತ್ತದೆ.

• ಗೋಲ್ಡ್‌ಮನ್ ಅಪ್ಲನೇಷನ್ ಟೋನೋಮೀಟರ್‌ನಿಂದ ಇಂಟ್ರಾಕ್ಯುಲರ್ ಒತ್ತಡದ ಮೌಲ್ಯಮಾಪನ

• ಗೊನಿಯೊಸ್ಕೋಪಿ

• ಆಪ್ಟಿಕಲ್ ಕೊಹೆರೆನ್ಸ್ ಟೊಮೊಗ್ರಫಿಯಿಂದ ಮ್ಯಾಕ್ಯುಲರ್ ದಪ್ಪ

• ಬಿಳಿ ಹಂಫ್ರಿ ಫೀಲ್ಡ್ ವಿಶ್ಲೇಷಕದಲ್ಲಿ ಬಿಳಿಯರಿಂದ ಮಾಡಿದ ಕ್ಷೇತ್ರ ವಿಶ್ಲೇಷಣೆ

3. ಭಾಗವಹಿಸುವವನಾಗಿ ನನಗೆ ಏನು ಪ್ರಯೋಜನ?

ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆಯು ನಿಮ್ಮ ಕಣ್ಣಿನ ಸ್ಥಿತಿಯ ಅಂತಿಮ ಫಲಿತಾಂಶವನ್ನು ಬದಲಾಯಿಸದಿರಬಹುದು. ಆದಾಗ್ಯೂ, ಈ ಅಧ್ಯಯನದಿಂದ ಪಡೆದ ಜ್ಞಾನದ ಪರಿಣಾಮವಾಗಿ ಭವಿಷ್ಯದಲ್ಲಿ ರೋಗಿಗಳು ಪ್ರಯೋಜನ ಪಡೆಯಬಹುದು. ಸಂಶೋಧನಾ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಡೆಸಿದ ಯಾವುದೇ ಕಾರ್ಯವಿಧಾನಗಳಿಗೆ ನಿಮಗೆ ಹೆಚ್ಚುವರಿ ಶುಲ್ಕ ವಿಧಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನೀವು ಭಾಗವಹಿಸುವುದು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಮೊದಲು ನೀವು ಅರ್ಹರಾಗಿದ್ದ ಯಾವುದೇ ಪ್ರಯೋಜನಗಳ ದಂಡ ಅಥವಾ ನಷ್ಟವಿಲ್ಲದೆಯೇ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಗೌಪ್ಯತೆ

ನಿಮ್ಮ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯನ್ನು ಅಧ್ಯಯನ ವೈದ್ಯರು ಮತ್ತು ಸಿಬ್ಬಂದಿ ಗೌಪ್ಯವಾಗಿಡುತ್ತಾರೆ ಮತ್ತು ಸಾರ್ವಜನಿಕವಾಗಿ ಲಭ್ಯವಾಗುವಂತೆ ಮಾಡಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮೂಲ ದಾಖಲೆಗಳನ್ನು ನಿಮ್ಮ ವೈದ್ಯರು ಅಥವಾ ಎಥಿಕ್ಸ್ ರಿವ್ಯೂ ಬೋರ್ಡ್ ಪರಿಶೀಲಿಸಬಹುದು.

ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಭವಿಷ್ಯದಲ್ಲಿ ಯಾವುದೇ ದಂಡ ಅಥವಾ ಕಾಳಜಿಯ ನಷ್ಟವಿಲ್ಲದೆಯೇ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಿದ್ದಕ್ಕಾಗಿ ಹೆಚ್ಚುವರಿ ವಿತ್ತೀಯ ಪ್ರಯೋಜನಗಳು ಅಥವಾ ಹಣವನ್ನು ಪಾವತಿಸಲಾಗುವುದಿಲ್ಲ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿ / ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ದಯವಿಟ್ಟು ಸಂಪರ್ಕಿಸಿ

ಹೆಚ್ಚಿನ ಮಾಹಿತಿ / ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ದಯವಿಟ್ಟು ಸಂಪರ್ಕಿಸಿ

ಡಾ.ರವೀಣಾ.ಜೆ

ನೇತ್ರಶಾಸ್ತ್ರ ವಿಭಾಗದಲ್ಲಿ ನಿವಾಸಿ

SDUMC, ಕೋಲಾರ -563101

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 8921602069

ANNEXURE- III

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

INFORMED CONSENT FORM

Case no:

IP no:

TITLE: THE ASSESSMENT OF MACULAR THICKNESS VARIABILITY IN PRIMARY OPEN ANGLE GLAUCOMA USING OPTICAL COHERENCE TOMOGRAPHY

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

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

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

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

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

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

ಶ್ರೀ ದೇವರಾಜ್ ಯುಆರ್ಎಸ್ ಅಕಾಡೆಮಿ ಆಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ ಅಂಡ್ ರಿಸರ್ಚ್, ತಮಕಾ, ಕೋಲಾರ್ - 563101.

ಮಾಹಿತಿ ಕಾನ್ಸೆಂಟ್ ಫಾರ್ಮ್

ಪ್ರಕರಣ ಸಂಖ್ಯೆ: ಐಪಿ ಸಂಖ್ಯೆ:

<u>ಶೀರ್ಷಿಕೆ:</u> ಆಪ್ಟಿಕಲ್ ಕೋಹೆರೆನ್ಸ್ ಟೊಮೊಗ್ರಫಿಯನ್ನು ಬಳಸಿಕೊಂಡು ಪ್ರಾಥಮಿಕ ತೆರೆದ ಕೋನ ಗ್ಲುಕೋಮಾದಲ್ಲಿ ಮ್ಯಾಕ್ಯುಲರ್ ದಪ್ಪದ ವ್ಯತ್ಯಾಸದ ಮೌಲ್ಯಮಾಪನ

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

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

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

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

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆ ನನಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚ ಒಳಗೊಳ್ಳುವುದಿಲ್ಲ.

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

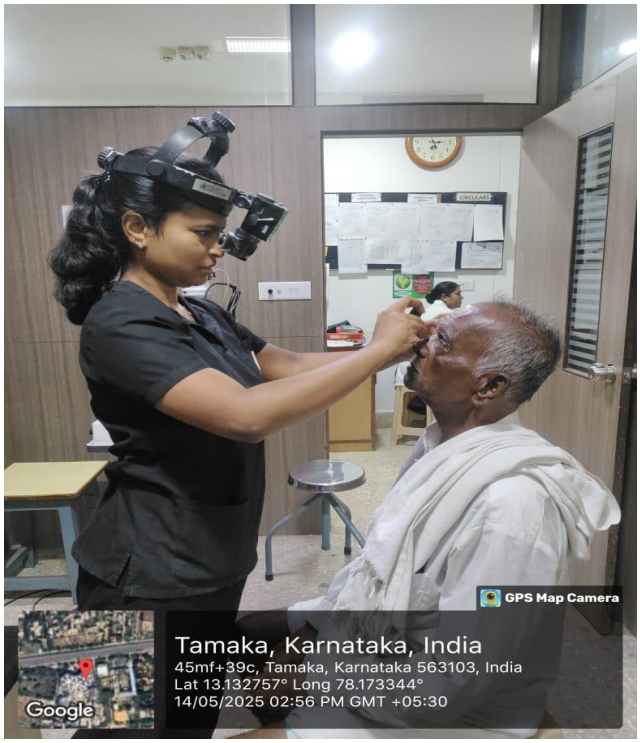
ANNEXURE- IV

PHOTOGRAPHS

PHOTOGRAPH 1 – SLIT LAMP EXAMINATION

PHOTOGRAPH 2: FUNDUS EXAMINATION BY 90 D LENS

PHOTOGRAPH 3: FUNDUS EXAMINATION BY IDO



PHOTOGRAPH 4: OCT EXAMINATION

ANNEXURE-V

KEY TO MASTER CHART

N- Normal

G- Glaucoma

SMT- Superior macular thickness

IMT- Inferior macular thickness

TMT-Temporal macular thickness

NMT- Nasal macular thickness

TMV- Total macular volume

IOP- Intraocular pressure

MASTER CHART

.

1	N/G	NAME	AGE	SEX	SMT(1-	IMT(1-3)	NMT1-3)	TMT(1-3)	SMT(1-6)	IMT1-6)	NMT(1	TMT(1-	TMV	IOP
2	N	shilpa	48	f	266	270	275	264	256	245	255	230	6.6	12
3	N	manjunatha	56	m	268	275	274	260	255	246	256	232	6.8	14
4	N	bhagyamma	60	f	260	268	270	258	252	240	253	228	6.65	16
5	N	ramappa	58	m	266	274	272	262	254	242	253	230	6.77	14
6	N	changamma	49	f	265	272	278	266	254	248	260	238	6.6	12
7	G	shivakumar	54	m	236	244	256	246	233	230	228	215	6.1	18
8	G	balappa	63	m	230	238	250	240	230	226	220	200	6.2	20
9	G	narasappa	61	m	232	237	251	242	230	224	220	204	6.33	16
10	G	chikkalappa	58	m	234	240	252	244	234	230	226	208	6.12	16
11	N	nagarathnamma	62	f	234	240	252	242	232	226	228	210	6.2	10
12	N	nagaraja	64	m	255	270	275	264	256	245	255	230	6.6	12
13	N	parvathamma	58	f	266	275	274	260	255	246	256	232	6.7	14
14	N	jayamma	55	f	268	268	270	258	252	240	253	228	6.8	12
15	N	suggaraju	56	m	260	274	272	262	254	242	253	230	6.7	10
16	N	babu	62	m	266	272	278	266	254	248	260	238	6.6	14
17	N	chandrashekaraih	60	m	265	244	256	246	233	230	228	215	6.9	12
18	N	lalithamma	54	f	236	238	250	240	230	226	220	200	6.7	12
19	G	papachamma	55	f	230	237	251	242	230	224	220	204	6.8	18
20	G	radhamma	56	f	232	240	252	244	234	230	226	208	6.3	20
21	N	peddaredappa	62	m	234	240	252	242	232	226	228	210	6.2	12
22	N	gangama	61	f	234	270	275	264	256	246	256	232	6.7	10
23	N	venkateshappa	60	m	255	275	274	260	255	240	253	228	6.6	14
24	N	seethamma	58	f	266	268	270	258	252	242	253	230	6.7	16
25	G	chinnamma	56	f	268	274	272	262	254	248	260	238	6.77	22
26	N	ramakrishnappa	60	m	232	240	270	242	232	246	256	232	6.8	10
27	N	chinnayallappa	60	m	234	270	272	264	256	240	253	228	6.6	12
28	N	nanjappa	57	m	234	275	278	260	255	242	253	230	6.7	12
29	G	rathnamma	58	f	255	268	256	258	252	248	260	238	6.7	18
30	G	reddappa	55	m	244	274	250	262	254	230	228	215	6.1	20
31	G	saraswathamma	56	f	268	272	251	266	254	226	220	200	6.2	22
32	G	sugumar	58	m	232	210	220	210	210	226	222	220	6.2	20
33	G	nagamma	55	f	230	215	225	220	206	230	210	222	6.1	24
34	G	venkateshappa	57	m	240	220	236	222	225	220	202	210	6.1	20
35	G	hanumanthappa	56	m	236	230	240	230	220	224	220	212	6.2	18
36	G	pappanna	55	m	220	234	255	232	212	230	230	202	6.3	22
37	G	ashwathamma	59	f	210	235	230	228	214	234	235	200	6.1	24
38	G	samakka	58	f	220	212	245	218	228	210	216	205	6	24
39	G	gurappa	56	m	228	214	250	238	228	218	218	203	6.2	22
40	G	somashekar	56	m	238	249	236	235	230	220	202	207	6.1	20
41	G	rathanamma	60	f	240	238	220	216	215	225	210	210	6.3	24