COMPARITIVE STUDY OF OPTICAL COHERENCE TOMOGRAPHIC ANALYSIS OF MACULA IN PREOPERATIVE AND POSTOPERATIVE DIABETIC PATIENTS UNDERGOING SMALL INCISION CATARACT SURGERY

BY

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LIST OF ABBREVIATIONS

BCVA	Best Corrected Visual Acuity
CME	Cystoid macular edema
CSME	Clinically Significant Macular Edema
CSMT	central sub field macular thickness
CME	Cystoid macular edema
DME	Diabetic Macular Edema
DR	Diabetic Retinopathy
DRT	Diffuse retinal thickening
ETDRS	Early Treatment Diabetic Retinopathy Study
FT	foveal Thickness
FA	Fluorescein Angiography
HTN	Hypertension
ID	Insulin Dependent
LogMAR	Logarithm of Minimum Angle of Resolution
ME	Macular Edema
NID	Non-insulin dependent
NPDR	Non Proliferative Diabetic Retinopathy.
OCT	Optical Coherence Tomography
PHT	Posterior Hyaloid traction
PDR	Proliferative Diabetic Retinopathy
SD	Standard Deviation
VA	Visual Acuity
SRD	Serous retinal detachment

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ABSTRACT

BACKGROUND

Diabetic Macular Edema (DME) is the Micro vascular complication of diabetes which leads to loss of vision. The underlying pathogenesis is disruption of inner blood retinal barrier which leads to fluid and lipid accumulation in the central part of retina. Which is seen clinically as increase in thickness of macular area with hard exudate formation. Diabetic patients pose a great challenge when there is diabetic retinopathy along with cataract which causes severe visual morbidity. So the risk of developing vision loss also depends upon the grade of diabetic retinopathy (mild, moderate, severe) after cataract surgery.

After surgical insult there is prostaglandin release and its association with preexisting diabetic retinopathy which might be the cause of macular edema after cataract surgery.

The relative increase in thickness of macula which is difficult to detect clinically.

OCT which a noninvasive modality particularly useful in measuring the macular thickness.

Hence this study is undertaken to measure the macular thickness in diabetic patients who are undergoing cataract surgery using macular analysis protocols scans on Cirrus HD OCT.

AIM AND OBJECTIVE OF THE STUDY

To compare and analyze pre-operative and post-operative OCT changes in macula of diabetic patients undergoing cataract surgery.

Methods: This was a hospital based prospective comparative study conducted from October 2018 to April 2020. Study participants are 65 diabetic patients undergoing cataract surgery. Each eye underwent fundus examination with indirect

ophthalmoscopy with 20D lens before surgery. If any changes are seen fundus photography is taken. Optical coherence tomography testing was performed before surgery i.e. preoperatively and at postoperatively at day 1, 1 week, 4 weeks and at 12 weeks. Best-corrected visual acuity (BCVA) was recorded at each visit.

Results: Out of 65 Patients, 59 patients (90.7%) completed the 3 months follow-up 59 patients (90.7%) completed the 3 months follow-up. mean age of the study population was 66.0 ±7.8years. 36 patients (61%) were males and 23 (39%) were males. Mean Age duration of DM is 4.8±2.9 yrs. No Diabetic retinopathy was in 26 patients.18 patients had Mild NPDR. 8 patients had Moderate NPDR. 4 patients had moderate NPDR with ME, Severe NPDR with macular edema in 1 patient and pre op macular edema is seen in 2 patients. By Postoperative 3 months 89.8% patients achieved a visual acuity of 6/6 – 6/12. The central subfield macular thickness increased in all patients irrespective of presence or absence of diabetic retinopathy of about 17.4±25.3µm and 29µm±38.8 at 1 month and 3 month follow up. Eyes with no diabetic retinopathy developed thickening of 15.2±µm and 21.2µm, the group with mild NPDR had increase in foveal thickness of 4.4±4µm and 8.4±4.4um, the group with moderate non proliferative diabetic retinopathy with macular edema had largest increase in foveal thickness of about 57.5±36.7um and 135±68.4µm at 4th & 12th week after surgery respectively. This increase in foveal thickness was correlated inversely with VA improvement in pre-op macular edema patients

Conclusions: There was a statistically significant increase seen in CSMT after cataract surgery especially in patients with preoperatively diagnosed macular edema. Associated retinopathy also acts as a risk factor. But there was no statistically significant increase in mild and moderate NPDR preoperatively and also in postoperative period after uncomplicated small incision cataract surgery

In patients with no DR Macular thickness changes are Subclinical. We implicate that influence of cataract surgery and the release of inflammatory mediators cause blood retinal barrier breakdown that could affect the thickness measurements.

INTRODUCTION

Cataract is a common cause of visual impairment in patients with diabetes. Cataract surgery is indicated when the visual function is significantly reduced as a result of the lenticular opacity or if the cataract reduces the view of the retina, thus impeding the diagnosis and treatment of diabetic retinopathy.

Cataract surgery is known to lead to increased levels of inflammatory mediators. Clinical studies are inconclusive as to the effect of cataract surgery on the onset of diabetic macular oedema (DMO) [(1)]

Macular oedema (ME) occurs in a variety of pathological conditions and accounts for different degrees of vision loss. Early detection of ME is therefore critical for diagnosis and management

A higher incidence of ME after cataract surgery is reported to occur in eyes with diabetic retinopathy (DR), and worsening of ME often occur after surgery in eyes with pre-operative diabetic macular edema (DME) [(2)]

The clinical evaluation of macular edema has been difficult to characterize, but evaluation has become more precise with the help of modern imaging such as FA and optical coherence tomography (OCT).

OCT is a method of analysing the in-vivo retinal architecture. It is particularly useful and accurate for measuring retinal nerve fibre layer (RNFL) thickness and macular thickness.

Need for the study: This study is undertaken to evaluate the quantitative changes in macular thickness using spectral domain optical coherence tomography in diabetic

patients undergoing cataract surgery pre and post operatively and its relation with diabetic retinopathy.

AIMS AND OBJECTIVE OF THE STUDY

To compare and analyze pre-operative and post-operative OCT changes in macula of diabetic patients undergoing cataract surgery.

REVIEW OF LITERATURE

The number of persons with diabetes worldwide is predicted to grow to 429 million by 2030, owing to the rising frequency of obesity, increasing life span, and improved detection of the disease[3][4]. In India, an estimated 32 million persons had diabetes in 2000, and roughly 79 million will be affected by 2030[5]. If the prevalence of complications remains unchanged, approximately 0.7 million Indians will have proliferative diabetic retinopathy and 1.8 million will have clinically significant macular edema[5].

Chronic hyperglycemia leads to the production of advanced glycation end products, increased oxidative stress, and increased activation of the polyol pathway, each of which has been implicated in the development of cataracts.

As a result, cataract develops and progresses more frequently, rapidly and at an earlier age in patients with diabetes[6].

The risk of cataract development in DM is fivefold higher than in the general population, and cataract is diagnosed twice as frequently in diabetic subjects[7]

At present, cataract surgery is the most commonly performed surgical procedure and diabetics represent a large percentage of patients undergoing cataract surgery.

Macular edema is an expected complication after cataract surgery[8] which can significantly decrease visual acuity.

Cataract surgery has been shown to cause release of inflammatory mediators into the vitreous and/or aqueous, with posterior diffusion that could precipitate a change in the macular anatomy[9].

Even a cataract surgery without complications may cause postsurgical inflammation and vitreous instability that may follow postoperative macular edema in normal individuals[10].

Background Cystoid macular edema (CME) is a well-known complication after cataract surgery, and diabetic retinopathy is reported to be an important risk factor for impaired visual recovery it is often difficult to distinguish cystic macular changes caused by surgery from edema or it is due to pre-existing diabetic maculopathy[11]

In patients with diabetic retinopathy, the blood–retinal barrier is often disrupted to a certain degree, which may cause them more susceptible to develop postoperative macular edema[12].

Pseudophakic cystoid macular edema (PCME), which is thought to be caused by Proinflammatory cytokine release[13].

In diabetic macular edema (DME), ME is induced by hyperglycemia-induced oxidative stress, deposition of advanced glycation end products (AGES), impaired blood flow, hypoxia, pericyte loss, endothelial cell loss, up regulation of vesicular transport, down regulation of glial cell–derived neurotropic factor and inflammation.

It has been shown that Diabetic Retinopathy progresses in almost 10–30% of patients after cataract surgery[14][15] although some studies have denoted that the progression of DR after cataract surgery is due to the natural course of the disease process, and that the progression is not associated with surgery[16][17] The most important predictor for progression of DR is based on the type and grade of the DR at the time of the cataract surgery[18].

Subtle macular changes are not noticed, especially when cataract is present[19].

There are different conventional methods for assessment of retina and macula which includes slit lamp bio-microscopy, indirect ophthalmoscopy, fluorescein angiography and fundus stereo-photography. SD-OCT is a new modality that allows excellent visualization of vitreomacular interface and intra retinal pathologies thus enabling us to study the vitreomacular abnormalities with high precision[20]

In a study conducted by Urban Eriksson, Macular Changes on OCT or FA are often seen without any obvious effect on VA. OCT is as good as FA at detecting a clinical CME, and is the technique recommended for follow-up before FA is considered[11].

Angiographic CME is diagnosed in patients who are otherwise asymptomatic with respect to visual acuity, but have detectable leakage from the Perifoveal capillaries on fluorescein angiography (FA). Clinical CME is diagnosed in those patients who have detectable visual impairment as well as angiographic and/or Bio microscopic findings. The clinical diagnosis has to be confirmed using optical coherence tomography (OCT) and FA[21].

A prospective study of patients evaluated by SD-OCT by Carlos showed that the incidence of ME found via OCT testing is 22% in the eyes of diabetic patients undergoing cataract surgery On the other hand, DR is known to progress in 10-30% of patients undergoing phacoemulsification[19].

NIDDM patients in particular appear to be at increased risk of developing macular oedema after cataract surgery, implying that postoperative follow- up should last at least 3 to 6 months as the few macular edemas reported and appeared after more than 3months observation[22].

The advent of spectral domain optical coherence tomography (SD-OCT) allowed axial imaging of the retina with a great level of detail, but visualization of the choroid was still difficult because of light dispersion induced by the retinal pigment epithelium. Such constraint was surpassed when Spaide et al described the "enhanced depth imaging" technique, which allowed reproducible and comparable choroidal imaging[9].

In a study which was done to assess the influence of cataracts on SD-OCT scans, to determine and to evaluate preoperative and postoperative macular and RNFL thickness measurements in healthy subject undergoing cataract surgery. It was found that presence of cataracts affects RNFL and macular measurements performed with SD-OCT. The repeatability of the images significantly improved after cataract phacoemulsification when using the Cirrus OCT[23].

Hayashi et al showed that the degree of diabetic macular edema generally increases after cataract surgery, and that the worsening of macular edema is more prominent in eyes with DR than in eyes without DR at the time of surgery. However, macular edema that occurs after cataract surgery resolves spontaneously in some patients for up to a year.

They quantitatively found two types of macular edema. That is, macular thickening generally occurs after cataract surgery, but this resolves with time in most diabetic patients. Another one is in some diabetic patients, diabetic maculopathy actually progresses. Both types of macular change are more prominent in eyes with DR than in eyes with no DR[18].

According to DRCR network (Protocol Q), the probability of developing central involved PME after cataract surgery in patients without preoperative DME is

extremely low. However, presence of non-central DME immediately prior to cataract surgery or history of DME treatment may significantly increase the chance of developing PME in these patients[(2)].

Hence it was said from the study was Clinicians should continue to maintain vigilance in diabetic patients after cataract extraction even when central ME is not present immediately prior to cataract surgery, especially in eyes with prior DME treatment or non–central-involved DME that may be at particularly high risk for development of central involved ME after cataract surgery.[(2)]

The UK Diabetic Retinopathy Electronic Medical Record (UK DR EMR) Users Group, Report 2 which is a Multicenter national diabetic retinopathy (DR) database study with data extraction across 19 centers from an electronic medical record system in which diabetic patients who are undergoing cataract surgery with no history of DMO prior to study start where the rate of 'treatment-requiring diabetic macular oedema (DMO)'is assessed for two years before and after cataract surgery. This large real-world study demonstrated that the rate of developing treatment-requiring DMO increases sharply in the year after cataract surgery for all grades of retinopathy, peaking in the 3–6 months postoperative period. Patients with moderate and severe NPDR are at particularly high risk[(1).]

It is commonly recognized that the clinical assessment of ME may be subjective and variable[(24)]. According to earlier reports, stereoscopic fundus examination usually only detects retinal thickening of ≈100 μm. contact lens bio microscopy is relatively insensitive for detection of mild foveal thickening which is apparent by OCT[(24)]

FA, which is known to be a sensitive method for the qualitative assessment of fluid leakage. But the interpretation of the results may be subjective. Actual macular

thickening is better correlated with loss of visual acuity. Ozdek et al found that the sensitivity of OCT was higher than that of FA, especially for the cystoid pattern of ME[24].

In a single center study of 50 eyes, Kim et al reported an incidence rate of 22% (95% CI, 13%-35%) for DME exacerbation (defined as ≥30% increase in OCT center-point thickness compared with presurgical OCT) 1 month after cataract surgery[25].

OCT is a new method for high-resolution, cross-sectional imaging that directly and reproducibly measures macular thickness. OCT has become an invaluable diagnostic tool to assess macular disease and offers several advantages over the gold standard of FA, including patient safety, comfort, and speed[26].

Simultaneous cataract surgery and intravitreal bevacizumab may play a role in the management of patients with cataract and diabetic macular edema[9].

In a study done at KFSH&RC Mean MFT preoperatively, 2 weeks, 1 month and 2 months postoperatively in diabetic patients with diabetic retinopathy (mild, moderate and proliferative DR) was statistically higher compared to patients without DR. There were statistical improvement of visual acuity after 1 and 2 month postoperatively in patient with no DR than those with DR. it reveals that disruption of inner blood retinal barrier may be the cause for more advanced vascular changes resulting from DR[27].

The prophylactic use of non-steroidal anti-inflammatory drugs (NSAIDs) preoperatively, and the combination of steroids and NSAIDs in the postoperative period, is recommended to reduce the incidence of pseudophakic CME[21].

A Prospective study carried out by Sam Buddha Ghosh, on patients who are without complications to measure post cataract surgery macular thickness on OCT the increase in macular thickness was sub-clinical and did not affect final visual outcome

in any patient. And it shows that in spite of the greater chance of iris and lens manipulation and theoretical risk of increased postoperative inflammation, there is no evidence of CME on OCT following MSICS[28].

Degenring et al conducted a study and compared the post phacoemulsification macular changes and visual outcomes between diabetic and non-diabetic eyes for 4 weeks after cataract surgery. Although they did not find a significant difference in macular thickness on OCT between the groups preoperatively and all time points postoperatively, they did find a trend toward a more prominent increase in foveal thickness in the diabetic eyes than the non-diabetic eyes at postoperative Week 4 ($p = 0.058$)[29].

It seems that patients with moderate to severe NPDR and/or macular edema prior to phacoemulsification are more likely to develop subsequent persistent macular edema or progression of diabetic retinopathy following surgery compared to patients with no or mild NPDR initially[30]

Khodabandeh et al showed that intravitreal injection of Bevacizumab during phacoemulsification would result in decreased macular thickness in patients with no diabetic retinopathy or NPDR and without macular edema in the early post-operative period, this effect would no longer persistent at 3 months[31].

A study conducted by Joanna Gołębiewska There is a significant increase in macular parameters after uneventful phacoemulsification. Despite downward trend, they remain elevated throughout the 6-month observation period. Diabetes and hypertension increase the risk of postoperative changes in macular thickness, especially if they coexist. Higher phaco power increases the risk of retinal thickening after cataract surgery. Early macular evaluation using the optical coherence tomography identifies patients at high risk of complications, who might benefit from

additional anti-inflammatory treatment[32]. The lack of preoperative OCT measurement is a limitation of this study.

Reliable, reproducible measurements of retinal thickness using OCT require obtaining high quality images, in which the external and internal retinal borders can be identified. This is difficult in most patients with cataract, especially subcapsular cataract, where scans are unreliable. cataract causes artifacts, which also renders the measurements less reliable [32,33].

Further literature review describes the anatomy and physiology of the retina and macula and the diagnostic tools to evaluate the structure, function of the macula. In addition, it provides the up-to-date findings on pathophysiology of diabetic eye disease, macular edema and discusses current diagnostic modalities mainly optical Coherence tomography and its scanning protocols.

SURGICAL ANATOMY

ANATOMY OF THE RETINA

The retina lines the inner surface of the eye with neuronal connections to the optic nerve and eventually to the central nervous system. It is a layered structure with neurons and interconnected synapses with principal light-sensitive cells at its outer aspect in the photoreceptor layer, containing rods and cones. There are approximately 6 million cones, most densely packed within the fovea and 125,000,000 rods spread predominantly throughout the peripheral retina.

The sensory retina extends to the oraserrata, where it is continuous with the non-pigmented ciliary epithelium of the parsplana. The oraserrata is 2.1 mm wide temporally and 0.7–0.8 mm wide nasally. It is located more anteriorly on the nasal

than on the temporal side. The nasal ora is about 6 mm posterior to the limbus, and the temporal ora is about 7 mm posterior to the limbus. The average distance from the oraserrata to the optic nerve is 32.5 mm temporally and 27 mm nasally, and 31 mm superiorly and inferiorly. The retina itself is a thin transparent tissue, which is thickest near the optic nerve, where it measures 0.56 mm. It thins to 0.18 mm at the equator and to 0.1 mm at the oraserrata. At the foveal area, it has thinned to about 0.2 mm. The nerve fiber layer increases at the edge of the disc and is the only retinal structure that continues into the disc to become the optic nerve. The sensory retina is composed of nine contiguous layers, linked to each other by synaptic connections between axons and dendrites in the inner and outer plexiform layers and to the ganglion cells[(34)]. The layers of the retina can be seen easily in cross-sectional histologic preparations[(35)]in order from the inner to outer retina (fig1)

- Internal limiting membrane (ILM)
- Nerve fiber layer (NFL; the axons of the ganglion cell layer)
- Ganglion cell layer
- Inner plexiform layer
- Inner nuclear layer
- Outer plexiform layer
- Outer nuclear layer (the nuclei of the photoreceptors)
- External limiting membrane (ELM)
- Rod and cone inner and outer segments

The **retinal pigment epithelium (RPE)** is a monolayer of pigmented cells located between the light-sensitive photoreceptor outer segments and the fenestrated endothelium of the choriocapillaris.[(36)] The RPE extends from the optic nerve to the oraserrata, and is continuous with the ciliary body pigment epithelium. The fovea

contains the highest density of RPE cells, with a progressive decrease in density to the periphery. Their apical microvilli interdigitate with photoreceptor outer segments, while their basal side attaches to the RPE basement membrane of Bruch's membrane. The RPE, with its brown melanin granules gives typical patterned fundus due to variations in RPE pigmentation. Typically, the highest concentration of pigment in the RPE is found in the peripheral retina, while the lowest is in the macula and the cells are tall and narrow. With age, the RPE gradually loses melanin granules, in part from the effects of photo-oxidation. The retina and RPE are separated by a potential space known as the sub retinal space. Every RPE cell is in diffusion through the para-cellular spaces and interaction with a mean of 23 photoreceptors. The RPE cells form a part of the blood–retina barrier. The RPE cells are connected by apical zonulae occludens (tight junctions) and adjacent zonulae adherens (adherens junctions), which form a barrier that regulates trans-epithelial transport[37].

NEUROSENSORY RETINA

Rod and Cone (Photoreceptor) Layer

There are **two** types of photoreceptor: rods and cones. Rods account for 95% of all photoreceptors. They are numerous, with slender outer segments, densely packed and specialized for high sensitivity under dark conditions.

Cones are larger, with tapering outer segments, and they are found in the top row of the ONL Cones make up only ~5% of photoreceptors but they provide high-acuity color vision in daylight conditions when photons are abundant. A lack of color vision is the hallmark of rod-mediated vision. There is a massive peak at the fovea where the density reaches around 200,000/mm2, approximately 100 times the density in the

periphery. Rods are absent within 350μm of the fovea but reach a peak density in an annular region at about 20° eccentricity cones are not evenly distributed.

Both rod and cone photoreceptors are elongated cells that have four subcellular compartments: the outer segment (OS), the inner segment (IS), the nucleus, and the synaptic terminal. The OS is where photons are captured and activation of the photo transduction cascade begins. The IS lies immediately proximal to the OS, and contains the cell's protein synthesis (Golgi apparatus and endoplasmic reticulum) and metabolic (mitochondria) machinery.

External limiting membrane

Zonular attachment between photoreceptors and Muller cells at this level creates the ELM, a structure visible by light microscopy**.** It separates the photoreceptor layer from the outer nuclear layer. The Muller cells course through almost the entire thickness of the retina.

The **outer nuclear layer (ONL)** contains the cell bodies of photoreceptors, both rods and cones. The inner nuclear layer (INL) contains the cell bodies of horizontal, bipolar, amacrine, and radial glial (or Müller) cells. The ganglion cell layer (GCL) contains displaced amacrine and ganglion cells. Ganglion cells are the projection neurons of the retina: their axons form the optic nerve and project to a variety of subcortical nuclei. The three nuclear layers are separated by two synaptic (plexiform) layers that contain the dendrites and synapses. The outer plexiform layer (OPL) lies between the ONL and the INL. This is where the photoreceptors, horizontal, and bipolar cell dendrites interact. The inner plexiform layer (IPL) separates the INL and the GCL and this is where the bipolar cell axons, amacrine, and ganglion cells interact. Bipolar cells take the signals from photoreceptors and transmit them to the

inner retina. Horizontal cells and amacrine cells are laterally extensive interneurons in the outer and inner retina, respectively. Ganglion cells receive input from bipolar and amacrine cells and form the output from the retina.[37]

Outer Plexiform Layer (OPL)

The terminations of the photoreceptor axons Cone Pedicles and Rod Spherules make contact with bipolar and horizontal cells in the OPL. Cone axons descend through the massed ranks of rod somas in the ONL to terminate in a two-dimensional array of cone pedicles at the OPL.

Inner Nuclear Layer (INL)

The INL is subdivided into the horizontal cell layer (HCL), bipolar cell layer (BCL), Müller cell layer (MCL) and amacrine cell layer (ACL)[36]. The bipolar cell represents the first order neuron and transmits the nerve impulse from the photoreceptors to the ganglion cells. The horizontal and amacrine cells have long branches which extend horizontally and probably act as integrating circuits. The glial Müller cells serve primarily a supportive and nutritive function, although they may participate in an as yet unknown way in transmission or modification of nerve impulses.

Inner Plexiform Layer (IPL)

This layer consists of synapses between axons of bipolar cells (first order neurons), dendrites of ganglion cells (second order neurons) and the process of integrative amacrine cells.

Fibers from Muller cells course vertically through this layer and their side branches form the horizontal extending reticulum. This layer is absent at foveola.

Ganglion Cell Layer (GCL)

The cell bodies and the nuclei of ganglion cells (second order neurons of visual pathway) lie in this layer throughout most of the retina, the ganglion cell layer is composed of single row of cells, expect in the macular layer where it becomes multilayered (6-8 layers of cells) and on the temporal side of the disc where it has 2 layers. GCL is absent at the foveola.

NERVE FIBRE LAYER

It consists of the unmyelinated axons of ganglion cells which converge at the optic nerve head, pass through lamina cribrosa and become ensheathed by myelin posterior to lamina.

Fibers from the nasal half of retina come directly as superior and inferior radiating fibers. Fibers in macular region pass straight in temporal part of the disc as papillomacular bundle. Fibers from the temporal retina arch above and below the macula and papillomacular bundle as superior and inferior arcuate fibers with a horizontal raphe in between.

Internal Limiting Membrane (ILM)

The ILM mainly consists of PAS positive true basement membrane that forms the interface between retina and vitreous. Muller cells play a role in its formation. The vitreal surface of the ILM is even but its retinal side is irregular, conforming to the irregularity of the basal plasma membranes (footplates) of the Muller cells.

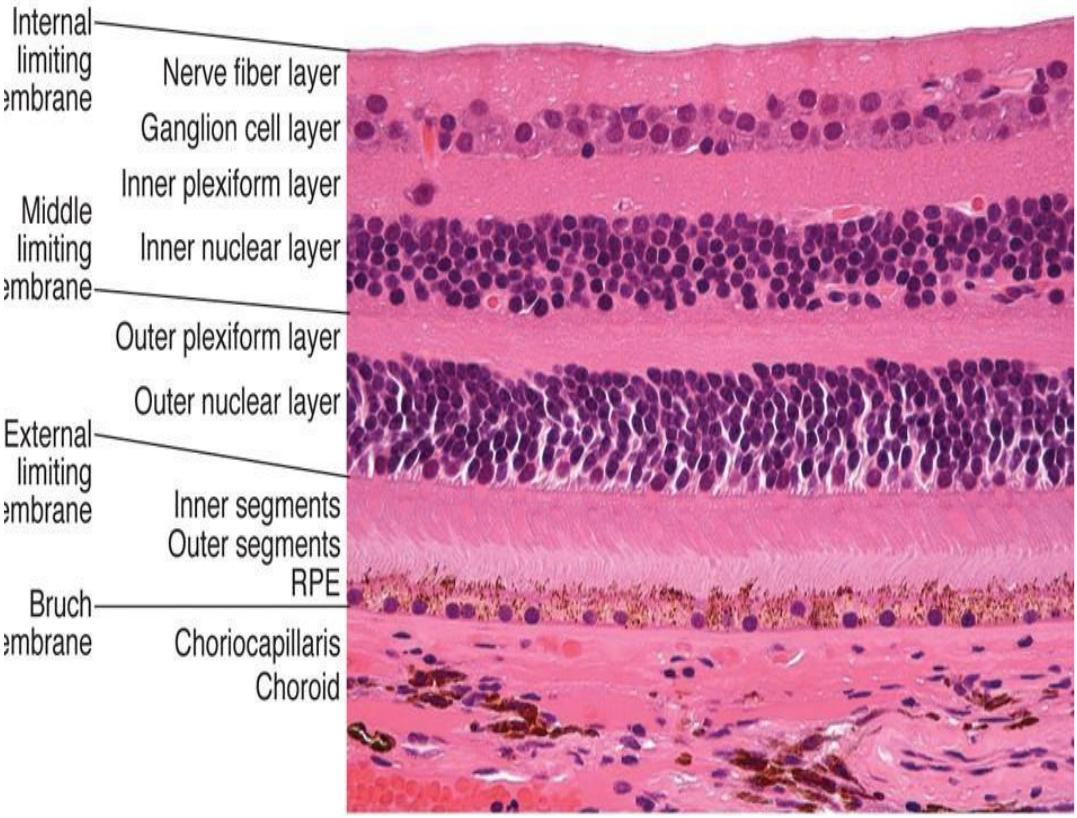


Figure 1 histological cross section of layers of retina

ANATOMY OF THE MACULA

The umbo, foveola, fovea, parafovea, and perifovea together constitute the macula, or central area. The fovea itself is a 1.5 mm depression in the center of the macula. It is located about 4 mm temporal and 0.8 mm inferior to the center of the horizontal plane of the optic disc. Fovea is an excavation in the retinal center and consists of a margin, a declivity, and a bottom. The bottom corresponds to the foveola, the center of which is called the umbo. The average thickness of the fovea is about 0.25 mm, roughly half that the adjacent para foveal area. The central 0.35 mm of the fovea is the foveola, which is located in a retinal capillary-free zone which measures about 0.5 mm in

diameter. A small protuberance in the center of the foveola is called the umbo, where there is a great concentration of cell bodies of elongated cones. A 0.5 mm wide annular zone surrounding the fovea is the area where the ganglion cell, inner nuclear layer, and outer plexiform layer of Henle are the thickest. This is referred to as the **Parafoveal area**. This area is surrounded by a 1.5 mm ring zone called the **Perifoveal area** where the ganglion cell layer is reduced from 5–7 layers to a single layer of nuclei, as seen elsewhere in the peripheral retina. There are several modifications in the retinal architecture in the macular area, beginning with the absence of retinal vessels in the perifoveal region. There are no rods in the foveola, and the cones have become so modified that they resemble rods in form. The external segments of the cones are long and approach the apical side of the RPE cells. At the edge of the fovea, the ganglion cell layer and the inner nuclear layer thicken, but both layers disappear within the fovea. In the foveolar area, only photoreceptor cells and Muller cell processes are present.

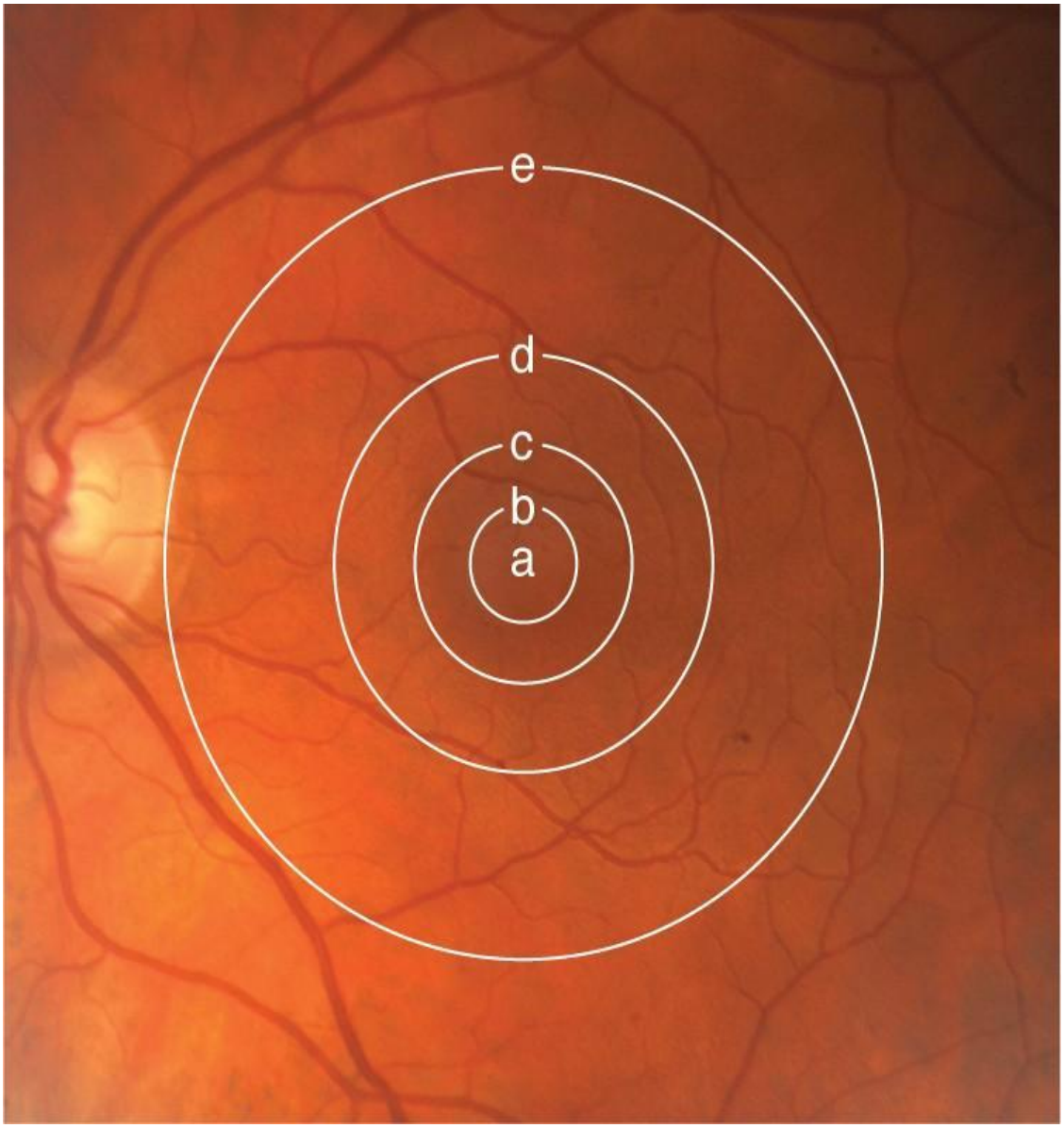


Figure 2 Anatomy of macula also called as posterior pole area centralis

a= umbo

b= foveola

c = fovea

C to d = parafoveal macula

D to e = perifoveal macula

e = macula

Xanthophyll is present in the fovea, located probably in the outer plexiform layer. These differences in pigmentation are the chief factors responsible for producing the characteristic dark zone in the macular region on normal angiograms. The absence of retinal vessels in the fovea (i.e., the perifoveal capillary-free zone), in most cases approximately 400–500 mm in diameter in the center of the fovea, is another cause of the dark appearance of the macula. Each cell is united with a single bipolar cell and possibly with a single ganglion cell, plus yielding maximal transmission of the visual stimulus.

The foveal cones result from the centripetal migration of the first neuron and the centrifugal lateral displacement of the second and third neurons during foveal maturation, which occur 3 months before and 3 months after term.

The fovea is the center of the macula and contains only four layers of the retina: (FIG 3)

(1) The internal limiting membrane;
(2) The outer plexiform layer;
(3) The outer nuclear layer; and
(4) The rods and cones.

No intermediate layers exist between the internal limiting membrane and the outer plexiform layer in the fovea, which in the macula is oblique. This is an important factor in understanding the stellate appearance of cystoid edema in the macula as opposed to the honeycomb appearance of cystoid edema outside the macula. Beyond the macular region the outer plexiform layer is perpendicular rather than oblique.

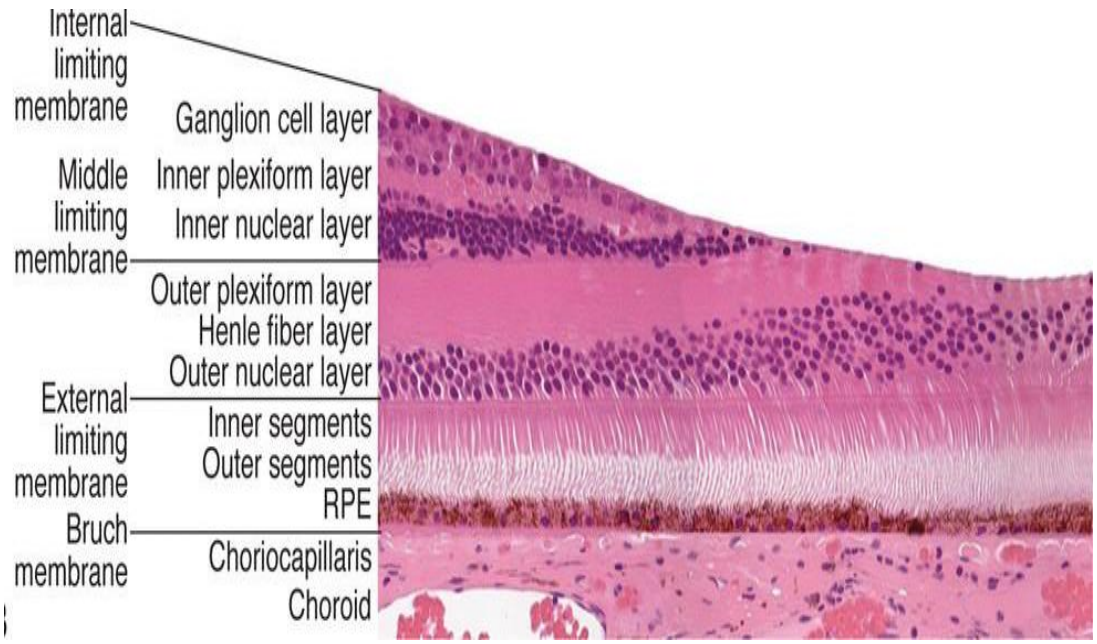


Figure 3 histological cross section of fovea showing laterally displaced inner retinal layers

VITREOUS

The vitreous is a transparent gel composed principally of water, collagen, and hyaluronic acid; it occupies 80% of the volume of the eye. The vitreous body is divided into 2 main topographic areas: the central, or core, and the peripheral, or cortical, vitreous. The vitreous gel is made up of collagen fibrils separated by hydrated hyaluronic acid molecules, which act as fillers and separators between adjacent collagen fibrils.

The anterior surface of the vitreous body is called the anterior cortical gel, made up of a condensation of collagenous fibers that attach to the posterior lens capsule, forming

the ligament of Wiegert. A prominent area of liquefaction of the premacular vitreous gel is called the premacular bursa, or precortical vitreous pocket. (fig4)

The retrolental indentation of the anterior vitreous is called the patellar fossa. The potential space between the peripheral posterior lens and the anterior cortical gel bordered by the Wiegert ligament is called the Berger space. In the vitreous base, the collagen fibers are particularly dense; they are firmly attached to the anterior retina and posterior pars plana, creating a ring like area that extends approximately 2 mm anterior and 3–4 mm posterior to the oraserrata. The vitreous is not only attached at its base; it is also firmly attached to the lens capsule, retinal vessels, optic nerve, and macula. (Fig 4)

Oxygen is derived from diffusion from choroidal and retinal circulation. Hyalocytes consume most of this, limiting the amount of oxygen that reaches the lens and anterior segment. The vitreous has high ascorbate levels, which protects against oxidative damage[35].

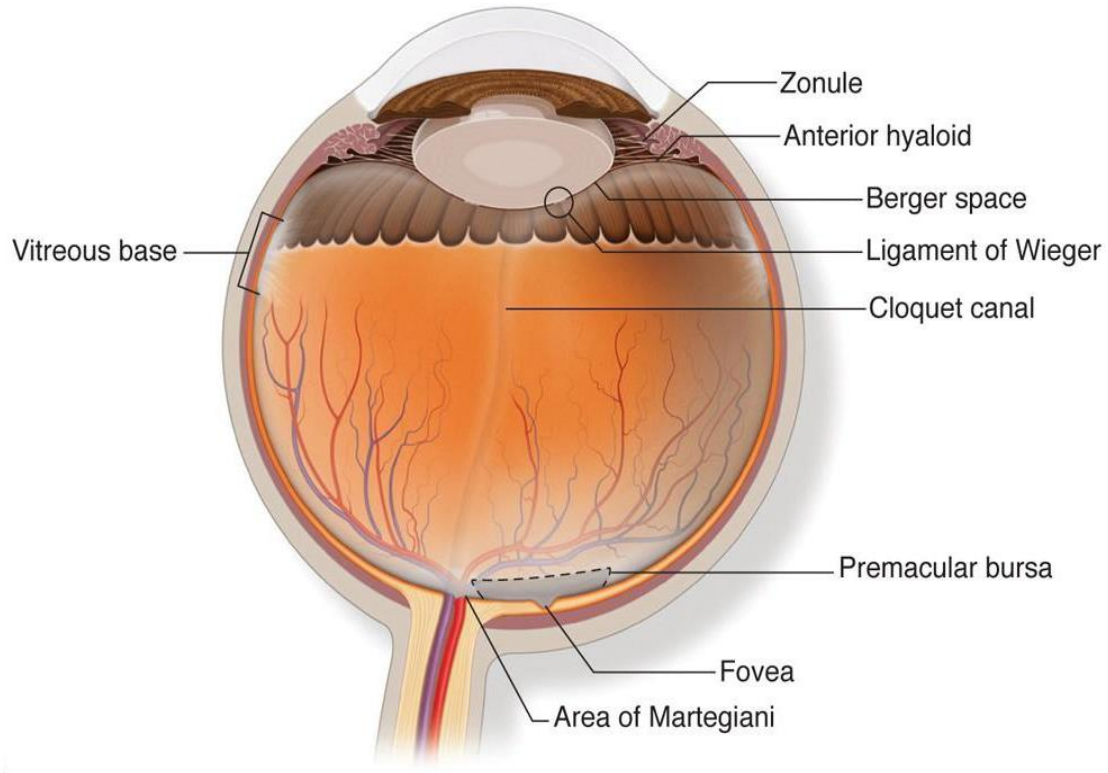


Figure 4 cross section of diagram of eye with emphasis on vitreous anatomical features

Pathophysiology:

Diabetes mellitus is the most common metabolic disorder. Type 2 diabetes mellitus comprises about 90% of all patients, and most of the rest have type 1 diabetes mellitus.

Glycemic control is critical for these patients because poor control, over time, leads to development of micro vascular complications. The micro vascular complications affect small blood vessels and include nephropathy, neuropathy, and retinopathy.

Chronic hyperglycemia is the primary factor leading to the development of diabetic retinopathy and other complications of the disease.

The duration of diabetes was strongly associated with frequency and severity of retinopathy.

Microglia represent the primary resident immune cell of the retina and, although normally quiescent, become activated by diabetes and have been reported to induce inflammatory changes underlying DR.

The pathogenesis of the disease is regarded multifactorial and complex:

- Capillary basement membrane thickening,
- Loss of Pericytes,
- Loss of endothelial cells
- Microaneuryms,

Blood retinal barrier breakdown and other anatomic lesions might contribute to macular edema and/or neovascularization the two major and sight threatening complications of diabetic retinopathy. VEGF is involved in pathogenesis and progression of the disease[38].

Prolonged hyperglycemia leads to the degeneration and loss of pericytes of the retinal capillaries. This eventually promotes a thickening of the capillary basement membrane.

These micro aneurysms are localized dilatations of the microvasculature which have been postulated to have developed as a result of localized weaknesses in the vessel wall, pressure disturbances, or glial retraction/death[39]

DR Starts from early damage to the small blood vessels in the retina. To compensate for impaired circulation and ischemia in the retina due to these damaged vessels, neovascularization may occur on the surface of the retina[40] These newly formed blood vessels as well as the existing damaged capillaries tend to have increased permeability, leading to accumulation of fluid in the macula and decreased visual

acuity. However, more recently, involvement of neuronal and glial components in early stages of retinopathy has been observed.[41]

Chronic inflammatory processes that occur in experimental models of DR include increased nitric oxide production, intracellular adhesion molecule 1 up regulation, leukostasis, and increased expression of proinflammatory cytokines associated with vascular damage and neuronal cell loss.[42]

DME can occur in any stage of nonproliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR), although its prevalence is increased in patients with advanced stages of DR. Risk factors for DME include hyperglycemia and hypertension[43].

DME is the thickening of the retina in the macular area due to DR. A primary cause of DME is an increase in retinal vascular permeability (RVP) that can occur both diffusely in the retina and in focal regions. DME is often accompanied by the appearance of hard exudates, which represent lipid deposition in areas where intra retinal fluid has resorbed. Increased retinal thickness has been correlated with worse visual acuity[44].

Elevated levels of RVP facilitate the diffusion of circulating proteins and lipids across the retinal endothelium into the interstitial fluid of the neuroretina.

The accumulation of plasma proteins in the retinal interstitium has been implicated in contributing to fluid retention, lipid deposition, and thickening of the macula[45].

Otani described classification of DME by OCT:

1) diffuse thickening type (sponge-like diffuse retinal thickening),

2) cystoid macular edema (CME) type (thickening of fovea with intra retinal cystoid change), and
3) serous retinal detachment (SRD) type (thickening of fovea with sub retinal fluid)[46]

Brian y. Kim et al have identified at least five different morphologic patterns of DME using OCT including:

- Diffuse Retinal Thickening,
- Cystoid macular edema,
- Serous Retinal Detachment without Posterior Hyaloid Traction,
- Posterior Hyaloid Traction without Tractional Retinal Detachment, and
- Posterior Hyaloid Traction with Tractional Retinal Detachment [47].

In DME, leakage from the deep capillary plexus suggests fluid migration into the outer plexiform layer; leakage from the superficial capillary plexus leads to fluid accumulation in the inner nuclear layer[48].

The arrangement of the cystoid cavities is determined by the Müller fibres, which are arranged vertically. The anatomy is clearly distinguished on OCT and also becomes very clear in late FA where one sees the typical petalloid leakage. Serous detachment of the retina is usually due to chronic edema[49]

When the post cataract macular edema is associated with a decrease in visual acuity, it can be categorized as clinical **Pseudophakic cystoid macular edema**. Disruption of blood aqueous barrier (BAB) and blood retinal barrier (BRB), causes increased vascular permeability. It usually appears as a **Petalloid** pattern of leakage in fluorescein angiography, and has been referred to as **Irvine–Gass syndrome.**

On FA, dye pooling within a **Petalloid** pattern in the fovea corresponds to cystic spaces in the **outer plexiform** layer, and a **honey-comb pattern** corresponds to cystic spaces in the **inner plexiform** layer. Fluorescein staining of non-cystoid edema is diffuse and irregular and not confined to well-demarcated spaces.

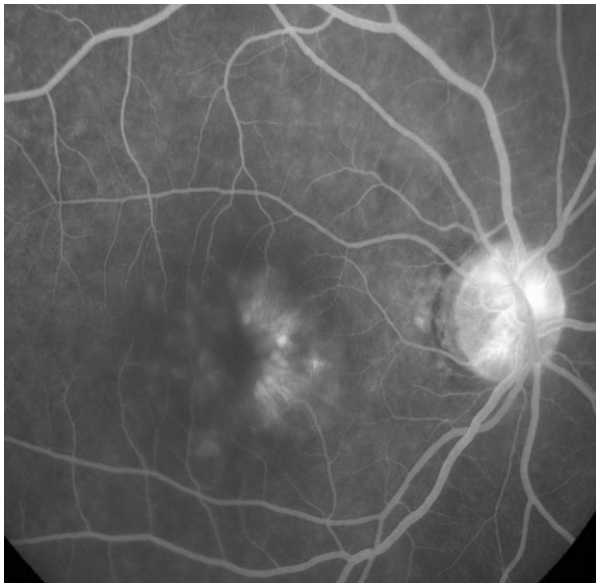


FIGURE 5 Showing Petalloid appearance in late phase on FFA with optic disc staining

Diabetic Retinopathy Disease Severity Scale [50]

Findings based on dilated ophthalmoscopy

No apparent retinopathy No abnormalities

Mild non proliferative diabetic retinopathy : Micro aneurysms only

Moderate non proliferative diabetic retinopathy: More than just micro

Aneurysms but less than severe non proliferative diabetic Retinopathy

Severe non proliferative diabetic retinopathy: any of the following:

- More than 20 intra retinal hemorrhages in each of 4 quadrants;
- Definite venous beading in 2 quadrants;

- Prominent intra retinal micro vascular abnormalities in 1 quadrant and
- No signs of proliferative retinopathy

Proliferative diabetic retinopathy: one or more of the following: neovascularization, vitreous/pre retinal hemorrhage

Vision – threatening Diabetic retinopathy (VTDR)

Diabetic macular edema (DME)

- Macular degeneration ,atrophy
- Sub foveal lipid deposition
- Foveal hemorrhage

Proliferative Diabetic retinopathy (PDR)

- Vitreous hemorrhage
- Tractional DME (Epimacular proliferation)
- Macular hole
- Tractional retinal detachment
- Neovascular glaucoma

TABLE 1 ABBREVIATED EARLY TREATMENT RETINOPATHY STUDY (ETDRS) CLASSIFICATION

CATEGORY	MANAGEMENT
NON PROLIFERATIVE DIABETIC RETINOPATHY (NPDR)	
NO DR	Review in 12 months
VERY MILD • Micro aneurysms only	Review most patients in 12 months
MILD Any or all of: Micro aneurysms, retinal hemorrhages, exudates, cotton wool spots	Review range 6-12 months, depending on severity of signs, stability, systemic factors, and patients personal circumstances
MODERATE • Severe retinal hemorrhages in 1-3 quadrants or mild IRMA • Significant venous beading in no more than 1 quadrant • Cotton wool spots	Review in approximately 6 months (PDR in up-to 26% , high-risk PDR in up-to 8% within a year)
SEVERE The 4-2-1 RULE • Severe retinal haemorrhages in all 4 quadrants • Significant venous beading in ≥2 quadrants • Moderate IRMA in ≥ 1 quadrants	Review in 4 months (PDR in up to 50%, high risk PDR in up to 15% within a year
VERY SEVERE ➢ ≥ 2 of the criteria for severe	Review in 2-3 months (High risk PDR in up-to 45% within a year)

TABLE2 ABBREVIATED EARLY TREATMENT RETINOPATHY STUDY (ETDRS) CLASSIFICATION

CATEGORY	MANAGEMENT
PROLIFERATIVE DIABETIC RETINOPATHY (PDR)	
MILD – MODERATE • New vessels on disc (NVD)< 1/3-disc area • New vessels elsewhere (NVE)< 1/2-disc area	• Treatment considered according to severity of signs, stability, systemic factors, and patient personal circumstances • If not treated, review in up-to 2 months
HIGH RISK • NVD >1/3-disc area • Any NVD with vitreous or preretinal hemorrhage • NVE > 1/2-disc area with vitreous or preretinal hemorrhage	• Laser photocoagulation • Intravitreal anti VEGF agents • Intravitreal triamcinolone • Parsplana vitrectomy • Lipid lowering of drugs

TABLE 3 The Proposed International Clinical Diabetic Retinopathy And Diabetic Macular Edema Disease Severity Scales with the corresponding levels from Early Treatment Diabetic Retinopathy Study (ETDRS), clinical findings.[50]

MEASURE	SCORE	OBSERVABLE FINDINGS
ICDR severity level		
No apparent retinopathy	0	No abnormalities (Level 10 ETDRS)
Mild non proliferative diabetic retinopathy	1	Micro aneurysm(s) only Level 20 ETDRS)
Moderate non proliferative diabetic retinopathy	2	More than just micro aneurysm(s) but less than severe non proliferative diabetic retinopathy(level 35,43,47 ETDRS)
Severe non proliferative diabetic retinopathy	3	Any of the following > 20 intraretinal hemorrhages in each of 4 quadrants, definite venous beading in > 2 quadrants, prominent intraretinal micro vascular abnormalities in > 1 quadrant, or No signs of proliferative retinopathy. (Level 53 ETDRS: 4-2-1)
Proliferative diabetic retinopathy	4	One or more of the following : neovascularization and/or vitreous or preretinal haemorrhages (Level 61,65,71,75,81,85 ETDRS)
Macular odema severity level		
No macular oedema	0	no exudates and no apparent thickening within 1-disc diameter from fovea
Macular oedema	1	Exudates or apparent thickening within 1 disc diameter from fovea

Optical coherence tomography (OCT) is a non-contact non-invasive imaging Technique which generates cross-sectional images of tissue with high resolution. Time-domain OCT technology (TD-OCT) offers very slow imaging speed and Poor image quality. The introduction of spectral domain OCT (SD-OCT) was able to overcome the limitations of TD-OCT. which is able to capture the whole depth information simultaneously.

It has become the standard tool for imaging in macula diseases, diabetic retinopathy and glaucoma.

PRINCIPLE

OCT is often compared to medical ultrasound because of the similar working principles.

The idea of low-coherence interferometry is the underlying principle.

The optical setup typically consists of an interferometer, Michelson type with a low coherence, broad bandwidth light source of wavelength 830nm.

Near-infrared Light is split into and recombined from reference and sample arm, which goes to the reference mirror and patients retina respectively.

The back-reflected light from the retinal tissue and reference mirror are combined and interferes if the optical path lengths match and therefore the time travelled by the light is nearly equal in both arms.

Modulations in intensity, also called interference fringe bursts, are detected by the photo detector.

The back reflected waves are analyzed and their delay is measured to reveal the depth in which the reflection occurred.

The delays of the back reflected waves cannot be measured directly, so a reference measurement is used. For each sample point, the reference mirror is scanned in depth (z) direction and the light intensity is recorded on the photo detector.

Fourier domain OCT (FD-OCT, also frequency domain OCT) is the second generation of OCT technology and provides a more efficient implementation of the principle of low-coherence Interferometry.

In contrast to TD-OCT, FD-OCT uses spectral information to generate A-scans without the need for mechanical scanning of the optical path length.

Two methods were established to acquire the spectral information of the interferometry signal.

Both record an interference spectrum, also called spectral interferogram, from which the A-scan is computed via Fourier transformation.

Spectrometer based FD-OCT, which is commonly referred to as spectral domain OCT (SD-OCT).The principle optical setup is similar to TD-OCT, but the point detector is replaced by a spectrometer.

Multiple interference patterns are created over the surface of the structure being imaged. As the instrument scans, a series of A‑scans is created.

These A‑scans are combined into a composite cross‑sectional image (B‑Scan).

Each A‑scan contains information on the strength of the reflected signal as a function of depth [51].

Reference mirror is fixed in Fourier transformation and spectrometer in the cross-sectional images. The spectral domain system provides faster acquisition times than time domain systems and its higher resolution allows visualization of more details.

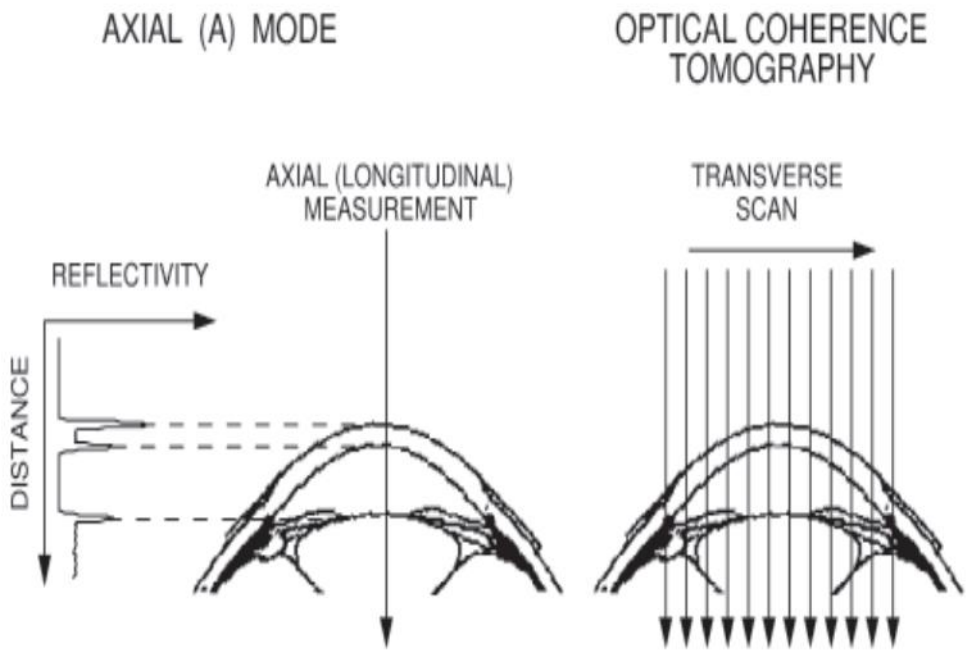


Figure 6 OCT Image Acquisition

Reflectivity: The normal ocular tissues which show high reflectivity are:

a) The retinal nerve fiber layer
b) The internal limiting membrane
c) The junction between the inner and outer segments of PRs, probably due to densely stacked disc membranes in the outer segments
d) Retinal pigment Epithelium-Bruch's membrane-choriocapillaries complex.

High reflectivity is also a feature of reduced retinal thickness (as in retinal atrophy) and scar tissue.

Lesions showing high reflectivity may be superficial, intra retinal or deep retinal. The superficial lesions include epiretinal and vitreal membranes, exudates and

hemorrhages and cotton wool spots which are exudates at the margin of ischemic areas. While the intraretinal lesions include; hemorrhages, hard exudates (these are lipoproteins located at the margin between healthy and edematous retina), and retinal fibrosis and disciform degenerative scars.

Hyper reflectivity patterns: suggested the following:

1) Hard exudates;
2) Hyper reflective shadows in the neurosensory retina that completely blocked the reflections from the underlying retina.

   Blood: If a thin layer, this was hyper reflective whereas a thick layer was

   Found to block the underlying reflections and
3) Scar tissue and neovascular membranes (showed varying Hyper reflectivity);

The lesions causing hypo or low reflectivity black, optically empty spaces include: a) atrophic RPE (loss of pigment); b) cystic or pseudo cystic areas containing serous fluid and c) cystoid edema, serous neural retinal detachment and RPE detachment. d) Optically empty space with absence of backscattering;

Scanning protocols

Macular and Optic Disc Cube Scans

Selectable in 512x128 and 200x200 mm fields, and generated by combining a series of A-scans taken at varying depths, Macular and Optic Disc Cube scans provide information about disc and fovea parameters including

- Size
- Cup, disc, rim area and volume
- Nerve fiber layer thickness

- Ganglion cell layer thickness (macular cube)

Macular Cube 512x128(512 horizontal A-scan and 128 vertical B scan lines with acquisition time of 2.4 secs) centered over the fovea.

This scan generates a cube of data through a 6 mm square grid by acquiring a series(512 horizontal A-scan and 128 vertical Bscan lines) centered over the fovea The early treatment diabetic retinopathy study (ETDRS) grid is routinely used for OCT recordings, and the software by default, automatically centered this on the fovea. The Macular Cube 512x128 is the default scan. Compared to the 200x200, this scan has greater resolution in each line from left to right, but the lines are spaced further apart, giving less resolution from top to bottom. This scan can be used to measure macular thickness and create a 3–D image of the data.

OTHERS

Raster Scans, HD 1 Line 100x, HD 21 Line, HD Cross, HD Radial

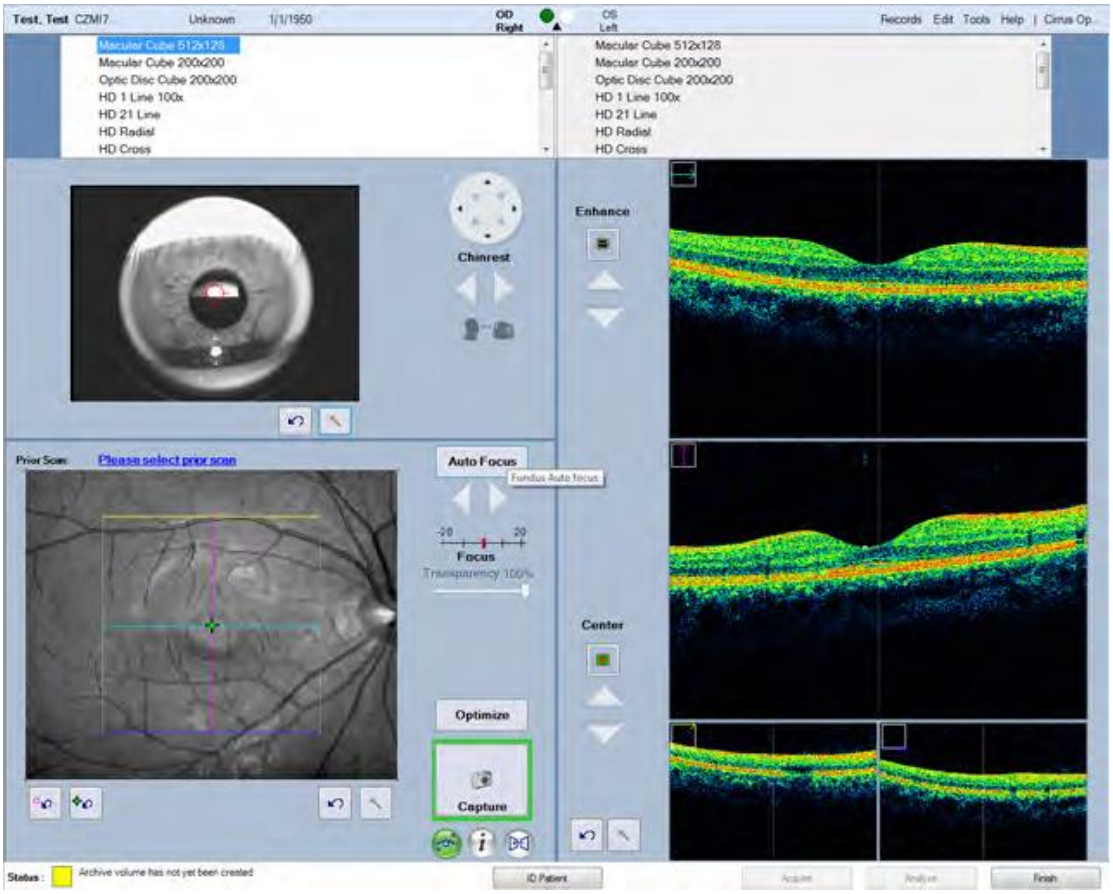


Figure 7 Acquire screen with fixation target in fundus view port

The line scans were 6 mm in the transverse direction, had a 2-mm axial depth, and were composed of 1,000 axial scans each. The cube scan was 6 × 6 mm, had a 2-mm axial depth, and was composed of 200 × 200 axial scans.

OCT is useful to diagnose of ANY type of DME and helps in deciding the management.

5 patterns seen on OCT

1) Sponge like retinal thickening
2) Cystoid macular edema: partial thickness cysts.
3) Cystoid macular edema: full thickness cysts.
4) Subfoveal serous RD

5) (TPHM) Taut posterior hyaloid membrane causing tractional foveal detachment

Involvement of macular center – OCT based classification

CENTRE INVOLVING DME: Cystic spaces /neurosensory

Detachment involving the center of fovea and CSFT > 300 Microns on SD-OCT.

NON CENTRE INVOLVING DME: Thickening of the macula in any other subfield falls under this category

MATERIALS AND METHODS

1. SOURCE OF DATA:

Study participants comprises cohort of diabetic patients with varying levels of retinopathy- mild , moderate and severe NPDR including the absence of retinopathy, who were scheduled for routine cataract surgery and admitted in ophthalmology department, B.L.D.E. (DU) Shri. B. M. Patil Medical College, Hospital and Research Centre VIJAYPURA**.**

2. METHOD OF COLLECTION OF DATA:

Study design: It was a prospective comparative study

Sample size: 65 patients

Duration: Two year.

SELECTION CRITERIA

INCLUSION CRITERIA:

Diabetic patients with senile immature cataract with varying levels of retinopathy including absence of retinopathy underwent uncomplicated small incision cataract surgery done by an experienced surgeon.

EXCLUSION CRITERIA:

- Diabetic patients where pre-op OCT is not possible.
- Subluxated lens
- Pseudo exfoliation

- Glaucoma
- Intraoperative any complication
- Diabetic patients with prior intraocular surgery in the same eye
- Uveitis
- Presence of any retinal or choroidal disease, other than diabetes, that could affect retinal thickness.

PREOPERATIVE EVALUATION:

1. Visual acuity testing for distance and near using Snellen's distant chart and near vision chart respectively.
2. Refraction and correction where required.
3. External ocular examination.
4. Slit lamp bio microscopic examination to grade the cataract.
5. Tonometry using Applanation tonometer.
6. Lacrimal patency test
7. Keratometry
8. A-scan and Intraocular lens power calculation by SRK-2 formula.
9. Fundus examination with indirect ophthalmoscopy with 20D Lens. The level of diabetic retinopathy was recorded as No, mild, moderate, and severe non proliferative; or proliferative, as described in the Early Treatment Diabetic Retinopathy Study.

Other investigations included: RBS, FBS, PPBS, HBA1C

Each study eye underwent fundus photography before surgery and OCT testing before surgery. Pupils were dilated for OCT examination in all cases with Itrop plus which contains 0.8% Tropicamide and 5% phenylephrine.

Subject characteristics including age, gender, and duration of diabetes, hemoglobin A1C, FBS, PPBS, RBS medication use, and type of diabetes were recorded. History of previous laser photocoagulation, prior intraocular surgery, and treatment with an intravitreal or sub–Tenon's capsule injection of triamcinolone acetonide was documented from review of the patient's chart. Age and gender was recorded at the time of the preoperative visit. Duration of diabetes was estimated by review of the patient's chart in addition to direct patient questioning and subdivided into 3 groups: ≤5 years, 6-10years or > 10 years. Hemoglobin A1 c and medication use were recorded. Type of diabetes was defined as insulin dependent or non–insulin dependent. Surgical complications, including posterior capsular rupture, vitreous loss, and need for additional surgery, were documented from review of the operative note. All cataract surgeries were small incision cataract surgery with posterior chamber intraocular lens implantation done under local anesthesia by an experienced surgeon. The surgical procedure routinely consisted of a Peribulbar anesthesia, 6-7 mm incision superior scleral incision, self-sealing sclera-corneal tunnel incision is made, Side-port entry is made with the help of 1.5mm valvular corneal incision at 9-0 clock position Anterior capsulotomy by continuous curvilinear capsulorrhexis of adequate size is done, Hydro dissection is done to separate cortico-nuclear mass from the posterior capsule, Nucleus was delivered, Cortical matter was removed by irrigation and aspiration and posterior chamber intraocular lens placement in the capsular bag, The viscoelastic was cleared from the anterior chamber, Subconjuntival gentamycin

and dexamethasone 0.5cc was given at the end of the procedure. Pad and bandage applied.

Postoperatively all the patients received a course of tapering dose of topical antibiotic and steroid eye drops (ofloxacin+dexamethasone), Topical Nepafenac as NSAID given 3 times daily for 1 month. Systemic antibiotic and analgesic was given for 3 days postoperatively.

Optical coherence tomography testing was repeated at the POD day1, POD1 week, and 4th and 12th week postoperative visits. Best-corrected visual acuity (BCVA) was recorded at each visit.

Fundus photographs of retina were taken with CANON CF-1 Digital retinal camera. Optical coherence tomography (ZEISS CIRRUS HD-OCT 5000, Carl Zeiss Meditec Inc., and Dublin, CA, USA) testing was performed and images were generated with the use of Macular cube 512*128 in 6 mm square grid according to the manufacturer protocol as described in the user's manual. CIRRUS software identifies the Fovea location automatically by looking for the reduced reflectivity below the retina. We can also change the Fovea location manually which will update the data table and the ETDRS grid thickness measurements. Macular Thickness OU Analysis provides interactive scan images, as well as the Fundus image with a scan cube overlay for both eyes together and includes:

- Colored thickness maps
- OCT Fundus image, including the identified fovea location with a red dot
- The ETDRS grid maps with normative data
- A table containing central subfield thickness, average thickness and volume Measurements for the entire cube

- Horizontal and vertical B-scans

Minimum 3 mm dilatation is required. Measurement of retinal thickness at selected points on the tomographs was obtained automatically by means of a computer algorithm, which assumes that the first highly reflective band corresponds to the Internal limiting membrane interface and the second corresponds to the retinal pigment epithelium. Thus retinal thickness measurement was made by evaluating the displacement between anterior surfaces of these two interfaces. The macular thickness and volume map was divided into nine sections and displayed as three concentric circles including a central circle, an inner ring and an outer ring with diameter of 1mm, 3mm, 6mm respectively, each ring being divided into four quadrants.

Optical coherence tomography scans were interpreted and assessed the quality of the OCT image according to signal strength. We took 5 and above as good signal strength.

We used the term macular edema to describe an increase in foveal thickness on OCT after cataract surgery in our diabetic cohort. An increase in foveal thickness on OCT after cataract surgery in this study is distinguished from several other definitions of ME, including the following: (1) typical cystoid ME (CME), noted on bio microscopic examination and on OCT after intraocular surgery, regardless of whether a patient had retinopathy; (2) DME, defined as ME noted clinically or on fundus photographs or OCT judged to be due to diabetes; and (3) clinically significant ME (CSME), defined as presence of retinal thickening and hard exudate at or within 500 μm of the center of the macula involving or threatening the center of the macula as defined by the Early Treatment Diabetic Retinopathy Study.

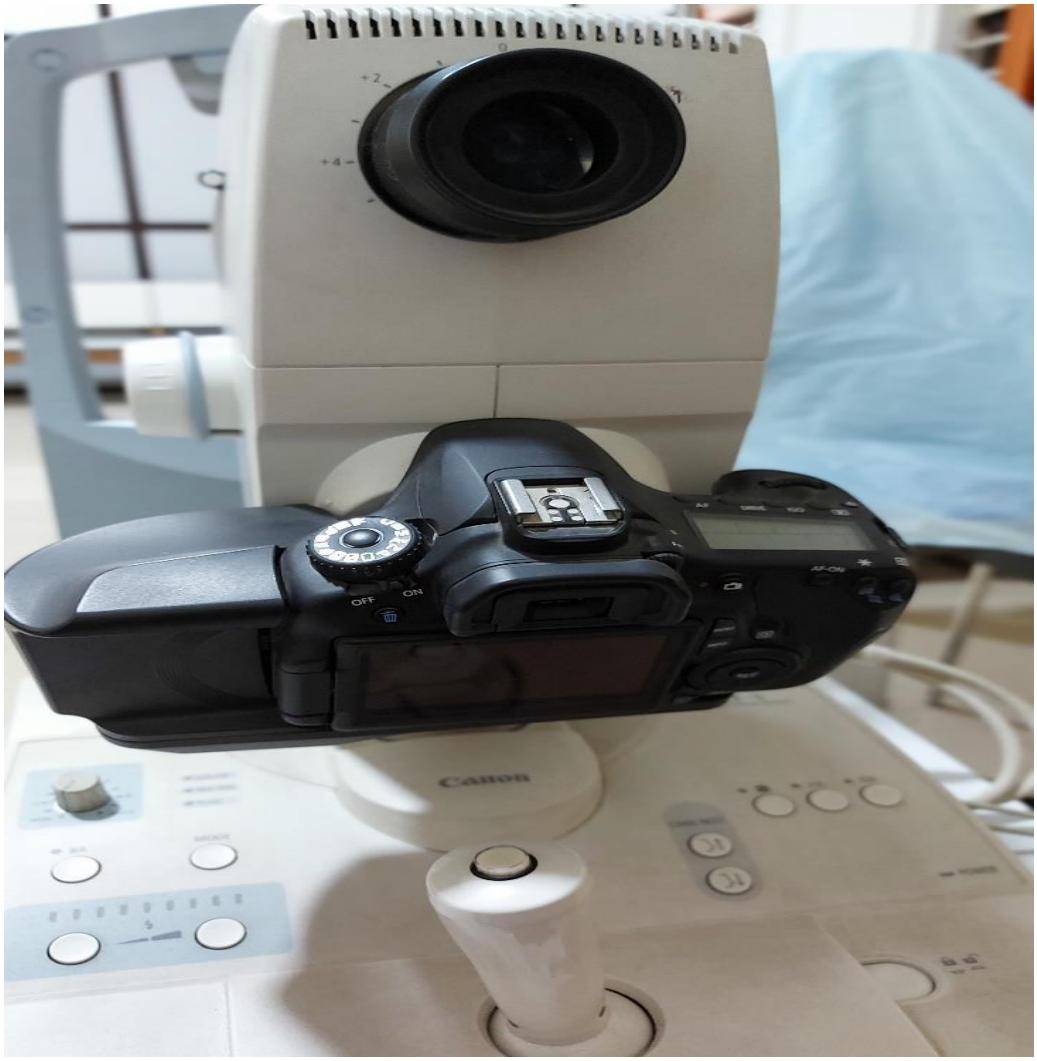


FIG 8 Fundus camera cf-1 digital retinal camera

FIG 9 ZEISS HD CIRRUS OCT 5000

SAMPLING:

- With Anticipated Mean Difference of best corrected visual acuity between the pre and post-operative diabetics as 0.17 and anticipated common SD as, 0.25 the minimum sample size is 65 patients with 90% power and 1% level of significance[25] Formula used

- $$n = \frac{(z_\alpha + z_\beta)^2 \, 2 \, SD^2}{MD^2}$$

Where Z= Z statistic at a level of significance

MD= Anticipated mean difference

SD= Anticipated Standard deviation

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean± standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square (χ^2) test was used for association between two categorical variables.

The formula for the chi-square statistic used in the chi square test is:

$$\chi_c^2 = \sum \frac{(O_i - E_i)^2}{E_i}$$

The subscript "c" are the degrees of freedom. "O" is observed value and E is expected value.

The difference of the means of analysis variables between two time points in same group was tested by paired t test.

T-Statistic

The T-Statistic is the value used to produce the *p*-value (Prob Level) based on the *T* distribution. The formula for the T-Statistic is:

$$T - Statistic = \frac{\overline{x}_{diff} - Hypothesized\ Value}{SE_{\overline{x}_{diff}}}$$

DF

The degrees of freedom define the T *distribution* upon which the probability values are based. The formula for the degrees of freedom is the number of pairs minus one:

$$df = n - 1$$

If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analyzed using SPSS software v.23 (IBM Statistics, Chicago, USA) and Microsoft office 2007.

RESULTS

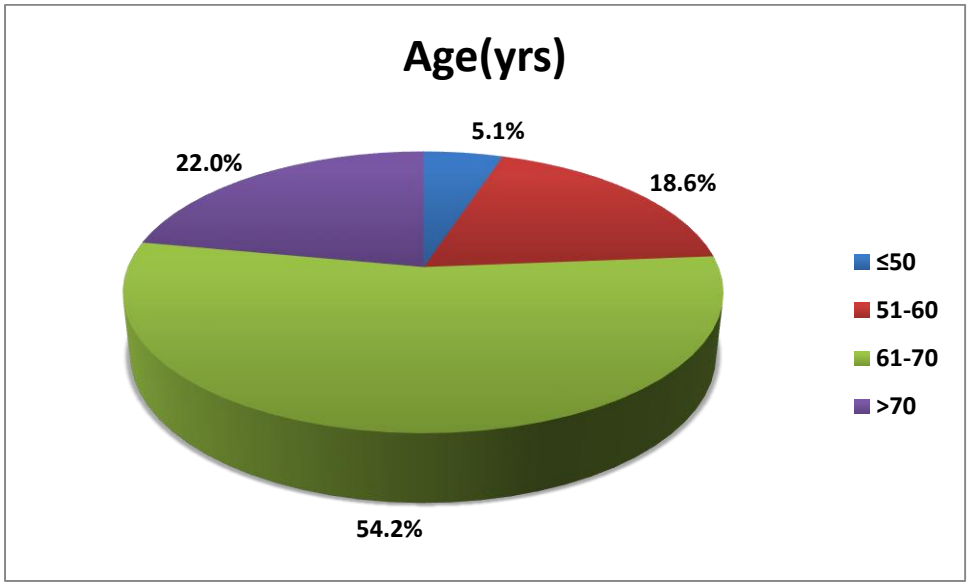
Of the 65 patients 4 were lost to follow-up. Two patients in the DR group had pseudophakic bullous keratopathy, did not appear for examination because of hazy media. Therefore, 59 patients (90.7%) completed the 3 months follow-up and were included for analysis. Patient characteristics are shown in following tabular columns and graphs.

Table 4: Distribution of Cases according to Age

Age(yrs.)	N	Percent
≤50	3	5.1
51-60	11	18.6
61-70	32	54.2
>70	13	22
Total	59	100

Descriptive Statistics	Min	Max	Mean	SD
Age(yrs.)	48	85	66.0	7.8

GRAPH 1: Distribution of Cases according to Age



The mean age of the study population was 66.0 ±7.8years.which is seen in fig1.

Among the 59 patients 36 patients (61%) were males and 23 (39%) were males which is depicted in and table

Table5: Distribution of Cases according to Sex

SEX	N	Percent
MALE	36	61
FEMALE	23	39
Total	59	100

Graph2: Distribution of Cases according to Sex

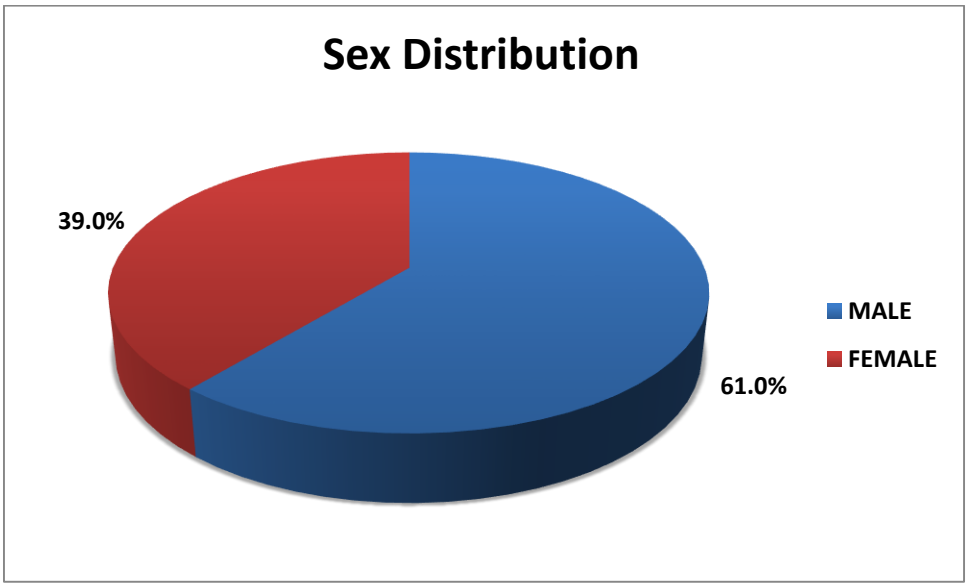


Table6: Distribution of Cases according to Duration of DM

Duration of DM	N	Percent
≤5	40	67.8
6-10	15	25.4
>10	4	6.8
Total	59	100.0

Descriptive Statistics	Min	Max	Mean	SD
Duration of DM	1	11	4.8	2.9

Among the 59 study group patients of type II diabetes mellitus patients, most of the patients are with DM ≤5 Years duration of about 67.8% patients. Mean Age duration of DM is 4.8±2.9 yrs. Which is shown in table

32 patients (54.2%) were systemic hypertensives table 3

GRAPH3: Distribution of Cases according to Duration of DM

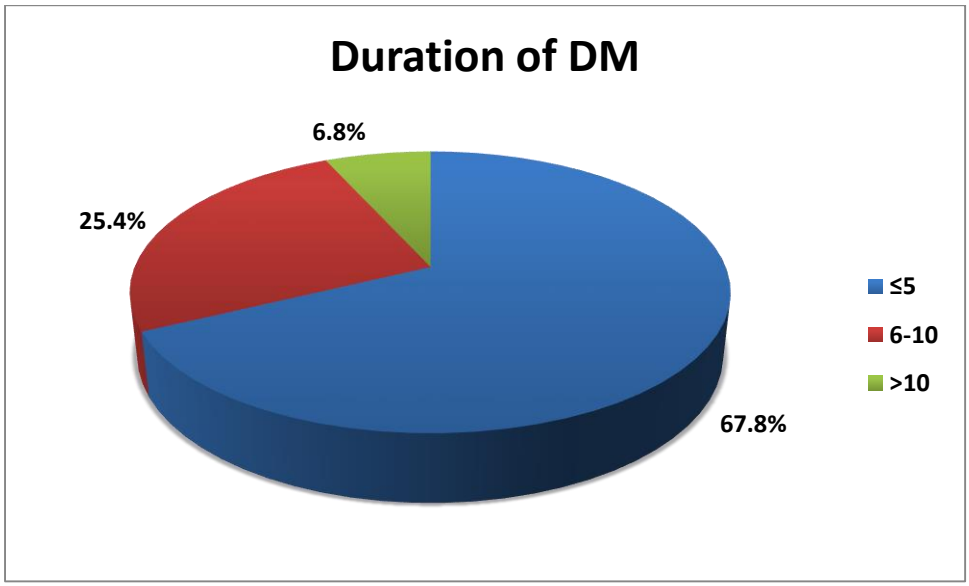
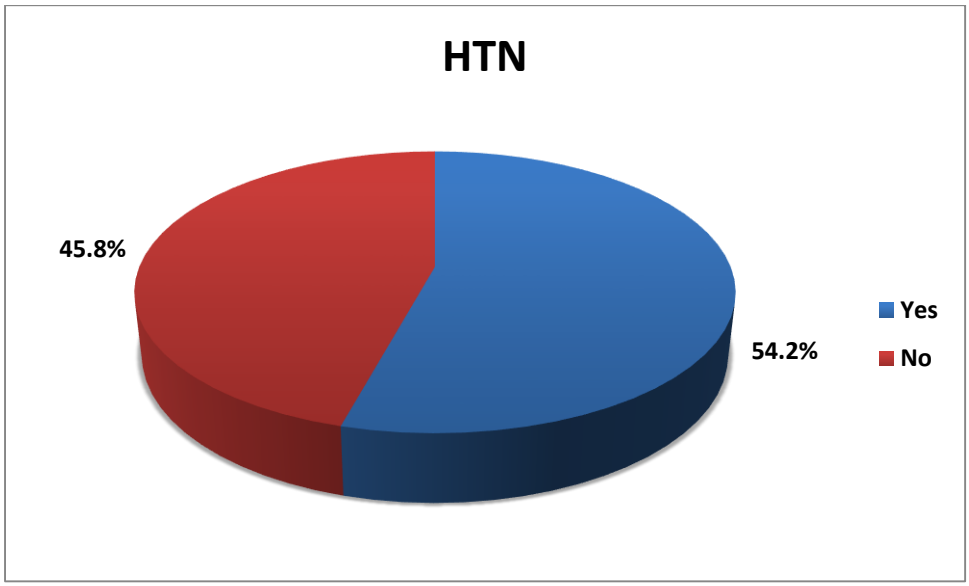


Table7: Distribution of Cases according to HTN

HTN	N	Percent
Yes	32	54.2
No	27	45.8
Total	59	100

GRAPH 4: Distribution of Cases according to HTN



Right eye cataract surgery was performed in 29 patients and left eye cataract surgery was performed in 30 patients.

No Diabetic retinopathy was detected preoperatively in 44% of diabetic eyes.

18 patients had Mild NPDR which comes to 30.5% of diabetic patients 8 patients had Moderate NPDR comprises 13% of diabetics

Severe NPDR With macular edema in 1 patient and pre op macular edema is seen in 2 patients.as shown in table 8 graph 5

Table8: Distribution of Cases according to Diabetic Retinopathy

Diabetic Retinopathy	N	Percent
NO	26	44.1
MILD NPDR	18	30.5
MODERATE NPDR	8	13.6
MODERATE NPDR ME	4	6.8
SEVERE NPDR ME	1	1.7
MACULAR EDEMA	2	3.4
Total	59	100

Graph5: Distribution of Cases according to Diabetic Retinopathy

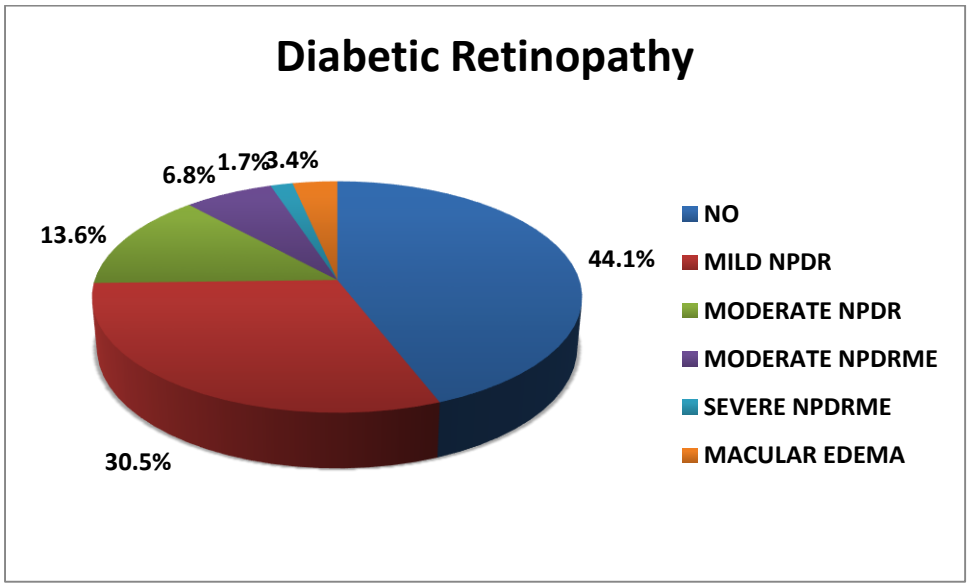


Table9: Distribution of Cases according to BCVA at Pre-op

Preop	N	Percent
6/24	1	1.7
6/36	2	3.4
6/36P	1	1.7
6/60	9	15.3
CF-1M	12	20.3
CF-2M	9	15.3
CF-3M	13	22
CF-4M	4	6.8
CF-5M	1	1.7
CF-CF	4	6.8
HM+	2	3.4
PL+ PR+	1	1.7
Total	59	100

Graph6: Distribution of Cases according to BCVA at Pre-op

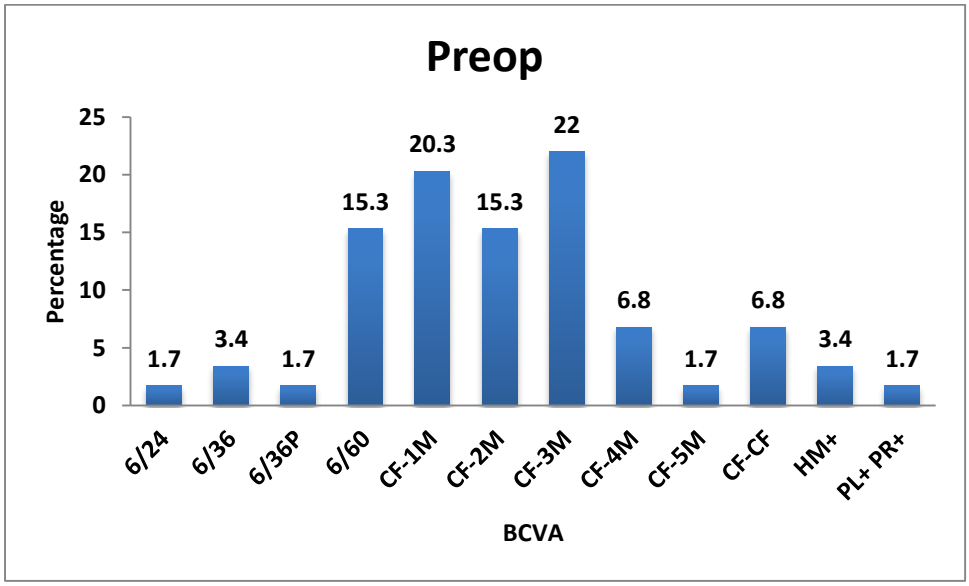


Table10: Distribution of Cases according to BCVA at POD 1

POD 1	N	Percent
6/6	1	1.7
6/9	1	1.7
6/12	23	39.0
6/18	14	23.7
6/24	16	27.1
6/36	1	1.7
6/60	1	1.7
CF-4M	1	1.7
CF-5M	1	1.7
Total	59	100.0

Graph7: Distribution of Cases according to BCVA at POD 1

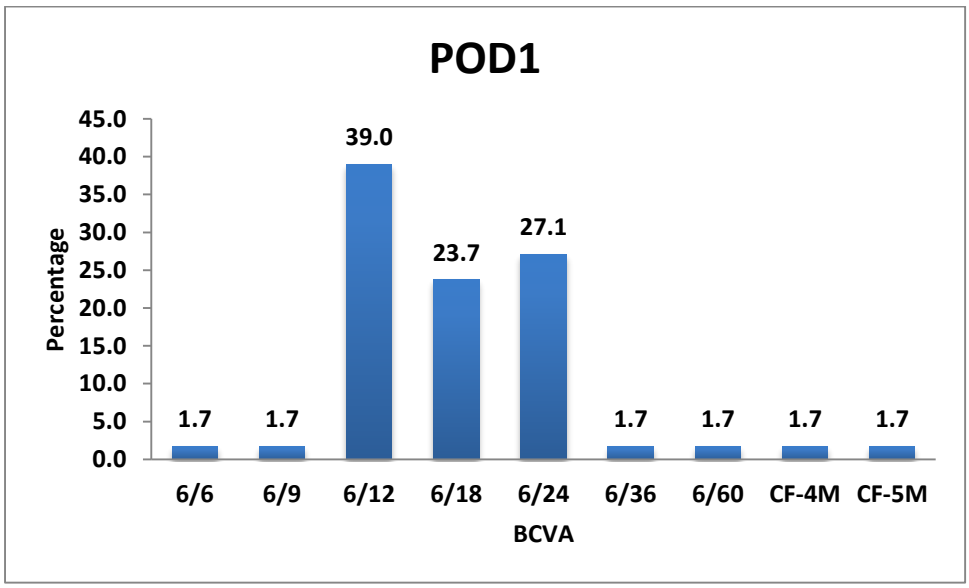


Table11: Distribution of Cases according to BCVA at POD 1wk

POD 1wk	N	Percent
6/6	2	3.4
6/9	8	13.6
6/12	21	35.6
6/18	16	27.1
6/24	10	16.9
6/60	1	1.7
CF-4M	1	1.7
Total	59	100.0

Graph8: Distribution of Cases according to BCVA at POD 1wk

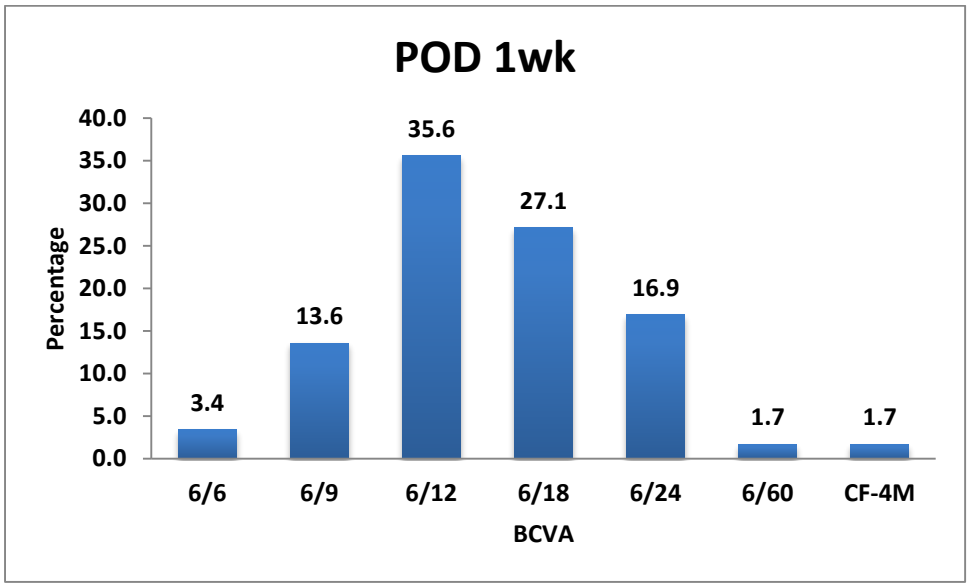
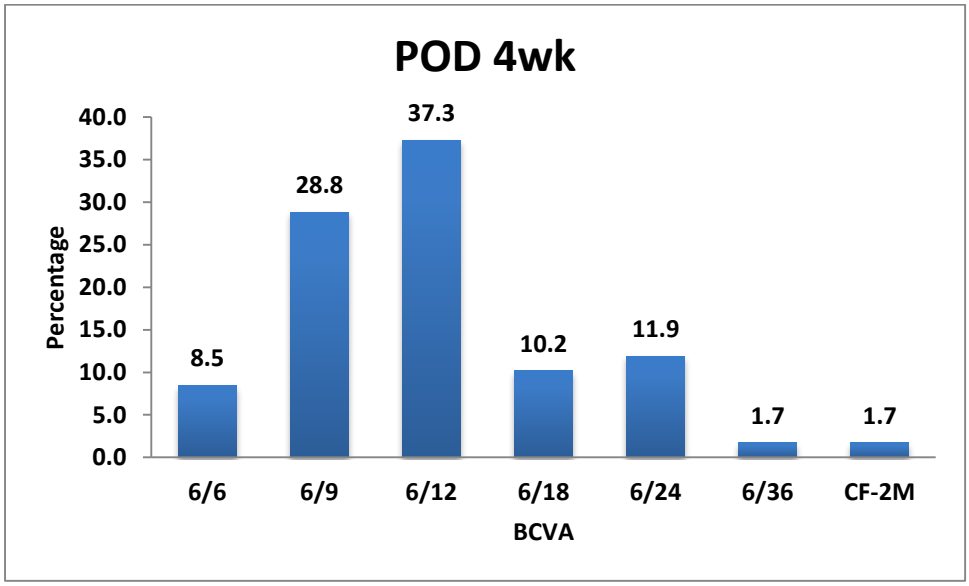


Table12: Distribution of Cases according to BCVA at POD 4wk

POD 4wk	N	Percent
6/6	5	8.5
6/9	17	28.8
6/12	22	37.3
6/18	6	10.2
6/24	7	11.9
6/36	1	1.7
CF-2M	1	1.7
Total	59	100.0

Graph9: Distribution of Cases according to BCVA at POD 4wk

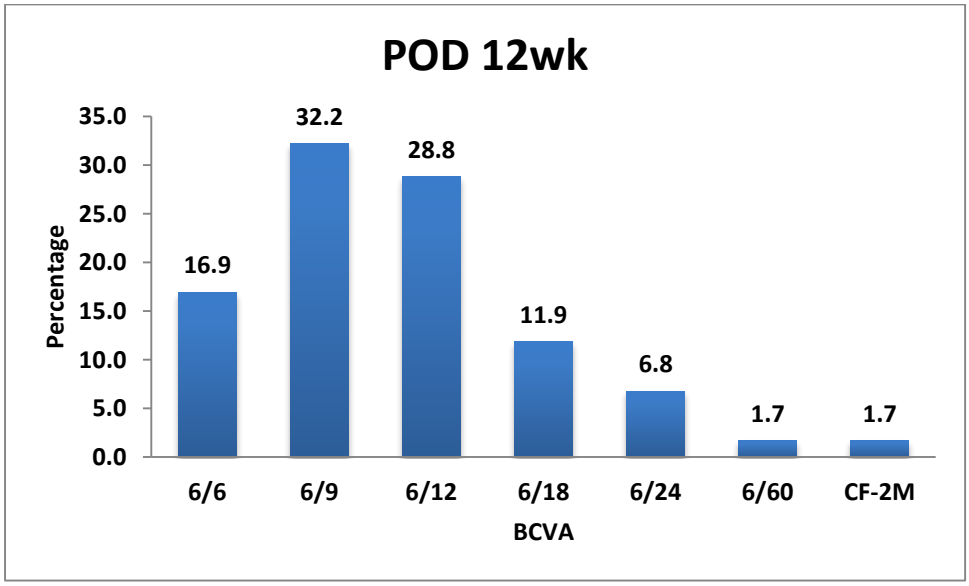


Preoperatively 93% patients had a vision of 6/60 or lesser. Over all visual acuity improved postoperatively at 4th and 12th week after small incision cataract surgery to 6/12 and 6/9 in majority of the patients. Post operatively 1 month 75% patients achieved vision of 6/6– 6/12. By Post-operative 3 months 89.8% patients achieved a vision of 6/6 – 6/12.as shown in table13 and graph 10

Table 13: Distribution of Cases according to BCVA at POD 12wk

POD 12wk	N	Percent
6/6	10	16.9
6/9	19	32.2
6/12	17	28.8
6/18	7	11.9
6/24	4	6.8
6/60	1	1.7
CF-2M	1	1.7
Total	59	100.0

Graph10: Distribution of Cases according to BCVA at POD 12wk



Mean central subfield macular thickness on OCT in diabetic patients of all grades of DR is increased with higher statistical significance at 1 and 3 months post operatively compared to 1st week postoperatively.

Visual acuity markedly improved from counting fingers to 6/12 and 6/9 in most of all patients post operatively.

The central subfield mean thickness in all patients irrespective of diabetic retinopathy increased 17.4µm and 29µm at 1 month and 3 month follow up. A statistically significant increase could be detected in central subfield macular thickness though the increase was mild. ($P<0.002$)

Eyes with preoperative macular edema after cataract surgery are high risk of developing macular edema.

However, the absolute changes in thickness were mild in all measurements.

The mean changes of CSMT at 3 months were shown in the

Mean Pre op CSMT 267.6±16.9µm. Mean Central subfield thickness increased from 267±16.9µm to 285±42.2 at 1 month and to 296.6±55.7 at 3 month follow up with

P-valve 0.002*($p<0.05$) among all the diabetic cases.as shown in table 14 and graph 11

Severity of DR was not significantly correlated to ME because of limitation in sample size. No statistical difference in macular thickness was revealed through severity of DR.

Table 14: Distribution of OCT according to POD among all cases

Total cases	PREOP		POD1		p value
	Mean	SD	Mean	SD	
OCT CENTRAL SUB FIELD THICKNESS	267.6	16.9	277.9	43.5	0.039*
	POD1		POD 1wk		p value
	277.9	43.5	280.4	38.5	0.321
	POD 1wk		POD 4wk		p value
	280.4	38.5	285.2	42.2	0.029*
	POD 4wk		POD 12wk		p value
	285.2	42.2	296.6	55.7	0.002*

Note: * significant at 5% level of significance (p<0.05)

The mean ±SD foveal thickness in all the patients groups are shown in and table14 and graph 11, respectively

Graph11: Distribution of OCT according to POD among all cases

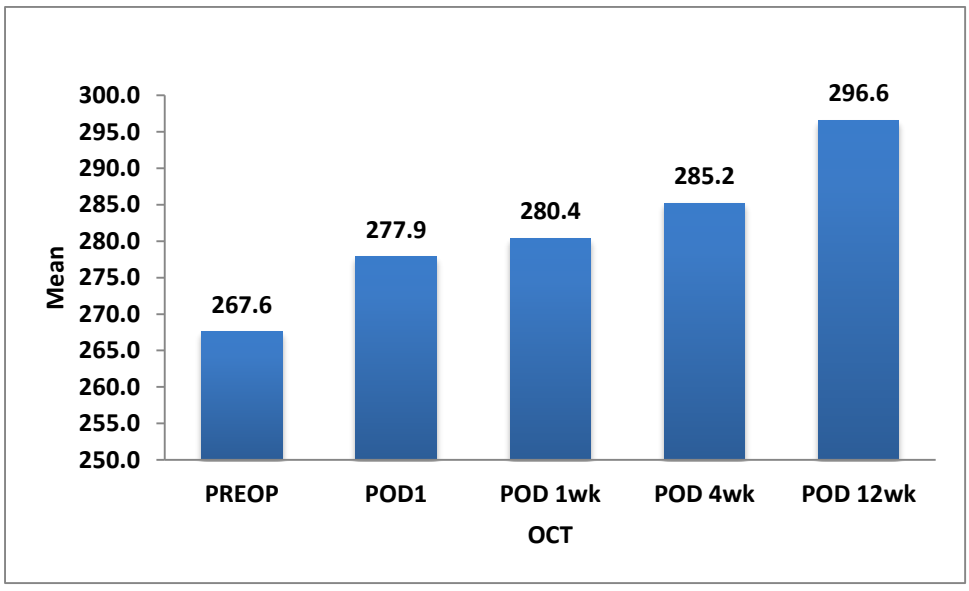


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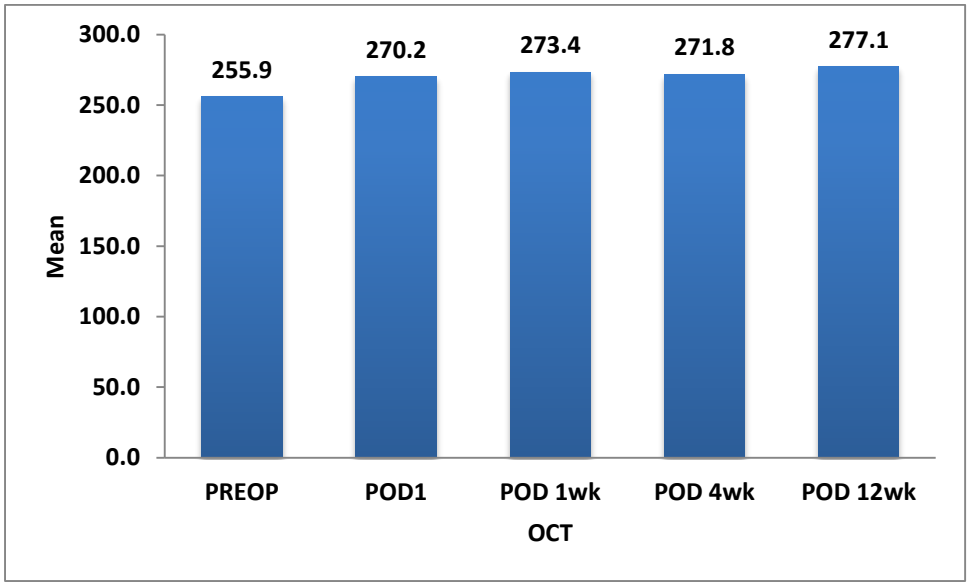
Table15: Distribution of OCT according to POD among No DR cases

Parameter	PREOP		POD1		p value
	Mean	SD	Mean	SD	
OCT CENTRAL SUB FIELD THICKNESS	255.9	12.0	270.2	15.0	<0.001*
	POD1		POD 1wk		p value
	270.2	15.0	273.4	11.3	0.332
	POD 1wk		POD 4wk		p value
	273.4	11.3	271.8	9.3	0.503
	POD 4wk		POD 12wk		p value
	271.8	9.3	277.1	12.2	0.030*

Note: * significant at 5% level of significance (p<0.05)

Graph12: Distribution of OCT according to POD among No DR cases



Among patients with no diabetic retinopathy developed thickening from a preoperative mean value of 255.9±12.0 to 277.1±12.2µm at 3rd month of follow up with a P-value of 0.030*(p<0.05) as shown in table 15 and graph15.

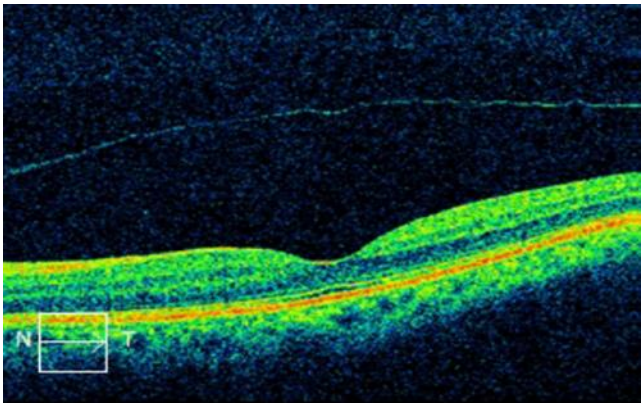


FIG10 POSTERIOR HYALOID TRACTION

- Posterior hyaloid traction seen in a patient Post operatively at 3 month

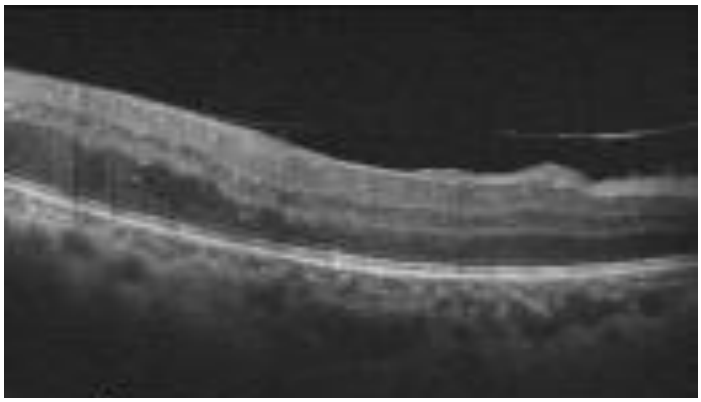


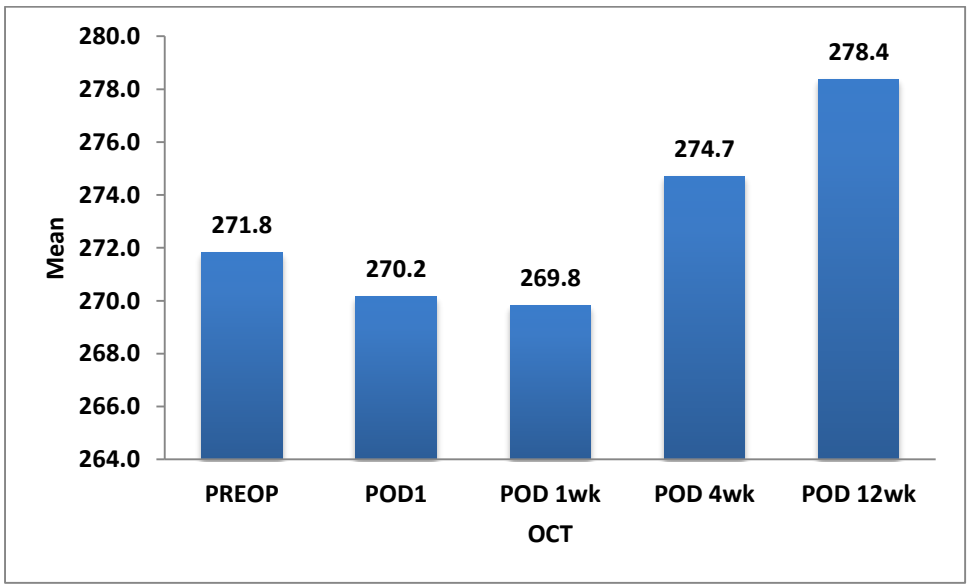
FIG 11 DIFFUSE MACULAR EDEMA

- At 3-month follow-up, 1 patient had diffuse type of macular edema with spongy thickness in a patient of moderate NPDR.

Table16: Distribution of OCT according to POD among Mild NPDR cases

Parameter	PREOP		POD1		p value
	Mean	SD	Mean	SD	
OCT CENTRAL SUB FIELD THICKNESS	271.8	10.8	270.2	8.9	0.656
	POD1		POD 1wk		p value
	270.2	8.9	269.8	13.6	0.933
	POD 1wk		POD 4wk		p value
	269.8	13.6	274.7	14.8	0.167
	POD 4wk		POD 12wk		p value
	274.7	14.8	278.4	15.2	0.163

Graph13: Distribution of OCT according to POD among Mild NPDR cases

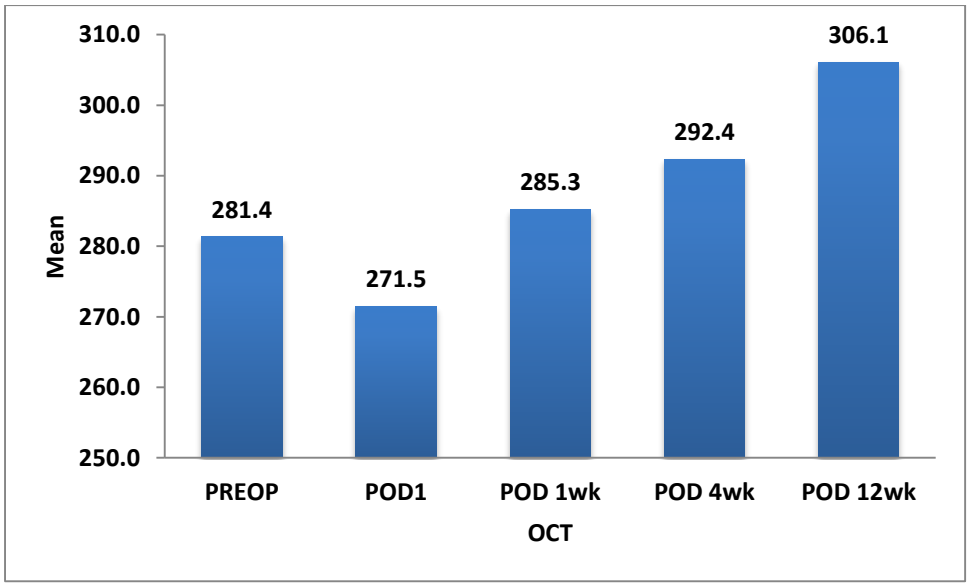


Patients with mild NPDR and moderate NPDR developed thickening but with low level of significance as shown in table 16,17 and graph 13,14.

Table17: Distribution of OCT according to POD among Moderate NPDR cases

Parameter	PREOP		POD1		p value
	Mean	SD	Mean	SD	
OCT CENTRAL SUB FIELD THICKNESS	281.4	6.6	271.5	11.8	0.123
	POD1		POD 1wk		p value
	271.5	11.8	285.3	14.3	0.135
	POD 1wk		POD 4wk		p value
	285.3	14.3	292.4	21.8	0.276
	POD 4wk		POD 12wk		p value
	292.4	21.8	306.1	35.9	0.281

Graph14: Distribution of OCT according to POD among Moderate NPDR cases



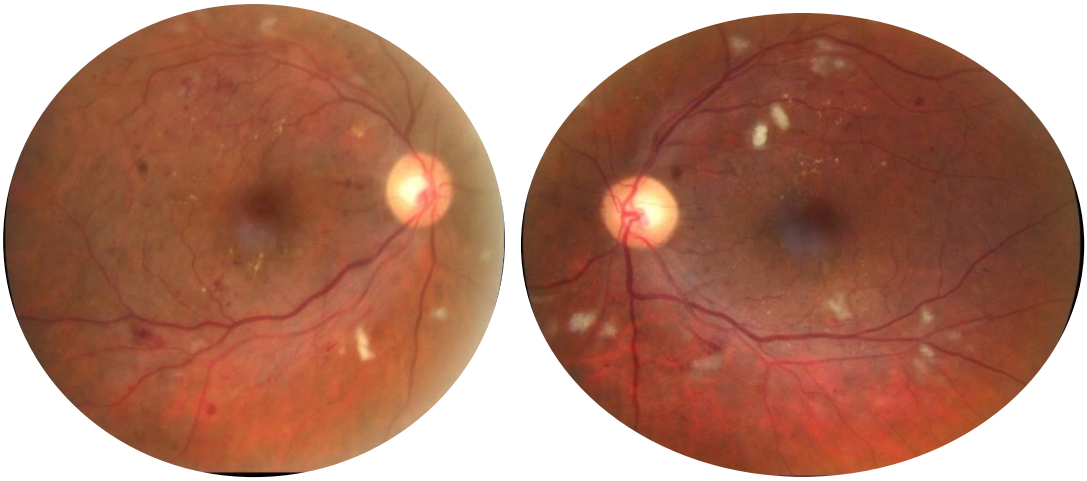


FIG 12 MODERATE NPDR WITH MACULAR EDEMA

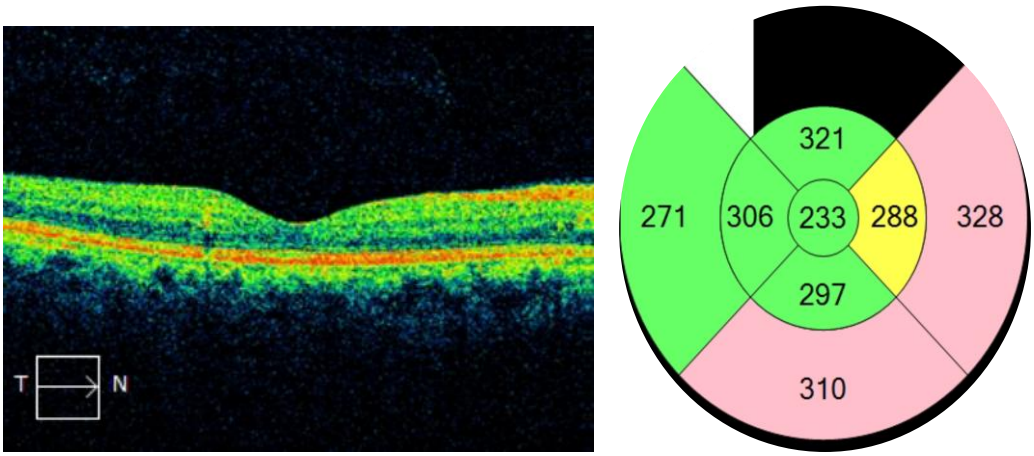


FIG 13 The MACULAR CUBE scan of the same patient in fig12 through the fovea at post operative day 1 where it shows para foveal thickening corelating with juxtafoveal oedema associated with hard exudates in fig 12

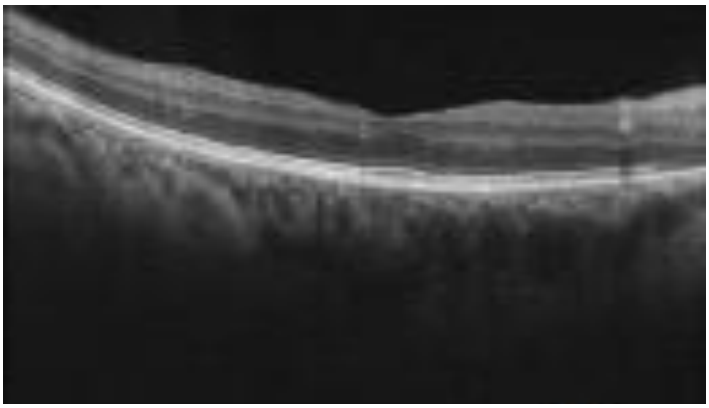


FIG 14 DIFFUSE MACULAR EDEMA

Where as this same patient fig12 devoloped diffuse spongy oedema at the end of 3 month follow up with increse in macular thickness.

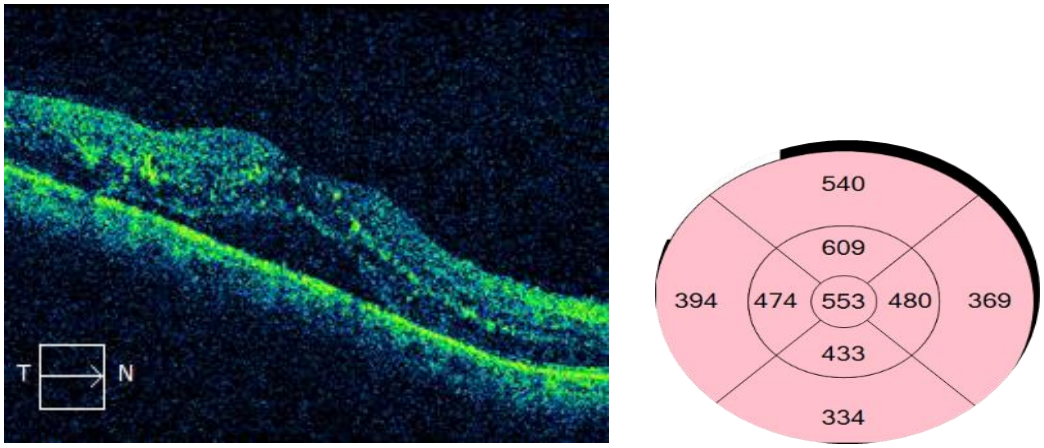


FIG 15 SEROUS RETINAL DETACHMENT AT 4TH WEEK

This is a macular scan of a patient with severe NPDR with severe macular edema where it is showing serous retinal detachment type with foveal thickening and sub retinal fluid accumulation. Cysts are seen in inner nuclear layer. Foveal contour is altered. This edema persisted till 3 months and visual acuity is further deteriorated. And the patient underwent PRP to prevent development of proliferative diabetic retinopathy. And strict metabolic control was advised for this patient

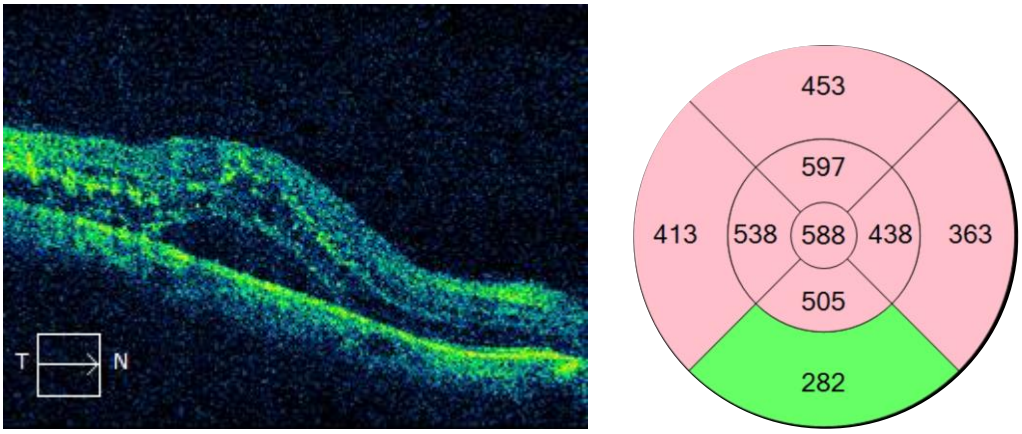


FIG 16 SEROUS RETINAL DETACHMENT AT 12TH WEEK

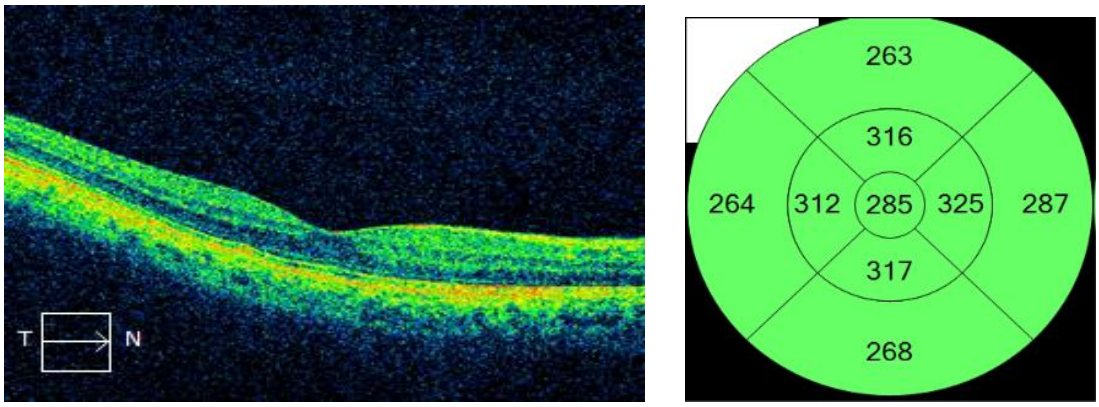
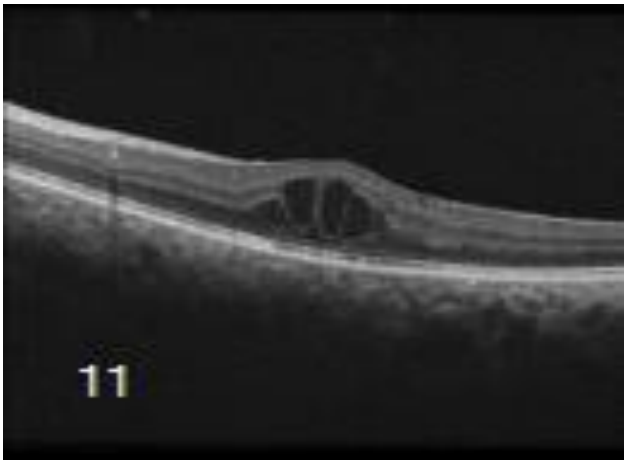


FIG 17 MACULAR CUBE SCAN OF A PRE OP PATIENT WITH MILD NPDR

This a mild NPDR patient with few micro aneurysms where the macular scan was normal with relatively high macular thickness valves.

patient developed cystoid macular edema with partial thickness hypo reflective cysts in inner nuclear layer- outer plexiform layer large cysts in the centre small cysts parafoveally with increase in foveal thickness at the 3 months of follow up.



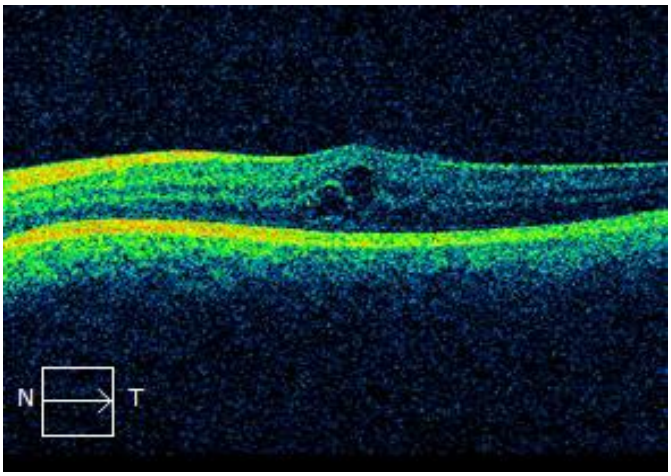


FIG 18 CYSTOID MACULAR EDEMA OF SAME PATIENT AT 3 MONTHS OF F/UP

Fig 19 moderate npdr with macular edema

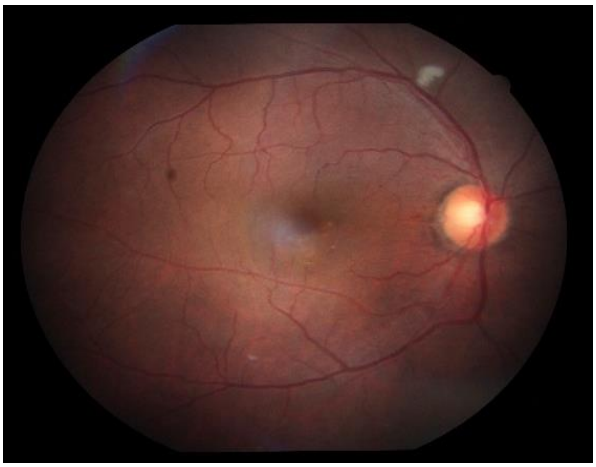
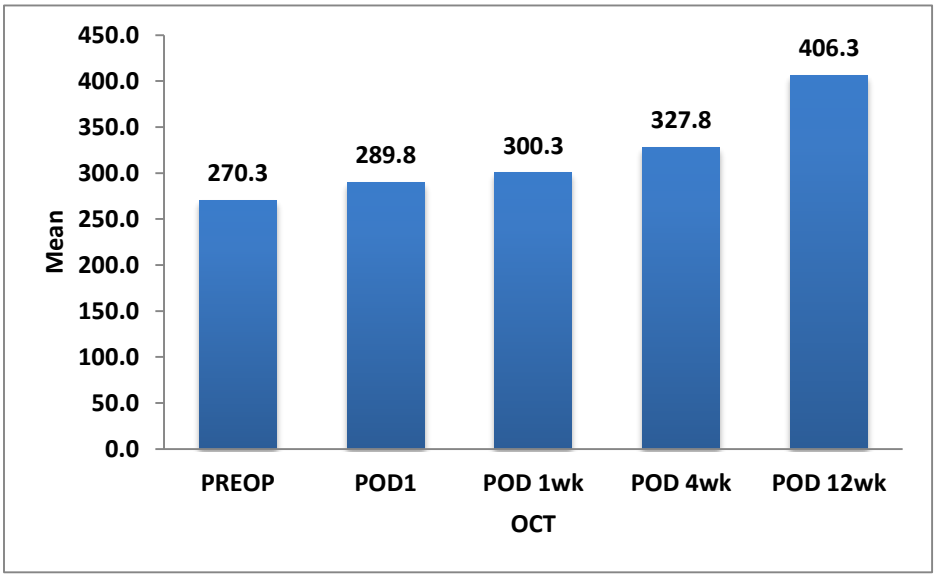


Table18: Distribution of OCT according to POD among Moderate NPDR ME cases

Parameter	PREOP		POD1		p value
	Mean	SD	Mean	SD	
OCT CENTRAL SUB FIELD THICKNESS	270.3	8.8	289.8	23.8	0.275
	POD1		POD 1wk		p value
	289.8	23.8	300.3	21.7	0.458
	POD 1wk		POD 4wk		p value
	300.3	21.7	327.8	44.7	0.128
	POD 4wk		POD 12wk		p value
	327.8	44.7	406.3	77.2	0.044*

Note: * significant at 5% level of significance (p<0.05)

Graph15: Distribution of OCT according to POD among Moderate NPDR ME cases

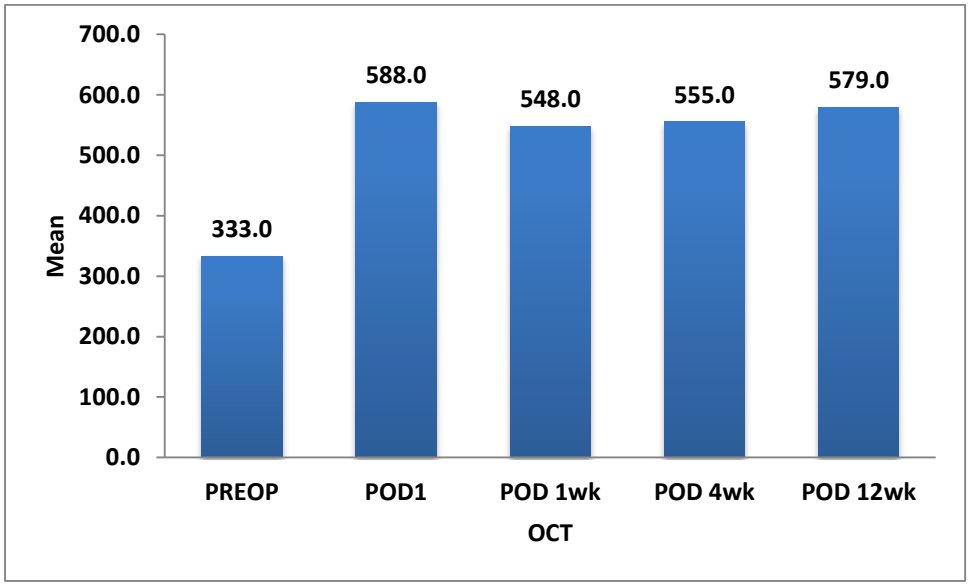


In eyes with preoperative moderate NPDR with macular edema the mean change of central subfield macular thickness was 136µm at 3 months follow up. (P value -0.044) The patients who developed macular edema had decrease in visual acuity

Table19: Distribution of OCT according to POD among Severe NPDR ME cases

Parameter	PREOP		POD1		p value
	Mean	SD	Mean	SD	
OCT CENTRAL SUB FIELD THICKNESS	333.0	0.0	588.0	0.0	-
	POD1		POD 1wk		p value
	588.0	0.0	548.0	0.0	-
	POD 1wk		POD 4wk		p value
	548.0	0.0	555.0	0.0	-
	POD 4wk		POD 12wk		p value
	555.0	0.0	579.0	0.0	-

Graph16: Distribution of OCT according to POD among Severe NPDR ME cases

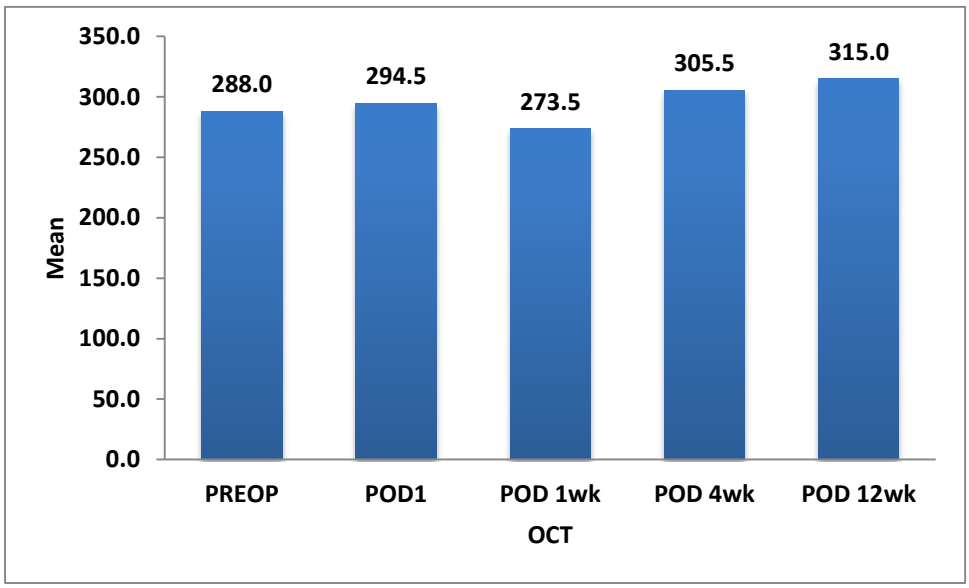


Due to single patient we couldn't quantify the statistical analysis.

Table20: Distribution of OCT according to POD among Macular Edema cases

	PREOP		POD1		p value
	Mean	**SD**	**Mean**	**SD**	
OCT CENTRAL SUB FIELD THICKNESS	288.0	2.8	294.5	4.9	0.144
	POD1		**POD 1wk**		**p value**
	294.5	4.9	273.5	9.2	0.283
	POD 1wk		**POD 4wk**		**p value**
	273.5	9.2	305.5	16.3	0.099
	POD 4wk		**POD 12wk**		**p value**
	305.5	16.3	315.0	1.4	0.586

Graph17: Distribution of OCT according to POD among Macular Edema cases



In eyes with preoperative macular edema the mean change of central subfield macular thickness was 27µm at 3 months follow up.

DISCUSSION

This prospective comparative study was undertaken to assess the effect of small incision cataract surgery with IOL implantation on Central Subfield Macular Thickness on OCT in diabetic patients. As cataract formation is common in people with diabetes, obtaining preoperative and postoperative OCT scans at 4^{th} and 12^{th} weeks allowed us to quantify changes in foveal thickness and to follow the progression. The CSMT was assessed with OCT preoperatively, and at weeks 1, 4 and 12 postoperatively, and comparisons were made.

The macular thickness in diabetic patients with and without DR was increased significantly at the end of the fourth week postoperatively compared to the preoperative results. This thickening persisted until 12 weeks postoperatively in all subjects and did not regress to preoperative levels till the last follow-up at 12 weeks. This study demonstrated that the influence of uncomplicated cataract surgery on CSMT in diabetic patients without diabetic retinopathy did not significantly differ from patients with diabetic retinopathy who are undergoing cataract surgery.

In other words, diabetics without DR and with DR patients showed similar intragroup thickening of the central macular subfield at weeks 4 and 12 after uncomplicated small incision cataract surgery, and the intergroup comparison was not statistically significant.

OCT has been able to demonstrate a moderate correlation between retinal thickness and best-corrected visual acuity, and it has been able to demonstrate 3 basic structural changes of the retina, i.e., diffuse retinal swelling, cystoid macular edema, and serous retinal detachment.

The limitation of this study is the small number of patients and a larger study is required to be done to confirm the results.

Most of the studies in the literature showed [52][53] [54] that mean CMT is statistically significant increase at 1 month after cataract surgery, which was maintained after 3 months, pointing out a possible leakage. Also, baseline CMT was thicker in eyes developing PCME, suggesting that increased CMT thickness may be a predisposing factor for PCME or the presence of subclinical PCME, which may not be detected by OCT imaging systems. This is witnessed in one of our patients. Inflammatory mediators may increase vascular permeability leading to an increase in macular thickness and cyst formation. [55,56] [57] Now a day's use of SD-OCT provides quicker, more objective, and noninvasive assessments of retinal thickness compared to FA. Compared to time-domain OCT, SD-OCT offers more accurate measurements, higher repeatability, and a lower rate of errors and false-negatives.

Duker et al showed that SD-OCT has enabled ophthalmologists to visualize and monitor the vitreomacular interface with better accuracy and repeatability[58].

In this study, similar to other recent studies, OCT was used to evaluate progression of ME following cataract surgery. Prior studies have shown reported rates of ME progression from other methods; such as FA and for example, the proportion of eyes manifesting Angiographic CME was 9% after cataract surgery using fluorescein angiography in people without diabetes[59].

Romero- Aroca et al reported that 6.06% of 132 eyes of diabetic patients developed DME on evaluation by fluorescein angiography and OCT following uneventful phacoemulsification[60].

In our study, level of diabetic retinopathy was associated with increased foveal thickening. The study group with no diabetic retinopathy developed minimal increases in foveal thickening, of 15.2 um and 21.2 um at 4th and 12th week after surgery, respectively. The worse the level of diabetic retinopathy at baseline, the more likely the foveal thickness increased at 4th and 12th week after surgery. The group with mild non proliferative diabetic retinopathy had increase in center point thickness—of 4.4um and 8.4 um at 4th and 12th week after surgery respectively. The group with moderate non proliferative diabetic retinopathy with macular edema had largest increase in foveal thickness 57.5um and 135 um at 4th and 12th week after surgery respectively. But it is not statistically significant in mild and moderate NPDR cases due to small number of patients. This increase in foveal thickness was correlated inversely with VA improvement.

Hayashi et al have shown that the foveal thickness and macular volume in diabetic patients increases after small incision cataract surgery in eyes both with and without DR: the percent increase from baseline was greatest at 3 months after surgery, and then decreased gradually.

Furthermore, the increase in foveal thickness was greater in eyes with DR than in eyes without DR. These results indicate that, on average, diabetic macular oedema worsens after cataract surgery, and the worsening is more pronounced in eyes with DR

Our study showed similar significant correlation between level of retinopathy, foveal thickness but the current study is limited by the duration of follow-up of patients to know the maculopathy progression or regression as described by hayashi.

Alastair K Denniston, Usha Chakravarthy et al reported that rate of developing treatment-requiring DMO increases sharply in the year after cataract surgery for all

grades of retinopathy, peaking in the 3–6 months' postoperative period. Patients with moderate and severe NPDR are at particularly high risk[(1)].

Though our study showed no significant statistical difference between no diabetic retinopathy and mild and moderate diabetic retinopathy, few other studies have shown a significant rise in the CMT postoperatively in diabetic retinopathy patients. Therefore, large-scale studies with a longer follow up period are likely required to accurately elucidate the role of DR status on the postoperative visual prognosis of patients undergoing uncomplicated small incision cataract surgery.

We found that the Mean MFT preoperatively, 1 week, 1 month and 3 months postoperatively in diabetic patients with diabetic retinopathy (mild, moderate and severe NPDR) was not statistically higher compared to patients without DR. but overall irrespective of DR macular thickness is increased There is improvement in visual acuity after 1 and 3 months postoperatively in patient with no DR than those with DR. this finding is consistent with the fact of deficient blood retinal barrier function in those patients with more advanced vascular changes resulting from DR [(61)]. Many previous studies [(62)(9)]showed the high risk of developing macular edema in patients with diabetic retinopathy but most of the studies included the patients with preoperative high macular thickness like ours. But the patients with increased macular thickness are less in our study (10%) to prove it is statistically significant.

Few other Studies have compared outcomes of post cataract surgery between diabetic eyes without retinopathy and non-diabetic eyes. Menchini et al [(63)]conducted a study in 1993 comparing the incidence of CME after extra capsular cataract extraction and intraocular lens implantation in diabetic patients without retinopathy and non -diabetic patients. They found a similar frequency of angiographic CME in the two groups 30

days after surgery, but a significantly higher frequency in the diabetic eyes at 90 days, 180 days, and 360 days. Final visual acuity, however, was similar in both groups. This suggests that, similar to our study, even with the older cataract surgical technique of extra capsular cataract extraction, the short-term macular structure and visual changes were possible in diabetic patients without diabetic retinopathy. Kim et al[(25)] also found only minimal increases in mean central macular thickness in patients with diabetes without retinopathy at 1 month and 3 months post phacoemulsification. The results of another study conducted by Katsimpris et al[(64)] in 2012 and Kim et al.[(25)] Where they found a significant post-phacoemulsification increase (48–78 μm) in CRT from 1 month to 12 months postoperatively in the diabetic without retinopathy eyes compared to the normal controls. The authors implied that the more prominent increase in postoperative CRT may account for the less satisfactory visual results following cataract surgery in the diabetic patients, and even in those without retinopathy. It is difficult to tell why the diabetics without retinopathy patients encountered such poor results in postoperative macular edema, as many conditions during or after the operation may affect the results of cataract surgery.[(52)]however, according to Eriksson et al [15] even in eyes with diabetic retinopathy, the OCT finding of macular change and the inferiority of visual outcome may only be transient in the short-term (6 weeks) post phacoemulsification period.

We used SD OCT for the measurement of macular thickness prior to and after cataract surgery. In the past, only qualitative or semi quantitative measurements of macular thickening by either Slit lamp bio microscopy examinations, 90D or FA could be used for the detection of macular thickness. OCT provides a more detailed qualitative examination of macular morphology, and also it gives us linear quantitative measurements of macular thickness. However, there has been some controversy

regarding whether postoperative macular thickening significantly correlates with postoperative visual outcomes after cataract surgery in normal individuals. One author found a correlation between VA and macular thickness[65] whereas others have not[66][67]

Kim et al [68]found that there is threshold of increase in macular thickening post cataract surgery which associated with clinically impaired visual outcomes. An increase of 40% or more in macular thickness[68] or a morphological Irvine-Gass pattern of cystic changes as detected by OCT[11] could be regarded as a threshold for reporting clinical vision-relevant post cataract macular edema.

In our study, there was no difference in median macular thickness between the groups, and no cases in either group had an increase in macular thickness to reach this threshold. We thus believe that the increases in macular thickness in all of our cases could only be regarded as subclinical changes.

Furthermore, several researchers have reported that increased retinal thickness correlates more strongly with visual acuity than the presence of leakage shown by fluorescein angiography. For these reasons, OCT offers an objective method to identify clinically relevant post cataract ME.

The limitations to this study are small sample size in sub group of patients with varying levels of diabetic retinopathy.

Another major limitation is that we did not compared the long term macular changes and the visual results.

In few patients because of the presence of significant media opacity in many patients interfering with good quality OCT scan. Macular thickness on the first postoperative

day was taken as baseline value in our study as no significant change in the foveal and Peri-foveal thickness values occurs on the first postoperative day[18][56]. Another shortcoming was the absence of control value from un-operated fellow eye. In most cases, fellow eye either had cataract or it was pseudophakic.

Macular thickness and the visual outcomes in diabetic patients without retinopathy and with retinopathy were good in both the groups for up to 4 weeks after small incision cataract surgery.

There is 2 or 3 line drop in visual acuity on snellens chart in patients who had increased macular thickness as PCME and DME at 3 months of follow up.

SUMMARY

A prospective comparative hospital based study conducted From October 2018 to April 2020. A sample of 65 diabetic patients undergoing cataract surgery in the dept. of ophthalmology in Shri B. M. Patil Medical College, were selected to assess the quantitative increase in macular thickness using Spectral Domain optical coherence tomography (OCT)

The results of the current study demonstrated that Central subfield macular thickness on OCT is increased after uncomplicated small incision cataract surgery both at 1 month and 3 months postoperatively in diabetic patients with and without DR; the difference between the two groups is not statistically significant.

There is statistically significant increase is seen in all diabetic patients undergoing cataract surgery irrespective of presence or absence of diabetic retinopathy at 1 month and 3 months of cataract surgery. The mean and standard deviation preoperative value was 267.6±16.9µm. At the 4^{th} week review, macular thickness had increased to 285.2±42.2µm (P-0.029*). At the 12th week review, the macular thickness was 296.6±55.7µm with a P valve of 0.002*($p<0.05$).

There is a statistically significant increase of central subfield macular thickness on OCT is seen in patients with no diabetic retinopathy with a mean preoperative value of 255.9±12.0µm which is increased to 277.1±12.2µm. with a P-valve of 0.030*($p<0.05$)

Where as in patients with mild NPDR and moderate NPDR there is significant increase in central subfield macular thickness and it was mild increase with less sample size and the raise in thickness was not statistically significant. In mild NPDR

patients mean preoperative value is 271.8±10.8 µm which is increased to 278.4±15.2 µm with a P value - 0.163 ($p<0.05$)

In patients with moderate NPDR and macular edema had highest increase in macular thickness with mean preoperative value of 289.8± 23.8 µm and at the end of follow-up the mean post-operative macular thickness at 12 weeks is 406.3±77.2 µm which is also proved statistically significant in our study with a P value-0.044*.($p<0.05$)

There was only one patient with severe NPDR and severe macular edema who underwent cataract surgery and developed serous retinal detachment with sub retinal fluid post 1 month of cataract surgery. This patient had poor visual outcome. Because of single patient we couldn't quantify statistical results

In conclusion, we have demonstrated that the diabetic macular edema generally worsens after cataract surgery, and that the worsening of macular edema is more prominent in eyes with DR. and the change in patients without diabetic retinopathy is subclinical without affecting the visual acuity.

Therefore by analyzing macula using OCT in our study we were able to diagnose minimal edema at earliest, we can conclude that OCT plays a major role in earlier management and visual acuity recovery in diabetic patients undergoing cataract surgery.

It is postulated that good diabetes control is needed to prevent an increase in CMT and postoperative macular edema after uncomplicated uneventful small incision cataract surgery. However, long term follow-up studies may be needed to formulate management protocol.

CONCLUSION

OCT has gained widespread popularity in detection of macular changes in various diseases. It allows early, accurate diagnosis and better follow-up.

It is a safe, noninvasive, and quick procedure which provides objective documentation of foveal, retinal and optic nerve head morphology.

In some situations, it is an alternative to FA in the follow-up of changes in retinal thickness. But now OCT is totally replacing FA except in few circumstances.

Our study shows that Patients with diabetes need a preoperative characterization of their retinopathy before cataract surgery, and advised about the risk of progression of retinopathy after surgery. To provide patients with DR the benefits of cataract surgery and avoiding the progression of macular edema it is advised that all patients with DR should be evaluated with OCT, particularly in the early postoperative period to detect macular changes, so that early diagnosis timely adequate management can be ensured. In our study patterns of macular edema seen in diabetics are DRT, CME, SRD, and PHT without TRD.

All diabetic patients need close observation for at least 6 months following surgery to intervene with laser photocoagulation and anti VEGF as and when required to prevent visual loss from diabetic maculopathy and other consequences of diabetic retinopathy.

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ANNEXURES

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103

IEC/NO:286/2018
17-11-2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : Comparative study of optical coherence tomographic analysis of macula in preoperative and postoperative diabetic patients undergoing small incision cataract surgery.

Name of P.G. Student : Dr Marati Shruthi
Department of Ophthalmology,

Name of Guide/Co-investigator: Dr.Vallabha.K, Professor & HOD of Ophthalmology,

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:

1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

SAMPLE INFORMED CONSENT FORM

TITLE OF THE PROJECT : **COMPARITIVE STUDY OF OPTICAL COHERENCE TOMOGRAPHIC ANALYSIS OF MACULA IN PREOPERATIVE AND POSTOPERATIVE DIABETIC PATIENTS UNDERGOING SMALL INCISION CATARACT SURGERY**

PG GUIDE : **DR. VALLABHA.K DOMS, M.S.**

PROFESSOR AND HEAD OF THE
DEPARTMENT OF OPHTHALMOLOGY
BLDE (DU)'S SHRI B.M.PATIL

MEDICAL COLLEGE, HOSPITAL AND
RESERCH CENTRE, VIJAYAPURA

PRINCIPAL INVESTIGATOR : **DR. MARATI.SHRUTHI**

DEPARTMENT OF OPHTHALMOLOGY

BLDE (DU)'S SHRI B.M.PATIL

MEDICAL COLLEGE, HOSPITAL AND

RESERCH CENTRE, VIJAYAPURA

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study comparitive study of optical coherence tomographic analysis of macula in preoperative and postoperative diabetic patients undergoing small incision cataract surgery. I understand and accept the risks, benefits and regarding the costs involved. I willingly give consent to take part in the study.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study to **Dr. VALLABHA.K** in the Department of Ophthalmology who will be available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that **Dr. Marati. Shruthi** may terminate my participation in the study after she has explained the reasons for doing so.

INJURY STATEMENT:

I understand that if I promptly report about any injury which occurred to me due to any participation in this study, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr. Marati Shruthi. Date
(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr. Marati Shruthi** has explained to me the purpose of research, the study procedure, that I will undergo and the possible discomforts as well as benefits that I may experience in my own language. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to signature) Date

PROFORMA FOR EXAMINATION

COMPARITIVE STUDY OF OPTICAL COHERENCE TOMOGRAPHIC ANALYSIS OF MACULA IN PREOPERATIVE AND POSTOPERATIVE DIABETIC PATIENTS UNDERGOING SMALL INCISION CATARACT SURGERY

NAME : AGE: SEX: IPno

CHIEF COMPLAINTS and HISTORY OF PRESENTING ILLNESS:

PAST HISORY OF OCULAR PROBLEMS:

TREATMENT HISTORY:

GENERAL PHYSICAL EXAMINATION and SYSTEMIC EXAMINATION:

H/O DIABETES and DURATION:

TREATMENT:

FBS/PPBS/RBS/HbA1c: H/O HYPERTENSION: TREATMENT:

ANY OTHER SYSTEMIC COMPLICATIONS: OCULAR EXAMINATION:

VISUAL ACUITY: DISTANCE: PINHOLE:

NEAR:

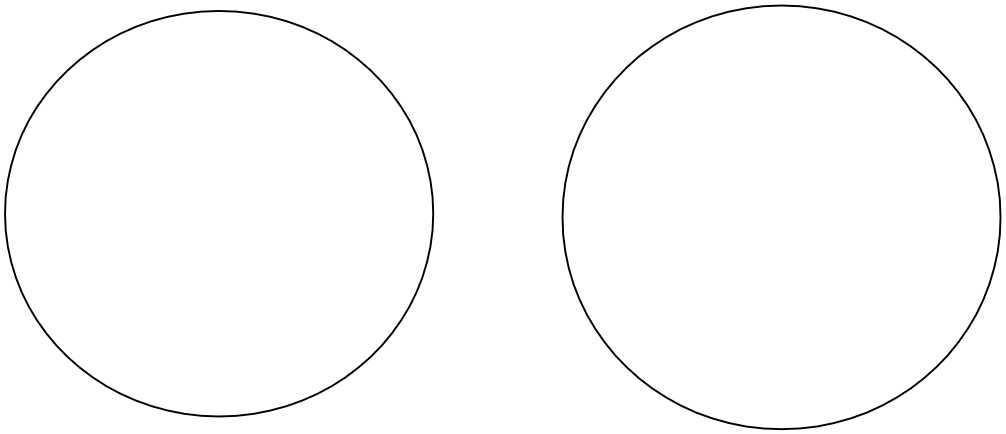
REFRACTION and CORRECTION (BCVA):

ANTERIOR EXAMINATION BY SLIT LAMP:

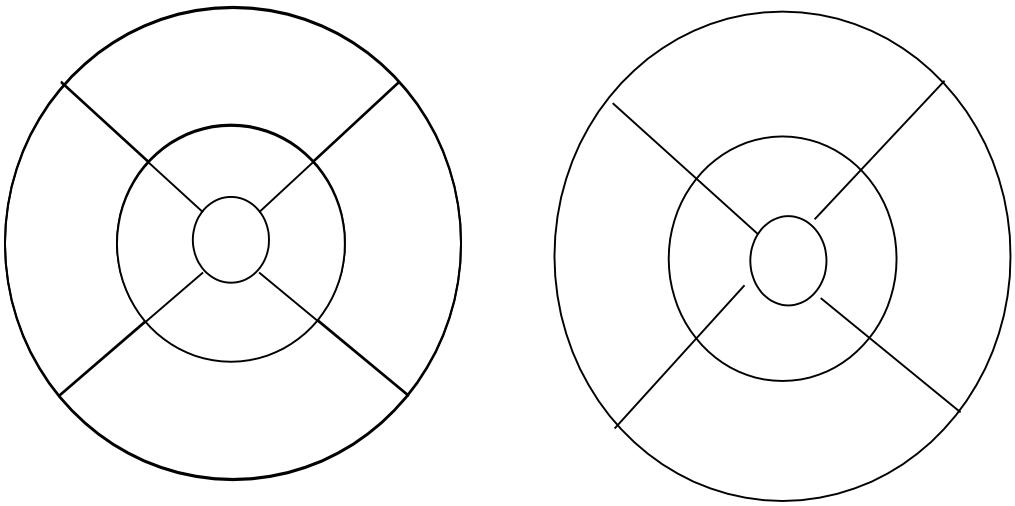
IOP (APPLANATION TONOMETRY): GONIOSCOPY:

FUNDUS EXAMINATION (DO, IDO, 90D, 78D):

RE LE



OPTICAL COHERENCE TOMOGRAPHY (OCT): OCT 1 ON: PRE-OP



INTERPRETATION:

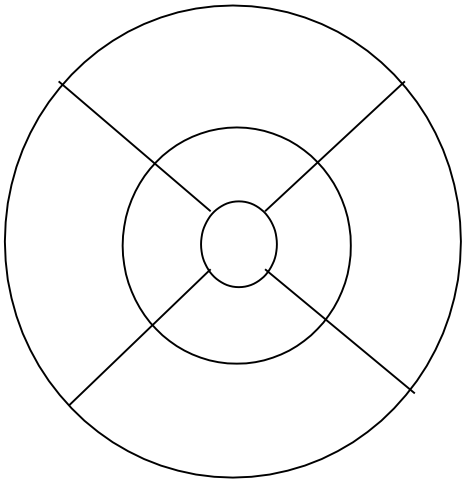
DIAGNOSIS:

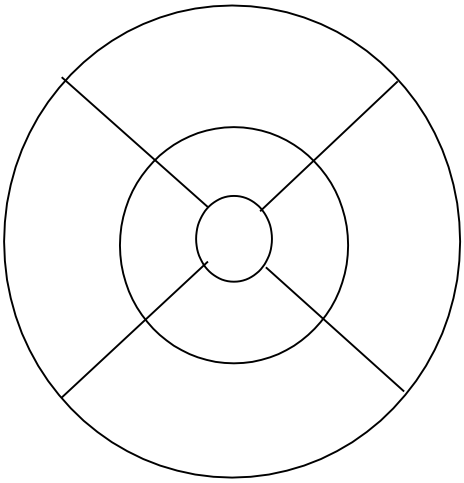
DATE OF OPERATION: TYPE OF SURGERY

VISUAL ACUITY:

DISTANCE: PINHOLE: NEAR:

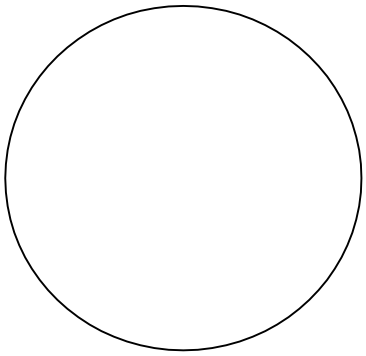
OCT 2 ON: DAY 1





INTERPRETATION: ANTERIOR SEGMENT: FUNDUS:

RE



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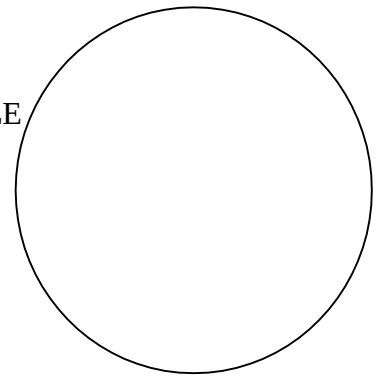
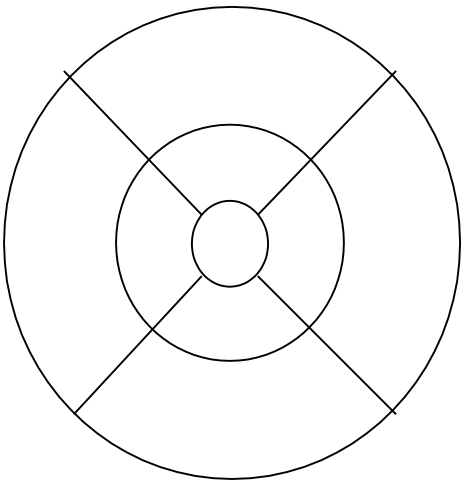


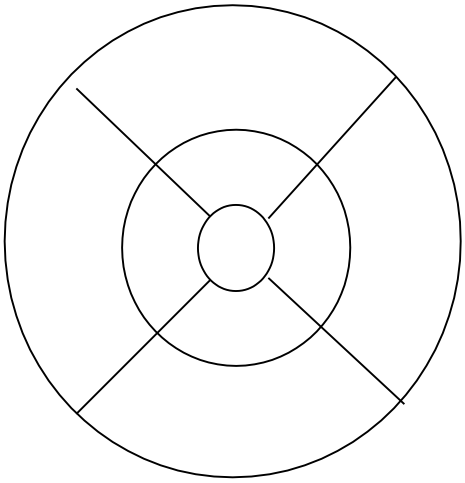
DATE OF DISCHARGE:

TREATMENT ON DISCHARGE:

FOLLOW UP VISITS:

OCT 3 ON: 1ST WEEK





INTERPRETATION:

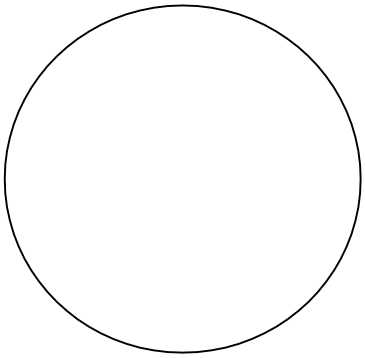
VISUAL ACUITY:

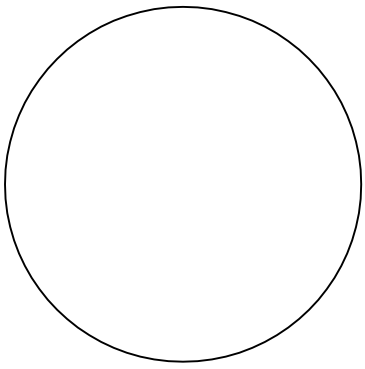
DISTANCE: PINHOLE: NEAR:

ANTERIOR SEGMENT:

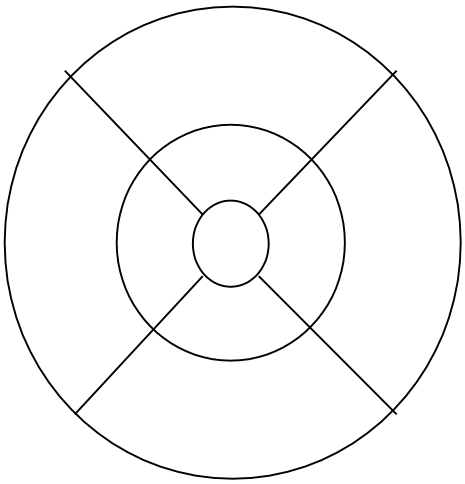
FUNDUS:

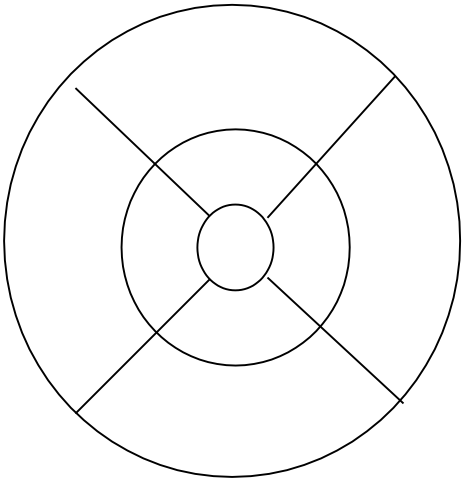
RE LE





OCT 4 ON: 4th WEEK





INTERPRETATION:

VISUAL ACUITY:

DISTANCE: PINHOLE: NEAR:

ANTERIOR SEGMENT:

FUNDUS:

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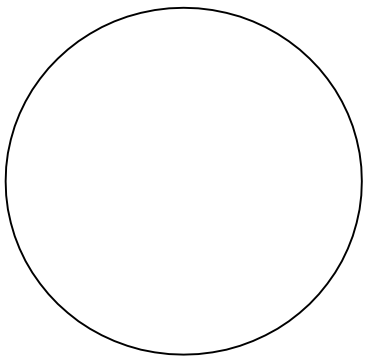
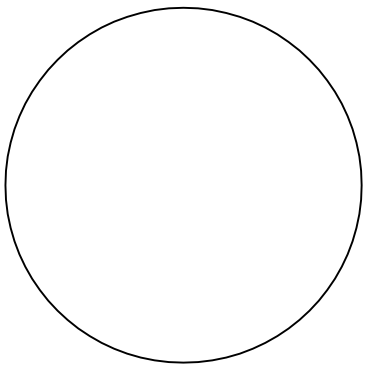
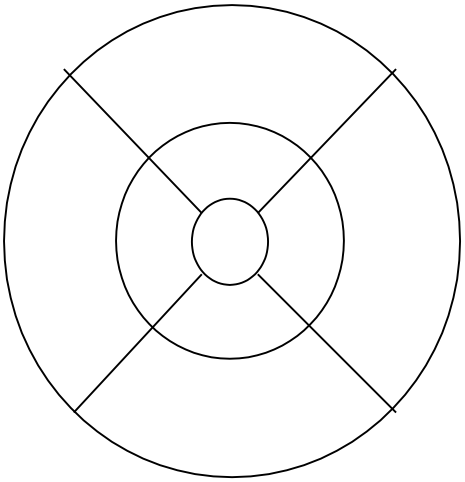
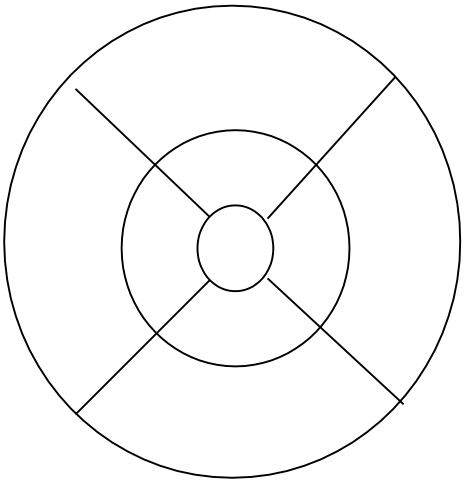




OCT 5 ON: 12th WEEK





INTERPRETATION:

VISUAL ACUITY:

DISTANCE: PINHOLE: NEAR:

ANTERIOR SEGMENT:

FUNDUS:

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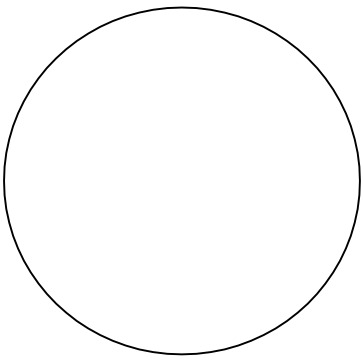
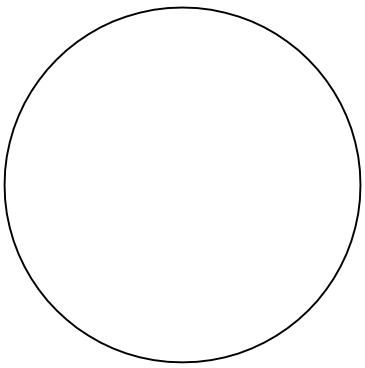
RE





KEY TO MASTER CHART

Sr. No. – Serial number

IP no. – In Patient number

M – Male

F – Female

FBS -FASTING BLOOD SUGAR

PPBS- POST PRANDIAL BLOOD SUGAR

BCVA - Best Corrected Visual Acuity

Cf - counting fingers

CF-CF – Counting finger close to face

CSMT – Central subfield macular thickness

DM- Diabetes Mellitus

HTN- hypertension

LE Left Eye

RE Right Eye

PL – Perception of light

PR – Perception of rays

Severe NPDR- Severe non proliferative diabetic retinopathy

Mild NPDR -Mild non proliferative diabetic retinopathy

Moderate NPDR- moderate non proliferative diabetic retinopathy

Moderate NPDR ME - Moderate non proliferative diabetic retinopathy Macular edema

ME- Macular edema

Severe NPDR

Pod - postoperative day

SEVERE NPDR ME - Severe non proliferative diabetic retinopathy Macular edema

MASTER CHART

S.No	NAME	AGE	SEX	IP No.	DM(Y/N)	FBS	PPBS	GRBS	Medication	HTN/others	Diagnosis	BCVA	BCVA	BCVA	BCVA	BCVA	Diabetic Retinopathy	OCT CENTRAL SUB FIELD THICKNESS				
												preop	POD 1	pod1 WK	pod 4 wks	pod 12 wks		PREOP	POD1	POD1 WK	pod4wks	POD 12 WKS
1	**Vittabai Biradar**	65	FEMALE	21749	6 MONTHS	111	140	157	**oral medications**	10 YEARS	LE SIMC	6/60	6/12	6/9	6/9	6/9	NO	253	259	258	250	267
2	**Sangappa**	77	MALE	70	4YEARS	72	140	126	INSULIN	NO	LE SIMC	6/36	6/12	6/12	6/9	6/9	NO	261	269	266	265	265
3	**Basamma**	75	FEMALE	20405	2 YEARS	91	153	178	**oral medications**	HTN 2 YEARS	RE SIMC	CF-2M	6/18	6/18	6/12	6/12	NO	253	249	286	278	282
4	**Sidram**	82	MALE	17004	5 YEARS	83	137	134	**oral medications**	NO	LE SIMC	CF-1M	6/12	6/12	6/9	6/6	NO	260	275	281	268	283
5	**Shyamu Rathod**	79	MALE	21433	1 MONTH	87	142	128	INSULIN	NO	LE SIMC	CF-3M	6/18	6/12	6/12	6/12	NO	251	274	249	275	280
6	**Lakkappa**	85	MALE	26355	10 YRS	72	137	145	**oral medications**	NO	RE SIMC	CF-3M	6/12	6/9	6/6	6/6	NO	253	260	276	267	286
7	**Karabasappa**	71	MALE	20943	DM 10 YRS	84	143	140	INSULIN	HTN 10 YRS	LE SIMC	HM+	.6/18	6/12	6/12	6/12	NO	263	251	265	275	281
8	**Kamalabai**	56	FEMALE	32993	DM 5 YRS	72	120	190	**oral medications**	HTN 1YR	LE SIMC	6/36P	6/18	6/12	6/9	6/9	NO	248	274	280	280	288
9	**Shivappa Bagewadi**	70	MALE	21810	DM 5 YRS	79	124	119	**oral medications**	HTN 3 YRS	RE SIMC	6/60	6/18	6/24	6/24	6/18	NO	249	257	256	266	275
10	**Kabir**	60	MALE	21799	DM 6YRS	82	128	125	**oral medications**	HTN 5 YR	RE SIMC	6/60	6/12	6/9	6/12	6/9	NO	246	255	264	275	257
11	**Shivkumar**	62	MALE	20397	DM 4YRS	93	132	96	**oral medications**	HTN 62YR	LE SIMC	6/60	6/20	6/24	6/9	6/9	NO	247	282	280	269	269
12	**Abdul Nasir**	48	MALE	30513	DM 3YRS	86	123	169	**oral medications**	NO	RE SIMC	CF-1M	6/12	/6/9	6/9	6/9	NO	249	282	287	275	262
13	**Mehaboob**	64	MALE	26345	7YEAR	99	129	78	**oral medications**	NO	LE SIMC	6/36	6/18	6/18	6/12	6/12	NO	250	286	287	260	283
14	**Brahamanand Biradar**	68	MALE	2368	8YEARS	77	147	146	**oral medications**	NO	RE SIMC	6/60	6/12	6/12	6/12	6/12	NO	249	287	272	270	284
15	**Siddanna**	62	MALE	33907	2YEAR	81	163	135	INSULIN	2 YRS	LE SIMC	6/24	6/6	6/6	6/6	6/12	NO	249	277	270	269	282
16	**Nandappa**	65	MALE	19450	1YEAR	73	128	106	INSULIN	2 YRS	RE SIMC	6/60	6/18	6/9	6/9	6/9	NO	247	237	268	268	288
17	**Gangabai**	65	FEMALE	31621	DM (4YRS)	94	120	155	**oral medications**	HTN 2 YRS	RE SIMC	HM+	6/18	6/24	6/12	6/24	NO	263	287	269	274	282
18	**Yallawwa**	70	FEMALE	22827	DM 3 YRS	91	173	193	**oral medications**	HTN 3 YRS	RE SIMC	PL+ PR+	CF5M	CF 4M	CF 2M	cf 2m	SEVERE NPDR ME	333	588	548	555	579
19	**Mahadev Daddimani**	70	MALE	22105	DM 1 YEAR	99	122	98	**oral medications**	NO	RE SIMC	CF-1M	CF-4M	6/24	6/18	6/24	MODERATE NPDRME	263	325	318	339	393
20	**Kamalabai**	56	FEMALE	32993	4YEAR	120	160	190	INSULIN	NO	RE SIMC	CF-CF	6/24	6/18	6/24	6/18	MODERATE NPDR	287	279	279	275	273
21	**Rambabai**	55	FEMALE	36969	7YEAR	87	124	88	INSULIN	NO	LE SIMC	CF-1M	6/12	6/12	6/9	6/9	MODERATE NPDR	287	276	268	290	260
22	**Yashwanth**	65	MALE	2320	2YEAR	83	161	136	**oral medications**	NO	RE SIMC	CF-2M	6/9	6/9	6/6	6/24	MODERATE NPDR	277	287	276	266	299
23	**Arjun Kamble**	68	MALE	31464	4YEAR	78	126	155	**oral medications**	NO	LE SIMC	CF-3M	6/18	6/18	6/18	6/18	MODERATE NPDR	273	277	279	274	331
24	**Prabhavathi**	64	FEMALE	37794	1 YEAR	83	141	153	**oral medications**	NO	RE SIMC	CF-2M	6/18	6/6	6/9	6/9	MODERATE NPDR	273	278	285	292	267
25	**Javed Mahammed**	48	MALE	38321	DM 4 YRS	92	132	96	**oral medications**	HTN 4 YEARS	LE SIMC	CF-2M	6/12	6/18	6/24	6/60	MODERATE NPDRME	267	273	320	386	499
26	**Satyawwa**	61	FEMALE	21045	DM 6 YRS	71	121	146	INSULIN	3 YRS	LE SIMC	CF-CF	6/12	6/12	6/18	6/60	MODERATE NPDRME	283	279	283	299	421
27	**Sharanappa**	60	MALE	36969	DM 3 YRS	91	149	139	**oral medications**	NO	RE SIMC	CF-1M	6/24	6/12	6/9	6/24	MODERATE NPDRME	268	282	280	287	312
28	**Sateppa**	69	MALE	4180	DM 3 YRS	72	155	120	**oral medications**	NO	LE SIMC	CF -2M	6/12	6/9	6/6	6/6	NO	285	280	274	275	264
29	**Kasturibai**	64	FEMALE	4162	DM 3 YRS	92	161	96	INSULIN	NO	RE SIMC	CF -3M	6/24	6/12	6/9	6/6	NO	254	253	276	281	274
30	**Nimvbewwa**	60	FEMALE	5052	DM 3 YRS	97	130	96	**oral medications**	NO	LE SIMC	CF -1 M	6/18	6/12	6/12	6/9	NO	275	276	270	281	279
31	**Mahadev Biradar**	65	MALE	22108	2 YEARS	75	162	116	INSULIN	HN 3 YRS	RE SIMC	CF-3M	6/24	6/18	6/12	6/12	MILD NPDR	283	275	264	276	297
32	**Mahadev Daddimani**	67	MALE	2367	DM 1 YEAR	79	130	83	**oral medications**	NO	LE SIMC	CF -3M	6/24	6/18	6/18	6/18	MILD NPDR	248	288	246	265	277
33	**Krishnachand**	70	MALE	21800	DM 6 YRS	72	132	175	INSULIN	HTN 6 YR	RE SIMC	CF -2M	6/12	6/18	6/18	6/18	MILD NPDR	289	263	247	263	269
34	**Kasturibai**	67	FEMALE	2466	3	72	157	99	**oral medications**	NO	LE SIMC	CF-CF	6/12	6/18	6/9	6/9	MILD NPDR	271	286	277	286	291
35	**Rakmabai Halagunaki**	75	FEMALE	42308	4	98	134	132	**oral medications**	HTN 4 YRS	RE SIMC	CF-2M	6/12	6/9	6/6	6/24	MILD NPDR	271	275	272	266	288
36	**Yashwanth**	49	MALE	37794	2	90	138	102	**oral medications**	HTN 1 YRS	LE SIMC	CF-4M	6/12	6/12	6/12	6/6	MILD NPDR	275	276	271	293	289
37	**Dondiba Bhimanna**	73	MALE	30056	1	81	138	97	INSULIN	HTN 3YRS	LE SIMC	CF-3M	6/24	6/18	6/9	6/6	MILD NPDR	280	266	269	288	293
38	**Prabhuling Pattar**	52	MALE	43129	2	90	129	164	INSULIN	HTN 2 YRS	LE SIMC	CF-4M	6/24	6/18	6/9	6/12	MILD NPDR	264	274	283	292	274
39	**Yammunawwa**	76	FEMALE	23680	3	77	165	98	INSULIN	HTN 2 YRS	RE SIMC	CF-3M	6/24	6/18	6/9	6/9	MILD NPDR	287	269	274	293	296
40	**Gangubai Hadimani**	62	FEMALE	15022	DM 9YRS	75	170	98	INSULIN	2 YRS	RE SIMC	CF-1M	6/24	6/18	6/9	6/6	MILD NPDR	263	265	275	287	276
41	**Rudrappa**	65	MALE	6064	2	93	131	119	**oral medications**	0	LE SIMC	CF-5M	6/12	6/12	6/12	6/9	MILD NPDR	268	265	285	278	286
42	**Hemalatha**	65	FEMALE	5950	4	90	162	152	INSULIN	0	RE SIMC	6/60	6/12	6/12	6/12	6/6	MILD NPDR	265	272	264	274	289
43	**Ratnabai**	75	FEMALE	6240	5	77	128	109	**oral medications**	2	LE SIMC	CF-3M	6/18	6/9	6/9	6/12	NO	268	288	281	272	278
44	**Kallappa**	60	FEMALE	6052	7	88	165	176	INSULIN	2	RE SIMC	CF-2M	6/18	6/18	6/12	6/9	NO	278	291	265	280	263
45	**Ashok**	74	MALE	6214	9	86	133	141	**oral medications**	NO	LE SIMC	CF-1M	6/18	6/12	6/12	6/12	NO	244	286	283	278	292
46	**Shantawwa**	60	FEMALE	5379	4	86	144	119	**oral medications**	2	RE SIMC	6/60	6/18	6/18	6/12	6/12	NO	231	250	273	279	276

47	**Parasappa walikar**	70	MALE	79	1	80	121	93	**oral medications**	6	RE SIMC	6/60	6/18	6/18	6/18	6/18	NO	271	267	303	290	310
48	**Ramachandra Madar**	70	MALE	608	9	94	133	117	**oral medications**	3	RE SIMC	CF-1M	6/12	6/12	6/12	6/24	MODERATE NPDR	287	264	307	297	345
49	**Gourabai birada**	65	FEMALE	2577	3	98	148	122	INSULIN	NO	LE SIMC	CF-3M	6/24	6/24	6/12	6/18	MODERATE NPDR	288	252	281	314	347
50	**Ranganna madar**	70	MALE	3241	5	75	128	122	**oral medications**	4	LE SIMC	CF-1M	6/18	6/12	6/12	6/12	MILD NPDR	259	255	288	284	276
51	**Bhimappa**	65	MALE	3541	11	70	146	120	**oral medications**	NO	LE SIMC	CF-CF	6/12	6/24	6/24	6/12	MILD NPDR	269	258	266	269	277
52	**Siddappa hojamani**	74	MALE	3554	11	75	143	118	**oral medications**	3	RE SIMC	CF-4M	6/24	6/24	6/24	6/18	MILD NPDR	286	269	276	279	275
53	**Bhimsingh rathod**	60	MALE	3547	6	72	140	116	**oral medications**	NO	LE SIMC	CF-1M	6/24	6/24	6/24	6/12	MILD NPDR	273	261	241	243	237
54	**danawwa**	75	FEMALE	5056	5	72	128	121	**oral medications**	6	RE SIMC	CF-3M	6/18	6/12	6/12	6/9	MILD NPDR	264	279	286	251	255
55	**Awanna kori**	70	MALE	5051	11	100	126	191	INSULIN	3	RE SIMC	CF-4M	6/36	6/24	6/24	6/12	MILD NPDR	278	267	273	258	266
56	**Itabai**	62	FEMALE	5079	11	70	150	94	**oral medications**	5	LE SIMC	CF-1M	6/24	6/18	6/12	6/9	NO	257	272	270	248	254
57	**Maruthi**	60	MALE	5083	5	93	123	86	INSULIN	5	RE SIMC	CF-2M	6/24	6/24	6/18	6/12	MACULAR EDEMA	290	298	267	294	316
58	**Draupadi shinde**	70	FEMALE	5094	4	70	140	85	**oral medications**	NO	LE SIMC	CF-3M	6/60	6/60	6/36	6/24	MACULAR EDEMA	286	291	280	317	314
59	**Dayanand loni**	63	MALE	5456	9	99	150	152	**oral medications**	NO	RE SIMC	CF-3M	6/24	6/18	6/18	6/18	MODERATE NPDR	279	259	307	331	327