"A ONE YEAR PROSPECTIVE STUDY OF VISUAL AND ANATOMICAL OUTCOME OF PARS PLANA VITRECTOMY FOR PROLIFERATIVE DIABETIC RETINOPATHY ADMITTED AT KLES DR. PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI".

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ACCEPTANCE LETTER

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LIST OF ABBREVIATIONS USED

DM	Diabetes Mellitus
DR	Diabetic Retinopathy
PDR	Proliferative Diabetic Retinopathy
DME	Diabetic Macular Edema
ETDRS	Early Treatment of Diabetic Retinopathy Study
iBRB	Inner Blood Retinal barrier
IRMA	Intra Retinal Microvascular Abnormalities
NPDR	Non-Proliferative Diabetic Retinopathy
AGEs	Advanced Glycation End products
DCCT	Diabetes Control Complications Trial
VEGF	Vascular Endothelial Growth Factor
PPV	Pars Plana Vitrectomy
NVD	Neovascularization on the Disc
NVE	Neovascularization Else where
PRPC	Pan Retinal Photocoagulation
PDGF	Platelet Derived Growth Factor
EPC	Endothelial Progenitor Cells
DRVS	Diabetic Retinopathy Vitrectomy Study
UKPDS	United Kingdom Prospective Diabetes Study

NVA	Neovascularization on the Angle
TRD	Tractional Retinal Detachment
FVM	Fibrovascular Membrane
BIOM	Binocular Indirect Ophthalmo microscope
EBIOS	Erected Image Binocular Ophthalmo microscope
ILM	Internal Limiting Membrane
FAE	Fluid Air Exchange
EL	Endolaser
SOI	Silicone Oil Injection
VH	Vitreous Hemorrhage
NVG	Neovascular Glaucoma
RRD	Rhegmatogenous Retinal Detachment
CSME	Clinically Significant Macular Edema
ICMR	Indian Council of Medical Research
PEDF	Pigment Epithelium Derived Factor
PVD	Posterior Vitreous Detachment
CKD	Chronic Kidney Disease
IHD	Ischemic Heart Disease

ABSTRACT

Purpose:

The purpose of this study was to assess the visual and anatomical outcomes of Pars plana vitrectomy for Proliferative Diabetic retinopathy patients and to assess the risk factors for poor visual outcome.

Methods:

This was a one-year prospective, longitudinal, interventional, non-comparative hospital-based study carried out over a period of one year. 30 eyes of 23 patients of who have been recommended to undergo Pars plana vitrectomy surgery for advanced PDR meeting the inclusion criteria and after thorough pre-operative examination.

Results:

30 eyes of 23 patients underwent Pars plana vitrectomy. There was significant improvement in visual acuity with preoperative log mar acuity of 1.72±0.52 improving to 1.02±0.67 ($p<0.0001$). There were 22(73.33%) eyes with initial VA of <6/60 and at the end of our study this number came down to 10eyes (33%) ($P=0.0032$ by Mc Nemara test) which was statistically significant. Out of the 30 eyes 26 eyes showed improvement in visual acuity, two eyes showed no change in visual acuity and two eyes showed worsening of visual acuity**.** Age of the patient, indication of surgery, combined or non-combined surgery were not found to be significant in predicting poor visual outcome. However initial VA of <6/60, receival of preoperative injection of bevacizumab and development of retinal tears were significant factors in predicting poor final VA of <6/60.

There were only two eyes with retinal detachment at the end of follow up. Three patients had some residual vitreous hemorrhage at the end of the study. Three patients had post operative tractional RD in which two patients needed re-surgery because the macula was involved in them. None of the patients developed neovascular glaucoma or phthisis. One of the patients who underwent resurgery needed silicone oil tamponade. However, three patients had residual TRD under silicone oil at the end of follow up. Some epiretinal membrane that did not affect the vision was noted in 10 patients (33.33%).

Conclusion:

This study showed that undergoing Pars plana vitrectomy gave a good over all result with a success rate of 93.33% flat retina and there was a significant improvement in visual acuity with preoperative log mar acuity of 1.72±0.52 improving to 1.02±0.67 ($p<0.0001$), at the end of three months of study. However significant number still had poor visual outcome of final VA<6/60 mainly due to advanced PDR in many cases. Initial VA of<6/60, intraoperative retinal tears and preoperative injection of bevacizumab were risk factors for poor visual outcome of final VA of <6/60.

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INTRODUCTION

All of us, from individuals to families to entire civilizations, are affected by diabetes in one way or another. As one of the 10 leading causes of adult mortality, it was anticipated that 4 million people died from it worldwide in 2017[(1)].Nearly 8.5% of adults above 18 years were suffering from diabetes mellitus in the world as in 2014.463 million people aged 20-79 are living with diabetes mellitus as in 2019. According to estimates, 642 million people worldwide will be diabetic by 2040.[(2)]

Diabetes mellitus is expected to affect up to 79.4 million people in India by 2030.7.3% percent of population aged more than 20 years had diabetes mellitus according to ICMR survey in 15 states in India.[(3)]

The major factor leading to blindness in the working population is due to Diabetic Retinopathy (DR). According to global scenario Diabetic retinopathy was seen in 34% of diabetic patients according to a large meta-analysis –one in 3 will have DR,10% of these patients will have sight threatening diabetic retinopathy.[(4)]

In India 18.1% of Diabetes mellitus patients aged >50yrs had some diabetic retinopathy according to a meta analysis study[(5)] .Visual impairment due to diabetic retinopathy was seen in 4% of 1414 individuals with diabetes mellitus in one study.[(6)]

In Asian Type2 DM patients, the prevalence of DR was found to be 28%. Another meta analysis showed that prevalence of DR was as high as 42%.[(7)]

After 15 years of diabetes, it is to be noticed that nearly 25 % of type-1 patients and, 15.5 % of type-2 patients will develop Proliferative diabetic retinopathy.

Type 1 patients have the highest rate of progression to PDR, with a cumulative risk of 42 percent over 25 years.[8]

In the Asian population as a whole, the prevalence of PDR in DM patients was 6%. PDR was found to be 17% prevalent in Indian populations. In terms of the prevalence of PDR in DR patients, this study found that 17 percent of Asian DR patients and 26 percent of Indian DR patients had PDR according to a large meta-analysis study[7]

It is the most common vascular disorder of the retina and is primarily a microvascular complication. It is characterized by progressive microvascular occlusion leading to retinal hypoxia. The changes in retina progress to the proliferative diabetic retinopathy gradually from non-proliferative diabetic retinopathy. The hallmark of proliferative diabetic retinopathy (PDR) is the progression of newly developed blood vessels over the surface of retina.[9]

The new vessels being fragile can bleed resulting in vitreous or preretinal hemorrhage. This vitreous hemorrhage may be absorbed over period of time or may persist if it is dense. This will affect the vision in the affected eye. Also, the new blood vessels grow along the posterior hyaloid using it as a scaffold and later can undergo fibrotic changes resulting in contractile fibrocellular membrane. If the fibrocellular membranes are extensive they pull the underlying retina causing tractional retinal detachment. If the traction involves macula it leads to deterioration of visualacuity. Excessive traction on the retina may cause a retinal tear which in turn may induce to rhegmatogenous detachment. Both non resolving vitreous hemorrhage and tractional retinal detachment involving or threatening the macula need surgical

treatment by Parsplana vitrectomy. PPV for PDR is also indicated where a macular hole occurs due to traction on macula.[9]

As complications of PDR are one of the most common indications for PPV. There is a need in our institute to study the results of procedure and surgical outcomes. Such a study has not been conducted so far in our institute and hence we aim to conduct the study and report the results.

REVIEW OF LITERATURE

HISTORY OF DIABETES

Araetus of Cappodocia introduced the term "diabetes" from a Greek word that means "to flow through." Thomas Willis added the word "mellitus" from Latin, which means "honey."[10]

Paul Langerhans discovered the Langerhans Islets in 1869.

Frederick Banting and Charles Best discovered insulin in the 1920s, which was further refined by James Collip.

Carbutamide, the first oral hypoglycemic drug, was invented in 1955.

Dean Kamen pioneered the insulin pump in the 1970s, and HbA1c testing was adopted in 1977.

Humulin, the first biosynthetic human insulin, and Novopen, the first insulin pen delivery method, were also invented by Novo Nordisk in 1985[11]

CLASSIFICATION AND DIAGNOSIS OF DIABETES MELLITUS

Definition- Diabetes Mellitus is a metabolic disorder characterized by chronic hyperglycemia and related abnormalities in carbohydrate, protein, and lipid metabolism, which is caused by a lack of insulin synthesis, decreased insulin action or both.

Classification: Diabetes can be classified as

1. Type 1 - autoimmune beta cell destruction, culminating in complete insulin insufficiency.

2. Type 2 - progressive reduction of insulin secretion, usually accompanied with insulin resistance.

3. Gestational diabetes - diabetes discovered in the later trimesters of pregnancy.

4. Additional monogenetic syndromes

Diagnosis

Can be made by

1. A fasting plasma glucose level of ≥ 126 mg/dl .

2. ≥200 mg/dl plasma glucose after 2 hours of meals.

3. HbA1C level of 6.5 percent

4. A random plasma glucose ≥200 mg/dl in a patient with specific features of hyperglycemia

LONG TERM COMPLICATIONS OF DIABETES

The micro- and macrovascular consequences of diabetes were first discovered after the introduction of insulin therapy, when the condition was no longer regarded as a disease with a 100 percent mortality rate.[12]

Microvascular complications

1. Diabetic retinopathy

It is regarded as the most prevalent microvascular problem. It is determined by the insufficiency of glycemic management as well as the duration of diabetes.

2. Diabetic nephropathy

Diabetic patients with microalbuminuria (30-299 mg of albumin excretion per day) typically develop nephropathy if not well treated.

Pathologic abnormalities in the kidney include increased glomerular basement membrane thickness, microaneurysms, and Kimmel stein- Wilson bodies in the mesangium, and other changes.

Poor glycemic management has been linked to the development of diabetic nephropathy. The goal of treatment should be to achieve the lowest safe blood glucose level that can either prevent or delay the progression of diabetic nephropathy.

Patients can also benefit from hypertension medications, which have reno protective properties in addition to antihypertensive effects. In around 60-70 percent of patients, they slow the progression of microalbuminuria to macroalbuminuria.

3. Diabetic neuropathy

It is referred to as an exclusionary diagnosis. In diabetic patients, it is characterised as the presence of symptoms and/or signs of peripheral nerve dysfunction that cannot be attributed to any other cause.

The risk is closely associated to poor glycemic management as well as diabetes duration. Chronic hyperglycemia can cause peripheral neuropathy by the accumulation of polyol such as sorbitol, Advanced glycation end products (AGEs), and oxidative stress.

It might present as sensory, focal/multifocal, or autonomic. The most frequent type of neuropathy is chronic sensorimotor distal symmetric polyneuropathy.

Macrovascular complications

Diabetes-related cardiovascular disease is the leading cause of death among diabetic patients.

Diabetes is also associated with an increased risk of developing cerebrovascular disease and stroke (around 150-400 percent increased risk)[13]

Both of these ailments are caused by hypercoagulability and high platelet adherence.

HISTORY OF DIABETIC RETINOPATHY

Appolinaire Bouchardat, a French ophthalmologist, discovered poor vision in diabetic patients who did not have any cataractous alterations in the lens in 1846. This was somewhat reversible, and with proper glycemic management, it got much better.[14]

Diabetic maculopathy

Eduard Jaeger observed round or oval yellowish dots in the macula of a diabetic patient in 1855, accompanied with full and partial thickness excavations.[15]

In 1877, Nettleship and Sir Steven Mackenzie published an article documenting the aberrant changes in diabetic retina[16]

Proliferative diabetic retinopathy

Wilhelm Manz documented precise diagrammatic representations of fibrovascular degeneration of the optic nerve head and vitreoretinal traction in his paper 'Retinitis Proliferans' in 1876.[17]

Although a misnomer, the word retinitis, which implies an inflammatory cause, was used for numerous years.

The evolution of therapeutic modalities for diabetic retinopathy

After witnessing the effects of a solar eclipse on the retina in a student, German ophthalmologist Gerhard Meyer- Scwickerath described the treatment of retinal diseases with photocoagulation in 1950.[18]

In 1979, the Diabetic Retinopathy Study Research Group demonstrated that both the Xenon ARC and the argon pan retinal laser photocoagulation significantly reduced visual impairment.[19]

The early treatment diabetic retinopathy study (ETDRS) also demonstrated that argon laser photocoagulation can reduce risk of moderate visual loss due to clinically significant macular edema(CSME).[20]

For the management of vitreous hemorrhages in proliferative diabetic retinopathy, Robert Machemer advised pars plana vitrectomy.[21]

PATHOGENESIS OF DIABETIC RETINOPATHY

The primary factors underlying development of diabetic retinopathy are hyperglycemia and microangiopathy, inflammation, neuronal loss in the retina, and oxidative stress.

BIOCHEMICAL MECHANISMS

1. **The Aldose reductase theory**

An increase in the activity of the aldose reductase pathway can be caused by an increase in intracellular glucose concentrations. Aldose reductase makes use of the reduced form of the Nicotinamide Adenine Dinucleotide Phosphate ((NADPH).

Several aldose sugars are reduced to their corresponding sugar alcohols using NADPH as a cofactor.

The enzyme aldose reductase converts glucose to its alcohol form, sorbitol. Sorbitol dehydrogenase then oxidizes sorbitol to fructose. However, this is a sluggish response that can result in intracellular sorbitol buildup. The aldose reductase pathway is redundant in a euglycemic environment, and glucose's high binding constant renders it an inappropriate substrate. Anyhow, in a hyperglycemic state, this route becomes active as other metabolic pathways become saturated.(22)

The hyperglycemic diabetes state which occurs with an increased usage of the aldose reductase results in a reduction in the intracellular Nicotinamide Adenine Dinucleotide Phosphate (NADPH), which affects the balance of the cellular redox and may reduce nitric oxide synthesis in endothelial cells.(23) Similarly, increased sorbitol dehydrogenase utilization can result in a rise in the Nicotinamide Adenine Dinucleotide Phosphate (NADPH)/ (NAD+ ratio), which can disrupt the cellular redox balance and lead to oxidative stress and further damages the cells.(24)

2. Advanced glycation end product (AGE) Theory

Nonenzymatic glycation and crosslinking of proteins expedites ageing, has been suggested as a mechanism to explain the complications of diabetes.(25) AGEs are proteins, lipids and nucleic acids which are irreversibly altered by either reducing sugars or their products. The Maillard reaction refers to the chain of chemical reactions that result in the development of AGEs. Early glycation is the first chemical reaction to occur which involves nonenzymatic reversible binding of a sugar to amino acid groups on proteins, lipids, or nucleic acids. They create Schiff bases

which can be rearranged to produce more stable Amadori products. The development of AGEs may cause direct injury to cells by interfering with the function of a variety of proteins, including both extracellular and intracellular proteins such as Collagen.[26]

The most well-known Amadori compounds are fructosamine and the glycated hemoglobin (HbA1c), which have been used as a clinical indicators of the blood glucose control. Although they are not Advanced Glycation End Products (AGEs), additional reactions might result in the production of the Advanced Glycation End Products (AGEs). Although they are not AGEs, they can undergo further reactions that will eventually result in the production of AGEs.

3. Photoreceptor metabolism theory

Pseudohypoxia of the retina caused by hyperglycemia, along with anoxia of the inner retina during dark adaptation (rods consume high oxygen levels during dark adaptation, resulting in decreased pO2 in the inner retina), leads to an increase in VEGF synthesis.[27]

4. Reactive oxygen intermediates theory

Chronic hyperglycemia causes diabetic complications with the increased oxidative strain.

The normal glucose metabolic process involves 2 factors, of which the first is glycolysis and the second is tricarboxylic acid cycle. The tricarboxylic acid cycle takes place in the mitochondria and results in the production of reducing equivalents, which are further utilized to steer the production of adenosine triphosphate through oxidative phosphorylation. Free radicals, such as the superoxide anion, are produced as byproducts of oxidative phosphorylation, which is facilitated by high glucose levels in the bloodstream.[28] Glucose autoxidation produces free radicals that might

harm mitochondrial DNA[29] and cellular proteins. Excessive oxidative strain will decrease the levels of nitric oxide,[30]increases the leukocyte adhesion further to endothelium, and minimizes the function of the endothelial cell barrier, and destroys cellular proteins.

5. Protein kinase C theory

Protein Kinase C (PKC) is a widely distributed enzyme that seems to expedite the formation of a variety of diabetes complications when aldose reductase pathway is inhibited or absent. Because of the activation of phospholipase C, there is an increase in Diacyl glycerol and the intracellular Ca2+, which in turn promotes the activity of PKC.[31] Pathologic PKC activation may occur as a result of hyperglycemia. Elevated glucose levels stimulate the glycolytic process, which results in elevated levels of the glyceraldehyde3phosphate in the cells. Glyceraldehyde-3-phosphate has the potential to increase again DAG production via the activation of PKC by glycerol-3-phosphate.[32] Pathologic consequences of PKC activation include increased vascular permeability, disruption in regulation of the of nitric oxide,[33] enhanced leukocyte adherence to walls of the vessel, and alterations in the flow of the blood are the pathologic outcomes of the Protein Kinase C (PKC) activation which induce vascular injury.

Overactivation of PKC causes VEGF overexpression, resulting in alterations in retinal vascularity

Hyperglycemia and microangiopathy

Blood vessel dilatation and hemodynamics are the main hallmarks of retinal vasculature as a result of chronic hyperglycemia, which serve as an autoregulatory mechanism to optimize retinal metabolism.[34] Hyperglycemia has also been linked to

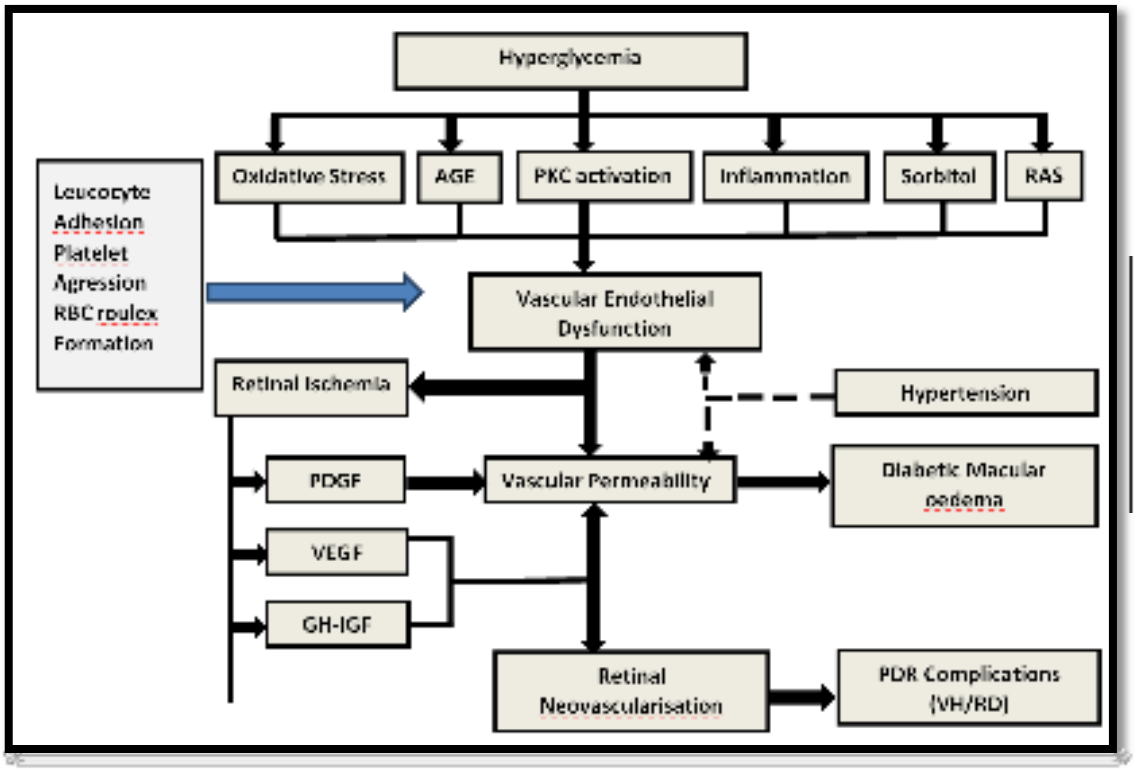
the death of pericytes,[35] which offer structural support to capillaries. This causes capillary wall outpouchings and the development of microaneurysms. This, together with endothelial cell death and basement membrane thickening, leads to disruption of the inner blood retinal barrier (iBRB), capillary obstruction, and ischemia.

This causes the upregulation of Vascular endothelial growth factor (VEGF) by Hypoxia inducible factor 1 (HIF-1),[36] which leads to enhanced permeability of the retinal microvasculature via phosphorylation of tight junction proteins. It also promotes endothelial cell proliferation by activating mitogen-activated protein (MAP).

Angiopoietins 1 and 2 are two more pro-angiogenic factors that have been implicated.

Retinal neurodegeneration

Proapoptotic molecules such as cleaved caspase-3, Bax, and Fas are upregulated in retinal neurons of diabetic animals.[37] The retinal neurodegeneration can also be caused by mitochondrial malfunction, which is caused by pro-apoptotic mitochondrial proteins such as cytochrome c and apoptosis inducing factor (AIF).



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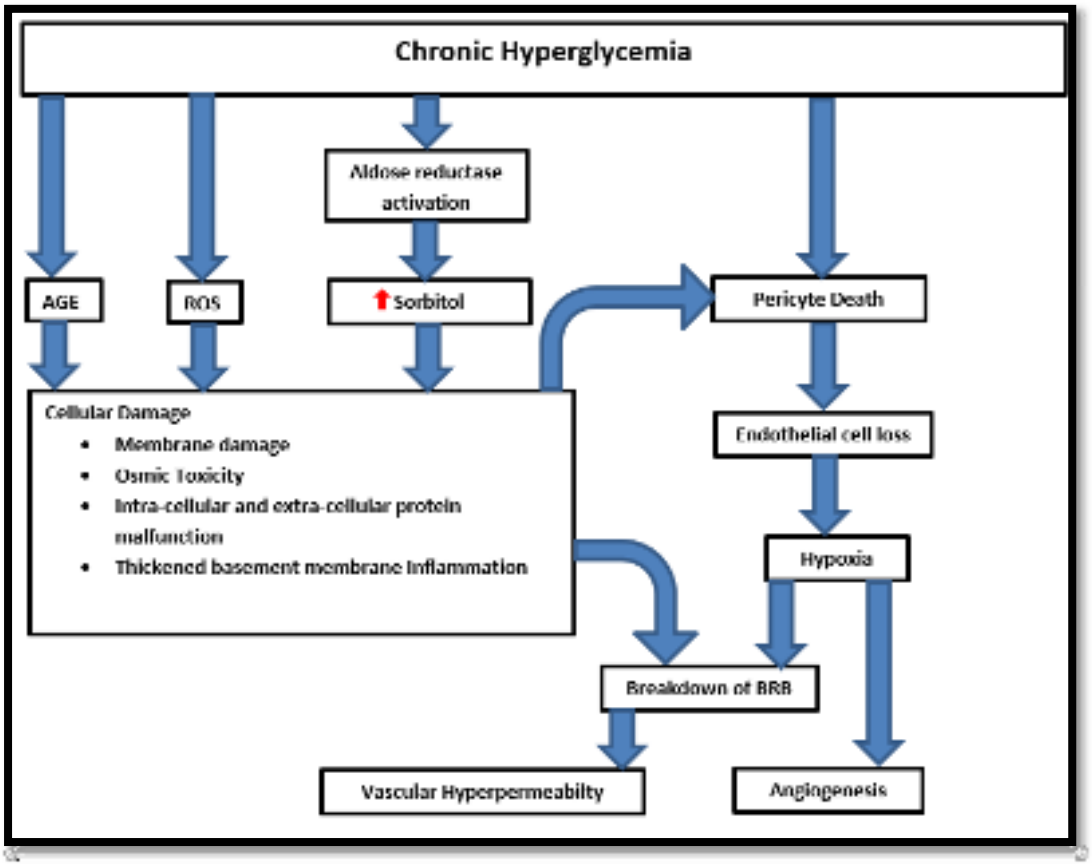

Genetic mechanisms in the diabetic retinopathy pathogenesis

The Family clustering cases of diabetic retinopathy, especially the more severe forms, was discovered in a study by the Diabetes control complications trial (DCCT) research group, which identified that families with a high number of diabetic members were more likely to develop the condition.[38]

In pathophysiology of the diabetic retinopathy, the VEGF gene has garnered the most attention. Analyses on the role (beneficial/risk) for single nucleotide polymorphisms (SNPs) in the VEGF gene have not provided definitive results because the relationships derived had contradictory values in different groups.

CLASSIFICATION OF DIABETIC RETINOPATHY

MODIFIED AIRLIE HOUSE CLASSIFICATION

This classifies diabetic retinopathy into 85 levels ranging from level 10 (no retinopathy) to level 85 (severe vitreous hemorrhage or macula involving retinal detachment).[39]

An **abbreviated ETDRS classification** of diabetic retinopathy is given below[40]

Diabetic retinopathy can be classified into non proliferative, proliferative and advanced diabetic eye disease

I. Non proliferative

1. Very mild- when there are only microaneurysms.
2. Mild - microaneurysms, retinal hemorrhages and cotton wool spots can be present, but not more than in moderate NPDR. Intraretinal microvascular abnormalities (IRMA) and beading should not be present.

3. Moderate –
    (i) Medium (20) to large hemorrhages per quadrant in 1-3 quadrants or mild IRMA
    (ii) Prominent venous beading, but only in a single quadrant
    (iii) Cotton wool spots
4. Severe – When one or greater of
    (i) The Severe hemorrhages in all the quadrants
    (ii) significant venous beading in two (2) or greater quadrants.
    (iii) Moderate Intraretinal microvascular abnormalities (IRMA) in one or more quadrants
5. Very severe – When two or greater of the above-mentioned criteria.

II. Proliferative

1. Mild to moderate- when the disc has new vessels (NVD) or no elsewhere (NVE), still less than high risk criteria
2. High risk –
    (i) NVD greater than (1/3) one-third of the disc area
    (ii) If Any NVD with the vitreous hemorrhage
    (iii) If NVE greater than that of the half disc area, and with the vitreous hemorrhage.
3. **Diabetic eye disease in its advanced stages comprises of**
    i. Retro or intrahyaloid hemorrhage
    ii. Tractional RD
    iii. Rubeosis iridis**.**

Management

- No DR- Annual review.
- Mild NPDR-advised 4-6 months
- Moderate NPDR-advised 3-4 months.
- Severe and very severe NPDR are asked to review in 2- 4 months.
- The risk of developing PDR increases with increasing severity of NPDR.
- Low risk PDR can be advised photocoagulation or anti-VEGF. High risk PDR usually requires Pan retinal photocoagulation (PRPC).Advanced PDR may need vitrectomy.

The ETDRS defines **Diabetic macular edema (DME)** as

- "Thickening of the retina at or within 500 µm of the Centre of the macula"; or
- "Hard exudate at or within 500µm of the Centre of the macula associated with thickening of adjacent retina"; or
- "A zone of retinal thickening 1 disc area or larger , any part of which is within 1 disc diameter of the Centre of the macula".[39]
- Diabetic Macular Edema(DME) can also be focal and diffuse.[41]

Focal DME is edema due to the focal leakage from microaneurysms. Microaneurysm clusters are usually surrounded by a circinate pattern of hard exudates.

Diffuse DME is due to diffuse breakdown of iBRB. This involves microaneurysms, capillaries as well as arterioles. Hard exudates are usually not seen.

Based on microaneurysmal fluorescein leakage in angiography, ETDRS classified diabetic macular edema as

1. Focal-≥67 % of leakage originating from the microaneurysms
2. Intermediate-microaneurysmal leakage between 33 to 66%.
3. Diffuse- ≤33 %microaneurysmal origin leakage.

The natural progression of non-proliferative diabetic retinopathy

Studies have indicated that in the early stages of diabetes, the retinal parenchymal cells undergo modifications such as reactivity of the glial cell, abnormalities of the glutamate metabolism, and loss of neuron.[42] Minimal changes in color perception and contrast sensitivity have also been documented.[43]

1. Microaneurysms

Microaneurysms which are dots in red deep color have a size range between 25- 100 microns and are typically the very first and most apparent indication of the DR, and also a distinguishing feature of the NPDR.

The Microaneurysms are formed on the capillary walls as the hypercellular saccular outpouchings, which are easily visible in trypsin-digest retinal mounts. Their lumina sometimes clogged by the agglutinated red blood cells or thrombus. Acellularity can develop in them over time in the same way as damaged retinal capillaries can develop into "ghost" vessels that are devoid of endothelial cells and pericytes.

Microaneurysms enhance the chance of diabetic retinopathy becoming more severe as the number of them increases.[44]

2. Retinal vascular hyperpermeability

Increased VEGF levels cause tight junction dysfunction at the BRB, resulting in increased vascular permeability and leakage. This may be accompanied by localized areas of retinal thickening.

Extravascular deposits of lipid-rich material termed as hard exudates are also found. Intraretinal hemorrhages begin to appear, particularly in the posterior pole and periphery. Superficial-flame shaped hemorrhages, deep-dot and blot hemorrhages.

3. Diabetic macular edema

DME in areas of vascular incompetence is caused by leaking from microaneurysms or more diffuse leakage from capillaries with enhanced permeability.

Retinal thickening can occur in locations of capillary nonperfusion without concomitant vascular leakage.

Macular oedema can be related with cyst development and subretinal fluid.

DME's natural history is highly diverse, ranging from years of persistence to spontaneous resolution over time.

4. Capillary closure, microvascular remodeling, and retinal ischemia

Trypsin digest retinal vascular mounts in advanced diabetic retinopathy show areas called as ghost vessels, where capillaries have lost their lining endothelial cells and pericytes.

Excessive count and conjoining of these can result in blockage of the terminal arterioles responsible for perfusion.

Increasing capillary closure and the resultant ischemia in the retina are usually accompanied by intraretinal microvascular abnormalities, intraretinal hemorrhages and venous beading. In some situations of severe hypoperfusion, the retina might take on a featureless look, with very few visible arteries, hemorrhages, or microvascular abnormalities.[22]

5. Alterations in the vitreous and vitreoretinal interface

The vitreous gel is critical in the fibrovascular growth of PDR, but it may also have an influence on the development of retinopathy in the beginning stages of disease. The development of Epiretinal membrane, produced by the vitreous gel liquefaction and the resulting consequences at the interface of the vitreoretinal, may arise with age and in otherwise healthy eyes, yet it is more prevalent in diabetic eyes due to the increased risk of diabetes complications.[45]

Posterior cortical vitreous shows an increased adherence to the retina. Increased collagen cross-linking, AGEs that improve vitreoretinal adhesion and drive glial cell reactivity, as well as a change in the quantity of certain soluble proteins are all biochemical changes in diabetic patients' vitreous.[46]

Pathogenesis of proliferative diabetic retinopathy

The following provides an in-depth illustration of the natural course of PDR, with particular emphasis on four elemental processes: (1) New vessels go through a cycle of proliferation and regression.; (2) New vessels are accompanied by a growth of fibrous tissue.; (3) adhesions are formed between the fibrovascular proliferations and the posterior vitreous surface; (4) contraction caused by the posterior vitreous surface and related proliferations.[22]

Diabetes-related hyperglycemia and metabolic abnormalities cause changes to the retinal vasculature, leading to a decreased perfusion to retinal tissue.[47] And the condition of relative retinal ischemia is assumed to be the major angiogenic trigger involved in the pathophysiology of PDR.

On the basis of both in vivo and in vitro research studies, VEGF appears to be the main culprit in PDR's ischemic-driven angiogenic disease.[48] Studies show elevated levels of VEGF in the vitreous of PDR patients, which correlate with disease activity. The concentrations of the vitreous VEGF are minimal or untraceable after laser treatment or in naturally quiescent PDR.

The Intraocular retinal new vessels resulting from active PDR and the diabetes-induced iris neovascularization are very responsive to the VEGF inhibitors, with many of them regressing within a single day of treatment.[49] However, while VEGF seems that it is the most important angiogenic factor in PDR, other factors are likely involved in the complicated mechanisms governing in vivo angiogenesis. Angiopoietin/Tie-2 system directly influence not only the retinal pericytes but also endothelium cells, that are hypothesized to be pathogenic mechanisms of PDR.[50]

Increased blood glucose levels leads to decrease in the survival-promotion action of the Platelet derived growth factor (PDGF) causing not only pericyte apoptosis, but also diabetic vasculopathy. This process is activated by hyperglycemia, which results in increased expression of a protein tyrosine phosphatase termed Src homology-2 domain containing phosphatase-1 (SHP-1). Resistance to PDGF is mediated by SHP-1 activation, which results in the loss of cellular survival

mechanisms and increased pericyte death.[51] SHP-1 inhibition may protect against early retinal alterations that lead to PDR development.

VEGF-independent mechanisms, such as those mediated by EPO, have also been linked to PDR development.[52] In a modest genetic investigation of three different patient populations, single nucleotide polymorphisms (SNPs) that boost EPO expression were linked to the development of PDR and severe renal impairment.[53] Antiangiogenic mediators including pigment epithelium-derived factor (PEDF) are reported to be lower in diabetes and active PDR patients than other retinopathies.[54] At various phases of retinopathy, an interplay between angiogenic and antiangiogenic pathways may be significant in the eye.

THE NATURAL PROGRESSION OF PROLIFERATIVE DIABETIC RETINOPATHY (PDR)

i. **Proliferation and development of new vessels-**For the majority of time, they appear on the disc. They have the ability to cross over both arterioles and veins. At first, they may be coupled with thickening of the optic nerve head and neighbouring retina. Later on, these vessels will be surrounded by fibrous tissue.

ii. **Contraction of both vitreous and fibrovascular proliferation -** As a result, there is a posterior vitreous detachment and vitreous hemorrhage

iii. **Distortion of retina and tractional detachment-** The contraction of the vitreous, combined with the proliferation of fibrovascular tissue, causes tractional retinal detachment, which may or may not affect the macula.

iv. **Involutional or Quiescent Proliferative Retinopathy-** The vitreous contraction is finished at this point, and vitreous detachment has happened in all places except where vitreoretinal adhesions prohibit it.

The frequency and severity of vitreous hemorrhages decreases, and the hemorrhage clears up within a few months.

It is likely that visual acuity will be reduced if the detachment occurs in the macula.

Both arterioles and veins become smaller because of the reduction in the number of visible branches. On rare occasions, hemorrhages and microaneurysms can be found, Fibrous tissues become thinner and more transparent, allowing better visualization of the retina.

Relationship of Proliferative Diabetic Retinopathy to Type and Duration of Diabetes

A population-based stereophotographic study conducted by Klein et al., showed that the prevalence of PDR in insulin-taking patients under 30 years of age (exclusively or mainly type 1)was almost 0% when *duration of* diabetes was less than 10 years but rose to 50% in those with diabetes of 20 or more years.[55]

Joslin 50-Year Medalist Study reveals that PDR prevalence persists around 50% in people who had insulin-dependent diabetes for 50 years or longer.[56]

In population with older onset of diabetes (30 or more years of age) and those taking insulin only 2% had PDR if the duration of diabetes was less than 5 years but rose to 25% in those with DM for 20 years or more duration.[57]

In those with DM at older age (after 30 years of age) the prevalence of PDR was less common if the persons were not on insulin treatment. In this group the prevalence of PDR increased only slightly with duration, from less than 2% before 5 years of DM to 15.5% after duration of DM of 15 years or more.[57]

A meta-analysis of 28 prospective interventional or observational trials involving 27,120 diabetic patients with at least 10 years of follow-up found that those newly diagnosed with diabetes had reduced rates of PDR and severe visual loss.[58] This paper indicates that the newly diagnosed diabetic patients had lesser duration of diabetes and less prevalence of PDR at the end of 10 years follow up.

Type of DM-

Diabetes that develops after the age of 30 (typically type 2) is 8–10 times more common than diabetes that develops before the age of 30 (typically type 1), and in clinical practice, PDR is seen with roughly equal frequency in the younger- and older-onset groups

The Diabetic Retinopathy Vitrectomy Study (DRVS) observed a significant variation in PDR severity by diabetes type among people with vitreous hemorrhage severe enough to reduce visual acuity to 5/200 or less for at least 1 month.[59] The severity of new vessels, fibrous proliferation, and vitreoretinal adhesions was significantly lower in type 2 diabetes, according to this study.

Klein et al. estimated that 43% of patients with PDR were younger-onset, 42% were older-onset insulin-taking, and 15 percent were noninsulin-taking in the population they surveyed.[57]

During a 5-month period, Aiello and coworkers studied 244 PDR patients at the Joslin Clinic and found that age of diagnosis of PDR was less than 20 years in 53%, between 20–39years in 25% and 40 years or older in 22%.[60]

Yau et al analyzed the results of 35 studies to study the risk factors for DR AND PDR. They observed that PDR was 15 times more likely to be present in patients with type I DM of 20 years duration compared to patients with type II DM of <10 years duration.[4]

Proliferative Diabetic Retinopathy and Blood Glucose Control

The diabetes control and complications trial (DCCT study) and the United Kingdom prospective diabetes studies (UKPDS) proved the effectiveness of the intensive control of the blood glucose in lowering the risk of appearance and worsening of DR in both of type-1 patients and type-2 patients respectively.[61]

The Early Treatment Diabetic Retinopathy Study (ETDRS) provides further evidence that improved glycemic management in persons with severe NPDR or early PDR minimizes their likelihood of future advancement.

The level of HbA1c at the start of the study was a significant risk factor. Patients with HbA1c levels more than 12 percent were at the greatest risk of advancement, while patients with HbA1c levels less than 8 percent were at the lowest risk.[62] Even among those with the lowest A1c levels, the five-year risk of acquiring high-risk PDR from severe NPDR was as high as 50 percent. The reports highlighted that the chances of development of PDR or severe NPDR were greater if the baseline

HBA1c was high and blood sugar control had not been good prior to starting of strict control of blood sugar.

Systemic diseases and PDR

Presence of hypertension, diabetic nephropathy and other diabetic complications have been associated with higher prevalence of PDR. Presence of systemic complications of diabetes in general indicate that the diabetes is poorly controlled or present for long duration. So, it is not surprising that one or more complications of diabetes may be present simultaneously.

Systemic hypertension has been shown to increase the risk of any DR and PDR in DCCT and UKPDS trials. Bek et al showed that increase in diastolic blood pressure increased the risk of PDR.(63)

Studies have shown that presence of renal failure or nephropathy is a risk factor for PDR.

Hsei et al in a study of 2135 patients with type II DM measured the baseline data like serum creatinine level, estimated glomerular filtration rate and urinary albumin and creatinine ratio and followed up the patients over 8 years. The study found that higher baseline serum creatinine level, lower estimated glomerular filtration rate and higher urinary albumin and creatinine ratio were associated with higher incidence of PDR.(64)

Atherosclerotic cardiovascular disease is an important cause of morbidity in type II DM. A study showed that PDR was an important risk factor for atherosclerotic cardiovascular disease.(65)

Another study also showed presence of PDR increases the risk of cardiovascular disease including coronary heart disease, stroke and death and hence presence of PDR marks an important marker for these diseases.[66]

Systemic Medications and Proliferative Diabetic Retinopathy

In the case of diabetes mellitus, systemic drugs are frequently employed to provide optimal glucose control and treating concomitant problems. All these medications may have either the positive or negative impacts on the diabetic eye disease development or progression. Oral systemic drugs may minimize microvascular consequences by mechanisms other than glycemic management, blood pressure, and cholesterol lowering, according to growing evidence.

Thiazolidinediones are a kind of oral hypoglycemic medication that stimulates the activity of the (PPAR-γ) Peroxisome Proliferator Activated Receptor-Gamma, a transcription element that regulates the genes expression primarily found in the adipose which is a tissue but can also be found in other different tissues like the retina.[67] Rosiglitazone, a thiazolidinedione, was shown to delay the start of the PDR, possibly due to the antiangiogenic effects, mediated through (PPAR-γ)-agonist activity.[68] According to the review by Shen and the colleagues[68], the medical records of patients (124) who were given rosiglitazone and, patients (158) who haven't been given rosiglitazone as the controls had a match on the characteristics of the baseline such as HbA1c levels.

At 3 years, the progression of the relative risk to PDR has a reduction of 59% (where p=.045) in patients with severe NPDR who received rosiglitazone, and this effect lasted for 5 years of follow-up.

However, these drugs have not been subjected to large clinical trials and hence their efficacy in prevention of PDR is not proven

Peripheral Diabetic Retinal Lesions and the Risk of Retinopathy Progression

Multiple independent studies have revealed that the presence of peripheral retinal lesions as detected on ultrawide-field imaging may indicate an increased severity of DR in 9–15 percent of eyes.[69] Predominant peripheral lesions (PPL), defined as presence of more than 50% of a particular DR lesion situated outside typical ETDRS fields, may be present in up to 50% of eyes. The presence of PPL is related with a 3.2-fold greater chance of 2 or more step DR progression and a 4.7-fold increased risk of PDR development according to prospective longitudinal 4 - year data.[70] The emergence of DR lesions in the peripheral retina is thought to be caused by underlying capillary nonperfusion and could be linked to DR progression. Furthermore, it has been noted that the amount of capillary nonperfusion in the midperiphery increases in conjunction with the severity of DR.[71] Capillary nonperfusion is assumed to be related to tissue hypoxia and is thought to be an irreversible process that causes overall ischemia in the eye.

Additional risk factors for PDR

Increased NPDR severity, decreased visual acuity, and higher HbAlc are all important risk factors for the development of PDR. Diabetic neuropathy, a low hematocrit with a high serum triglyceride, and a low albumin plasma level were all additional risk factors.[62] The link between raised serum lipids and an increased chance of developing high-risk PDR, as well as increased hard exudates and impaired visual acuity[72] adds to the case for lowering the frequently elevated lipid levels seen

in diabetic individuals. In the ACCORD-EYE study, the progression rates of DR were reduced from 10.2% with placebo to the 6.5% with fenofibrate medication for the dyslipidemia, despite the fact that the benefit from fenofibrate treatment appeared to be independent of its effects on lipid levels in this study.[73]

Serious anemia is an issue that diabetes individuals face on a less frequent basis, but it has been linked to an increased risk of severe retinopathy in ETDRS analyses and three other studies.[62]

Management of proliferative diabetic retinopathy

Management of PDR include pan-retinal photocoagulation, intravitreal Anti-VEGF injection and vitreous surgery in advanced cases.

Pan retinal photocoagulation was the first treatment that was shown to have efficacy in reducing the severe visual loss due to PDR in a randomized control study in late 70s(Diabetic Retinopathy study -DRS).[74] The study showed that if the patients had the following characteristics of PDR and were subjected to PRPC treatment then it reduced the risk of severe visual loss (defined as visual acuity of 5/200 on two successive 4 monthly visits) by half compared to patients who were not given PRPC treatment.

The high-risk characteristics (HRC) of PDR have already been mentioned above. Since then the pan retinal photocoagulation has become the gold standard treatment of PDR with HRC.

The PRPC is a destructive procedure and leads to reduction of peripheral visual field apart from other side effects like- macular edema, exudative retinal detachment, and increased fibrosis of neovascular complexes leading to TRD.

As the driving force for neovascularization of retina is production of VEGF by the hypoxic retina, intravitreal injection of anti VEGF is being tried to treat PDR with HRC. A randomized control study compared PRPC with intravitreal injection of anti VEGF ranibizumab for eyes with PDR with HRC[(75)]. The results of the study showed equal efficacy of anti VEGF injection with advantages of better preservation of peripheral visual fields, less need for vitrectomy surgery and less chances of macular edema when compared to PRPC treatment arm. However, the intravitreal injection of anti VEGF needs to be given monthly – about 8 injections in the first year. This is economically and logistically difficult in our population and hence most PDR cases with HRC are treated with PRPC.

However, some patients do not respond to PRPC and develop severe vitreous hemorrhage or TRD. In such cases treatment with PRPC or anti VEGF treatment may not be possible or effective.

Surgical treatment of PDR is necessary in cases with non-resolving vitreous hemorrhage, tractional retinal detachment involving or threatening the macula and combined tractional and rhegmatogenous RD. There are other less common indications of PPV for PDR.

Surgical management- Machemer invented pars plana vitrectomy in the year 1971 which was a closed system that allowed for secure intraocular manipulations and continual vision of the retina. Non-clearing vitreous hemorrhage lasting more than a

year and macular involvement with complex retinal detachments were the most common indications at that time.

Fig 1: Front view of a new constellation vitrectomy unit, providing phacoemulsification,vitrectomy,photocoagulation,diathermy,and air/gas/silicone tamponades in one machine.

The most important pathologic feature of diabetic retinopathy is microangiopathy that leads to closure of capillary beds leading to hypoxia of the affected retina. Retinal hypoxia can lead to macular edema and/or retinal and iris neovascularization. Local proangiogenic factors such as IGF-1 referred as Insulin Growth Factor-1, also bFGF referred as Basic Fibroblast Growth Factor, and other

factors initiate these processes. The recruitment and the proliferation of the retinal vascular endothelial cells, as well as the locally activated cytokines like the Vascular Endothelial Growth Factor (VEGF), were thought to be involved in the switching from non-proliferative to the proliferative diabetic retinopathy. Endothelial cell proliferation and permeability are induced by this cytokine, which is linked to elevated counting of white blood cells and also other markers such as inflammatory markers. The VEGF protein is expressed by i) the Retina Glial cells, ii) the optic nerve, iii) the retinal astrocytes, iv) the pigment epithelial cells, v) the vascular endothelial cells, vi) and the ganglion cells. By working as the chemoattractant protein, VEGF is thought to mobilize and raise number of Endothelial Progenitor Cells (EPC) in the bone marrow. Circulating EPCs are thought to proceed straight to the areas of ischemia to start the development of new vessels and fibrous tissue formation. The new vessels grow from the retinal vessels and pierce the internal limiting membrane (ILM) and then grow on the surface of posterior hyaloid using it as scaffold. The new vessels being fragile may rupture easily and cause sub hyaloid or vitreous hemorrhage. Extensive sheets of new vessels undergo fibrosis with time and because of firm adhesion between retina and the posterior hyaloid the retina may be pulled in the form of tractional retinal detachment. Both vitreous hemorrhage and TRD involving the macula cause reduction of vision. Tractional forces may result in retinal tear leading to rhegmatogenous retinal detachment in addition to existing tractional retinal detachment causing severe visual loss if macula is involved

INDICATIONS AND TIMING OF SURGERY

The standard indications of vitrectomy surgery for PDR are as mentioned before 1) non clearing vitreous hemorrhage 2) tractional retinal detachment involving the macular or threatening the macula and 3) combined tractional and rhegmatogenous retinal detachment. As the use of PPV became more widespread, other indications for PPV in PDR have been included -fibrovascular or fibrous membrane obscuring the macula, dense premacular hemorrhage, macular hole due to traction, severe PDR that is not responding to PRPC with extensive proliferations and iris/angle neovascularization with vitreous hemorrhage have been included.

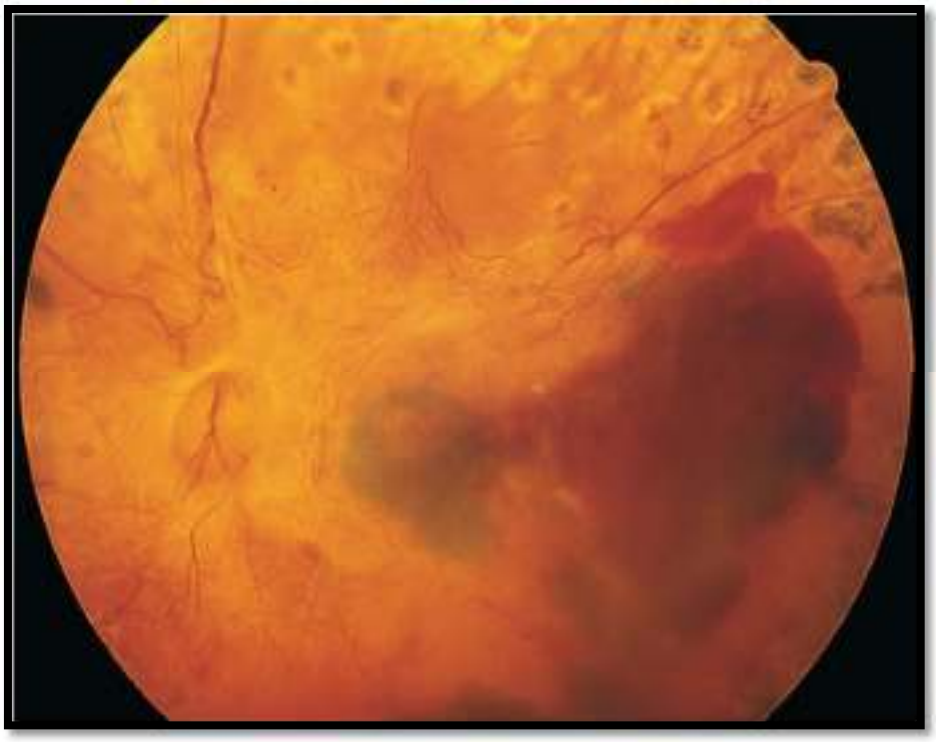

Fig 2: Dense premacular hemorrhage. Extensive fibrovascular proliferation, contraction, and distortion of vessels present.

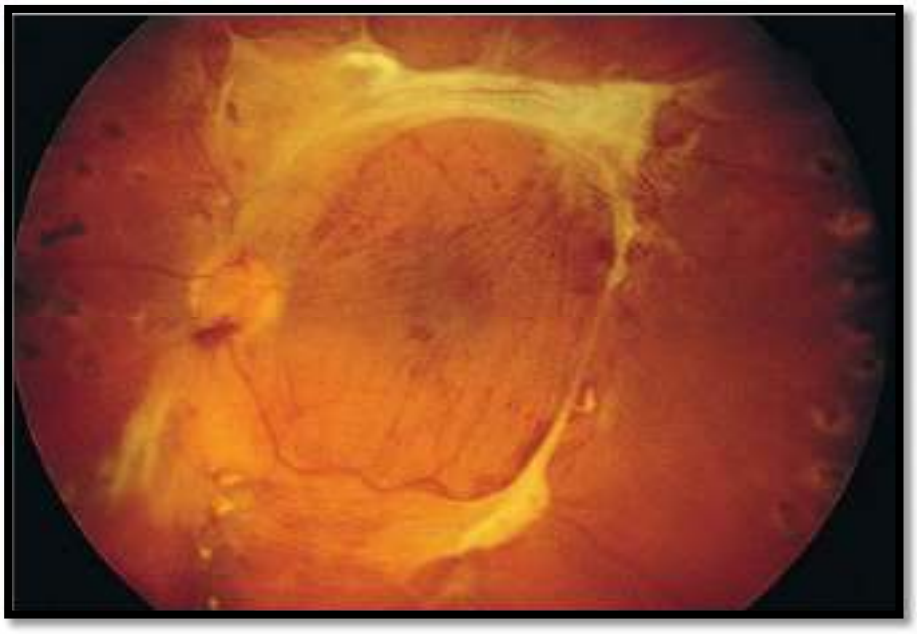

Fig 3: Tractional retinal detachment involving the entire macula

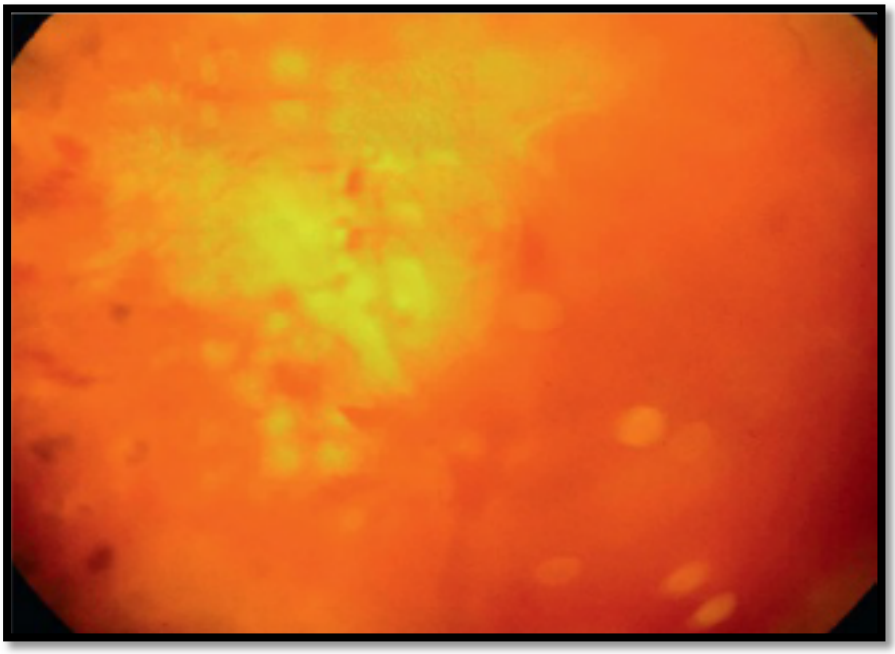

Fig 4: Combined tractional-rhegmatogenous detachment with dense fibrovascular proliferations over optic nerve and arcades.

Timing of surgery for Vitreous Hemorrhage

In the 1970s, the non-clearing vitreous hemorrhage in DR was the most common indication for the vitrectomy, accounting for 70% of cases. In many cases of vitreous hemorrhage, the blood may clear enabling PRPC to be carried out thus avoiding a major surgical intervention. So, the question arises as to how long one should wait before subjecting the patient to vitrectomy surgery?

The influence of timing of surgery for vitreous hemorrhage was studied in a randomized clinical trial known as Diabetic retinopathy Vitrectomy Study -DRVS.

The study randomized patients with vitreous hemorrhage for at least a month to immediate vitrectomy or deferred vitrectomy done after one year.

The results at 2[(76)] and 4 years[(77)] showed that early vitrectomy results in better visual gains than deferred vitrectomy. However, the effect of early vitrectomy was more pronounced in type I DM patients

However, the deferral of PPV for vitreous hemorrhage in type II DM patients is not done for one year anymore and most surgeons perform a vitrectomy between 1-3 months of vitreous hemorrhage.

Immediate PPV is usually carried out in cases with 1) poor visual acuity in the other eye due to PDR or any other cause 2) bilateral vitreous hemorrhage with poor visual acuity and 3) NVI or NVA with vitreous hemorrhage.

The timing of surgery for TRD involving macula or threatening the macula. Many studies have shown that delayed surgery in cases of TRD involving the macula has poor visual outcomes.[(78)]

So, patients with TRD involving macula are advised surgery as soon as possible. Patients with TRD and combined rhegmatogenous RD should undergo PPV on an urgent basis as the chances of severe fibrosis may make surgery difficult it there is delay in surgery.

Surgical aspects of PPV

SURGERY

Preoperative assessment

An optimum control of diabetes, hypertension and other systemic factors is important before we proceed to surgery. The use of anticoagulants is usually stopped 3-5 days before surgery as it may lead to bleeding during surgery.[79]

However other articles did not find any difference in rates of bleeding during or after surgery even when the anticoagulants were not stopped.[80]

Surgical Equipment

PPV is an instrument driven procedure and needs excellent microscope, wide angle viewing systems with invertor like BIOM or EBIOS attached to the microscope to view the retina and a vitrectomy machine with excellent fluidics and high-speed cutters.

The vitreoretinal surgery has evolved over the last five decades and is becoming less and less invasive. The pars plana approach to vitreous body was first attempted by Robert Machemer who used a single 17 g instrument that contained cutter, lighting and aspiration. Later the three functions were separated in to three

instruments by Connors O'Malley and Ralph Heintz with 20 g instruments that included infusion canula fixed inferotemporally, light pipe and vitrector instruments inserted through superotemporal and superonasal entries in pars plana. Trocar cannula fixed in pars plana to insert instruments was devised by Robert Machemer and Dyson Hickingbotham and later 23 g cutter and instruments by Gholam Peyman and Claus Eckardt were introduced. Twenty-five g instruments by Gildo Fujii and Eugene de Juan have further improved the vitrectomy procedure and made it less invasive, faster and safer. In 2010, 27 g instrumentation was introduced by Yusuke Oshima and colleagues. Presently the vitrectomy procedures for PDR and other surgeries use 23 or 25g instruments so that it does not need conjunctival opening and is less time consuming. Also, the small gauge vitrectors with improved tips with opening close to the tip allow them to be used as scissors and forceps and allow surgery to be done very close to the retina. Figures of 20,23 and 25 g instruments

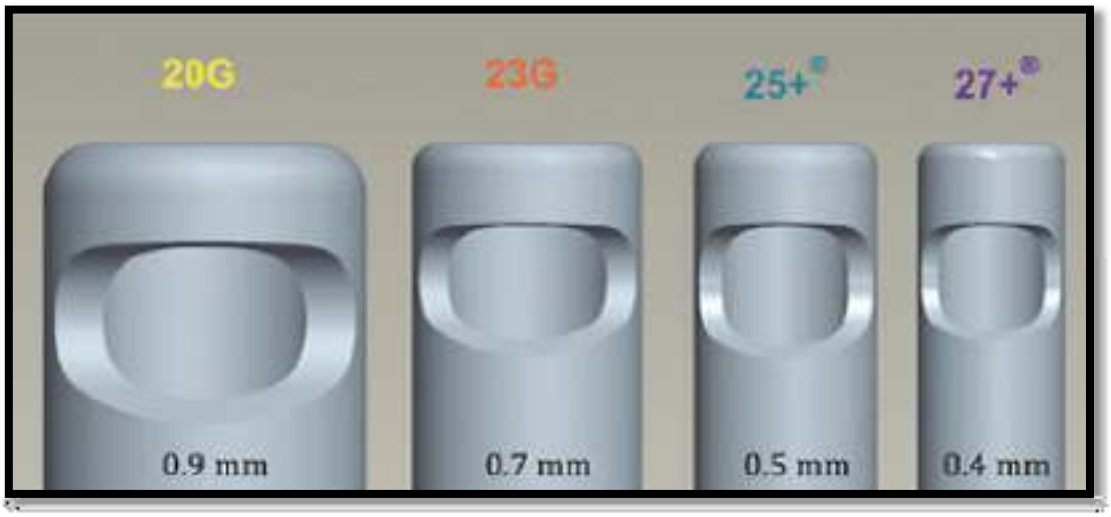

Fig5: Gauge and diameter of the probe

Fig 6: Robert Machemer

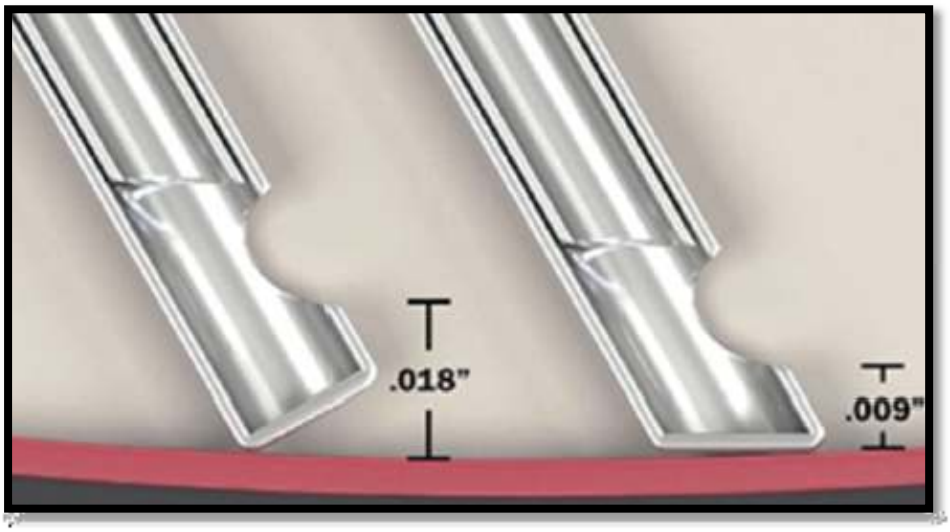

Fig 7: Tip-to-port distance enables the beveled cutter port to be closer to the retina

Surgical procedure

Pars plana vitrectomy

Preparation of entry sites

The most common procedure is a three-port vitrectomy, in which two trocars are fixed in superotemporal and superonasal quandrants , and a third inferotemporal quadrant to fix infusion cannula. Trocars are fixed at a distance of 3.5–4.0 mm from

the limbus. Blades for sclerotomy must be directed parallelly to the limbus, and the trocars must be placed 20° –30° oblique to the surface of the scleral in order to avoid intra and/or dehiscence of postoperative wound.

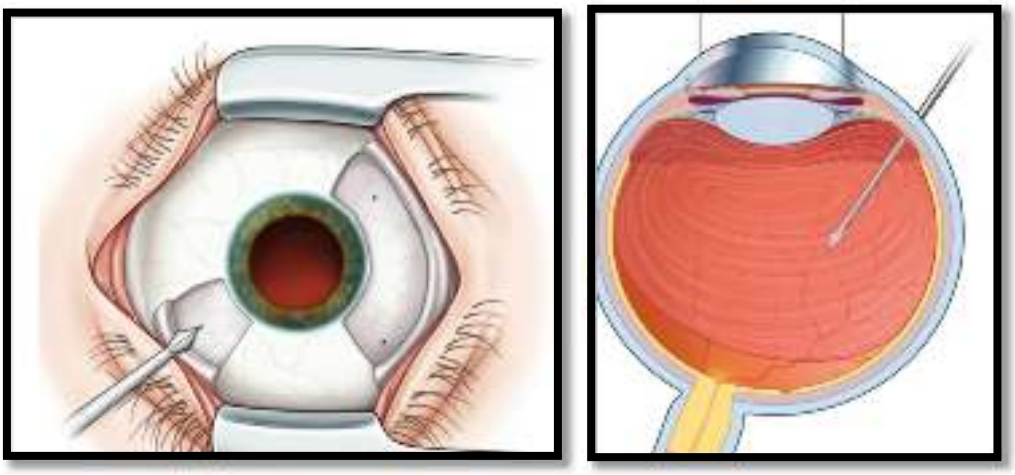

Fig 8: Blade or trocar is directed toward the Centre of the vitreous cavity to avoid contact with the lens or retina

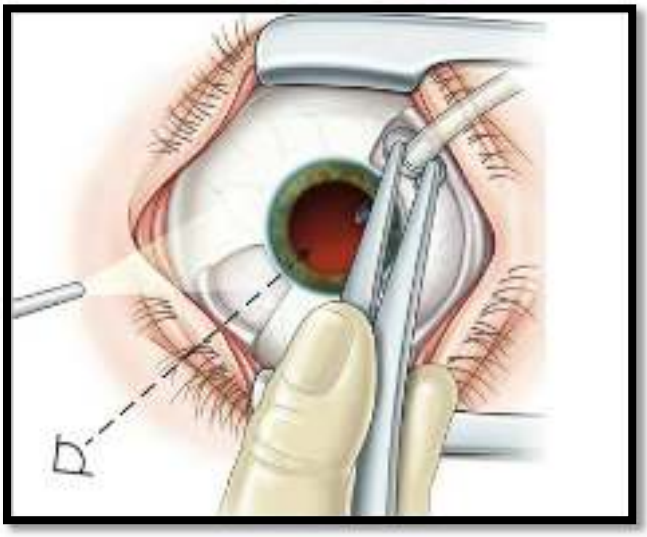

Fig 9: The infusion cannula is inserted and secured with a suture in the inferotemporal pars plana sclerotomy

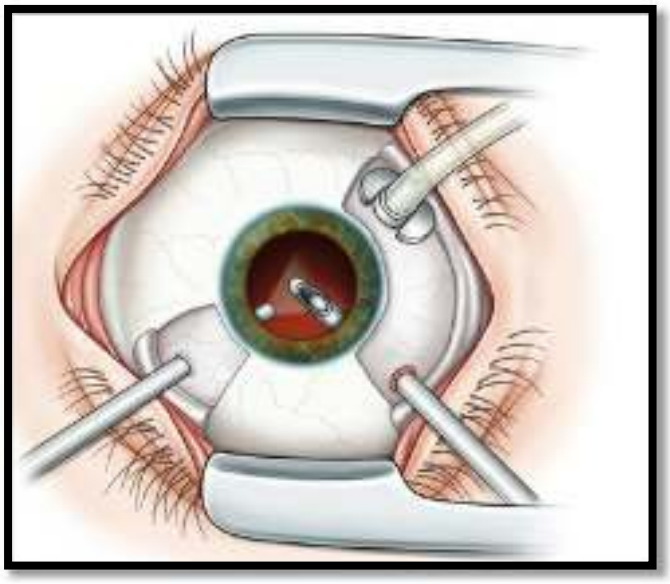

Fig 10: The vitreous cutter and fiberoptic illuminator are positioned in the anterior vitreous cavity and visualized through the pupil.

The procedure involves core vitrectomy and remove the media opacities. If there is peripheral posterior hyaloid detachment an opening is made in it and extended circumferentially 360d. If there are no membranes and TRD the posterior hyaloid is detached with suction, blood removed, peripheral vitreous is removed with help of peripheral scleral depression, fluid air exchange done and endolaser PRPC is completed if not done already and surgery completed without any tamponade usually. However, if there is TRD then the tractional membranes are approached and removed by a combination of segmentation and delamination depending upon the cleavage plane available. Horizontal scissors, vitrector, forceps are used as needed to remove the membranes. Many a times there may be vitreoschisis in which case the remnant hyaloid is recognized by staining it with triamcinolone acetonide injection in vitreous and removal of remnant hyaloid is done with help of a forceps. In some cases the posterior pole is s stained with brilliant blue dye and internal limiting membrane is

peeled from the posterior pole. This ensures removal of posterior hyaloid and also facilitates visual recovery**.**

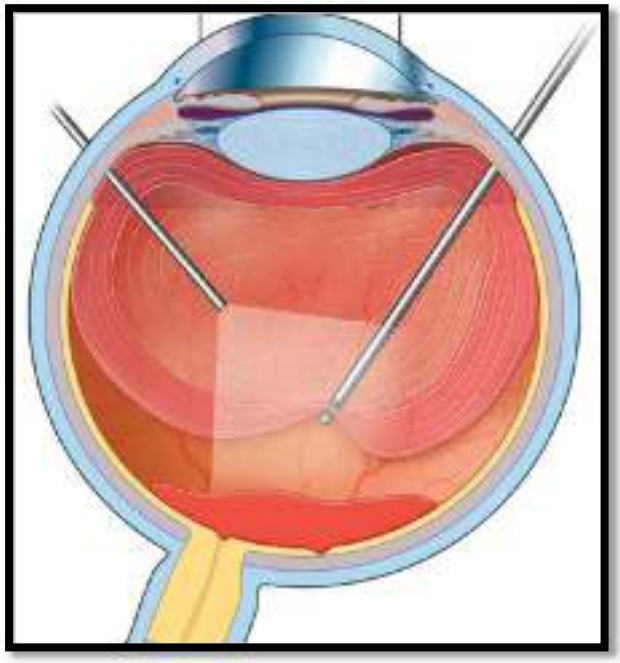

Fig 11: The vitreous cutter easily incises the posterior hyaloid face since a complete posterior vitreous detachment is present.

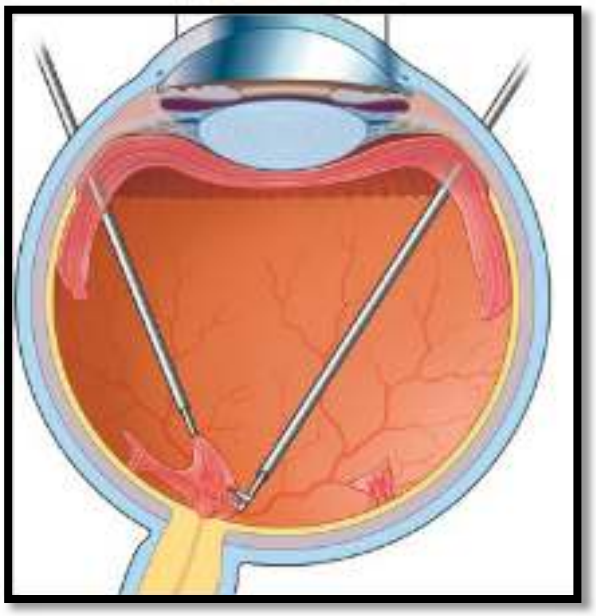

Fig 12: In Delamination,fibrovascular adhesions to the posterior hyaloid are excised parallel to the retinal surface with horizontal scissors.

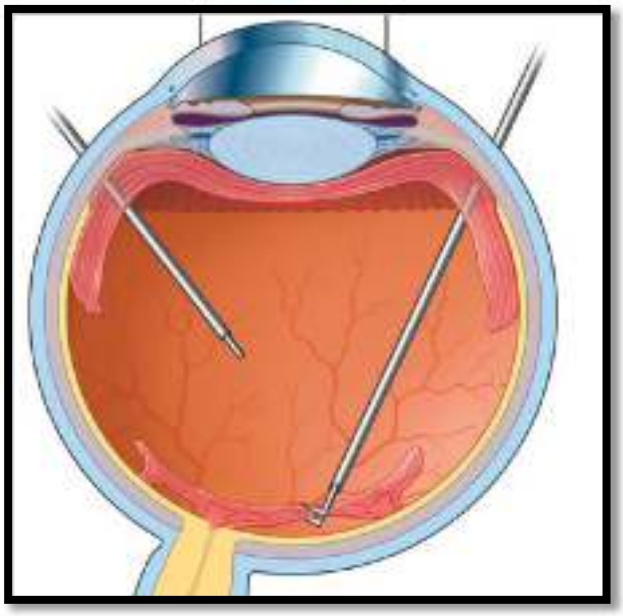

Fig 13: Segmentation with vertically cutting scissors

In case iatrogenic tears occur during surgery then they are marked with endo diathermy. The bleeding points are usually controlled with increasing infusion pressure or gentle pressure on bleeding points or applying endo diathermy to the bleeding points. In cases where very adherent membranes are noted then a chandelier light is fixed in fourth trocar fixed inferiorly and bimanual dissection is used to remove the membranes. After complete removal of membranes fluid air exchange, endolaser to all breaks and PRPC are done. Suitable tamponade is injected depending upon the condition of the retina at the end of surgery. Trocars are removed and wounds are sutured if there is any leakage or silicone oil has been used as tamponade.

Results of PPV for PDR have been reported extensively from 1980s though there are few reports from India. In the beginning of vitrectomy era the main indication used to be non-resolving vitreous hemorrhage (70%) and TRD used to be less common indication for PPV(20%).[81]

However, with advances in instrumentation and experience, the vitreoretinal surgeons are operating more and more cases of TRD with better visual results. Smiddy et al reported that 46% of the cases undergoing PPV for complications of PDR the indication was TRD.[82]

The results of PPV for complications of PDR are varied and depend upon the period during which surgery was done, indications of PPV, pre- operative status of the PDR, complications during surgery and length of follow up. Thomson et al reported results of 1007 patients undergoing surgery for PDR. Among the patients with non-clearing vitreous hemorrhage 79% of patients achieved final visual acuity of 5/200 or better and 48% achieved visual acuity of 20/100 or better. However among patients with TRD 64% achieved VA of 5/200 or better and only 27% achieved VA of 20/100 or better.[83]

Gupta et al from UK reported results of 185 eyes undergoing PPV for complications of PDR (TRD in 59%, non-clearing vitreous hemorrhage in 37%). The VA of 6/12 or better was achieved in only 29% of eyes with TRD compared to 65% in eyes with vitreous hemorrhage.[84]

In another study from UK in which 420 eyes underwent PPV without need for membrane removal improvement in visual acuity of two lines or more was achieved in 63.6% and in 519 eyes that needed delamination improvement in visual acuity of two or more lines was seen in 62%.[85]

The complication rate of PPV for complications of PDR vary between studies and can affect the anatomical and visual results. The complications include intraoperative bleeding, post operative vitreous hemorrhage, redetachment,

reproliferations, neovascular glaucoma and need for resurgery. The rates of complications can be 13% in cases where delamination or segmentation is not needed to 30% where these procedures are needed.(85)

Takayama et al reported that resurgery was needed in 26% of 452patients undergoing surgery for complications of PDR.(86)

Kumar et al observed redetachment in 23% after PPV for complications of PDR in type I DM patients. (87)

The prognostic factors for poor visual results of vitrectomy for PDR include age, chronicity of TRD, initial visual acuity, preoperative presence of NVI, lack of preoperative PRPC and other factors.

AIMS AND OBJECTIVES

PRIMARY OBJECTIVE: To report the results of visual and anatomical outcome of Pars plana vitrectomy for proliferative diabetic retinopathy at the end of 3 months of surgery**.** Anatomical outcome will be concluded as good if retina remains flat at the end of follow up. Visual outcome will be concluded as good if the visual acuity after 3 months follow-up is 6/60 or better.

SECONDARY OBJECTIVE: To study the demographic and clinical characteristics of patients who need Pars plana vitrectomy for proliferative diabetic retinopathy.

MATERIALS AND METHODS

METHODOLOGY

This present study was conducted at Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi on patients who are diagnosed with Proliferative diabetic retinopathy to assess the visual and anatomical outcome using 23guage or 25 gauge pars plana vitrectomy.

METHOD OF DATA COLLECTION

STUDY POPULATION: All patients attending the OPD of the Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre who have been diagnosed with proliferative diabetic retinopathy and have been recommended to undergo pars plana vitrectomy for advanced PDR meeting the inclusion criteria**.**

STUDY DESIGN: A one-year prospective, longitudinal, interventional, non-comparative and hospital based study.

STUDY DURATION: 1st January 2020 to 31st December 2020

SAMPLING PROCEDURE: Considering the prevalence, the formula for calculating the minimal sample size is

$$n = \frac{Z\alpha^2 P(1-P)}{d^2}$$

where P represents the percentage in improvement and d represents the percentage difference in P.zα is related with the significance level. For significance level 5%, zα = 1.96.

Ref: Where P = 84.3%, d = 16% of P = 13.48%, the sample size is 30

Method of Collection of Data

Selection criteria:

Inclusion criteria:

1. Type I or II Diabetic patients above 18 years of age.

The above patients having non-resolving vitreous hemorrhage or dense vitreous hemorrhage

2. Tractional retinal detachment-threatening macula
3. Tractional retinal detachment (TRD) involving macula.
4. Combined tractional and rhegmatogenous retinal detachment
5. Thick posterior hyaloid covering the macula
6. Macular hole due to tractional forces of proliferative diabetic retinopathy.
7. Patients with vision of perception of light or better in the eye to be operated.

Exclusion criteria:

1. Patients who did not complete the follow up of three months after surgery
2. Patients who refused consent to be part of the study
3. Patients having uncontrolled neovascular glaucoma

PROCEDURE:

1. After approval from the institutional review board, patients who satisfy the above-mentioned criteria enrolled into study and demographic data wash noted in a pre-designed proforma.
2. A detailed written and informed consent obtained from all patients after explaining the procedure and the associated risks.
3. Prior to surgery the following investigations will be done:
    a) An in-depth medical and ocular history was obtained at the time of the initial visit, which includes the diabetes duration and its treatment history, duration of hypertension and its treatment history, history of ischemic heart disease and the treatment received for IHD, the history of chronic kidney disease, dialysis history of oral anticoagulants, amputation history. Ocular history includes duration of diminution of vision (DOV) gradual/ sudden in onset, DOV worsening or improving, associated with pain, redness, watering. Any treatment taken after diminution of vision started laser, injection history.
    b) The best corrected visual acuity noted using Snellen's visual acuity chart.
    c) Detailed Slit lamp examination to document other ocular co-morbidities.
    d) Posterior segment evaluation using slit lamp bio microscopy and indirect ophthalmoscopy.
    e) If fundus view is not possible because of vitreous hemorrhage or cataract then ultrasound b scan of the posterior segment done
    f) wherever possible optical coherence tomography was done to document tractional changes in the posterior pole
    g) Basal parameters such as pulse rate, blood pressure, HBA1C

4. Surgical fitness obtained from the treating physician

5. In some cases where there was florid neovascular membranes preoperatively, intravitreal bevacizumab (0.05ml containing 1.25mg) injection was given about 3-5 days before the day of surgery only after surgical fitness was obtained.

6. The steps of surgery: The surgery was conducted under local or general anesthesia depending upon the age of the patient and his/her preference. The eye was cleaned draped, and speculum inserted. Surgery was done with 23 or 25 g vitrectomy using Constellation machine by Alcon company. The surgical steps include core vitrectomy, removing all blood in the vitreous, removing all the membranes that are contributing to traction, ensure hemostasis, perform pan retinal photocoagulation to treat retinal ischemia and seal all tears or holes and inject appropriate tamponade at the end of surgery. The procedure of surgery, findings of TRD and the number of quadrants involved, intraoperative complications and how they were tackled were noted.

More details about procedure have been mentioned under review of literature section. The procedure of surgery, findings of TRD and the number of quadrants involved, intraoperative complications and how they were tackled were noted.

The following tests were performed at each review visit

1. Determination of best corrected visual acuity for distance and near vision.
2. Slit lamp bio microscopy.
3. Fundus examination (+90D lens) and indirect ophthalmoscopy.
4. Intraocular pressure measurement (Goldman’s applanation tonometry).
5. Fundus photography.

The main outcome measures at the end of three months of study were following

1. final visual acuity of 6/60 or better -good visual outcome
2. Improvement of visual acuity post operatively compared to preoperative vision
3. retina flat at the end of three months of follow up was regarded as good anatomical outcome
4. RISK FACTORS FOR final VA of less than 6/60

Risk factors studied for bad visual outcome were- age of the patient (above or below 60 years), initial visual acuity, indication for surgery, preoperative bevacizumab injection given or not, TRD involving 2 quadrants or more than 2 quadrants, combined phaco PPV or only PPV and occurrence of retinal tear during surgery.

Statistical analysis: Data is analyzed using statistical software R version 4.1.1 and Microsoft Excel. Variables (which are continuous) were indicated by the average or mean± SD, while the categorical variables represented in terms of frequency. Shapiro-Wilk's test is used to determine the normalcy of the variables. Two sample t-test used to compare the mean between the groups. Chi-square test will be used to check the association between two categorical variables. Mc-Nemar test is used to compare the pre-operative and post-operative vision, Paired T-test is used to compare the baseline mean log mar vision and post-operative log mar vision. Statistical significance will be established If the p-value is lower than or equal to 0.05.

RESULTS

The study included 30 eyes of 23 subjects. The mean age of the 23 subjects was 56.43±7.5 years, ranging from 45 to 70 years. There were 18(60%) males in the study. All the 23 subjects were suffering from type II DM. The duration of DM ranged from 4 to 30 years with mean of 14.74±7.85years. There were 13 subjects (56.52%) with DM of more than 15 years of duration. Many patients had comorbidities like hypertension in 12(52.17%), IHD in 4(17.39%) and h/o taking anticoagulants in 8(34.78%). The mean HbA1C in 23 subjects as seen preoperatively was 9.09±1.52(range;7.1 to 13.2) indicating poor control of DM. These demographic data are summarized in table 1 and in respective figures. Eighteen (78.26%) patients were on oral hypoglycemics and 5 were receiving insulin injection and oral hypoglycemic agents. None of the patients had chronic kidney disease (CKD).

Table 1: Demographic details.

Below table gives the distribution of demographic details.

Variables		Number of subjects (%)
Age (in years)	45-49	5 (21.74%)
	50-54	3 (13.04%)
	55-59	6 (26.09%)
	60-64	6 (26.09%)
	65-69	1 (4.35%)
	70-74	2 (8.7%)
Age (in years)	56.43±7.5	57 (45, 70)
Gender	Female	5 (21.74%)
	Male	18 (78.26%)
Duration of T2 DM	≤ 5 years	3 (13.04%)
	6-10 years	7 (30.43%)
	> 15 years	13 (56.52%)
Duration of T2 DM	14.74±7.85	14 (4, 30)
Hypertension	Absent	11 (47.83%)
	Present	12 (52.17%)
IHD	Absent	19 (82.61%)
	Present	4 (17.39%)
H/O Anticoagulants	Absent	15 (65.22%)
	Present	8 (34.78%)
HbA1c	9.09±1.52	8.9 (7.1, 13.2)

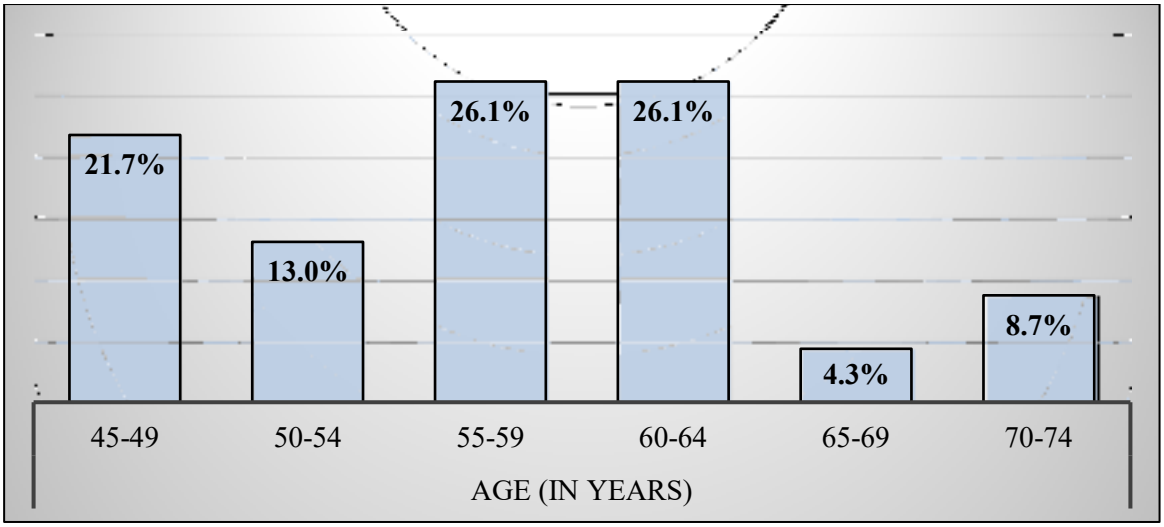

Table 1 Figure 1a: Distribution of subjects by age.

In this study, 60% (14 individuals) falls to the age group just under sixty years (60 yrs), while 39% (9 individuals) falls to the age group greater than sixty years (60 yrs). The mean age was found to be 56.43+7.5 years. The ages ranged from 45 to 74 years.

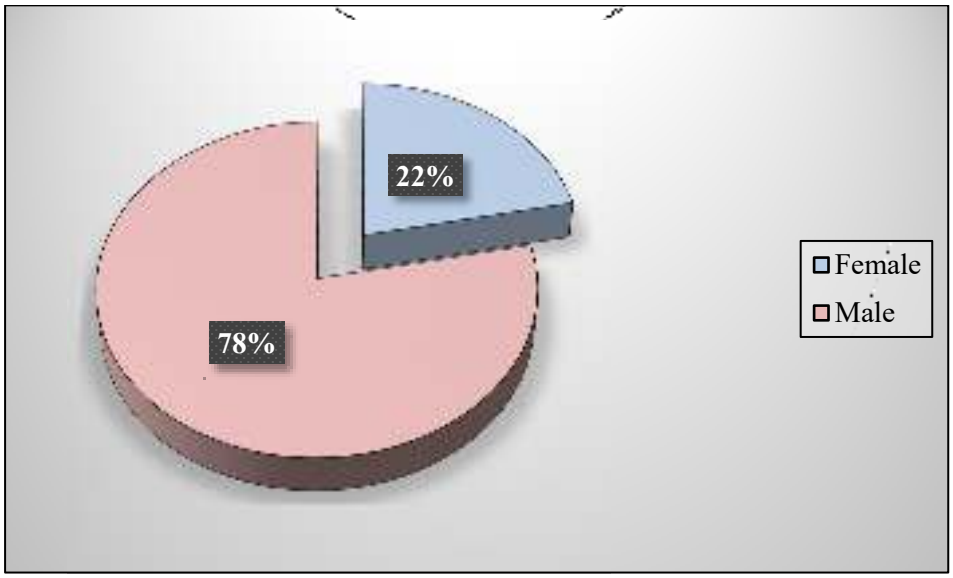

Table 1 Figure 1b: Distribution of subjects by gender. Males constituted 78% of the patients (18), with females being the remainder 22% of the patients (5).

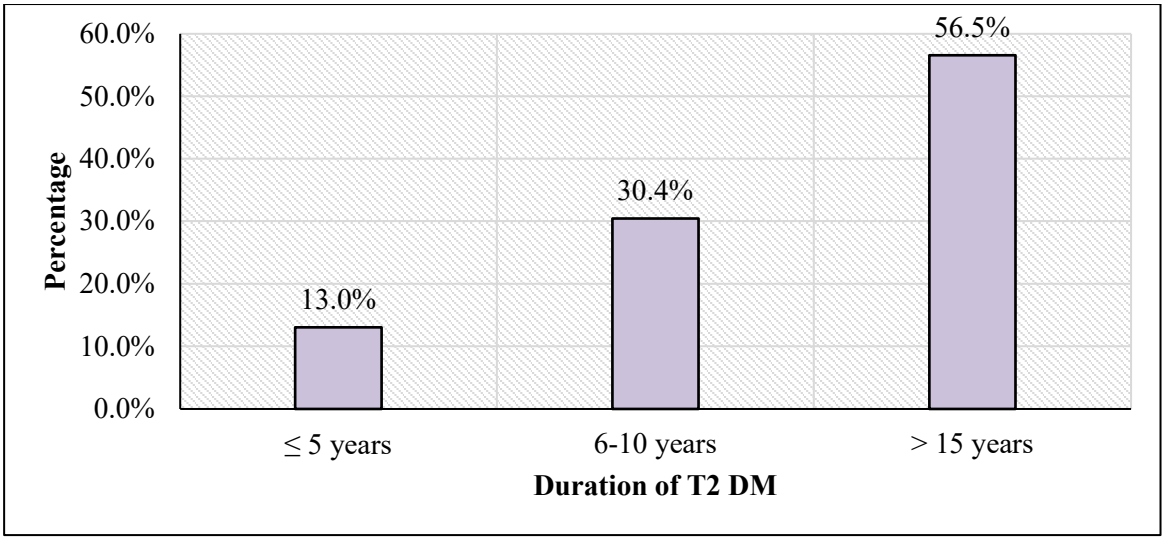

Table 1 Figure 1c : Distribution of subjects by duration of type 2 diabetes mellitus.

In this study there were 3(13.0%) subjects with duration of diabetes less than or equal to 5 years,7(30.4%) subjects with duration of diabetes since 6-10years, 13(56.5%) subjects with duration of diabetes more than 15 years. Mean duration of diabetes was found to be 14.74± 7.85yrs. Lowest and highest duration of diabetes mellitus was found to be 4 and 30 yrs respectively**.**

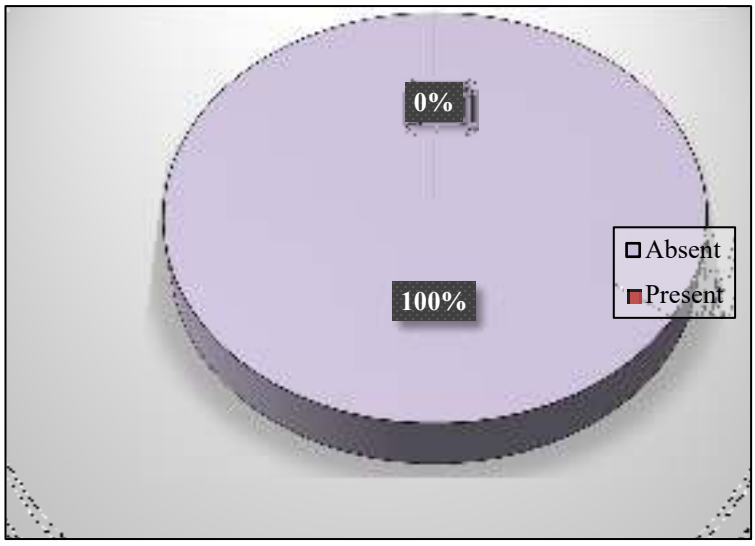

Table 1 fig 1d: No subjects had CKD

Below plot shows mean and standard deviation values for HbA1c

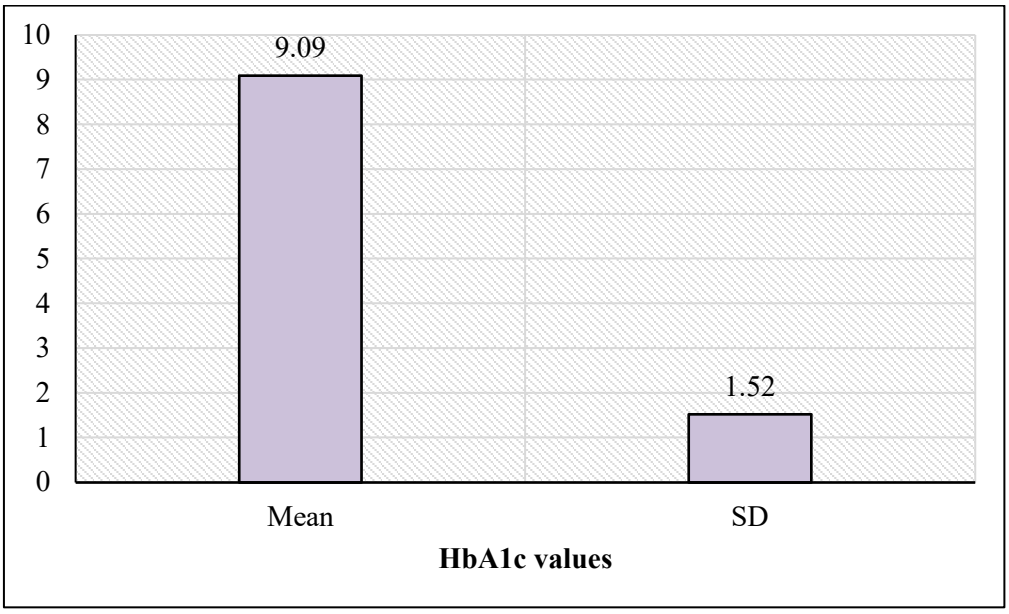

Table 1 Fig 1e:The mean HbA1C in 23 subjects as seen preoperatively was 9.09±1.52(range;7.1 to 13.2)

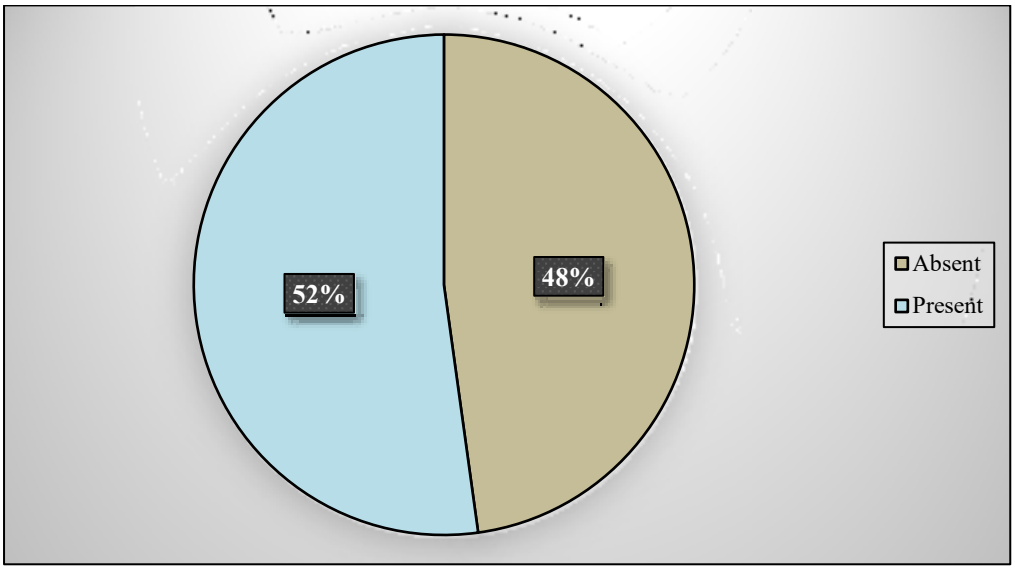

Table 1 Fig1 f: Distribution of subjects by hypertension.

52 percent of the patients (12 patients) were found to be hypertensive

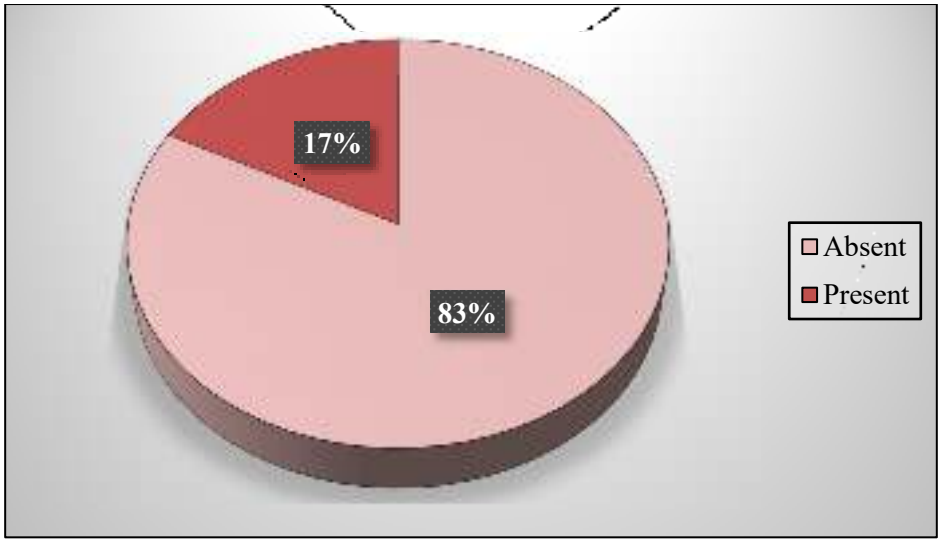

Table 1 Fig 1g: Distribution of subjects by IHD.

17 percent of the patients (4 patients) were found to be having IHD.

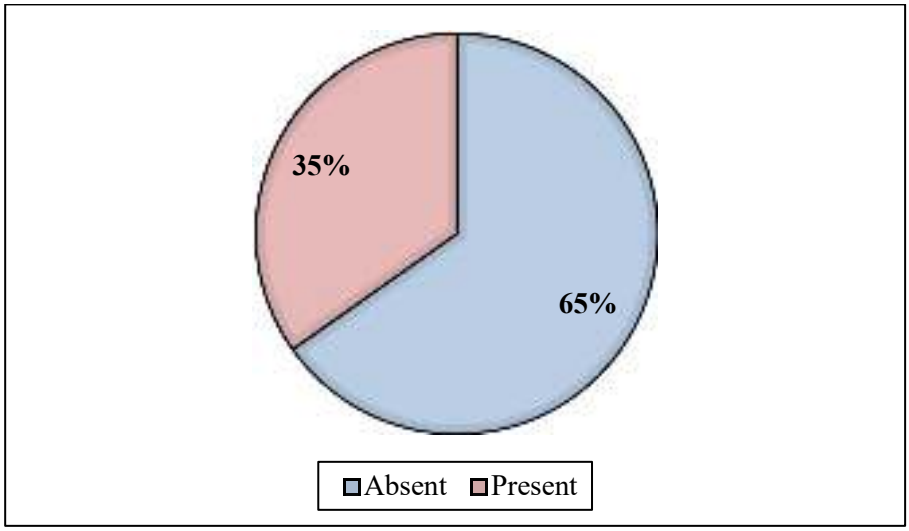

Table 1Fig 1h: Distribution of subjects by H/O Anticoagulants.

8 individuals accounting for 35% of the total were on anticoagulant therapy.

Table2: Distribution of subjects by treatment of T2DM.

Treatment taken	Number of subjects (%)
Oral Hypoglycemic agent	18 (78.26%)
Insulin alone	0 (0%)
Both	5 (21.74%)

Below plot visualizes the above table.

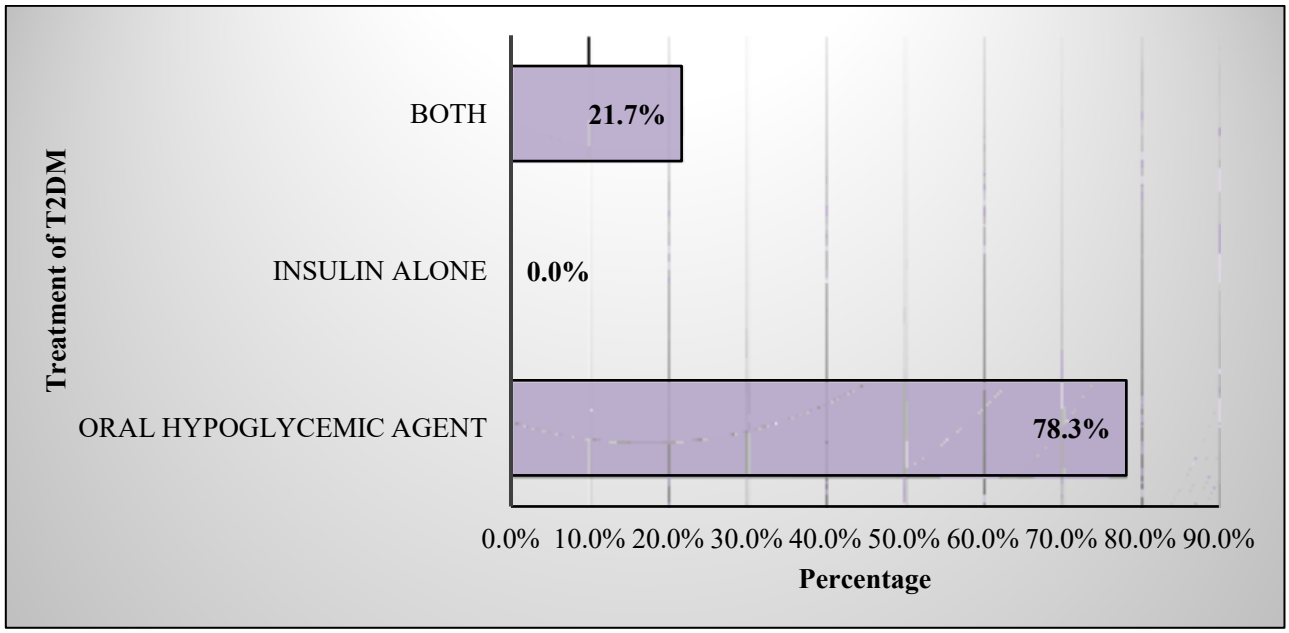

Table 2Fig 2a: Distibution of subjects by treatment of Type 2 DM.

Eighteen (78.26%) patients were on oral hypoglycemics and 5 were receiving insulin injection and oral hypoglycemic agents

Table 3: Distribution of different parameters of eyes.

The dispersion of different parameters related eyes were shown in table-3 below.

Variables		Number of eyes (%)
Duration of DOV (in months)	0-3 months	19 (63.33%)
	4-6 months	9 (30%)
	> 12 months	2 (6.67%)
Duration of DOV (in months)	4.10±5.73	2 (0.5, 24)
Indication of Surgery	Vitreous Hemorrhage	15 (50%)
	TRD	6 (20%)
	Vitreous Hemorrhage+ TRD	8 (26.67%)
	TRD+ RRD	1 (3.33%)
Pre-operative TRD	1 Quadrant	9 (30%)
	2 Quadrants	7 (23.33%)
	3 Quadrants	2 (6.67%)
	4 Quadrants	2 (6.67%)
	No	10 (33.33%)
Pre-operative FVM	1 Quadrant	11 (36.67%)
	2 Quadrants	8 (26.67%)
	3 Quadrants	3 (10%)
	4 Quadrants	2 (6.67%)
	Epiretinal Membrane on Macula	1 (3.33%)
	No	5 (16.67%)

Below plots visualizes the same.

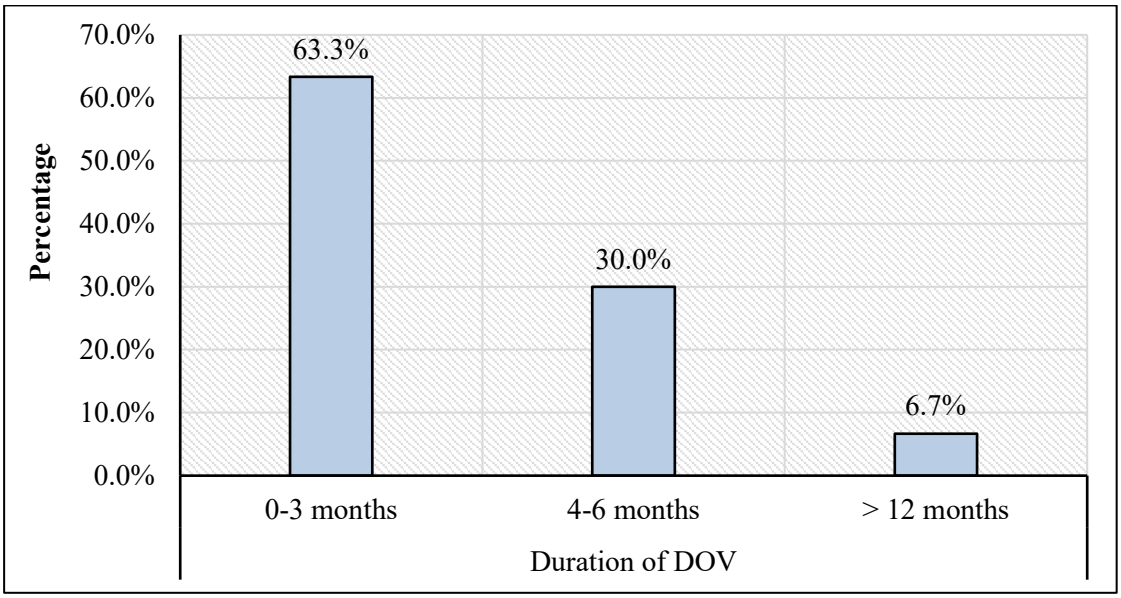

Table 3 Fig 3a: Distribution of eyes by duration of DOV

In this study mean duration of diminution of vision observed was 4.10± 5.73 yrs. Maximum and minimum duration of diminution of vision observed was 15 days and 24 months respectively.

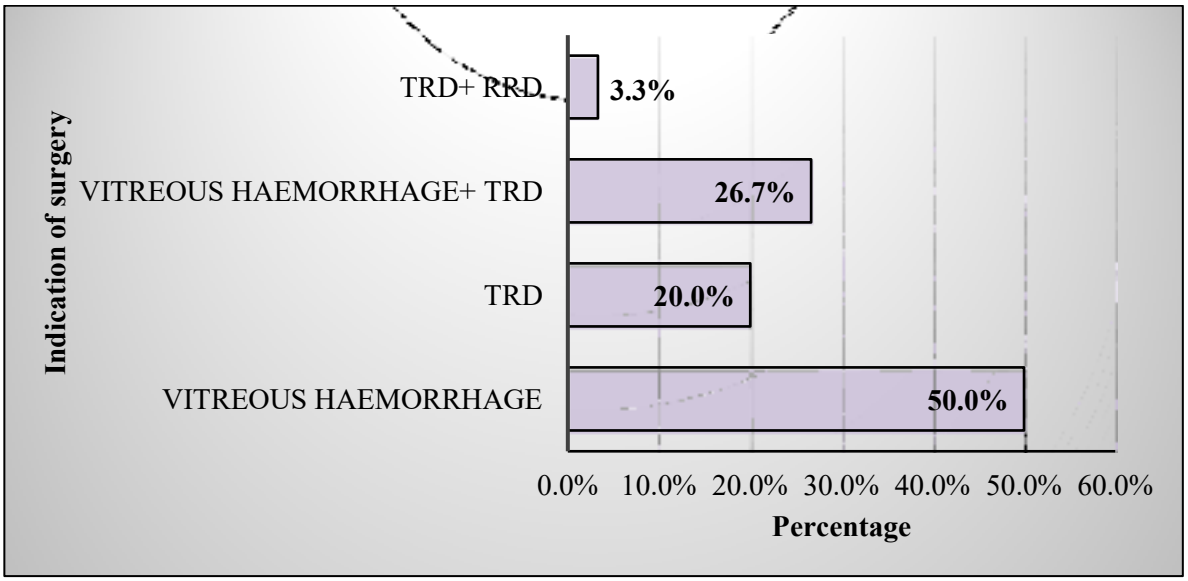

Table 3 Fig 3b: Distribution of eyes by indication of surgery.

In this study indication of surgery observed as vitreous hemorrhage in 50% of the subjects, only TRD observed in 20% of the subjects, vitreous hemorrhage +TRD observed in 26.7% of the subjects, TRD+RRD observed in 3.3% of the subjects

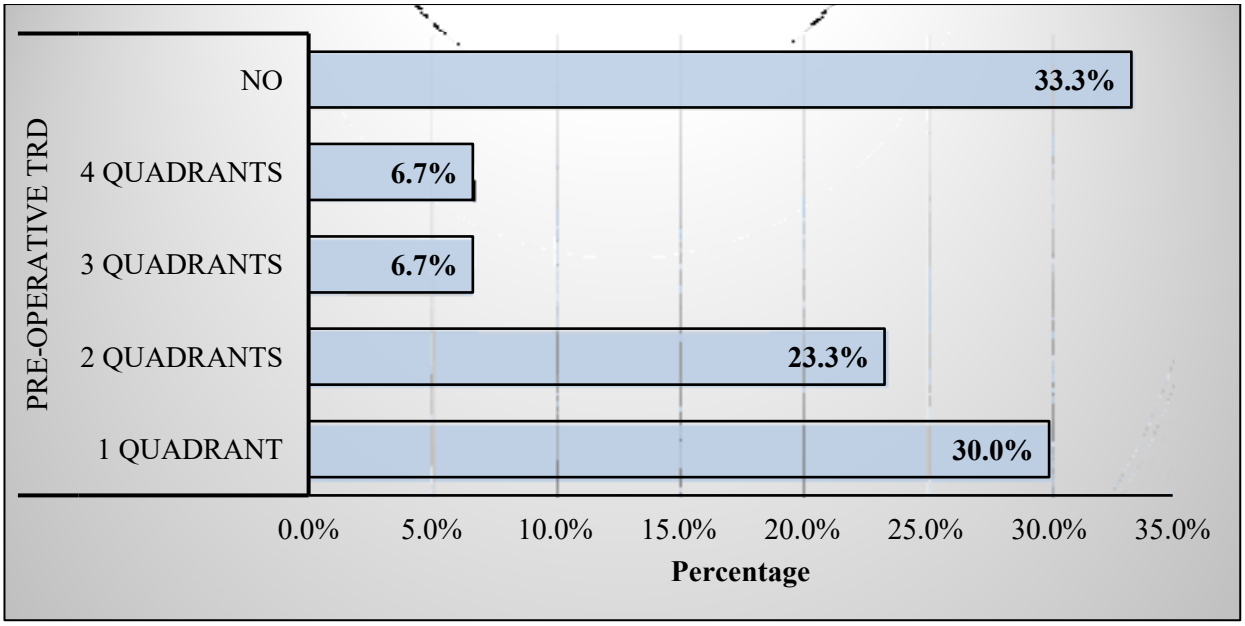

Table 3 Fig 3c: Distribution of eyes based on pre-operative TRD quadrants.

In this study it was observed as preoperative Tractional retinal detachment (TRD) 1 quadrant in 30% of the subjects (9 patients), 2 quadrants in 23.33% of the subjects (7 patients), 3 quadrants in 6.67% of the subjects (2 patients), 4 quadrants in 6.67% of the subjects (2 patients), and not present in 33.33% of the subjects (10patients)

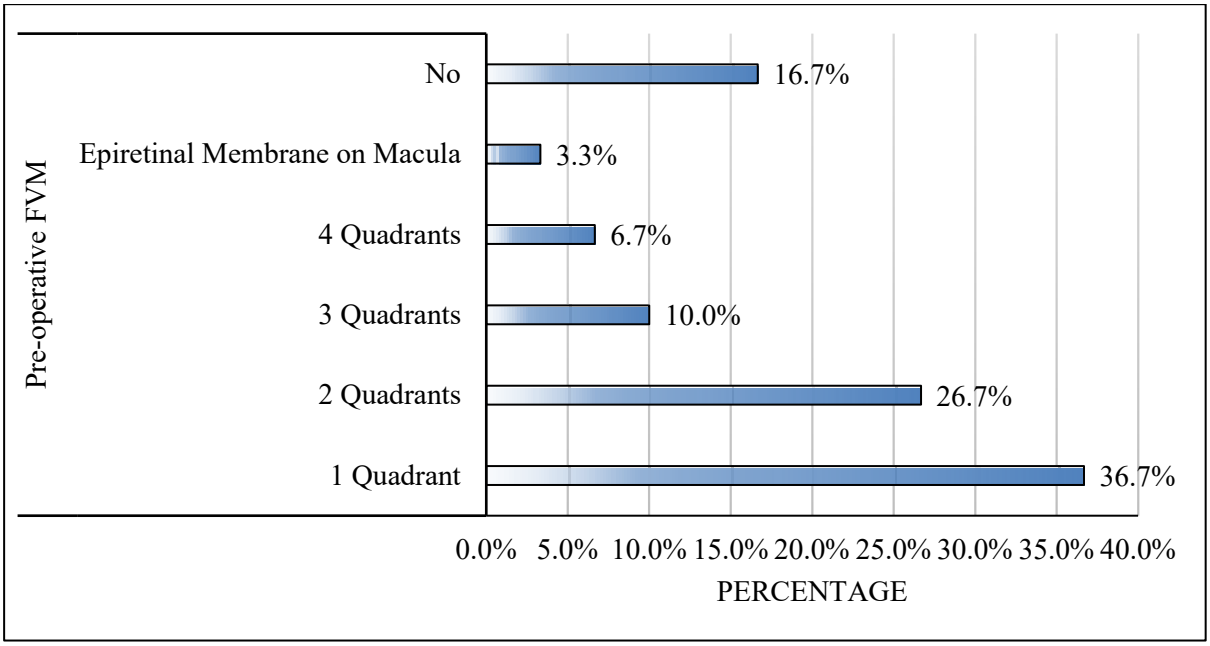

Table 3 Fig 3d: Distribution of eyes based on pre-operative FVM quadrants.

In this study it was observed as preoperative Fibrovascular membrane in 1 quadrant 36.7% of the subjects (11 patients), 2 quadrants in 26.7% of the subjects (8 patients), 3 quadrants in 10% of the subjects (3 patients) 4 quadrants in 6.67% of the subjects (2 patients), epiretinal membrane on macula in 3.33% of the subject (1 patient) and not present in 16.67% of the subject (5 patients).

The mean duration of diminution of vision (DOV) was 4.10±5.73 and ranged from 0.5 months to 24 months. Majority of the patients had DOV of less than 6 months (93.33%). Indications of surgery was non resolving vitreous hemorrhage in 15(50%), TRD in 6(20%) and TRD with vitreous hemorrhage in 8(26.67%) and combined TRD with RhRD in one patient (3.33%). The preoperative visual acuity varied from PL/HM+- to 6/18. Majority of patients 22 (73.33%) had initial VA of <6/60 and only 8 patients (26.66%) had initial VA of >6/60.

The initial VA in log mar units was mean of 1.72+0.52.

As per the preoperative and per operative assessment the TRD involved 1 quadrant in 9(30%), 2 quadrants in 7(23.3%), three and four quadrants each in 2 patients (6.67%).

Five eyes the TRD was noted only during surgery after the vitreous hemorrhage was cleared and it was not macula involving or macula threatening. Fibrovascular membrane with or without TRD was noted in 24 eyes (80%) and their quadrant wise involvement is shown in the table 3.

Four patients needed bimanual surgery because of extensive membranes. One patient had epiretinal membrane on the macula.

Table 4: Relationship between Avastin injection and post-op vision.

Below table compares the relationship between Avastin injection and post-operative vision.

Nine (30%) eyes had received preoperative intravitreal injection of bevacizumab for presence of florid neovascularization

		Post-operative vision		p-value
		< 6/60 (n=10)	≥ 6/60 (n=20)	
Avastin Injection	Given	6(60%)	3(15%)	0.01699*[MC]
	Not given	3 (30%)	18 (90%)	

Abbreviations: MC: Monte-Carlo's simulation used in Chi-square test.

By Chi-square test, there is significant association present between Avastin injection with post-operative vision.

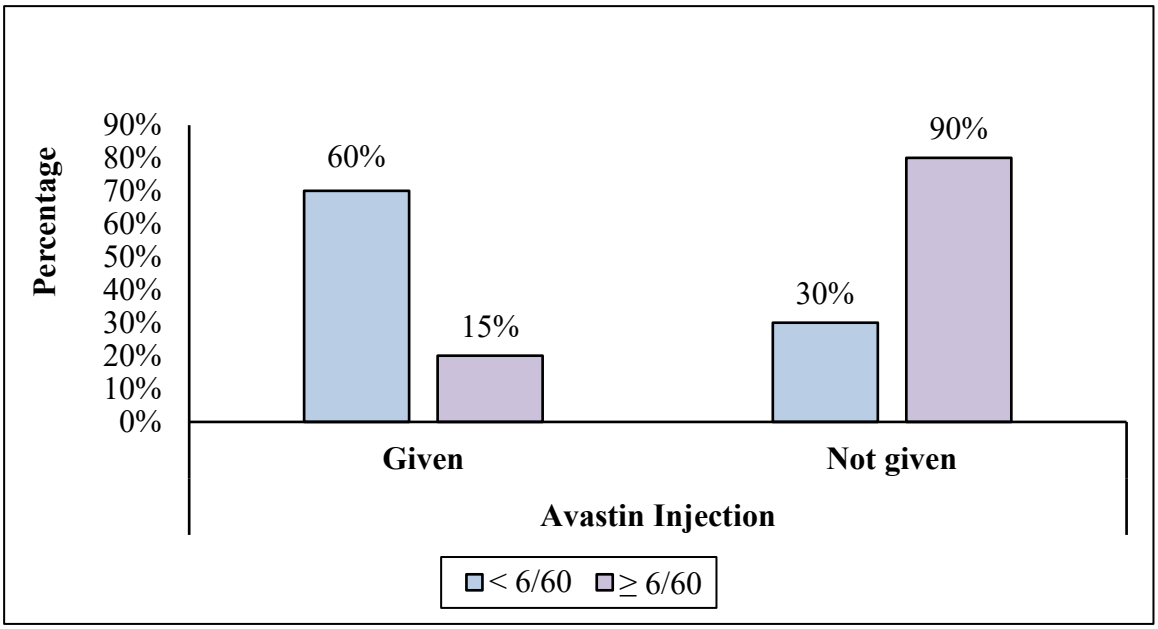

Table4 fig4a: Comparison of Avastin injection over postoperative grading

In the present study it was observed that Preoperatively Avastin injection was given, 6(60%) eyes had postoperative vision <6/60 and 3(15%) eyes had postoperative vision >6/60. Preoperatively Avastin injection was not given, 3eyes (30%) had postop vision<6/60 and 18 (90%) had postop vision more than 6/6

Table 5: Distribution of surgical steps among subjects

Fluid air exchange was done in 29 eyes and endolaser was done in all except one to complete the PRPC and /or treat retinal breaks. Intravitreal tamponade was used in 25 eyes (83.33%) and none in 5 eyes. In 15 eyes (50%) the tamponade used was either perfluoropropane gas(C3F8 -14%) in 9(30%) or sulphur hexafluoride gas in 6 eyes(20%). In ten eyes we used silicone oil as tamponade because of the severity of the TRD or because of incomplete removal of membranes in some cases.

ILM Peeling	Done	12 (40%)
	Not Done	18 (60%)
FAE and EL	Done	29 (96.67%)
	Not Done	1 (3.33%)
SF6/C3F8	C3F8	9 (30%)
	SF6	6 (20%)
	No	15 (50%)
Silicone oil	Done	10 (33.33%)
	Not Done	20 (66.67%)

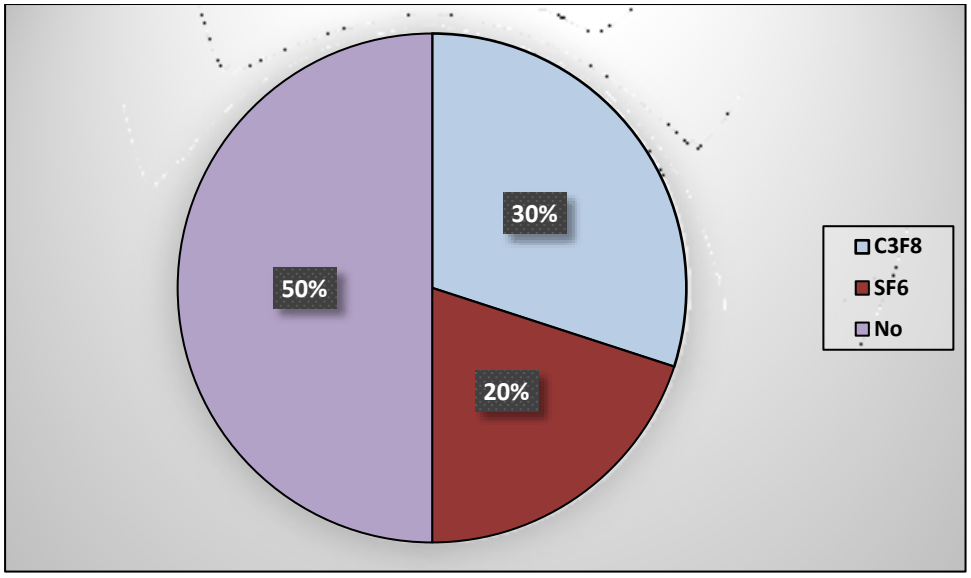

Table5 Figure 5a: Distribution of eyes by C3F8/SF6.

In this study it was observed as C3f8 injected in 30% of the subjects, SF6 injected in 20% of the subjects.

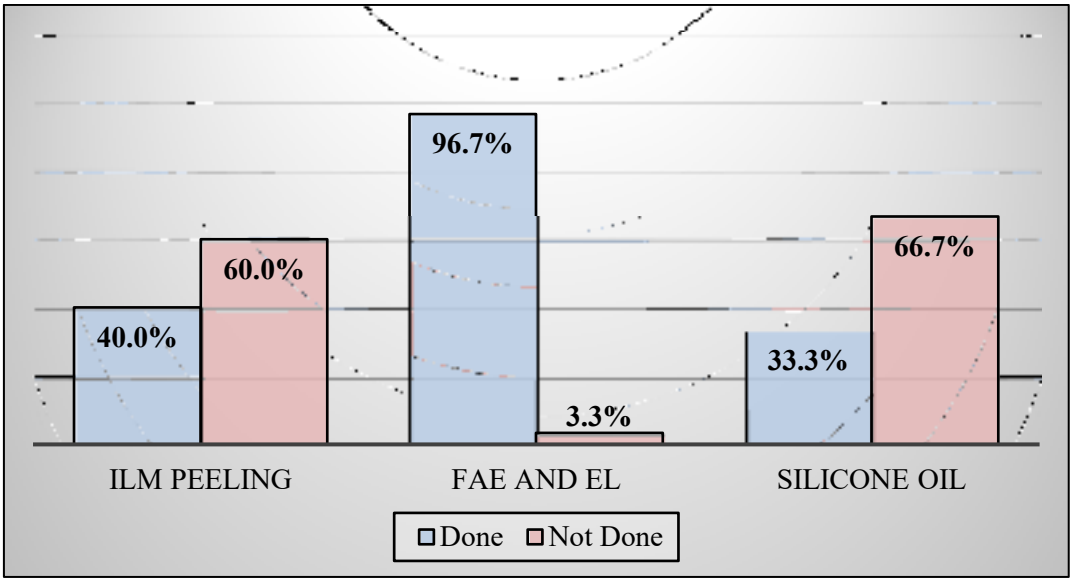

Table 5 Fig 5b: Distribution of eyes by ILM peeling, FAE & EL, Silicone oil.

In this study it was observed as Internal limiting membrane peeling done in 40% of the subjects (12patients), Fluid air exchange and endolaser was not done in 3.3% of subjects (1patient), silicone oil was injected in 33.33% of the subjects (10 patients).

Table 6: Distribution of surgery.

Surgery	Number of eyes (%)
PPV+MP+FAE+EL+SOI	4 (13.33%)
PPV+MP+FAE+EL+GAS	6 (20%)
Phaco Fragmentation +PPV+MP+FAE+EL+SOI	1 (3.33%)
PPV+MP+FAE+EL+AIR	1 (3.33%)
PHACO+IOL+PPV+MP+FAE+EL+AIR	3 (10%)
PPV+MP+ILM+FAE+EL+GAS	9 (30%)
Bimanual Dissection +PPV+MP+FAE+EL+SOI	2 (6.67%)
Bimanual Dissection +Phaco +IOL+ PPV+MP +FAE+EL+ SOI	1 (3.33%)
Bimanual Dissection +PPV+MP+PFCL INJECTION+ PFCL AIR EXCHANGE+EL	1 (3.33%)
PPV+FAE+EL+AIR	1 (3.33%)
PHACO+IOL+PPV+MP+FAE+EL+SOI	1 (3.33%)

Table 7: Distribution of intra-op complications over indication.

Indication	Intra-op complications				
	No	**Retinal tear**	**Bleeding**	**Bleeding +Tear**	**Membrane Left**
TRD	1 (16.67%)	3 (50%)	1 (16.67%)	0 (0%)	1 (16.67%)
TRD+RRD	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0 (0%)
VH	4 (26.67%)	2 (13.33%)	7 (46.67%)	0 (0%)	2 (13.33%)
VH+TRD	1 (12.5%)	2 (25%)	2 (25%)	2 (25%)	1 (12.5%)

There were twelve intraoperative bleeding cases and ten iatrogenic retinal breaks (two from vitreous hemorrhage group and six from tractional retinal detachment group) in this study. Membranes were left in four (13%) eyes. Six (20%) eyes had no intraoperative complications.

These occurred in significant number of eyes in our series. Intraoperative bleeding was noted in 12 (40%) eyes and was controlled with intraoperative diathermy or by rasing the infusion pressure. Intraoperative retinal breaks occurred in 10 eyes (33%). Corneal clouding due to epithelial edema occurred in 23 eyes necessitating the debridement of the epithelium in them. However all the eyes showed healing of the corneal epithelial defect with bandage contact lens and had no residual corneal complications.

Table 8: Distribution of post-op complications over indication.

Indication	Post-op complications						
	Increased IOP+ Macular Edema	Macular edema	No	Silicone Oil in Anterior Chamber	Subretinal Fluid	VH	VH+ Increased IOP
TRD	0 (0%)	0 (0%)	3 (50%)	0 (0%)	1 (16.67%)	2 (33.33%)	0 (0%)
TRD+RRD	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)
VH	1 (6.67%)	0 (0%)	10 (66.67%)	0 (0%)	0 (0%)	4 (26.67%)	0 (0%)
VH+TRD	0 (0%)	1 (12.5%)	3 (37.5%)	1 (12.5%)	0 (0%)	2 (25%)	1 (12.5%)

A total of eight (26.67%) eyes had recurrent vitreous hemorrhage, one (3.33%) eye had macular edema, one (3.33%) eye had elevated IOP and macular edema, one (3.33%) eye had silicone oil in the anterior chamber, and two (6.66%) eyes had subretinal fluid**.**

The post operative complications included vitreous hemorrhage in 9 eyes (30%) and in all eyes it cleared with time.

Table 9: Distribution of post-operative outcomes observed in the study.

Below table gives the distribution of post-operative outcomes observed in the study.

Post-operative Outcomes		Number of Eyes (%)
Retina	Not Flat	2 (6.67%)
	Flat	28 (93.33%)
Any Membranes	Absent	20 (66.67%)
	Present	10 (33.33%)
Vitreous Hemorrhage	Absent	27 (90%)
	Present	3 (10%)
Re-surgery	Done	2 (6.67%)
	Not Done	28 (93.33%)
TRD	Absent	27 (90%)
	Present	3 (10%)
NVG	Absent	30 (100%)
	Present	0 (0%)
Silicone Oil in AC	Absent	30 (100%)
	Present	0 (0%)
Silicone Oil	Absent	19 (63.33%)
	Present	11 (36.67%)

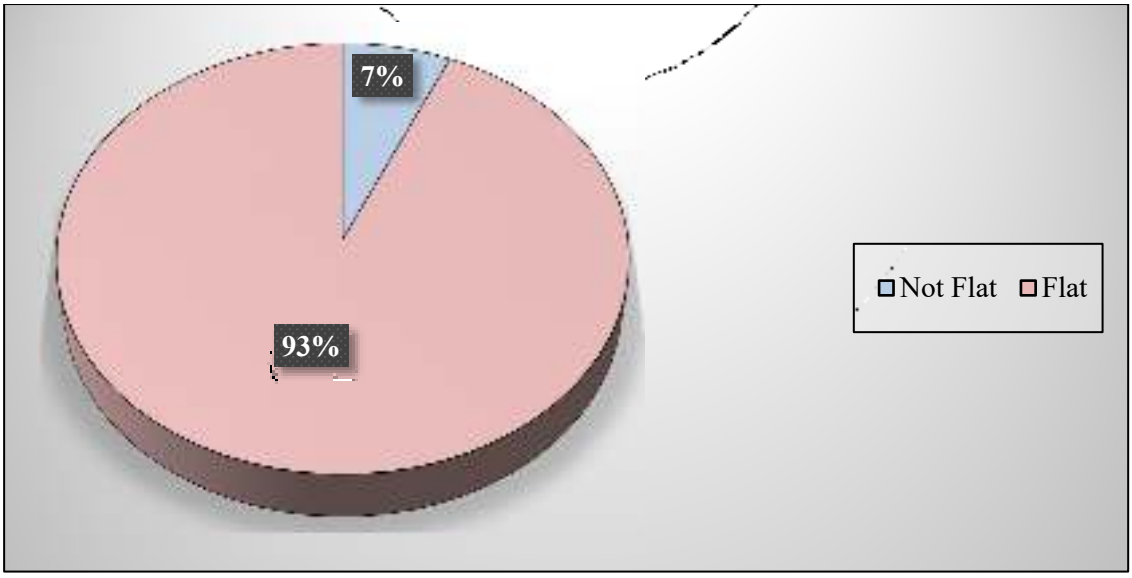

Table 9 Figure 9a: Distribution of eyes by post-operative retina category.

In this study it was found that retina was flat in 93% of the subjects at 3 months follow up,not flat in 7% of the subjects.

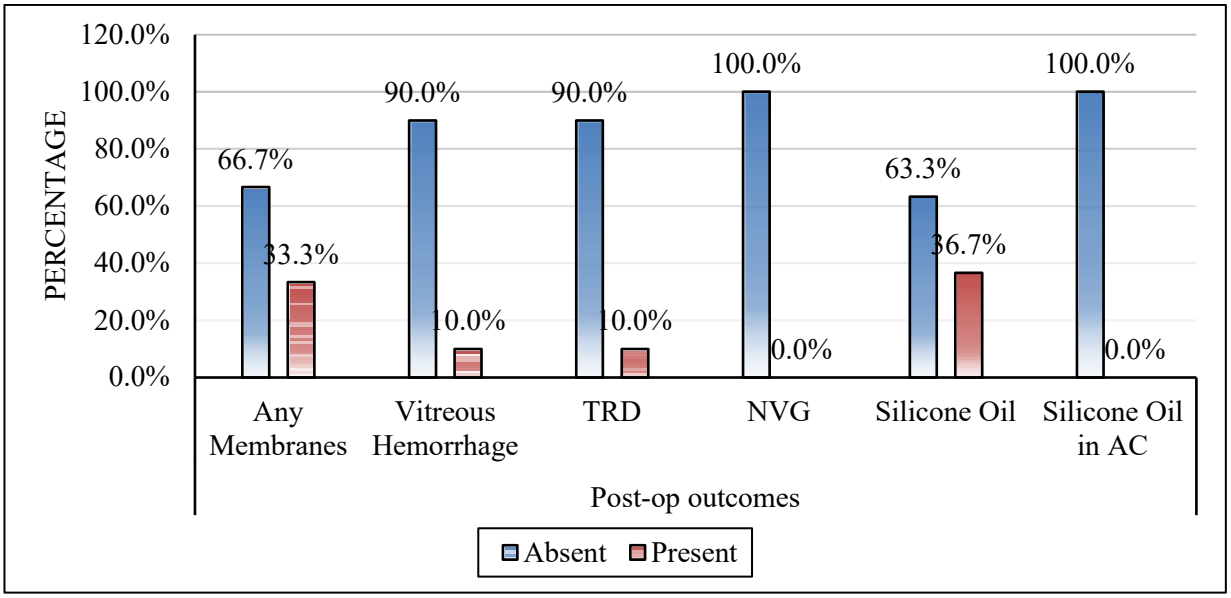

Table 9Figure 9b: Distribution of eyes by different post-operative outcomes.

In this study it was observed that postoperatively at 3 months follow up any membranes was absent in 66.7% of the subjects, present in 33.3% of the subjects. vitreous hemorrhage was absent in 90% of the subjects, present in 10% of the

subjects. Tractional retinal detachment was absent in 90% of the subjects, present in 10% of the subjects. Neovascular glaucoma was absent in 100% of the patients, silicone oil was absent in 63.3% of the subjects, present in 36.7% of the subjects, silicone oil in anterior chamber was absent in 100% of the subjects.

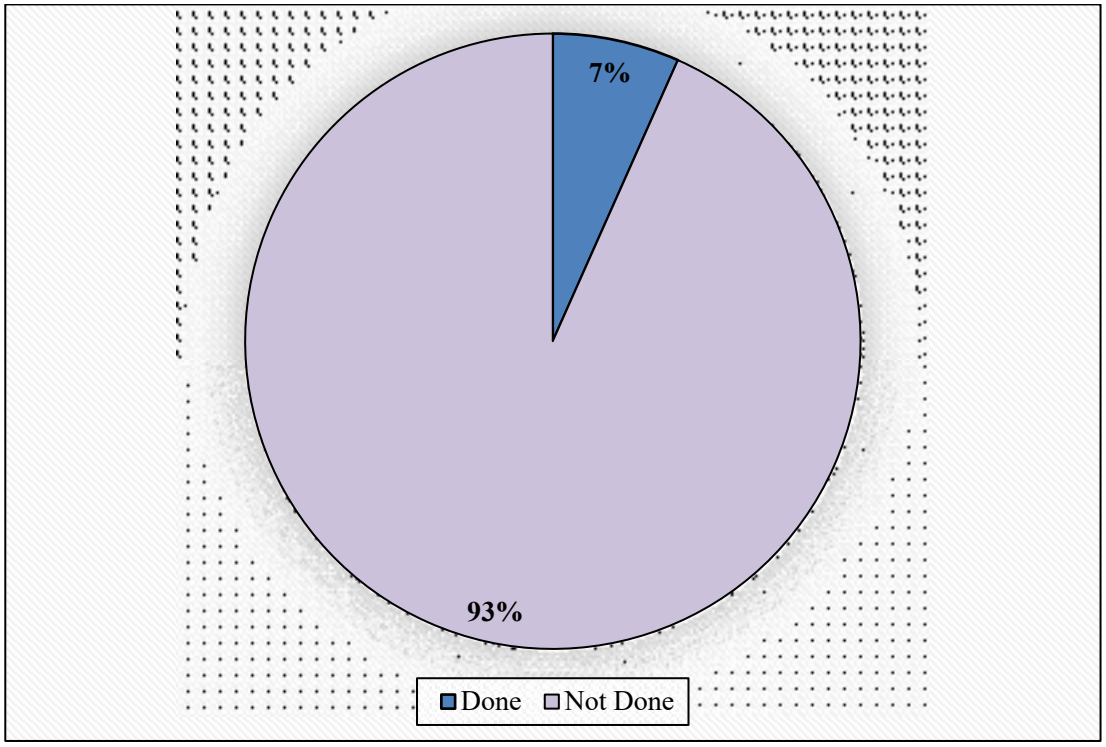

Table 9 Figure9c: Distribution of eyes by post-operative re-surgery.

In this study it was found that resurgery was done for 7% of the subjects at 3 months follow up, not done for 93% of the subjects.

Three patients had some residual vitreous hemorrhage at the end of the study. Three patients had post operative tractional RD in which two patients needed re-surgery because the macula was involved in them. None of the patients developed neovascular glaucoma or phthisis. One of the patients who underwent resurgery needed silicone oil tamponade. Hence at the end of our study 11 eyes were silicone filled. However,

three patients had residual TRD under silicone oil at the end of follow up. Some epiretinal membrane that did not affect the vision was noted in 10 patients (33.33%).

Table 10: Comparison of visions at baseline and at 3 months.

Below table compares pre-operative vision and post-operative visions in the study.

		Post-op Vision		p-value
		< 6/60	**≥ 6/60**	
Baseline vision	**< 6/60**	9 (30%)	13 (43.33%)	0.003283*
	≥ 6/60	1 (3.33%)	7 (23.33%)	

In the above table we can see that, 30% of the subjects had vision less than 6/60 before and after the surgery. However, 13 (43.33%) of the subjects had vision less than 6/60 before the surgery and it was improved to greater than or equal to 6/60.

By Mc-Nemar's test, there is significant difference in the distribution of baseline vision and post-operative vision.

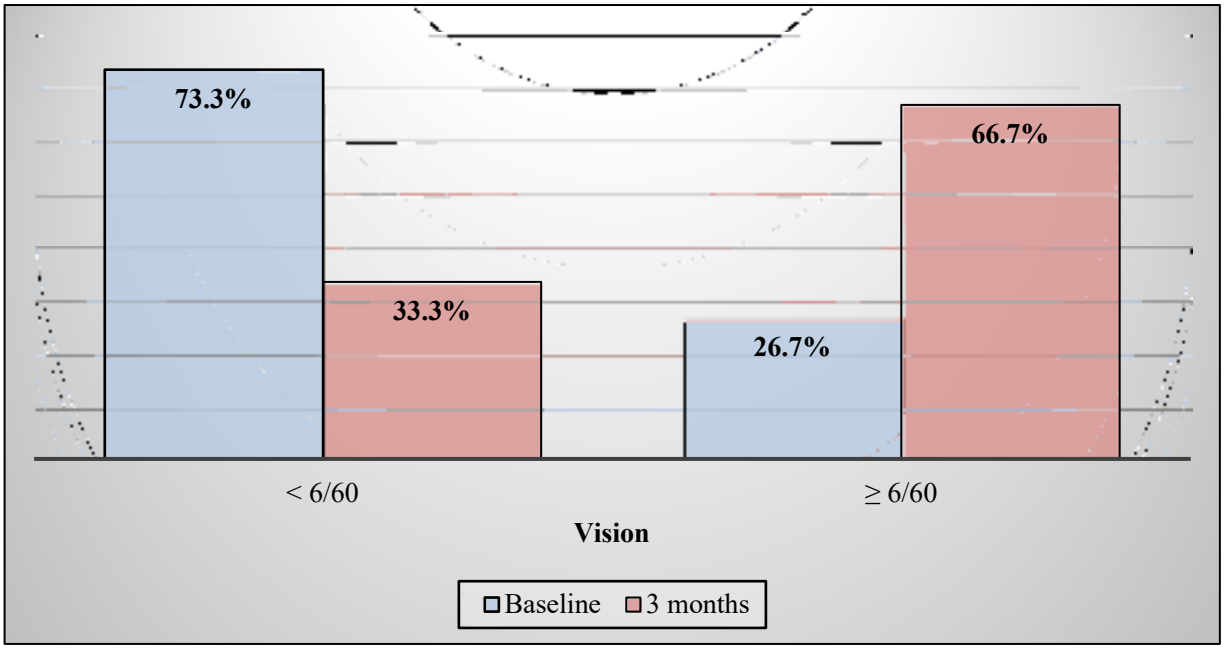

Table 10 Figure 10a: Comparison of visions at baseline and 3 months.

Table 11: Comparison of Log mar visions between the time points

Below table compares the baseline log mar vision and post-operative log mar vision.

	Baseline	**Post-op**	**p-value**
Log Mar vision	1.72±0.52	1.02±0.67	<0.00001*

By paired t-test, mean log mar vision is significantly large at baseline compared to post-operative log mar vision.

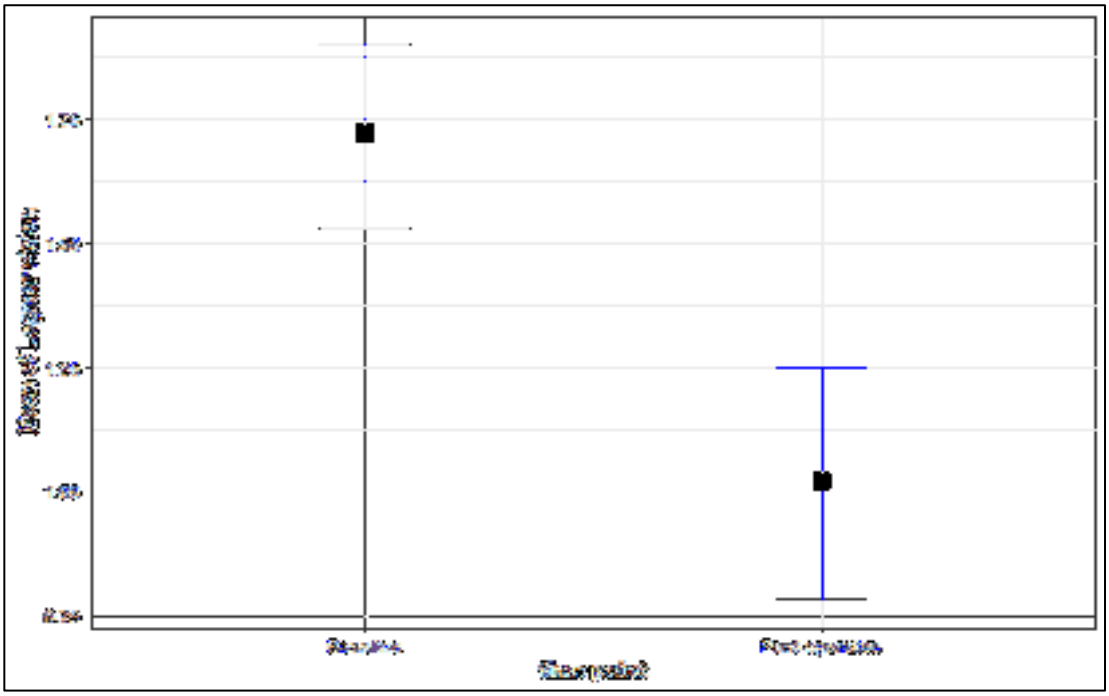

Table 11 Fig 11a: Mean of Logmar vision over time point.

Table 12: comparision of preoperative and post operative vision in snellens

VISION	PL+, HMCF	CF TO <6/60	6/60 TO 6/36	6/24 TO 6/18	6/12 and above
PREOPERATIVE	5	17	7	1	NONE
POSTOPERATIVE	1	9	7	8	5

Visual results of surgery:

The preoperative and post operative visual acuity distribution in Snellen acuity is shown in the above table. There was significant improvement in visual acuity with preoperative log mar acuity of 1.72±0.52 improving to 1.02±0.67 ($p<0.0001$). There were 22(73.33%) eyes with initial VA of <6/60 and at the end of our study this number came down to 10 eyes (33%) ($P=0.0032$ by Mc Nemara test) which was statistically significant. Out of the 30 eyes 26 eyes showed improvement in visual acuity two eyes showed no change in visual acuity and two eyes showed worsening of visual acuity.

Table 13: Comparison of age with post-op vision.

Below table compares the age distribution with vision category.

		Post-op Vision		p-value
		< 6/60	≥ 6/60	
Age	< 60 years	8 (80%)	12 (60%)	0.4198[MC]
	≥ 60 years	2 (20%)	8 (40%)	

Abbreviations: MC: Monte-Carlo's simulation used in Chi-square test.

By Chi-square test, there is no significant difference in the distribution of age group over post-operative vision.

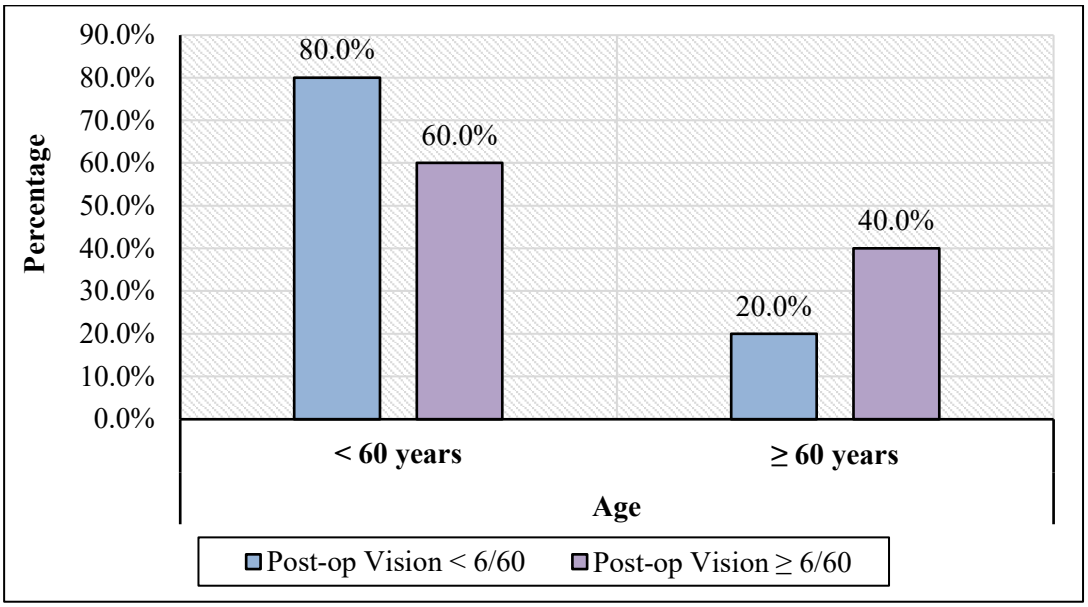

Table13 Figure13a: Distribution of age over post-op vision.

Table 14: Comparison of different parameters over post-operative vision grading.

Below table compares the different pre-operative parameters with post-operative vision grading. Post-operative vision divided into two categories as vision greater than or equal to 6/60 or vison less than 6/60

Variables		Post-operative vision		p-value
		< 6/60 (n=10)	**≥ 6/60 (n=20)**	
Age (in years)		56±5.93	55.55±7.74	0.8731[t]
Age (in years)	40-49	1 (10%)	6 (30%)	0.1504[MC]
	50-59	7 (70%)	6 (30%)	
	60-69	1 (10%)	7 (35%)	
	≥ 70	1 (10%)	1 (5%)	
Indication of Surgery	TRD	3 (30%)	3 (15%)	0.953[MC]
	Vitreous Hemorrhage	4 (40%)	11 (55%)	
	Vitreous Hemorrhage+ TRD	3 (30%)	5 (25%)	
	TRD + RRD	0 (0%)	1 (5%)	
TRD pre-op Quadrants	< 2	6 (60%)	13 (65%)	1[MC]
	≥ 2	4 (40%)	7 (35%)	
Retinal tear	Absent	4 (40%)	17 (85%)	0.02849*[MC]
Intraop	Present	6 (60%)	3 (15%)	
Surgery type	Combined	1 (10%)	3 (15%)	1[MC]
	Single	9 (90%)	17 (85%)	

Abbreviations: t: Two sample t-test; MC: Monte-Carlo's simulation used in Chi-square test.

By two sample t-test, mean of age is not significantly differ between the vision grading. Similarly, there is no association present between age group, indication for surgery, TRD pre-op quadrants and type of surgery with post-operative vision by Chi-square test.

However, by Chi-square test, there is significant association present between intraoperative retinal tear and post-operative vision.

Odds of having post-operative vision greater than or equal to 6/60 is 8.5 (CI: 1.4585-49.5386) times mores for the subjects who don't had retinal tear compared to the subjects who had retinal tear.

Below plots visualizes the above table.

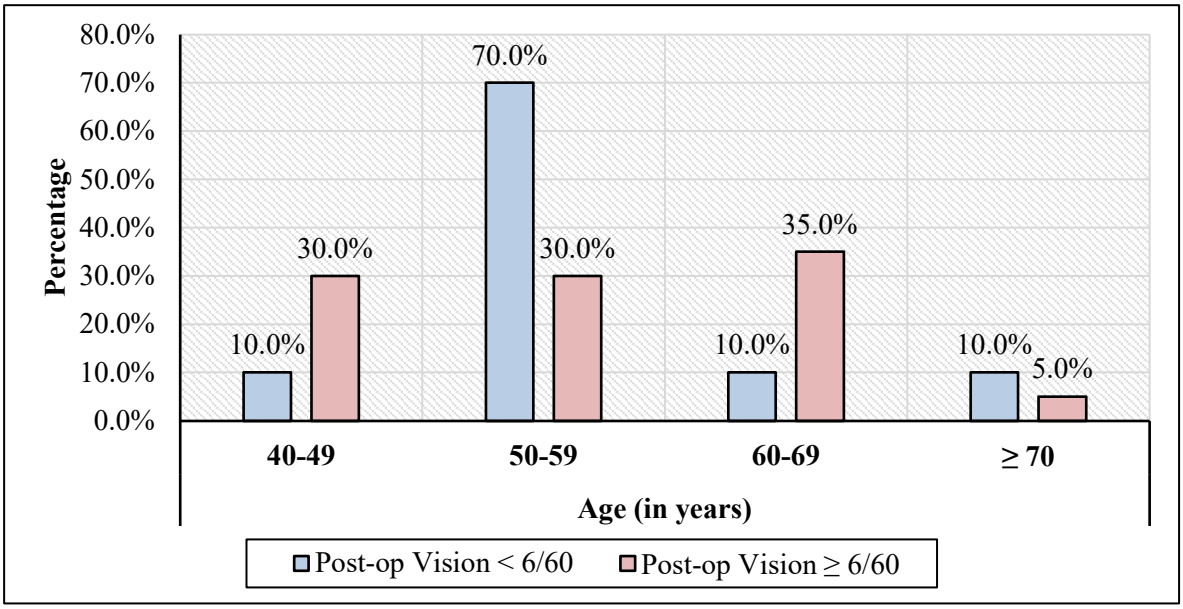

Table 14Figure 14a: Distribution of eyes by age and post-operative vision.

In this study it was observed by two-sample t-test, mean of age is not significantly differ between the vision grading. Mean age group in $\leq$ 6/60 was 56$\pm$ 5.93yrs,while in $\geq$ 6/60 was 55.55$\pm$ 7.74yrs**.**

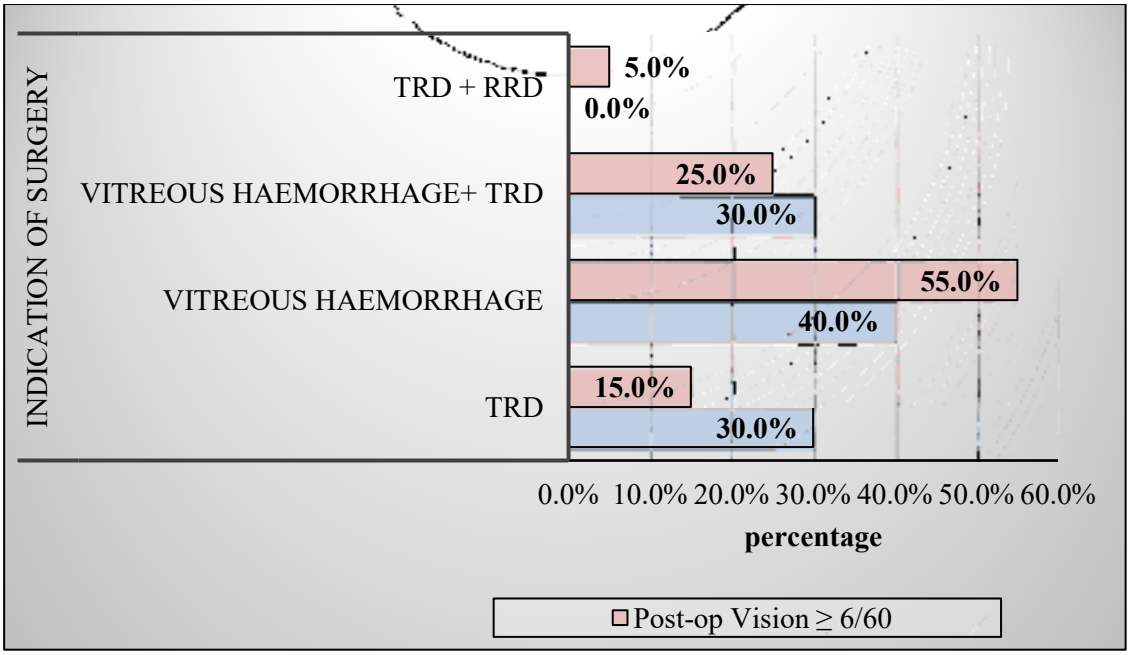

Table 14 Figure 14 b: Distribution of eyes by indication of surgery and post-operative vision.

In this study it was observed that post operative vision was < 6/60 in 30% of the subjects,>6/60 in 15% of the subjects with preoperative Tractional retinal detachment(TRD).Postoperative vision was <6/60 in 40% of the subjects,>6/60 in 55% of the subjects with preoperative vitreous hemorrhage. Postoperative vision was <6/60 in 30% of the subjects,>6/60 in 25% of the subjects with preoperative vitreous hemorrhage +TRD. Postoperative vision was <6/60 in 0% of the subjects,>6/60 in 5% of the subjects with preoperative TRD+RRD.

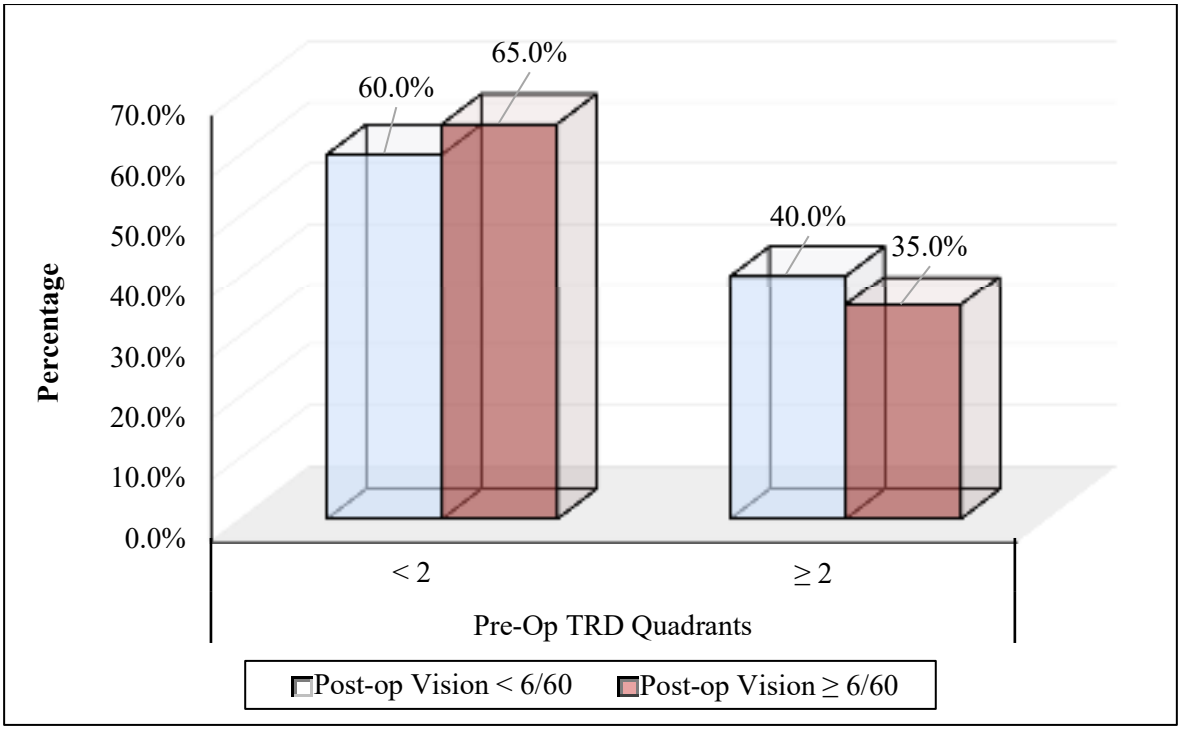

Table 14 Figure 14 c: Distribution of eyes by Pre-op TRD quadrants and post-operative vision.

In this study it was observed that Post operative vision < 6/60 in 60% of the subjects,>6/60 in 65% of the subjects with preoperative Tractional retinal detachment < 2 quadrants, while postoperative vision <6/60 in 40% of the subjects,>6/60 in 35% of the subjects with preoperative Tractional retinal detachment > 2 quadrants.

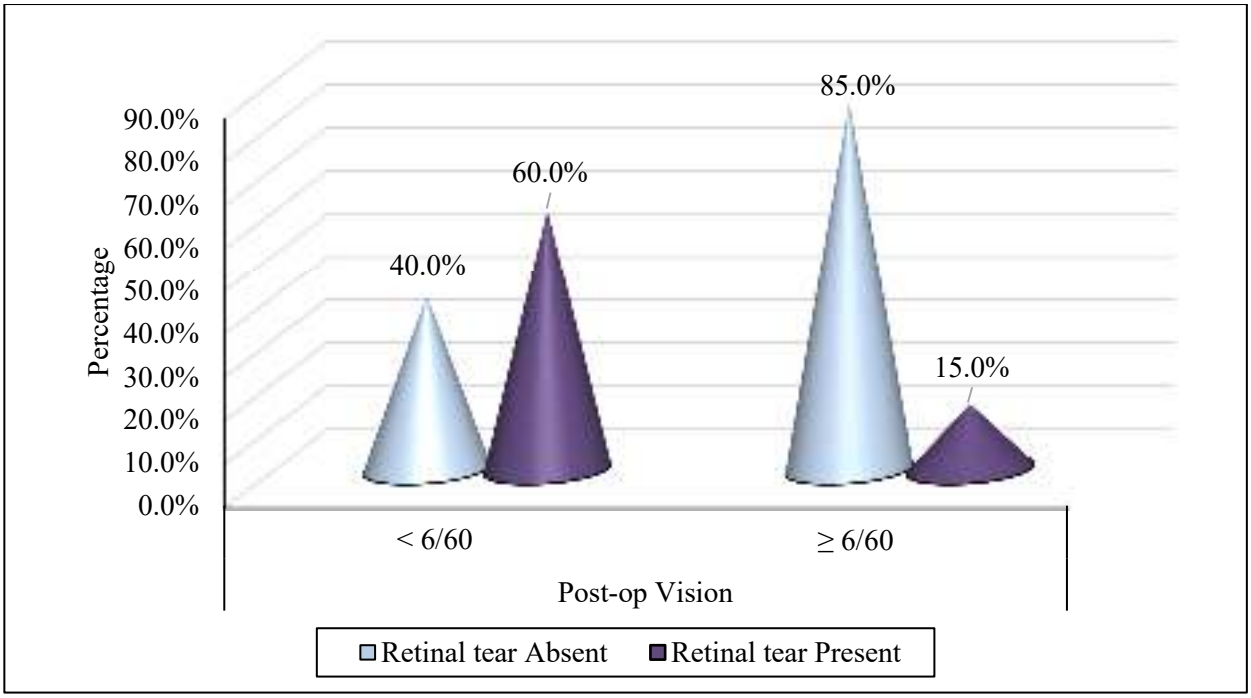

Table 14 Fig 14d: Distribution of eyes by Intra operative retinal tear and post-operative vision.

In this study it was observed that postoperative vision was <6/60 in 40% of the subjects,>6/60 in 85% of the subjects with no intraoperative retinal tears, while postoperative was <6/60 in 60% of the subjects,>6/60 in 15% of the subjects with intraoperative retinal tears.

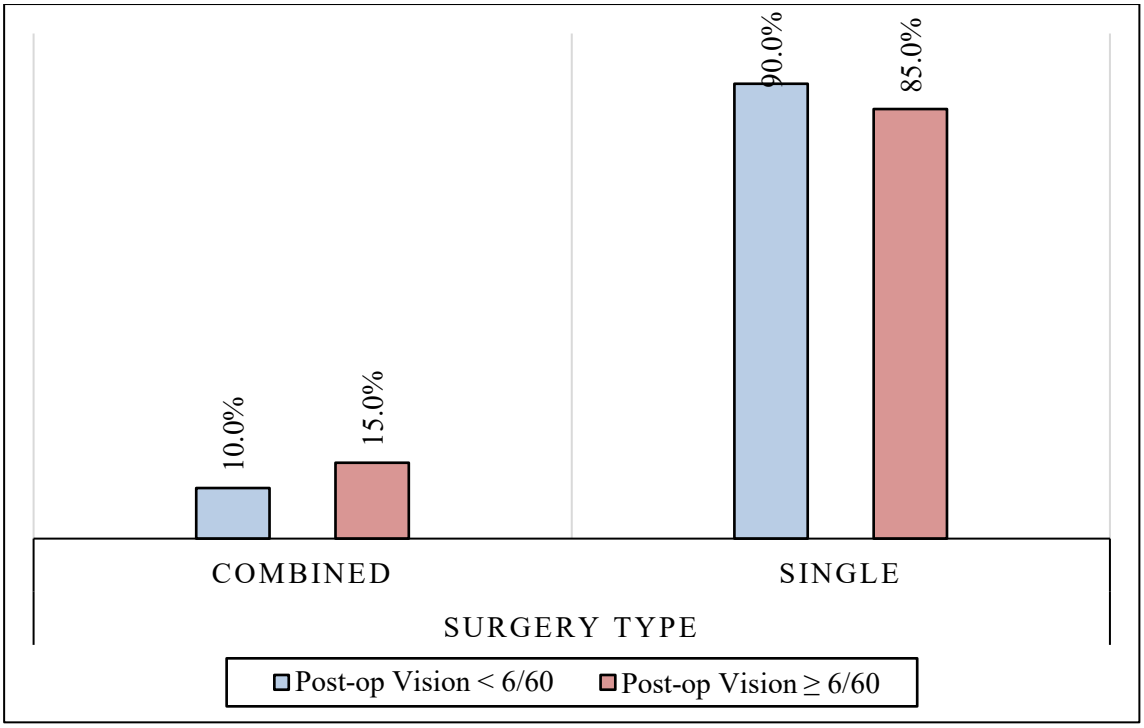

TABLE 14 Figure 14e: Distribution of eyes by type of surgery and post-operative vision.

In this study it was found that postoperative vision <6/60 in 10% of the subjects,>6/60 in15% of the subjects with combined surgery type, while postoperative vision <6/60 in 90% of the subjects, >6/60 in 85% of the subjects with single surgery type.

Twenty six (86.6%) eyes were phakic and four were pseuedophakic at the beginning of our study. Six eyes underwent combined phaco and PPV surgery and 24 eyes underwent PPV procedure only.

Risk factors for visual outcome of <6/60:

We studied the following variables to see if they had any predictive value- age of the patient, initial visual acuity, indication of surgery, involvement of quadrants by TRD, combined phaco and PPV surgery versus only PPV, preoperative bevacizumab injection, development of intraoperative retinal tears. The results of this analysis are summarized in the above tables. Age of the patient, indication of surgery, combined or non-combined surgery were not found to be significant in predicting poor visual outcome. However initial VA of <6/60, non-receival of preoperative injection of bevacizumab and development of retinal tears were significant factors in predicting poor final VA of <6/60.

There were only two eyes with retinal detachment at the end of follow up. Because of the small number of cases statistical analysis could not be done to find predictive factors for poor anatomical outcomes**.**

DISCUSSION

Proliferative diabetic retinopathy is a sight threatening disease and the vision is affected by vitreous hemorrhage, TRD involving the macula, combined TRD and rhegmatogenous RD and other conditions mentioned earlier. Some of these conditions if left untreated may lead to neovascular glaucoma, atrophy of the macula and or ghost cell glaucoma and destroy the vision permanently. Pars plana vitrectomy is the only method to treat non resolving vitreous hemorrhage, TRD involving or threatening macula, combined TRD and Rh RD and other conditions mentioned earlier. Ever since Dr. Robert Machemer performed vitrectomy for a non-resolving case of vitreous hemorrhage due to PDR, the surgery, instrumentation and viewing systems have evolved over the last five decades to make the surgery less invasive, safer, faster and more rewarding for the patient. Some of the advances in the field of vitrectomy include high speed cutters(10000 cuts per minute as against 400 in the initial vitrectomy cutters), smaller g instruments and vitrectors of 23 and 25 g which enable suture-less transconjunctival surgery, modified vitrector tips with port of aspiration and cutting very close to the tip, trocar system to introduce vitrector and other instruments in the eye without disturbing the vitreous base, better wide field viewing systems like BIOM, facility of endolaser, more sophisticated vitrectomy machines with controlled fluidics that allow stable maintenance of intraocular pressure, availability of various tamponade options, preoperative injection of anti VEGF and better auxiliary instruments.[88]

Because of the advances in the field of vitrectomy more and more patients are being treated by PPV for the complications of PDR and the results of surgery have improved. Also, more and more complex cases of TRD, which were deemed inoperable in the past are being operated with relatively better results than before.

With this background we have analyzed results of 30 eyes of 23 patients who underwent PPV for various complications of PDR in our hospital.

In this study, the demographic characteristics are as follows:

1. **Age distribution.**

The average age of the patients in this study was 56.43+7.5 years, ranging from 45-70yrs. Previously conducted studies yielded similar results. According to a study conducted by B Gupta et al, the average age of patients with PDR was 54.08+14.15 yrs.[84] Shroff et al. reported the mean age of patients with PDR to be 51.7+9.5yrs in another study.[89]

2.SEX

Ours was a small study with preponderance of male patients (78.26%). The high rate of males in our study could be due to small sample size or it could be due to males seeking medical care more than females as it happens in less developed economies. Other studies have recorded relatively lower proportions of male patients undergoing PPV for PDR. In a previous study conducted by shroff et al males formed 66% of the cohort whereas in a study by Gupta et al the males formed 54.1% of the study population. Ozawa et al have shown that the diabetic retinopathy is more severe in males than females at least the time of diagnosis of type 2 DM.[90]

2. **DURATION OF DIABETES**

In line with global epidemiological statistics on DR, the duration of diabetes remains the most significant risk factor for the development of diabetic retinopathy. In this study it was found that 56.52% of the patients had type 2 diabetes for more than

15 years, mean duration of diabetes was 14.74±7.85. The mean HbA1C in 23 subjects as seen preoperatively was 9.09±1.52(range;7.1 to 13.2) indicating poor control of DM.

In a similar study, GUPTA et al found that the average duration of diabetes was 23.12 +8.82 years. Ferreira et al. found that the average duration of diabetes was 18 years in another study.[91]

ASSOCIATED RISK FACTORS

In our study all the subjects had type 2 diabetes mellitus and significant number of subjects had associated comorbidities. Hypertension was seen in 52.17%, ischemic heart disease in 17.39% and need for anticoagulant treatment in 35% of the subjects. None of the patients had chronic kidney disease in our study.

Majority of our subjects were on oral hypoglycemic agents (78.26%).

Rukmangathan et al reported hypertension in 34%, IHD in 2% and chronic kidney disease in 4% of their subjects. Similar to our study majority (91%) were on oral hypoglycemic agents.[92]

Similar findings have been reported by Gupta et al in which 110(69.6%) had hypertension, 96(60.7%) were on treatment for dyslipidaemias, 13(8.2%) were on dialysis for CKD and 42(26.5%) were having IHD in their case series of 158 patients.[84]

Indications for PPV

Non resolving vitreous hemorrhage is one the most common indications for PPV in PDR cases.

In our study 50% eyes the indication for surgery was non resolving vitreous hemorrhage only. In 26.67% of the eyes there was both vitreous hemorrhage and TRD and in 20% of the eyes the indication was purely for TRD involving the macula. One case had combined TRD with Rh RD.

Compared to our study Thompson et al had vitreous hemorrhage as indication of surgery in 35%, TRD in 36% and combined TRD with Rh RD in 17%. [(83)]

As per research conducted by yorson et al 43% of their patients had non-clearing vitreous haemorrhage,32.8% had TRD affecting the macula,11.5% had combined tractional and rhegmatogenous retinal detachment. The presence of TRD in our series was nearly 46.6% if we consider presence of TRD in some cases of non-resolving vitreous hemorrhage.[(93)]

The surgical and visual results are less satisfactory in cases of PPV for TRD. Yang et al reported that anatomical success was achieved in all the eyes with vitreous hemorrhage where as 24% of eyes with TRD ultimately developed retinal detachment with poor visual results.[(94)]

Smiddy et al also reported less satisfactory visual results in patients undergoing PPV for TRD compared to eyes undergoing PPV for vitreous hemorrhage.[(82)]

The anatomical success rate in our series was 28 out of 30 eyes had flat retina at the end of follow up of three months. Two patients (6.6%) needed resurgery because of post operative retinal detachment. In one case it was due to a peripheral retinal tear that was missed, and, in another case, it was due to incomplete removal of membranes

that led to reproliferation and redetachment. The rate of retinal detachment after PPV for PDR varies in the literature.

Schreur et al reported resurgery in 16% by end of one year and it rose to 27% over a follow up of 10 years in cases that underwent PPV for complications of PDR.[95]

Smiddy et al also reported revitrectomy in 19% of the eyes in their study.

Jackson et al in a large study of 939 eyes reported resurgery in 13.5% eyes after a median of 2.8 months for various causes.

Our rate of resurgery of 6.6% is low because of short duration of follow up and low number of cases.

Gupta et al reported revitrectomy rate of 30.7% in eyes undergoing PPV for TRD and 8.3% of eyes undergoing surgery for non-clearing vitreous hemorrhage. In this series 11.35% of the eyes had detached retina or developed phthisis at the end of 12 months thus highlighting the risks involved in such complex surgeries.[84]

However, recently lower incidence of resurgery has been reported with the use of bimanual surgery. Shroff et al reported resurgery rate of only 7.6% of 315 eyes over a mean follow up period of 23 months.[89]

Takayama et al reported resurgery rate of 26.3% of452 eyes undergoing surgery for PDR.[86]

Other complications:

Retinal tears while dissecting the membranes is a very common complication during surgery for TRD or PDR. The causes of retinal tear in our experience during surgery are very adherent membranes to the retina, which is already thin due to traction, bleeding during surgery thus precluding the view of retina, attempt to remove the posterior hyaloid in relatively simple cases can sometimes result in formation of tears due to small patches of very adherent fibrovascular membranes. In our series we encountered retinal tears in ten out of 30 eyes (33%). They occurred in both TRD and VH cases. Retinal tears are a common complication during PPV for PDR and they have been reported by many researchers. Shroff et al reported retinal tears in nearly 52% of the eyes where they were using bimanual surgery for complicated cases of TRD in 315 cases.[89]

Jackson et al reported retinal tears in 19.4% of the eyes operated for advanced PDR.[85]

Choovuthayakorn et al reported retinal tears in 7.5% of the eyes operated.[96] Retinal tears may be successfully treated by laser photocoagulation and adequate tamponade if the posterior hyaloid is fully removed along with all the membranes. However, in some difficult cases it may not be possible to remove all membranes and under such circumstances the retinal tears may lead to development of rhegmatogneous retinal detachment.

VISUAL OUTCOME

In our study there was significant improvement in visual acuity with preoperative log mar acuity of 1.72±0.52 improving to 1.02±0.67 ($p<0.0001$) by paired T-test. In a study conducted by Shroff et al the mean log MAR vision improved from 1.67±0.63 preoperatively to 0.95±0.66 at 3 months. This was in a series of advanced TRD where bimanual surgery was used for all the cases. Raman et al also reported improvement of visual acuity from preoperative log mar units of 1.73 to post operative 0.82.[97]

There were ten (33%) eyes with final VA of <6/60 in our study compared to 22 (73.33%) eyes preoperatively. Our cases included TRD in 46% of eyes. Raman et al reported similar results of 33.2% of eyes having final VA<6/60. Jackson et al reported <6/60 final visual acuity in 25% of the cases in a multicenter study. However, in this study a significant number of patients had no recorded postoperative vision or had not followed up for more than 3 months. Smiddy et al reported final VA of 6/60 or less in 17% of their patients. Sokol et al reported VA of >6/60 in 29% of the eyes operated for PDR.[98] The variation in such gains in visual acuity are due to selection of cases, rate of complications, period of follow up and number of cases**.**

Post operative visual acuity improved from preoperative vision in 26 (86.66%) out of 30 eyes in our study and remained same in 2(6.66%) and worsened in 2(6.66%) eyes. Mason 3rd et al reported improvement in VA in 73% and worsening visual acuity in 27% post operatively.[99]

Raman et al reported improved visual acuity in 91.5% of the eyes they operated upon. Shroff et al reported improved visual acuity in nearly 94% and worsening of VA in 5.6% of the eyes operated for PDR.

Final visual acuity outcome of 6/12 or better is less common after vitrectomy for PDR especially if it involves cases of TRD. This is unlike surgery for cataracts where final vision of 6/12 is usually taken for granted. In our series none of the 30 eyes had initial vision of 6/12 or better but five (16.66%) eyes had final vision of 6/12 or better. Many studies report similarly low proportion of eyes achieving final VA of 6/12 or better. Gupta et al reported final VA of 6/12 or better in 29% in cases of TRD, 65% in cases of non-clearing vitreous hemorrhage and 100% of other cases. Raman et al also reported relatively higher proportion of final VA of 6/12 or better in 30% of the cases. At the end of three months Shroff et al reported final VA of 6/12 in 17.3% eyes. Jackson et al reported 6/12 or better final visual acuity in 43-45% of eyes depending upon delamination was done or not. But as mentioned earlier the visual data was missing from more than half of the cases. Smiddy et al reported final VA of 6/12 or better in 22% of their series of 213 eyes. Choovuthayakorn et al reported final VA of 6/12 or better in 12.1% to 36.1% depending upon indication for surgery.

Recent improvement of visual acuity after PPV are probably due to advancements in instrumentation and surgical technique, such as: (1) avoiding the removal of a clear crystalline lens; (2) effective dissection and removal of most of the fibrovascular tissue using bimanual dissection in few cases. (3) improved haemostasis methods; and (4) enhanced experience detecting and treating retinal breaks.

RISK FACTORS FOR POOR FINAL VISUAL ACUITY OF <6/60

It is interesting to see what factors contribute to poor visual outcome after PPV for advanced PDR in our series. This will help clinicians to counsel patients prior to surgery and prepare them for acceptance of probable poor visual outcomes if any of any significant factors are present in these affected eyes.

Our study observed 10 eyes (33%) had final visual acuity of <6/60. Several factors were found to be significant for poor visual outcomes. They were **1) initial visual acuity of <6/60, 2) preoperative bevacizumab intravitreal injection and 3) occurrence of intraoperative retinal tears.**

Other factors like age, indication of surgery, combined surgery or only PPV, quadrants of TRD were not found to be significant for poor visual outcomes.

Initial visual acuity is an important predictor of visual outcomes after PPV for PDR as it may indicate the damage sustained by macula due to the effects of long-standing diabetic retinopathy prior to surgery. In such cases it is unlikely that the final visual acuity will improve substantially.

In our study poor initial visual acuity (VA<6/60) was significant risk factor for final poor visual acuity. Raman et al, Choovuthayakorn et al and Yorson et al also have observed that poor initial visual acuity to be a predicting factor for poor final visual acuity. Mason et al also found that initial visual acuity was a predictor of poor visual outcome of only perception of light or no perception of light.[99]

Preoperative intravitreal injection of bevacizumab is supposed to reduce vascularity hence reduce bleeding during surgery and post-surgery. Some reports suggest better visual results when intravitreal bevacizumab is used preoperatively.

Pokroy et al and Oshima et al also found the intravitreal injection of bevacizumab preoperatively reduced the surgical time of PPV.[100] [101]

Shroff et al and Choovuthayakorn et al found lower incidence of post operative vitreous hemorrhage among patients who received preoperative bevacizumab compared to those who did not receive it.[89] [96] However, in both the above studies the preoperative bevacizumab injection had no bearing on the final visual results. Choovuthayakorn et al also observed that the intravitreal injection of bevacizumab also reduced the chances of formation of retinal tears during surgery.[96]

Gupta et al in contrast, did not find preoperative intravitreal injection of bevacizumab to reduce the incidence of post operative vitreous hemorrhage but it did reduce the occurrence of macular edema.[84] Rukmangathan et al also did not find any benefits on visual outcomes by giving preoperative intravitreal bevacizumab injection.[92] A meta-analysis concluded that preoperative intravitreal injection of bevacizumab reduces the surgical time, intraoperative bleeding, post operative vitreous hemorrhage and yields better visual outcomes.[102]

In contrast to many studies our study found preoperative intravitreal bevacizumab injection to be a significant risk factor for poor visual outcome($p=0.001699$). The reasons for this contrast could be small number of cases and selection of cases. We used intravitreal injection of bevacizumab in very advanced cases with extensive neovascularization which have usually poor prognosis. Because of this selection of cases the visual results might have been poor in our series.

As mentioned above intraoperative retinal tears can occur in up to 52% of cases of TRD.[89] In our series they occurred in 30% of the eyes and were found to be a significant factor for poor visual outcome(p=0.02849). Intraoperative retinal tears lead to increased incidence of post operative retinal detachment as observed by Gupta et al and affect the visual recovery.[84]

Choovuthayakorn et al also observed that intra op retinal tears to be a significant factor for poor visual recovery. Intraoperative retinal tears usually indicate presence of TRD with strong attachment of membranes to retina. So, while delaminating the membranes retinal tears may occur especially if the TRD is chronic. Hence the visual results are affected by the chronicity and complexity of TRD more than the occurrence of retinal tears.

In our study **age of the patient, indication of surgery, TRD preop quadrants, combined or single surgery** were not found to be a significant risk factors for poor visual outcome. Similarly, Raman et al also did not find age or gender of the patient to be significant risk factors for poor visual outcome.[97] In another study conducted by shroff et al found that postoperative VA was significantly better in < 40 yrs. age group. Schreur et al also found higher age to be poor prognostic factor for visual recovery in a long term study.[95]

In our study combined phacoemulsification and PPV or only PPV surgery had similar visual results. Raman et al and Shroff et al also observed similar findings in their studies.

Indication of surgery for PPV was not found to be significant risk factor for final visual outcome in our study. Mason et al, Jackson et al and Choovuthayakorn et

al also observed that TRD was not a significant risk factor for poor visual outcomes. However, Thompson et al observed that the patients undergoing PPV for vitreous hemorrhage only had better visual outcomes than those undergoing PPV for TRD.[83] Smiddy et al also reported that the rate of visual deterioration are less if the indication for surgery is vitreous hemorrhage compared to TRD.[82]

The extent of TRD should be a powerful indicator of visual outcomes because more extensive the TRD more complex the surgery will be, and chances of complications increase. Also, more extensive TRD indicate a long standing TRD with worse visual outcomes. We studied cases with TRD ≤2 quadrants and TRD> 2 quadrants regarding final visual outcome. However, we did not find it to be predictor of poor visual outcome. Shroff et al however found that when TRD involved four quadrants the visual results were worse.[89]

Shortcomings of the study.

The study is a small study involving only 30 eyes of PDR. The COVID epidemic affected recruitment of more cases to some extent. Also, the follow up period had to be shorter due to time limitation of thesis requirements. As the 30 eyes belonged to 23 cases the role of gender, duration of DM, control of DM and other factors that affect both eyes could not be studied for their significance in predicting final visual acuity.

CONCLUSION

Majority of our 30 patients had improved visual acuity after PPV for complications of PDR. However significant number still had poor visual outcome of final VA<6/60 mainly due to advanced PDR in many cases. Initial VA of<6/60, intraoperative retinal tears and preoperative injection of bevacizumab were risk factors for poor visual outcome of final VA of <6/60. Two cases (6.66%) needed repeat surgery in our series. There were no cases of NVG or endophthalmitis in our series.

SUMMARY

The study entitled ‘a one-year prospective study of visual and anatomical outcome of PPV for PDR was conducted at KLE Eye hospital Belgaum in the vitreoretinal clinic, in the months of January 2020 till December 2020. Eyes included in the study were those with non-clearing vitreous hemorrhage (50%), TRD with vitreous hemorrhage (26.67%), TRD (20%) and TRD with combined rhegmatogenous RD (3.3%) which were all consequences of PDR. The study included 30 eyes from 23 individuals (18 males, 5 females). The patients average age was 56.43+7.5 years, arranging from 45 to 74 years. All the 23 subjects were suffering from type II DM. The duration of DM ranged from 4 to 30 years with mean of 14.74±7.85years. In this study, it was discovered that 56.52 percent of the patients had type 2 diabetes for more than 15 years. The mean HbA1C in 23 subjects as seen preoperatively was 9.09±1.52 (range; 7.1 to 13.2) indicating poor control of DM**.** Many patients had comorbidities like hypertension in 12(52.17%), IHD in 4(17.39%) and h/o taking anticoagulants in 8(34.78%), and 78.26 percent of the patients were treated for diabetes with oral hypoglycemic agents, and 5 were receiving insulin injection and oral hypoglycemic agents. Nine eyes had received preoperative intravitreal injection of bevacizumab for presence of florid neovascularization.

All the patients underwent either 23 or 25 G PPV. We reported indications of surgery, preoperative and post operative visual acuity, complications of surgery and factors predicting poor visual outcome.

Pars plana vitrectomy with membrane peeling, internal limiting membrane peeling as well as Fluid air exchange + endolaser and gas injection, was the most common surgical method done in this study. Intraoperatively 10 eyes developed

retinal breaks, and 12 eyes developed bleeding out of 30 eyes. The preoperative log mar vision was 1.72±0.52, while the postoperative log mar vision was 1.02±0.67 the improvement was statistically significant (P=0.0001) showing good results in our study.

Majority of the patients had vitreous hemorrhage (26.67%) as postoperative complication which resolved on its own in all cases. At three months, 28 eyes (93.33%) had a flat retina, ten eyes (33.33%) had residual membranes, vitreous hemorrhage was seen in 3 eyes (10%) of the patients. Resurgery has been done in 2 eyes (6.67%) patients. None of the patients developed NVG or endophthalmitis. Final VA was >6/60 in 66.67% of the eyes operated. Five eyes achieved final VA of 6/12.

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ANNEXURE –I - ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to-be-University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
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Ref: MDC/DOME/ 254 **Date: 24/12/2019**

To,

Dr.
PG student in Ophthalmology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A ONE YEAR PROSPECTIVE STUDY OF VISUAL AND ANATOMICAL OUTCOME OF PARS PLANA VITRECTOMY FOR PROLIFERATIVE DIABETIC RETINOPATHY "**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE II: CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

STUDY ID NO: _____

TITLE OF THE STUDY: "A ONE YEAR PROSPECTIVE STUDY OF VISUAL AND ANATOMICAL OUTCOME OF PARS PLANA VITRECTOMY FOR PROLIFERATIVE DIABETIC RETINOPATHY ADMITTED AT KLES DR. PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI".

Principal Investigator:
Dr.
Post Graduate student,
Department of Ophthalmology,
Jawaharlal Nehru medical college,
K.L.E University, Belagavi-590010

Guide:
DR.
Professor,
Department of Ophthalmology,
Jawaharlal Nehru medical college,
K.L.E University, Belagavi-590010

CO GUIDE:
DR.
Assistant professor,
Department of ophthalmology,
Jawaharlal Nehru medical college
Vitreoretina Consultant,
KLES Dr Prabhakar Kore Hospital
K.L.E University, Belagavi-590010

Introduction and Purpose: The purpose of this study is to assess the visual and anatomical outcomes of Pars plana vitrectomy for Proliferative diabetic retinopathy patients. As complications of proliferative diabetic retinopathy are one of the most common indications for pars plana vitrectomy there is a need in our institute to study the results of procedure and surgical outcomes. Such a study has not been conducted so far in our institute and hence we aim to conduct the study and report the results.

Procedure: If I agree to be a part of this research study, then I will be asked the relevant history and will be subjected to relevant clinical examination and investigations.

Risks and Benefits

BENEFITS: Results will help to study the visual and anatomical outcomes and to assess the risk factors associated with poor visual outcome.

RISKS: were explained and consent taken.

ALTERNATIVES: If patient is not willing to take part in the study, his/her treatment or any other further investigations the patient wants to undergo, in future, in KLE will not be affected by his/her decision.

VOLUNTARY PARTICIPATION/WITHDRAWAL: Taking part in this study is voluntary. I may choose not to take part in this study, or if I decide to take part, you can later change my mind and withdraw from the study. My decision will not change the present or future healthcare or other services that i receive. The study doctor or the sponsor may stop my participation in this study. I will tell of any important new findings that may change my willingness to continue to take part. If I choose not to

take part in the study, I will receive the standard treatment for patients with my condition.

COSTS: As per norms of the hospital

COMPENSATION: In the event that I become injured as a result of taking part in this study, treatment will be offered to me. No reimbursement, compensation or free medical care is given.

Confidentiality: All information collected about me during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify me in this research record. Information from this study may be published but my identity will be confidential in any publication.

Authorization to publish the results: The results of the study would be forwarded to KLE University, Belgaum as part of requirement towards the compensation of my M.S degree, review and publishing.

CONSENT TO PARTICIPATE IN RESEARCH TRIAL

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form, or it has been read to me, and has been explained to me in my vernacular language and had all my questions answered. I will be given a copy of this consent form. Signature/Left Thumb print of the participant or legally authorized representative.

Participant's Name: _____

Name of legally authorized representative/guardian: _____

Signature / Left Thumb impression of participant or

legally authorized representative: _____

Witness name: _____

Signature or Left Thumb impression: _____

Investigator's name: **Dr.**

Date: **Place:**

Signature: _____

Guide: Dr.

Signature: _____

Co- Guide:Dr.

Signature of co-Guide: _____

Information from this study may be published but my identity will be confidential in any publication.

In case of any queries during the study or in the future you may contact following person.

QUESTION:

If any enquiries in the future or in case of research related injury illness, you may contact following person.

1) PRINCIPAL INVESTIGATOR: Dr. _____, Post Graduate student, Department of Ophthalmology, J N Medical College, Belagavi.

2) GUIDE: Dr. _____ M.B.B.S, M.S., Professor, Department of Ophthalmology, J N Medical College, KLE Academy of Higher Education and Research, Belagavi.

3) CO-GUIDE: DR._____M.B.B.S., M.S., FRCSED, Assistant Professor, Department of Ophthalmology, J N Medical College, KLE Academy of Higher Education and Research, Belagavi. Consultant Vitreo-Retina Surgeon, Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Center, Belagavi

ANNEXURE - III - PROFORMA

PATIENT INFORMATION:

IP NUMER: OP NUMBER:

NAME: _____

AGE: _____ GENDER: F/M

PHONE NUMBER: _____

ADDRESS: _____

DATE OF ADMISSION: _____ DATE OF DISCHARGE: _____

Is patient eligible for study?

Has informed consent been given?

MEDICAL HISTORY: 1) DIABETES MELLITUS TYPE2/ ☐ TYPE1 ☐

DURATION ☐ MONTHS/ YEARS ☐

TREATMENT_____

2) HYPERTENSIVE YES 1 / ☐ NO 2 ☐

DURATION ☐ MONTHS/ YEARS ☐

TREATMENT_____

3) DYSLIPIDEMIA/LIPID PROFILE YES 1 / ☐ NO 2 ☐

TREATMENT_____

4)ISCHEMIC HEART DISEASE. YES 1 / ☐ NO 2 ☐

STENT / CABG

MEDICAL TREATMENT _____

5) CHRONIC KIDNEY DISEASE YES 1 / NO 2 ☐

ON DAIALYSIS ☐

MEDICATION_____

6)HISTORY OF TAKING ANTICOAGULANTS YES 1/ NO 2 ☐

MEDICATION _____

7) ANY OTHER ILLNESS

8) HISTORY OF AMPUTATION. WHICH LIMB? ————

WHEN

OPHTHALMIC HISTORY:

1) PRESENTING COMPLAINTS: A) WHICH EYE LEFT EYE / RIGHT EYE

DIMINUTION OF VISION

B) DURATION DAYS / MONTHS / YEARS. ————

C) ONSET GRADUAL / SUDDEN IN ONSET.————

D) WORSENING OR IMPROVING

E) PAIN PRESENT / ABSENT

REDNESS PRESENT / ABSENT

WATERING PRESENT / ABSENT

F) ANY TREATMENT TAKEN AFTER DIMINUTION OF VISION STARTED

1) LASER _____

2) INJECTION_____

3) USAGE OF DROPS_____

OTHER EYE VISION COMPLAINTS YES/NO

PAST OPHTHALMIC HISTORY

ANY EPISODES OF DIMINUTION OF VISION IN EITHER EYE YES 1 /NO 2 ☐

HISTORY OF LASER INJECTION OR PPV IN EITHER EYE YES 1 /NO 2 ☐

HISTORY OF INTRAVITREAL INJECTION IN EITHER EYE (if so when)YES1/NO2 ☐

_____

HISTORY OF CATARACT SURGERY IN EITHER EYE IF SO WHEN YES 1 /NO 2 ☐

HISTORY OF GLASSES YES 1/NO 2 ☐

HOSTORY OF TRAUMA YES 1/NO 2 ☐

FAMILY HISTORY:

Similar complaints:

Any other significant family history:

PERSONAL HISTORY:

Smoking: Yes/No

Duration _____ days/months/years

Alcoholism: Yes/No

Duration _____ days/months/years

Other addiction: Yes/No

Duration days/months/years

OCCUPATION: Education: Illiterate

Below 10 standards

Above 10 below degree

Above degree

GENERAL PHYSICAL EXAMINATION:

Appearance: well-built/moderately built/poorly built/emaciated

Pallor: Present/Absent

Pulse: _____ beats/minute BP: _____mm HG

FBS ☐ HBA1C ☐

AMPUTATION

SYSTEMIC EXAMINATION:

CVS: Normal/Abnormal

Specify: _____

RS: Normal/Abnormal

Specify: _____

CNS: Normal/Abnormal

Specify: _____

GIT: Normal/Abnormal

Specify: _____

OCULAR EXAMINATION:

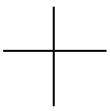
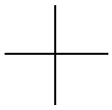
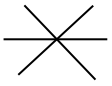
Head posture: Erect/Tilted

Visual axis: Parallel/Deviated

Facial symmetry: Symmetrical/Asymmetrical

Extra-ocular movements: Normal/Restricted/Partially restricted

RE LE Binocular

VISUAL ACUITY:

	RE	LE
DISTANT		
PINHOLE		
NEAR		
WITH GLASSES		

ANTERIOR SEGMENT:

	OD	OS
LIDS CONJUNCTIVA CORNEA ANTERIOR CHAMBER IRIS-SPECIFICALLY LOOK FOR NVI ANGLE EXAMINATION AND CHECK FOR NVA (SOS) LENS STATUS-NORMAL/CATARACT/ PCIOL/ACIOL ANTERIOR VITREOUS ANY CELLS/HAEME IOP-APPLANATION(PREOP)		

FUNDUS EXAMINATION	OD	OS
GLOW		
MEDIA CATARACT PRESENT/NOT VITREOUS HAEMORRHAGE		
DISC NVD COVERED BY MEMBRANE NOT SEEN		
C:D RATIO		
BLOOD VESSELS AV CHANGES BEADING SCLEROSED VESSELS		
BACKGROUND		

MEMBRANES INVOLVING 1 QUADRANT 2/3/4 TRD INVOLVING 1 QUADRANT 2/3/4 SUBHYALOID HAEMORRHAGE		
MACULA FLAT ON FUNDUS EXAMINATION TRD PRESENT/NOT TRD THREATENING MACULA		

DIAGNOSIS_____

_____

INVESTIGATIONS: FUNDUS PHOTOGRAPHY

B SCAN

HBA1C

INDICATIONS FOR SURGERY: 1) VITREOUS HAEMORRHAGE

2) VITREOUS HAEMORRHAGE+TRD

3) TRD ONLY

USB (BSCAN): IN CASE OF HAZY MEDIA

PARS PLANA VITRECTOMY SURGERY DETAILS:

1) COMBINED SURGERY OR ONLY PPV

2) ANAESTHESIA LOCAL OR GENERAL

3) STEPS PERFORMED:

4) MEMBRANE PEELING - QUADRANTS INVOLVED

A) WITH CUTTER ONLY

B) CUTTER+SCISSORS

C) BIMANUAL DISSECTION

5) PVD INDUCED

6)FLUID AIR EXCHANGE

7)ENDOLASER

8)GAS INJECTION/SILICONE OIL INJECTION

9)FINAL STATUS

10)COMPLICATIONS (intraoperative)

A) BLEEDING

B) RETINAL TEAR

C)MEMBRANES LEFT

D)LENS TOUCH, CORNEAL EPITHELIUM SCRAPED

POST OPERATIVE (COMPLICATIONS)

1. INCREASED IOP
2. VITREOUS HAEMORRHAGE
3. GAS IN ANTERIORCHAMBER
4. SILI CONE IN ANTERIORCHAMBER

FOLLOW UP – PLAN ONE MONTH POST OPERATIVELY

VISUAL ACUITY:

VISUAL ACUITY	RE	LE
DISTANT		
PINHOLE		
NEAR		
WITH GLASSES		

IOP _____

ANTERIORSEGMENT STATUS

_____

_____

_____

_____

FUNDUS

_____

_____

_____

_____

FOLLOW UP – PLAN THIRD MONTH POST - OPERATIVELY

VISUAL ACUITY	RE	LE
DISTANT		
PINHOLE		
NEAR		
WITH GLASSES		

IOP _____

ANTERIORSEGMENT STATUS

_____

_____

_____

_____

FUNDUS

1.RETINAL FLAT/NOT

2.SILICONE OIL PRESENT/NOT

3.ANY MEMBRANES

4.VITREOUS HAEMORRHAGE

5.RESURGERY

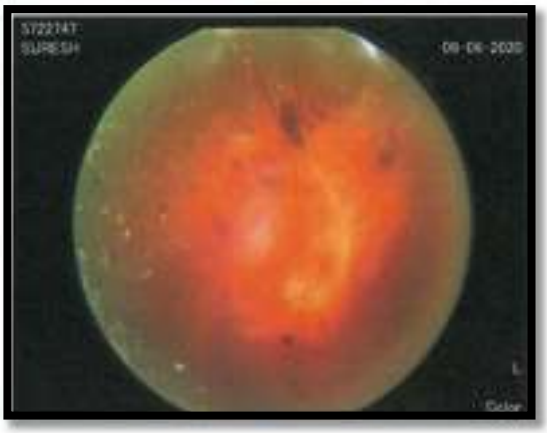
6.TRD

7.NEOVASCULAR GLAUCOMA

ANNEXURE - IV- LIST OF CLINICAL PHOTOGRAPHS:

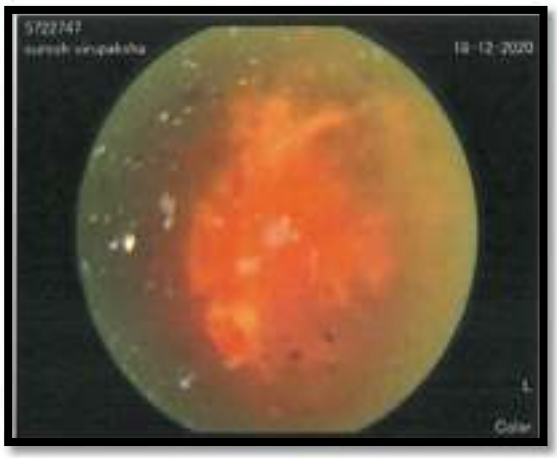
MASTER CHART No: 1

PRE-OPERATIVE



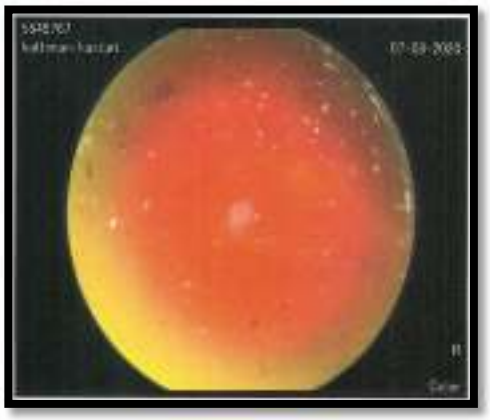
INTRA-OPERATIVE

POST-OPERATIVE



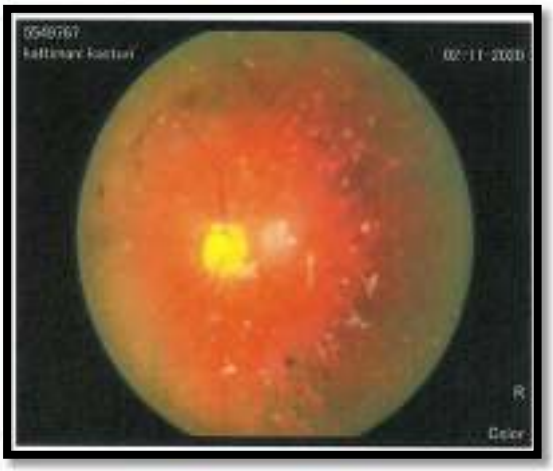
MASTER CHART No :2

PRE-OPERATIVE



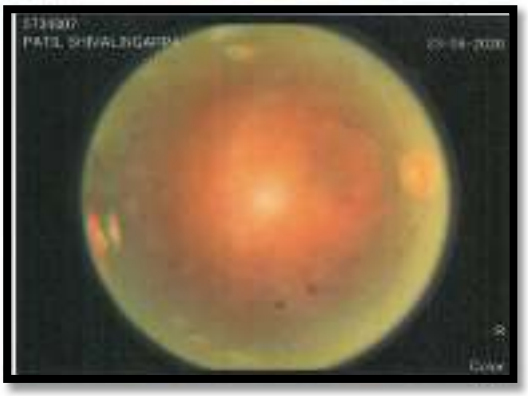
INTRA-OPERATIVE

POST-OPERATIVE

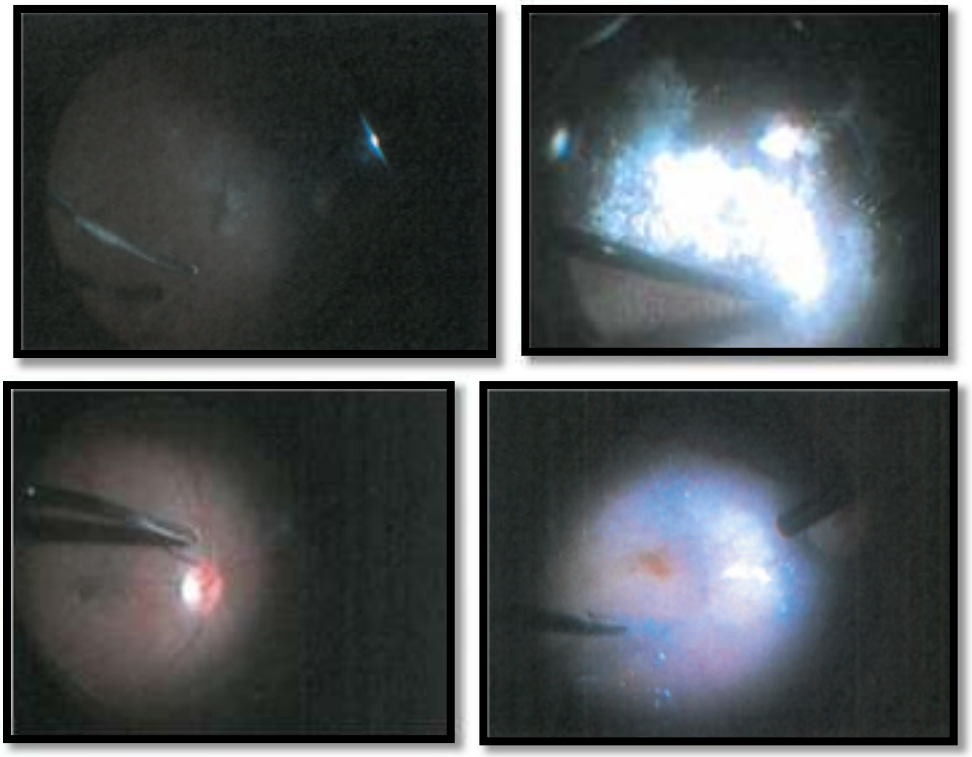


MASTER CHART No:3

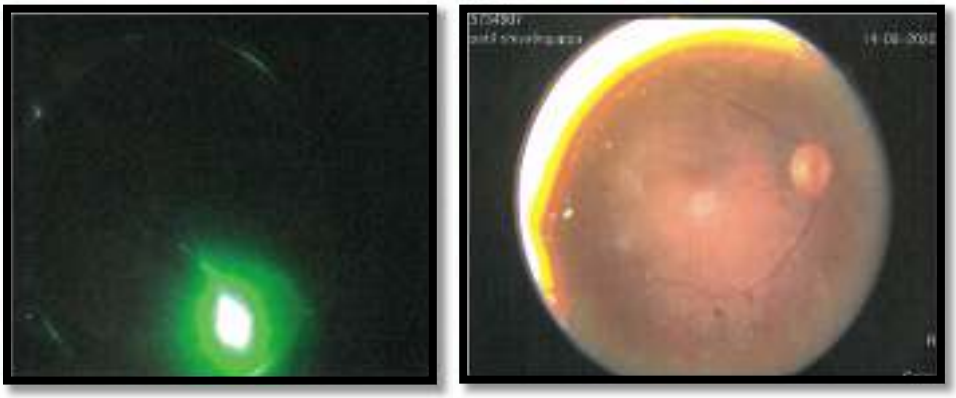
PRE-OPERATIVE



INTRA-OPERATIVE

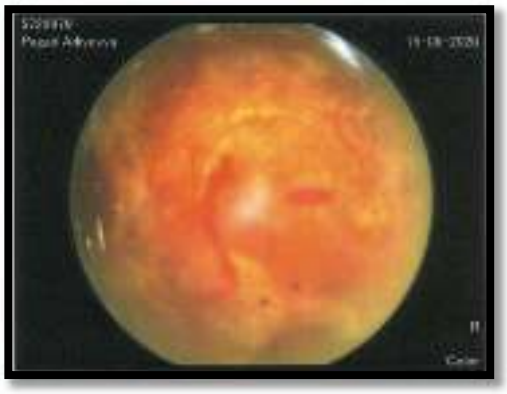


POST-OPERATIVE



MASTER CHART No:4

PRE-OPERATIVE

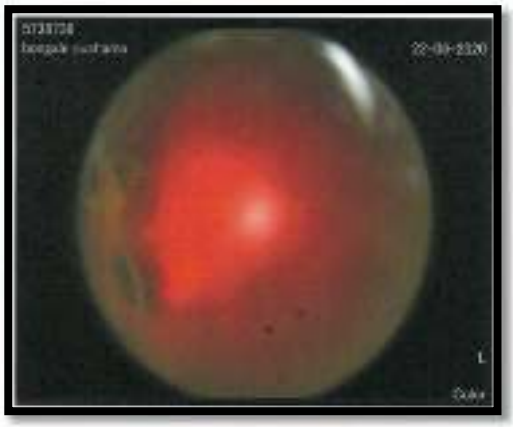


INTRA-OPERATIVE

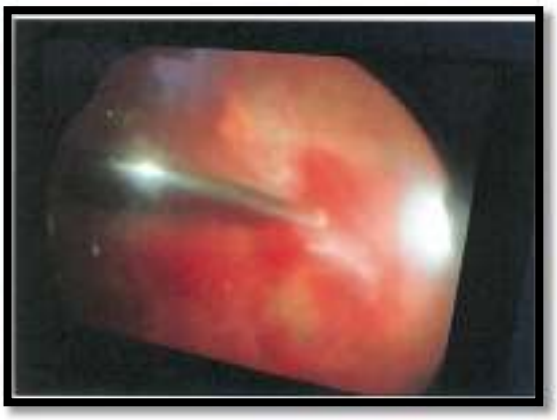
POST-OPERATIVE

MASTER CHART NO:5

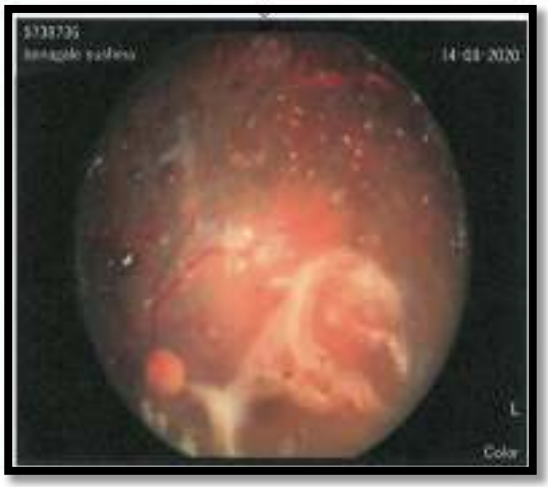
PRE-OPERATIVE



INTRA-OPERATIVE

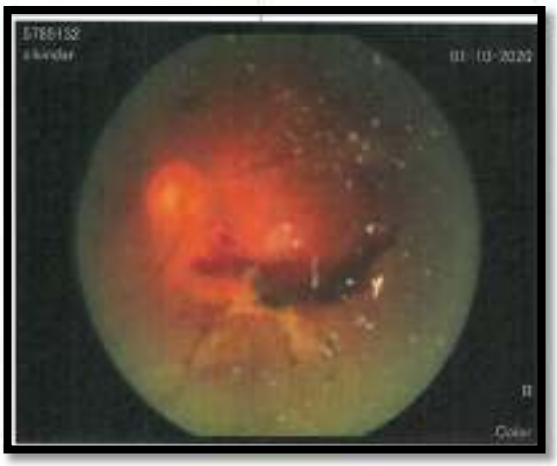


POST-OPERATIVE

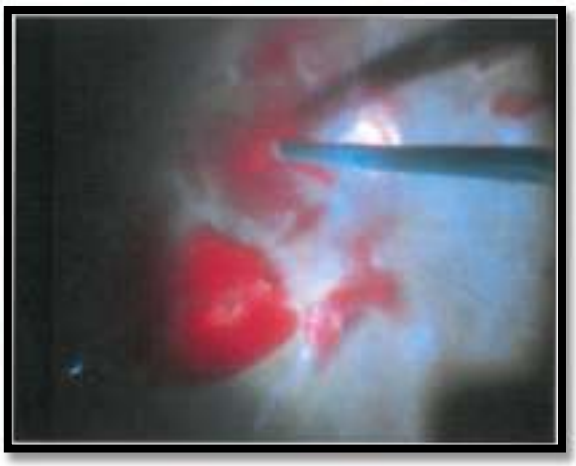


MASTER CHART No: 6

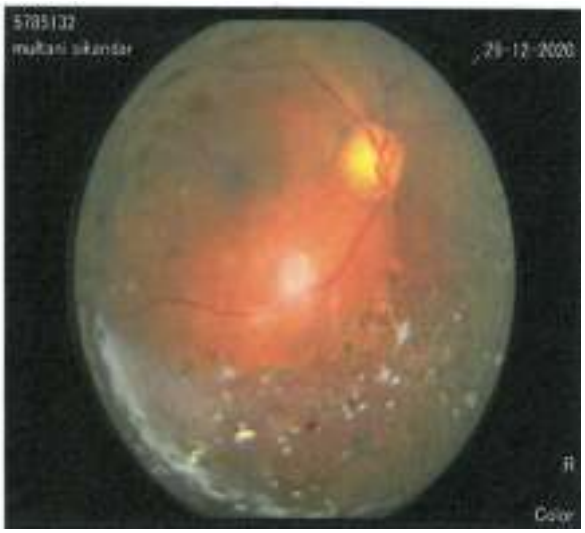
PRE-OPERATIVE



INTRA-OPERATIVE



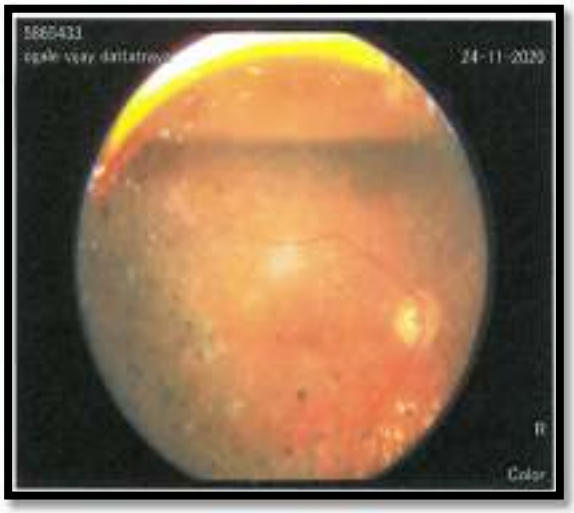
POST-OPERATIVE



MASTER CHART No :7

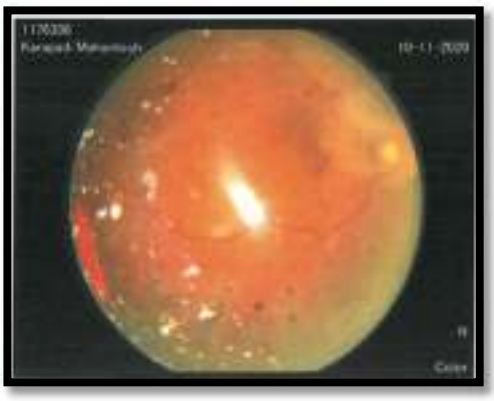
PRE-OPERATIVE **INTRA-OPERATIVE**

POST-OPERATIVE



MASTER CHART No: 8

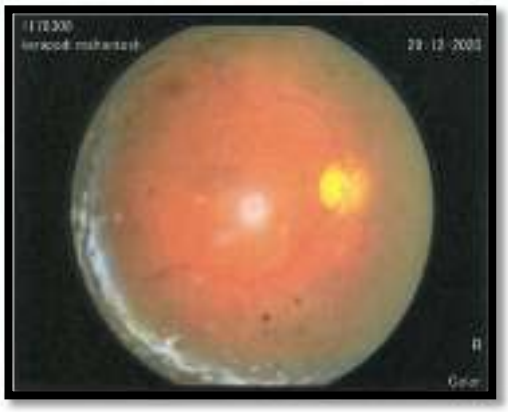
PRE-OPEARTIVE



INTRA-OPEARTIVE

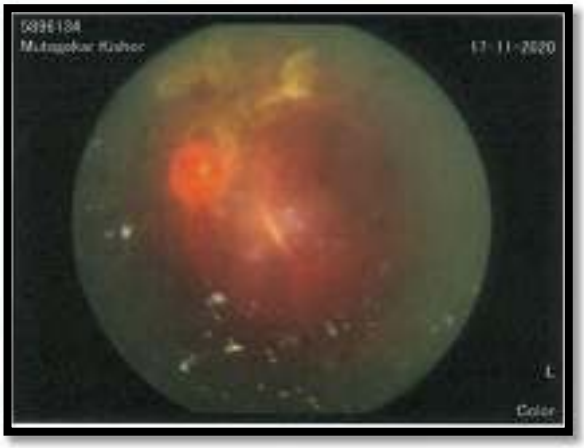
INTRA-OPERATIVE

POST-OPERATIVE



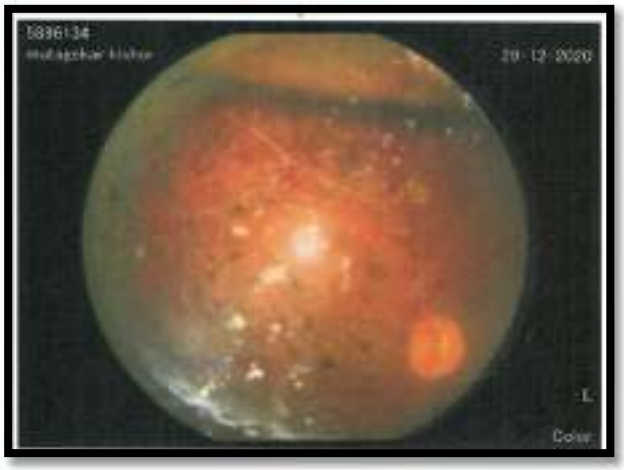
MASTER CHART No :9

PRE-OPERATIVE



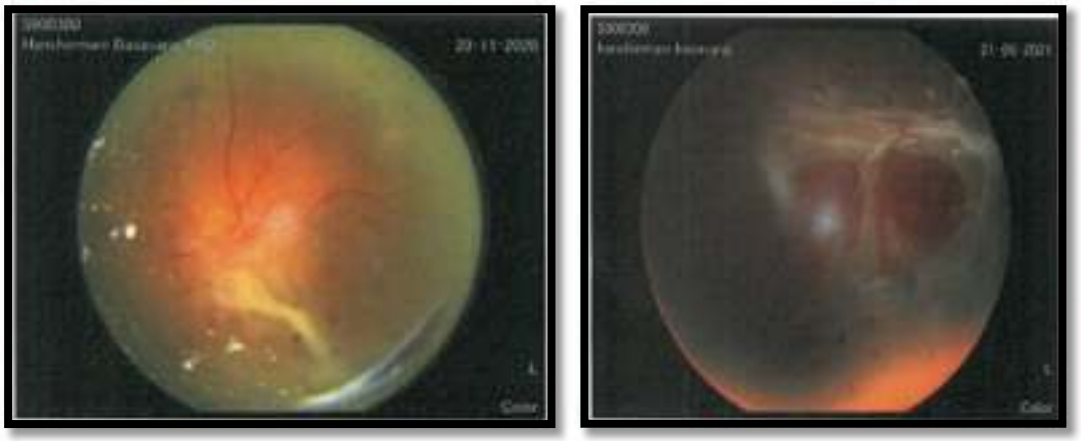
INTRA-OPERATIVE

POST-OPERATIVE



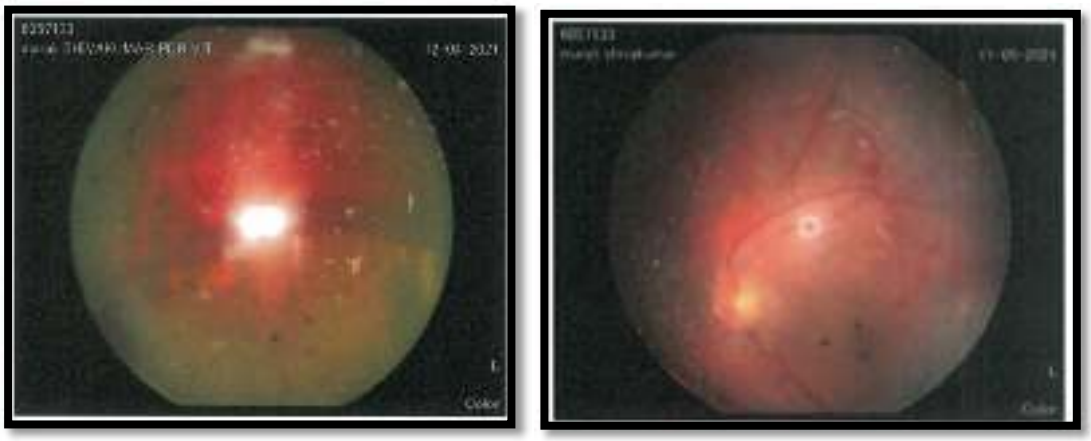
MASTER CHART No:14

PRE-OPERATIVE **POST-OPERATIVE**



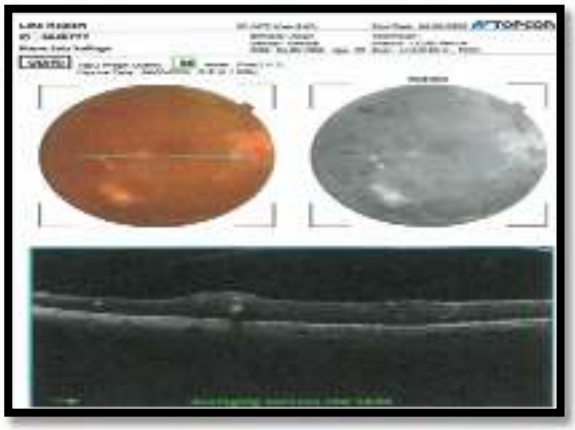
MASTER CHART NO:18

PRE-OPERATIVE **POST-OPERATIVE**



MASTER CHART NO:19

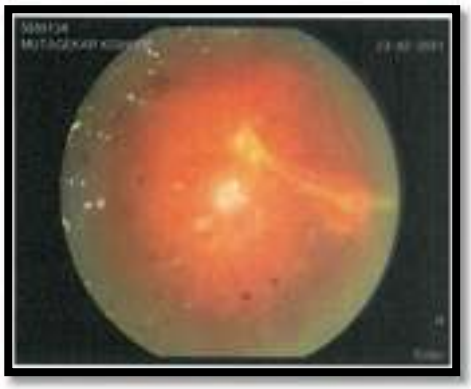
PRE-OPERATIVE



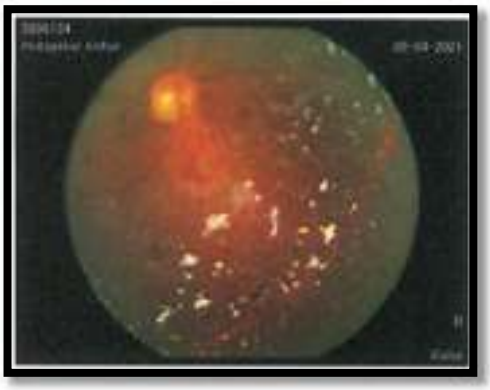
POST-OPERATIVE

MASTER CHART No :22

PRE-OPERATIVE



POST-OPERATIVE



OT set up: PPV in progress

ANNEXURE - V- MASTER CHART

S.NO	Name	Age	Sex	OP NO	IPNO	DOA	DOD	TY 2 DM	TX	HBA1C	HTN	TX	IHD	CKD	H/O ANTICOAGULANTS
1	Suresh virupaksha	55 years	Male	5722747	1014632	9.6.2020	14.6.2020	since 25 yrs	sitagliptin phosphate tab(JANUVIA 100mg1-0-0) Human actrapid 25-25-0 40iu/ml Human mixtard 0-0-25 40 iu/ml	9.50%	since 12yrs	LN BETA 2.5 1-0-0 Telmisartan40mg 0-0-1	yes,stent 2yrs ago	no	JUPIROS GOLD 20 TM 0-1-0
2	Kasturi Kattimani	45 years	Female	5549767	1021113	17.8.2020	20.8.2020	since 25 yrs	Metagid P2 tablets 1-0-1/2 glimepride 2mg+pioglitazone 15mg+metformin 500 mg)	9.20%	No	##########	No	No	No
3	Shivalingappa patil	70 years	male	5734807	1016289	18.6.2020	21.6.2020	since 20yrs	sitagliptin phosphate and metformin hydrochloride JANUMET500MG(0-0-1),GLIMEPiRIDE M1 O-O-1	7.10%	Since 20yrs	Starpress XL 50(Metoprolol succinate release tablets 0-0-1, Dilnip-T40(cilindipine and telmisartan tablets) 1-0-0	No	No	JUPIROS GOLD 10 0-0-1
4	AdivevvaPagad	57 years	female	5729978	1015059	15.6.2020	17.6.2020	since 14 yrs	Glimeperide and Metformin hydrochloridetablets POSIMET G1 FORTE 1-0-1	12.80%	Since 8 yrs	Nifedipine sustained release tab.10mg	No	N0	JUPIROS GOLD 10 0-0-1
5	sushma bonagale	60 years	female	5738736	1016282	20.6.2020	26.6.2020	since 20yrs	Gliclazide and metformin Hydrochloride extended release tablets (DIAMICRON XR MEX 500 1-0-1),INSULIN GLARGINE 100 IU/ML 20-0-20U ,NOVOPEN 6U-6U-6U	10.70%	Since 3 yrs	Telmisartan chlorthalidone and metoprolol succinate tablets (MET XL 30 1-0-1)	No	No	No
6	Sikandar multani	47 years	male	5785132	1024472	1.10.2020	4.10.2020	since 5 yrs	Galvus met 50/500 mg ,novonor 18u-0-12u	8.70%	since 2 months	Telma-H	No	No	No
7	Vijay Dattatraya ogale	62years	male	5865433	1026017	22.10.2020	22.10.2020	since 15 yrs	Glycomet GP3/850 TAB,Tab.Tenepride M 500mg		No	########	No	No	Ecosprin 75mg since 3-4yrs
8	Mahantesh s Karapudi	47 years	male	1176306	1028202	17.11.2020	20.11.2020	since 10 Yrs	Glycomet 1000 mg 1-0-1 Metformin hydrochloride sustained release tablets	10.10%	No	########	No	No	No
9	Kishore Mutagekar	50 years	male	5896134	1028420	19.11.2020	22.11.2020	since 7 years	GLIMESTAR M1 0-0-1,GLIMESTAR M2 1-0-0	7.40%	Since 6 months	Nebivolol 5 mg 1-0-0,clindipine 10mg (LN BETA 5)	No	No	No
10	Babu mutnale	58 years	male	5878560	1028712	20.11.2020	24.11.2020	Since 15yrs	PIOPLUS-2 1-0-1 Glimepride 2mg,Metformin 500mg,pioglitazone15mg	8.70%	No	No	No	No	No

S.NO	CHIEFCOMPLAINT	VISION	LASER	AVASTIN INJ	ANTERIOR SEGMENT	POSTERIOR SEGMENT	B SCAN
1	DOV in LE since 20 days	OD-HMCFPL+VEPRACC PH-NI OS-CF2M PH-NI	LE laser done6 months ago	LE inj on 10.6.20	within normal limits lens-mild cataractous changes in both eyes	OD-old vitreous haemorhage +,thick FVM along ST arcade-1 QUADRANT, Temporal retina seen partially OS- **Dense vitreous haemorhage+** , thick FVM present above the disc-1 QUADRANT subhyaloid haemorrhage temporally,nasal retina seen.	###########
2	DOV in RE siince 2 months	OD-HMCFPL+VEPRACC PH-NI OS-6/18 PH-6/12(P)	Laser done in right eye 3 times in one month	No	within normal limits lens-mild cataractous changes in both eyes	OD-fibrous bands seen running through disc along ST quadrant -1 QUADRANT, macula not seen.OS-media clear,disc fine NVD+,fibrous membrane above the disc- 1 QUADRANT,multiple microaneurysms+,periphery shows haemorrhages,cottonwool spots.	There is thick fibrovascular stalk coming with thickened posterior hyaloid with subhyaloid hage,there is TRD along the suprachoroidal membrane
3	DOV in RE since 2 years	OD- UA- CF2.5M PH-NI OS-CF3M PH-NI	Laser sine 2yrs- 5 times	H/o RE injection-2 times	within normal limits lens-mild cataractous changes in both eyes	OD-old vitreoushaemrorhage+,superotemporal vein dilated,PRPC marks+,few hage+,epiretinal membrane on macula.OS-Vitreous opacity+,venous looping+,Incomplete laser PRPC+nasally,superiorly,FVM around temporal arcade- 2 QUADRANTS, epiretinal membrane on macula.	No
4	DOV in RE since 2 years	OD-UA-CF2M PH-NI OS-6/36 PH-NI	Both eyes laser done 1 time	RE avastin inj on 16/6/2020	with in normal limits ,PSC+ in RE,mild cataract in LE.	OD- media clear,inferiorly vitreous hage+,Thick FVM on disc along the arcade and nasally- 4 quadrants with TRD.NVD+,DISC-seen hazily.Superiorly retina-flat,Laser marks+ . OS-Media-clear,vitreous haemorrhage temporally,DISC seenNVD+,Thick FVM present superior arcade and nasal to disc-2 QUADRANTS, superior retina -flat.	#########
5	DOV in LE since 4 months.	OD-UA-CF2M PH-NI OS-CF1M PH-NI	Both eyes laser done 1 time	LE avastin inj on22/6/2020 0.05 ml containing 1.25 mg in ST quadrant.	within normal limits,OD-mild lens changes+,NS+1,PSC+, OS-PSC+cortical cataract+	OD-inferiorly vitreous Hage+,Hage temporally on disc,partially detached posterior hyaloid in the posterior pole,FVM present nasal to disc- 1 QUADRANT, macula seen flat OS- **Vitreous Hage+**,FVM over the disc and outside the disc,nasally posterior hyaloid attached with thick FVM running from disc superiorly and inferiorly,inferiorly disc seen.FVM - 2 QUADRANTS superiorly and inferiorly,superotemporally-TRD+-1 QUADRANT,macula is covered by membrane and blood	###########
6	DOV in RE since 1 month 15 days	0D-UA-6/60 PH-NI OS-UA-6/36 PH-NI	H/o laser done for both eyes-4 times	No	mild lens changes+---psc+ inboth eyes,Anterior vitreous cells+in both eyes.	OD-glow-present,media-slightly hazy,DISC-NORMAL,fibrovasular membrane inferonasal to disc- 1 QUADRANT,marked AV changes,verynarrow arteries,sclerosed vessels inferiorly,**inferior vitreous Hage+,subhyaloid Hage inferiorly with FVM**,macula shows Hard exudates. OS-glow-present,Hardexudates present temporal to macula,PRPC marks present,traction inferonasal to disc,small vitreous haemorrhage inferiorly,subhyaloid Hage over macula,Inferior retina flat.	##########
7	DOV in RE since 5-6 months	OD-UA--6/60 PH-6/36 OS-6/18(p) PH-6/18	H/o laser done in both eyes	No	within normal limits PCIOL in bag, in both eyes	OD-Glow-present,media-hazy,**vitreous Hage inferiorly,NVD+**,laser marks+,fibrovasular membrane present- 4QUADRANTS -nasal to disc,inferior arcade temporal to posteriorpole with partial PVD,TRD+ below disc and temporal to macula,macula-flat,epiretinal membrane present thin fold. OS-glow-present,media-clear,inferiorly vitreous Hage+,DISC-NORMAL,PRPC MARKS+,inferotemporally and inferiorly,FVM -present inferior to disc >3 DD away- 1 QUADRANT,fovea-pigment+,superotemporal to fovea,Hage present temporal to macula.	##########
8	DOV in RE since 4-6 months.	OD-UA-CF2M PH-NI OS-6/24 PH-6/12(p)	H/o laser for both eyes done -3 times	No	Within normal limits,mild cataractous changes in both eyes	OD-glow-present,media-clear,FVM on disc,FVM on macula causing macular TRD,-2 QUADRANTS,TRD-1 QUADRANT,PRPC marks+.OS-glow-present,media-clear,inferiorly vitreous Hage+,FVM on disc-1 QUADRANT,Hard exudates and Hages in Posterior pole,Macular reflex lost	########
9	DOV in LE since 1 month	OD-UA-OD-6/36(p) PH-NI OS-CF<4m	H/o laser done in both eyes - 1time and in right eye 2 nd time	No	Withinnormal limits mild lens changes in both eyes	OD-Glow-present,vitreous Hage present,FVM covering disc,Laser marks+,TRD nasal to the disc pulling macula.-2 QUADRANTS,OS-Glow-Present,media-clear,Posterior hyaloid detachment+,FVM on disc extending superiorly,superotemporally causing TRD 2 QUADRANTS,**Subhyaloid Hage extending to periphery.**	###########
10	DOV in LE since 3-4 months.	OD-UA-OD-6/18 PH-NI OS-CF-3m PH-NI	H/o laser done for RE 1 time	No	within normal limits both eyes	OD-GLOW-present,Media-clear,DISC-NORMAL,midperiphery shows HAGES,cottonwoolspots+,PRPC marks seen,Hardexudate ring superior to fovea.OS-GLOW-present,media-not clear,**vitreous Hage+**,DISC-seen,Inferior retina not seen,FVM + superior to disc - 1 QUADRANT,subhyaloid Hage just above the disc.	#######

S.NO	DIAGNOSIS	INDICATION OF SURGERY	DOS	PPV WITH MP	ILM PEELING	FAE AND EL	SF6/C3F8	SILICONE OIL	INTRAOP COMPLICATIONS (Bleeding, Retinal tear, Membranes left)
1	LE eye PDR with vitreous haemorrhage	VITREOUS HAEMORRHAGE	13/6/2020	yes	No	yes	No	yes	No
2	RE vitreous haemorrhage with tractional retinal detachment	VITREOUS HAEMORRHAGE WITH TRD	20/8/2020	yes	No	yes	SF6	NO	**Bleeding+,small hole+.**
3	Right eye PDR with vitreous haemorrhage with epiretinal membrane on macula	VITREOUS HAEMORRHAGE	27/6/2020	yes	Epiretinal membrane removal	Yes	SF6	No	No
4	Right eye advanced PDR with vitreous Hage+with TRD.	VITREOUS HAEMORRHAGE WITH TRD	20/6/2020	Yes BIMANUAL DISSECTION	No	yes	No	Yes	**3 RETINAL TEARS superior to superior arcade and superotemporally**
5	Left eye advanced PDR with vitreous haemorrhage	VITREOUS HAEMORRHAGE	25/6/2020	YES	No	Yes	No	YES	Subretinal blood present, **one tear present.**
6	Right eye proliferative diabetic retinopathy with vitreous haemorrhage+	VITREOUS HAEMORRHAGE	10-03-2020	YES	YES	No	SF6 25%	No	**Very small tear superiorly**
7	Right eye pseudophakia with PDR with vitreous haemorrhage with TRD with epiretinal membrane	VITREOUS HAEMORRHAGE WITH TRD.	22.10.2020	YES	No	Yes	C3F8	No	some thin membrane remained temporal to macula
8	Right eye fibrovascular membrane over macula with macular TRD due to PDR.	TRD	19.11.2020	YES	YES	YES	C3F8	No	**Tears occurred internally at the edge of vitreous base**
9	LE TRD +SUBHYALOID HAEMORRHAGE SECONDARY TO PDR.	TRD	22.11.2020	YES	YES	YES	C3F8 14%	No	Bleeding+.
10	Left eye advanced PDR	VITREOUS HAEMORRHAGE	21.11.2020	YES	No	YES	C3F8	No	BLEEDING+

S.NO	VISION POD 1MONTH	VISION POD 3 MONTH	IOP POD 1 MONTH	IOP POD 3 MONTH	ANTERIOR SEGMENT POD 1 MONTH	ANTERIOR SEGMENT POD 3 MONTH
1	UA-CF2M WITH +4-6/36(P) PH-CF3M	UA -CF3M PH-NI	12mmhg	13mmhg	Within normal limits,mild catarctous changes in both eyes	within normal limits NS in OD,densePSC-OS
2	UA-OD- CF2M PH-6/60 OS-6/18(P) PH-NI	UA-OD-6/60 PH-6/36 OS-6/18(P) PH-NI	OD-18.0mmhg OS-19mmhg	OD-20.4mmhg OS-19.2mmhg	within normal limits,mild catarctous changes in both eyes	within normal limits,mild cataractous changes in both eyes.
3	UA-OD-CF3m PH-6/36 os-6/36 PH-6/24(p)	UA-OD-6/36 PH- 6/24 OS-CF2M PH-6/24	OD-12.5 mmhg OS-13 mmhg	OD-12.6mmhg OS-14.8mmhg	within normal limits,lens-PCIOL in RE,cataract in LE	Within normal limits,lens-PCIOL in RE,cataract in LE
4	UA-OD-CF1M PH-NI With +5-6/36 OS-UA-6/36 PH-6/24	UA-OD-CF1M PH-NI OS-6/36 PH-6/24	OD-16 mmhg,OS-18 mmhg.	OD-28 mmhg OS- 16 mmhg by applanation tonometry.	Within normal limits,RE -PSC+,LE-mild cataract	within normal limits,RE-psc,LE-cataract.
5	UA-OD-6/60 PH-NI OS-CF1.5m PH-NI	UA-OD- 6/36(P) PH-NI OS-CF2M PH -NI	OD-18.6mmhg OS-18.9mmhg	OD-17.6mmhg OS-17.8mmhg	with in normal limits lens-OD-Cataract OS- peripheral anterior synechiae superiorly,aphakia.	with in normal limits lens-OD-Cataract OS-peripheral anterior synechiae superiorly,aphakia.
6	UA-OD-6/24 PH-6/18 OS-6/18 PH-NI	OD-UA-6/18(P) PH-6/12(p) OS-UA-6/60 PH-NI	OD-19.3mmhg 0S-17.2 mmhg	OD-16 mmhg OS-16.9mmhg by NCT	Within nrmal limits mild PSC in both eyes.	within normal limits mild PSC in both eyes.
7	UA-OD-CF3m PH-NI OS-6/24(p) PH-NI	UA-OD-6/60 PH-NI OS-6/24 PH-NI	OD-19mmhg OS-23 mmhg	OD-18mmhg OS-16mmhg	Within normallimits LENS-PCIOL+	Withinnormal limitsLENS-PCIOL+
8	UA-OD-CF2M PH-6/36(P) OS-6/24 PH-6/12(P)	UA-OD-6/24(P) PH-NI OS-6/12(P) PH-6/9(P) WITHGLASSES-OD-6/12(P) OS-6/9(p)	OD-15.4mmhg OS-14.7 mmhg	OD-18.4 mmhg OS-15.5 mmhg	Within normal limits lens-PSC+in right eye,OS-within normal limits lens-mild cataract.	Within normal limits lens-PSC+in right eye,OS-within normal limits lens-mild cataract.
9	UA-OD-6/36(P) PH-NI OS-CF3M PH-6/36	UA-OD-6/24 PH-NI OS-6/36 PH-6/24(P)	OD-18.5mmhg OS-20.4mmhg	OD-17.8 mmhg OS-18.9mmhg	Within normal limits lens -mild cataract in both eyes	within normal limits,mild lens changes in RE,PSC in LE
10	UA-OD-6/24 PH-NI OS-CF1M PH-NI	UA-OD-6/18 PH-6/12 OS-UA-6/36 PH-6/18	OD-12.3 mmhg os-14.8 mmhg	OD-16.2 mmhg OS-16 mmhg	Within normal limits Lens clear in RE,mild lens changes in LE.	Within normal limits,mild lens changes in both eyes.

S.NO	POSTERIOR SEGMENT POD 1MONTH	POSTERIOR SEGMENT POD 3MONTH
1	OD-media hazy,vitreous hage+,TRD+. **OS-media hazy,silicone full,mild traction superiorly membrane present nasally and superiorly**	OD-media hazy,vitreous hage+,subtotal retinal detachment present tractional and combined. OS-media hazy,mild traction superiorly,RETINA,MACULA-FLAT,membrane present nasal to disc and superior to superior arcade,around superotemporal arcade.
2	**OD-media clear,disc-normal,macula shows multipe small hard exudates over the posterior pole with few haemorrhages,sclerosed vessels,PRPC marks+,foveal reflex lost,macular edema+.** OS- media hazy,multiple haemrrhages on posterior pole,few hard exudates,fine sheen on macula,periphery PRPC marks+	OD-media clear,disc-normal,hard exudates on macula,few hage+ on macula,PRPC marks+,retina flat.OS-media-clear,DISC-normal,there are hage on posterior pole,PRPC marks+,fovea-Dull.
3	**OD-media -clear,disc-normal,there is some fine membrane temporal to macula,RETINA-FLAT,MACULA-FLAT,PRPC marks+,no gas** OS-Media-clear,disc-normal,there is FVMalong ST arcade,fovea shows some hard exudates,PRPC marks+,active new vessels superior to superotemporal arcade.	OD-Media clear,Disc-normal,fine EPIRETINAL MEMBRANE NEA MACULA,RETINA-FLAT.OS-Media-clear,FVM along ST arcade,Macula-shows some thin meebranes.PRPC marks+
4	**OD-media -clear,sclerosed vessels on disc,silicone full,Haemorrhage and fibrosis ST,inferiorly vessels sclerosed,RETINA-FLAT**. OS-vitreous hage+,NVD+,Hage over macula.superior part of macula is detached also retina superior to arcade detached as nasal to the disc.Inferior vitreous hage+,Laser marks nasally,detached hyaloid inferiorly and temporally,superotemporally near the arcade retina is pulled up	OD-Media is clear,DISC-normal,there are ILM folds over the macula,sclerosed vessels temporally-both major temporal arteries,Laser scars temporally,silicone oil-full. OS-Tractional RD superotemporally along the arcadethen superior to disc,superonasally there is large FVM,Inferiorly partial PVD present.
5	OD-media -hazyview,inferiorly vitreous Hage+,large subhyaloid Hage+inferiorly, small fibrous membrane on disc,**OS-superiorly Hage+,silicone full,retina flat,lasermarks+.**	OD-inferiorly vitreous Hage+,TRD along ST arcade and superiorlyDetached posterior hyaloid on theposteriorpole,Attached hyaloid on the disc,lasermarks seen,hardexudate in centre.OS-media clear,DISC-NORMAL,there is some membrane superior to the disc,haemorrhage nasal to disc,macula flat,PRPC marks+.OS-
6	**OD-media clear ,DISC-NORMAL,shiny membrane on macula with mild edema,PRPC MARKS+,RETINA-FLAT** OS-FVM nasal to disc,localized traction ,macula flat with hard exudates,temporal to macula	OD- DISC-NORMAL,FOVEA-NORMAL,some edema temporal to fovea,Hages in the POSTERIORPOLE+,PRPC MARKS+,RETINA -FLAT.OS-Vitreous Hage +,Disc faintly seen,Hardexudates temporal to macula,PRPC marks+,subhyaloid Hage temporal to retina.
7	**OD-Good glow,gas around 40%,small Hage temporal to fovea,RETINA-FLAT,PRPC-marks+**.OS-media clear,DISC-NORMAL,few Hage temporal to fovea,PRPC marks+,fibrosis inferiorly,small subhyaloid Hage inferiorly,fovea-dull,pigment temporal to fovea.	OD-DISC-little pale,RPE chages around fovea,fine membrane on fovea,small gas bubble superiorly,PRPC marks+,OS-DISC-NORMAL,pigments near fovea,PRPC marks.
8	**OD-Media-clear,DISC-NORMAL,very narrow arteries white,? Subretinal lines seen in macula nasal part,fovea dull but flat,PRPC marks+.** OS-membrane on disc which is regressed NVD,Macula-fovea-dull,Hages and exudates temporal to macula.Inferiorly old vitreous Hage+,PRPC marks+.	OD-Media-clear,subretinal whitelines present,macular edema+,microaneurysms near fovea,PRPC marks+. OS-Thin membrane on disc (regressed NVD),Hages temporal to fovea+,Microaneurysms+,Inferiorly vitreous Hage+,Sclerosed vessels inferiorly,PRPC marks+.
9	OD-Subhyaloid Hage over macula,membrane on disc with attachment to ST arcade,retina not seen due to subhyaloid Hage. **OS-DISC-NORMAL,macula-normal,RETINA-FLAT,gas-10%,Laser marks+.**	OD-FVM from disc going ST ARCADE AND NASALLY, cause focal traction.NVD+,inferiorly vitreous Hage+,superiorly PVD with subhyaloid Hage+.OS-DISC-NORMAL,some residual membrane inferiorly,PRPC MARKS+,SCLEROSED vessels+,RETINA-FLAT.
10	OD-Glow-present,media-clear,DISC-NORMAL,subhyaloid Hage temporal to macula,edema temporal to macula.**OS-Dispersed vitreous Hage+,gas around 30%,RETINA-FLAT.**	OD-Media-clear,subhyaloid Hage+,infero and ST to macula,DISC-NORMAL,PRPC-marks+,macula-flat,few hardexudates near macula.OS-DISC-NORMAL,MACULA-FLAT,few microaneurysms around macula,PRPC marks+.

S.NO	RETINA FLAT	ANY MEMEBRANES	VITREOUS HGE	RESURGERY	TRD	NVG	SILICONE OIL IN AC	SILICONE OIL PRESENT OR NOT
1	Yes	present nasal to disc and superior to superior arcade,and around superotemporal arcade in LE	NO	NO	NO	NO	NO	FULL
2	Yes	No	N0	No	NO	No	No	No
3	RETNA-FLAT	Fine ERM near macula	No	No	No	No	No	No
4	RETINA -FLAT	ILM folds over macula,FVM present temporally	No	No	No	No	No	yes
5	RETINA-FLAT	there is some membrane superior to the disc,haemorrhage nasal to disc.	No	No	No	No	No	Yes
6	RETINA-FLAT	No	No	No	No	No	No	No
7	RETINA-FLAT	fine membrane on fovea	No	No	No	No	No	No
8	RETINA-FLAT	No	No	No	No	No	No	No
9	RETINA-FLAT	Some residual membrane inferiorly	No	No	No	No	No	No
10	RETINA-FLAT	No	No	N0	No	No	No	No

11	Narayan.M.shivat hankar	70 years	male	5879300	1027360	6.11.2020	9.11.2020	Since 10yrs	T.Glimepride 1mg,T.metformin 500mg	8.90%	No	No	No	No	No	DOV in LE since 3-4 months.	OD-UA-OD-6/9 PH-NI OS-CFCF PH-NI	H/o LE laser done 2 times	No	Within normal limits both eyes ,LENS-PCIOL+ in both eyes.	OD-GLOW-present,media-clear,Temporal disc -normal,crescent-normal,Laser marks-superior,inferior,nasal.Hemorrhages+. OS-GLOW-present,media-Hazy,old vitreous haemorrhage+,No view of fundus,superonasally fundus seen,some laser marks in nasal retina,membrane from disc coming inferonasally-1 QUADRANT.	#########	LEFT EYE OLD VITREOUS HAEMORRHAGE DUE TO PDR.	VITREOUS HAEMORRHAGE	7.11.2020	YES	YES	YES	C3F8	No	There was preexisting partial thickness hole superior to superior arcade.	UA-OD-6/12 PH-NI OS-CF2M PH-NI	UA-OD-6/12 PH-NI OS-HMCFPL+VE,PRACC PH-NI	OD-12.8 OS-10.7 mmhg	OD-13.8mmhg OS-7.8 mmhg	Within normal limits,PCIOL in both eyes.	Within normal limits PCIOL in both eyes	OD- DISC-NORMAL,MACULA-FLAT,PRPC marks+ OS-Macula detached,membrane on temporal aspect of macula,superior RETINA-FLAT,laser marks+,INFERIOR DETACHMENT+,peripheral retinal tear present.	OD-DISC-NORMAL,MACULA-FLAT,PRPC -marks+,OS-DISC-seen,media-clear,Hages nasal to disc,macula-flat,Inferiorly small amount of fluid+.after resurgery OS-Macula and superoir reina flat,Buckle effect seen.	No	YES-MEMBRANE PRESENTNASLLY,INFERIORLY	YES	YES-PPV+SCLERALBUCKLE+MEMBRANEPEELING+ILM PEELING+FAE+EL+SILICONEOIL INJ	YES-INFERIOR DETACHMENT+	No	No	yes
12	Kumargouda patil	51yrs	male	2847718	1028705	5.12.2020	5.12.2020	Since 9 yrs	Human INSULIN 30/70 ACTRAPID 20-0-20U LANTUS 0-0-12U GP1 (Glimepride 1mg+metformin 500 mg)	13.20%	No	No	No	No	Roseday-A ROSUVASTATIN 75-20,ASPIRIN	DOV in LE since 1 month.	OD-UA-OD-6/60 PH-6/18(P) OS-CF1M PH-NI	No	RE AVASTIN INJ 0.05 ML containing 1.25mg in ST quadrant.	Withinnormal limits both eyes LENS-PSC+ in both eyes.	OD-GLOW-present,Media-clear,veins-dialted,Hardexudates ++,midperiphery shows microaneurysms+++,macula shows subhyaloid Hage+. OS-GLOW-present,media-unclear,VITREOUS HAEMORRHAGE+,DISC-seen faintly,Nasal retina seen faintly.	LE shows vitreous haemorrhage ,Inferiorly memebrane seen has mobility,amplitude 50% mostly posterior hyaloid.	LEFT EYE PROLIFERATIVE DIABETIC RETINOPATHY WITH VITREOUS HAEMORRHAGE	VITREOUS HAEMORRHAGE	5.12.2020	YES	YES	YES	SF6	No	Some NVD was left for fear of causing haemorrhage	UA-OD-6/18(P) PH-6/18 OS-CF1.5M PH-NI	UA-OD- 6/18(P) PH-6/18 OS- CF2M PH -NI	OD-20.2 mmhg OS-27.6mmhg	OD-15.5 mmhg OS-14.4 mmhg	within normal limits LENS-PSC+ in both eyes.	within normal limits LENS-PSC+in both eyes	OD- DISC-NORMAL,there are haemorrhages superotemporal to fovea,few hardexudates over posterior pole.OS-medis hazy due to cataract,DISC-NORMAL,MACULA-FLAT,there are some RPE changes ST to fovea,PRPC marks+,RETINA-FLAT.	OD- DISC-NORMAL,there are haemorrhages superotemporal to fovea,few hardexudates over posterior pole.OS-medis hazy due to cataract,DISC-NORMAL,MACULA-FLAT,there are some RPE changes ST to fovea,PRPC marks+,RETINA-FLAT.	RETINA-FLAT	No	No	No	No	No	No	No
13	Basavanni R.kupati	59yrs	male	5942407	1033619	4.1.2021	8.1.2021	since 20yrs	T.Tenegliptin 20mg 0-1-0 ,Metformin hydrochloride 500 mg 1-0-1 since 2yrs.	7.70%	No	No	No	No	No	DOV in LE since 1.5 month	OD-UA-OD-6/60 PH-NI OS-HMCF PH-NI	H/o Laser done only for RE PRPC 4 times	No	Within normal limits both eyes LENS-OD-mild cataract+,LENS-OS-PCIOL+.	OD-Glow-present,inferiorly VITREOUS HAEMORRHAGE present,FVM -1 QUADRANT-present on the disc ,nasal to disc,localized TRD -1 QUADRANT,MACULA-edema with hardexudate ring.OS-Glow-present,media-Hazy view,old Vitreous haemorrhage+,subhyaloid haemorrhage+.	LE shows Vitreous haemoorhage ,tractional membrane withTRD seen near disc.	LEFT EYE OLD VITROUS HAEMORRHAGE WITH?TRD	VITREOUS HAEMORRHAGE	7.1.2021	YES	No	YES	C3F8 12%	No	Bleeding+	UA-OD-6/60 PH-NI OS-CF3M PH-NI	UA-OD-6/24(P) PH-6/18(P) OS-6/24 PH-6/18	OD-14.8 mmhg OS-15.1 mmhg	OD-12.2 mmhg OS-13.4 mhg	Within normal limits LENS-OD-mild cataract,OS-PCIOL+	Within normal limits Lens-OD-PCIOL+,mild lens capture temporally,OS-PCIOL+	OD-FVM on disc,fibrous membrane on macula with hard exudates on centre,inferior vitreous haemorrhage+,RETINA-FLAT,TRD present nasally-1 QUADRANT.OS-media-clear,hard exudates temporal to fovea,laser marks 360 degree,fovea-dull,gas bubble+.	OD- media clear,DISC-NORMAL,hardexudates near macula and temporal to fovea,PRPC marks+,RETINA-FLAT. OS-media-clear,DISC-NORMAL,mild edema+fine hard exudates in macula,PRPC marks+,RETINA-FLAT.	RETINA-FLAT	No	No	No	No	No	No	No
14	Basavanni hanchimani	51yrs	male	5900300	1033284	28.12.2020	1.12.2021	since 20yrs	GLIZID MU 500mg 1-0-1,on insulin lantus 0-0-14u	9.10%	since 17yrs	TELMIKIND BETA 501-0-0,TAB.AMLOKIND 5MG 0-0-1,VILACT 500MG 0-1-0	No	No	No	DOV in LE since 1 month	UA-OD-6/24 PH-NI OS-CF2M PH-NI WITHGLASSES OD-6/24 OS-6/60	H/O laser done for RE 3 times	No	Within normal limits-OD-mild cataract OS-PCIOL+.	OD-GLOW-present,smallVITREOUS HAEMORRHAGE ALONG ST ARCADE,Hages around ST arcade,PRPC marks+,NVE superiorly,FVM present ST to macula,present inferior to subhyaloid Hage,subhyaloid Hage present below inferotemporal arcade.FVM-2 QUADRANTS. OS-Glow-present,sclerosed vessels temporally,superiorly retinal Hages present,Thick FVM starting from the disc causing inferior TRD-1 QUADRANT.subretinal bands inferior to inferior arcade.MACULA-shows some RPE changes,lower part of macula is lifted.	########	LEFT EYE CHRONIC TRD SECONDARY TO PDR.	TRD	31.12.2020	YES,BIMANUAL DISSECTION	No	PFCL INJECTION+PFCL AIR EXCHANGE+ENDOLASER	N0	YES	RETINAL TEAR PRESENT INFEROTEMPORALLY,SUBRETINAL BANDS PRESENT INFERIORLY	UA-OD-6/60 PH-NI OS-CF2M with +4D PH-NI	UA-OD-CF2M PH-NI OS-CF2M WITH +4D PH-NI	OD-19.3 mmhg OS-14.2 mmhg	OD-18.8,OS-17.2 mmhg	within normal limitsLENS-mild cataract in RE,LENS-PCIOL in LE	within normal limitsLENS-mild cataract in RE,LENS-PCIOL in LE	OD- media-clear,posterior hyaloid detachment+,subhyaloid haemorrhage over macula.OS-media-clear,superior retina flat,preretinal haemorrhage present,some fluid below the disc,MACULA-FLAT,subretinal bands inferiorly,large tear inferiorly,laser marks+,silicone full.	OD- media-clear,posterior hyaloid detachment+,subhyaloid haemorrhage over macula.OS-media-clear,superior retina flat,preretinal haemorrhage present,some fluid below the disc,MACULA-FLAT,subretinal bands inferiorly,largetear inferiorly,laser marks+,silicone oil full.	SUPERIOR RETINA-FLAT.Inferior-retina-detached.	subretinal bands inferiorly	No	No	inferiorly large TEAR+	No	No	yes
15	Lalsab naik	61yrs	male	6028965	1047554	2.4.2021	3.4.2021	since 8yrs	T.metformin 500 mg BD ,T.GLIMEPERIDE 2mg1-0-0	9.80%	Since 5 yrs	T.AMLODIPINE 5mg1-0-0	No	No	No	DOV in RE since 1 month	UA-OD-CF2m PH-NI OS-6/18 PH-NI	H/o Laser done for LE 3 times	No	Within normal limits inRE,in LE-NVI+ OD-LENS-GREYISH,OS-LENS-GREYISH.	OD-Glow-present,posterior hyaloid detachment inferior to disc+,Thick,FVM present superior pole of disc,extending to vitreous-2QUADRANTS,TRACTIONAL RD superiorly AND TEMPORALLY - 2 QUADRANTS,subhyaloid Hage inferiorly.RETINA under subhyaloid haemorrhage flat.OS-Glow-present,media-clear,fine NVD on the disc along ST border,posterior pole shows multiple hard exudates and few Hages.There is FVM temporal to macula,superior to fovea.-1QUADRANT	#########	RIGHT EYE TRD WITH SUBHYALOID HAEMORRHAGE	TRD	2.4.2021	YES	No	YES	C3F814%	No	SOME TAGS OF MEMBRANE WERE LEFT ALONG THE SUPEROTEMPORAL ARCADE FOR FEAR OF CREATING TEARS.	UA-OD-CF3M PH-NI OS-6/12 PH-NI	UA-OD-6/9(P) PH-6/6(P) OS-6/12 PH-NI	OD-10.6 mmhg OS-12.2 mmhg	OD-16.2 mmhg OS-12.3 mmhg	Within normal limits ,LENS-PSC+,LENS-greyish.	Within normal limits ,LENS-PSC+,LENS-greyish.	OD-Media-clear,vitreous haemorrhage+ inferiorly,Laser marks seen,RETINA-FLAT,Macula-clear. OS-Media-clear,fine NVD on the disc superotemporal border,there is membrane temporal to macula.	OD-Media-clear,C:D ratio-0.4,Macula-flat,PRPC marks+,OS-Media-clear,fine NVD on the disc, superotemporal border,there is membrane temporal to macula.	RETINA-FLAT	No	No	No	No	No	No	No
16	Annasab naik	67yrs	male	5646873	1042099	2.3.2021	8.3.2021	since 10yrs	T.GLIMESTAR PM1 1-0-1	7.40%	Since 10yrs	Tab.TELMA-AM H 40,TELMI-H 40mg/12.5mg1-0-1	No	No	No	DOV in RE since 2months	UA-OD-CF1M PH-NI OS-CF2M PH-6/60	No	H/o INJ.AVASTIN FOR RE ON 10.2.2021 0.05ML containing 1.25mg in ST quadrant.	Within normal limits LENS-cortical cataract,PSC+in both eyes.	OD-Glow-present,media-hazy,vitreous Hage+.OS-GLOW-present,media-Hazyview,DISC-PALE,PRPC marks present,fibrous membrane over macula+.	#########	RIGHT EYE VITREOUS HAEMORRHAGE SECONDARY TO PDR	VITREOUS HAEMORRHAGE	6.3.2021	YES	YES	YES	C3F8 14%	No	BLEEDING+	UA-OD-CF1.5M PH-NI OS-CF2M PH-NI	UA-OD- 6/60 PH-NI OS-CF2M PH-NI	OD-15.3 mmhg,OS-17.3 mmhg	OD-14mmhg OS-17mmhg	Within normal limits ,LENS-CC,PSC+in BE	Within normal limits ,LENS-CC,PSC+in BE	OD-media-hazy,disc-normal,atrophy in centre,gasbubble -10%,PRPC marks present.OS-media hazy,FVM on the disc,fibrovascular scar+,Hage temporal to macula+.	OD-glow-present,DISC-NORMAL,RETINA-FLAT,PRPC marks+.OS-media hazy,FVM on the disc,fibrovascular scar+,Hage temporal to macula+	RETINA-FLAT	No	No	No	No	No	No	No
17	Shridevi kadonnavar	64yrs	male	6019899	1043867	13.3.2021	13.3.2021	since 30yrs	HUMAN MIXTARD 30/70 14u-0-12u,ISRYL M2 1-0-1	6.80%	No	No	No	No	No	DOV in LE since 2 months	UA-OD-6/9 PH-NI OS CF2M PH-NI	H/o laser done LE -2times.	No	Within normal limits PCIOL+in BE	OD-Glow-present,media-clear,DISC-NORMAL,C.D-0.3,few Hages+,cotton woolspots+ in periphery.OS-Glow-present,media-vitreous Hage+,DISC-seen hazily,superior and inferior-retina flat,macula not seen.	########	LE PDR WITH VITREOUS HAEMORRHAGE	VITREOUS HAEMORRHAGE	13.3.2021	YES	YES	YES	No	No	No	PT didn't folloup because of lockdown	UA-OD-6/9,PH-NI OS 6/9 PH-NI	pt didn't followupbecause of lockdown	OD-19.5mmhg,OS-15.6mmhg	PT didn't folloup because of lockdown	Within normal limits BE-LENS-PCIOL+	PT didn't folloup because of lockdown	OD-GLOW-present,DISC-NORMAL,Cotton wool spots+,peripheral Hages+ OS-DISC-NORMAL,tortuous vessels present,macula-flat,laser marks seen superiorly and inferiorly.	RETINA-FLAT	No	No	No	No	No	No	No
18	Shivakumar murali	45 yrs	male	6057133	1048894	12.4.2021	13.4.2021	since 5yrs	Tab.Tenegliptin 0-0-1 (AXEGLIPTIN M 5oomg/20mg) GLIRUM MF 1-0-1 (GLIPIZIDE 5mg,METFORMIN 500MG)		RECENTLY DIAGNOSED NOT ON MEDICATIONS	No	No	No	No	DOV in LE since 1 month	UA-OD- 6/18(p) PH-6/9 OS- 6/60 PH-NI	H/o Laser done in both eyes -3 times.	No	Within normal limits BE.LENS-cataract in both eyes.	OD-Glow-present,media-clear,DISC-NORMAL,C.D-0.3,Haemorrhages+,cottonwoolspots+,PRPC marks+,MACULA-FLAT OS-Glow-present,media-clear,PRPC -marks+,small FVM present nasal to disc-1QUADRANT,inferiorly subhyaloid Haemorrhage,partial detachment of hyaloid with haemorrhage over macula.	#########	LEFT EYE CATARCT WITH VITREOUS HAEMORRHAGE.	VITREOUS HAEMORRHAGE	15.4.2021	PHACO+IOL+PPV	YES	YES	No	No	BLEEDINGFROM FIBROVASCULAR MEMBRANE CAUTERIZED	UA-OD-6/9 PH-6/6(P) OS-6/12(P) PH-6/9	UA-OD-6/9(p)PH-NI OS-6/12(P) PH-6/18	OD-14.5 mmhg,OS-14.6mmhg	OD-13.7 mmhg OS-14.2 mmhg	Within normallimits BE-LENS-PCIOL+	Within normal limits,lens-OD-greyish,OS-PCIOL+	OD-Media-clear,DISC-NORMAL,veins-dilated,superiorly small subhyaloid Hage,PRPC marks+,MACULA-FLAT,hardexudates along ST ARCADE. OS-Few haemorrhages along ST area,laser marks seen.DISC-normal,MACULA-NORMAL,RETINA-FLAT.	OD-Media-clear,DISC-NORMAL,Fovea-normal,Haemorrhages+posterior pole,PRPC marks+,few haemorrhagesOS-Media-clear,DISC-NORMAL,Macula-few hard exudates+,No edema,PRPC marks+	RETINA-FLAT	No	No	No	No	No	No	No
19	Latha appasab Kalinge	55yrs	female	5648777	1008225	16.3.2020	22.3.2020	Since7-8 yrs	Tab.TENEGLIPTIN AND METFORMIN HYDROCHLORIDE EXTENDED RELEASE TABLETS (TENEGLIPTIN M 500) 1-0-1,METRIDE PLUS 2 1-0-1	9.30%	No	No	No	No	No	DOV in LE since 1 month	UA-OD-CF2M PH-NI OS-CF1.5M PH-NI	H/o laser done for RE - 1time	H/o LE AVASTIN INJ 0.05ML CONTAINING 1.25MG ON MARCH 1,MARCH 16	Both eyes IRIS-NVI,LENS-OD-MILD PSC+,OS-PSC+.	OD-Glow- present,Media-Hazy,Inferiorly Haemorrhages+ in all 4 quadrants,FVM over the DISC,and around temporal arcade,superior to disc-2 QUADRANTS.MACULA-shows haemorrhages,microaneurysms,partially detached posterior hyaloid over posteriorpole. OS-Glow-present,Media-Hazy,Thick vitreous haemorrhage inferiorly,FVM 3 QUADRANTS-over the disc,and along the superior arcade,and along the nasal and inferonasal part of retina.Inferiorly vitreous Hage+,superior retina look flat.MACULA-covered by blood and memebrane	#########	LEFT EYE ADVANCED PDR WITH VITREOUS HAEMORRHAGE WITH TRD	VITREOUS HAEMORRHAGE +TRD	21.3.2020	PHACO+IOL+PPV+MP	YES	YES	No	YES	BLEEDING+,NASALLY 3 TEARS OCCURRED LEADING TO RETINAL DETACHMENT.	PATIENT DIDN'T FOLLOWUP BECAUSE OF LOCKDOWN	UA-OD-6/36(P) PH-NI OS-CF3M PH-NI	PATIENT DIDN'T FOLLOWUP BECAUSE OF LOCKDOWN	OD-11.7 mmhg OS-11.2 mmhg	PATIENT DIDN'T FOLLOWUP BECAUSE OF LOCKDOWN	Within normal limits LENS-OD-mild cataract,OS-LENS-PCIOL+,PCO +.	PATIENT DIDN'T FOLLOWUP BECAUSE OF LOCKDOWN	OD-media-clear,FVM on the DISC AND AROUND TEMPORAL ARCADE+,SUPERIOR TO DISC+,NVD+,Haemorrhages+.OS-Media-clear,DISC-PALE,sclerosed vessels nasally,membrane in inferotemporal arcade,MACULA-FLAT.Superonasal arcade also has membrane.RETNA-FLAT,SILICONE -FULL.	RETINA-FLAT	MEMBRANE IN THE INFEROTEMPORAL ARCADE ,SUPERONASAL ARCADE ALSO HAS MEMBRANE	No	No	No	No	No	YES ,FULL

20	Uday Annasaheb patil	60yrs	Male	5659006	1007487	11.3.2020	17.3.2020	since 30yrs	INSULIN.HUMAN ACTRAPID 14-14-14U , ON TAB.GLIMI M2 1-0-1	8.30%	since 7 years	LOSAR BETA H1-0-0,LOSAR 0-0-1	YES,ANGIOPLASTY DONE IN 2011	No	ECOSPRIN earlier,now DEPLAT-CV since 6yrs	DOV in LE since 1 month	UA-OD-6/24 PH-NI OS-CF3M,PH-NI ,WITHGLASSES OD-6/12(P) OS-6/36	H/o LASER done for RE -4 times,LE-2times.	H/o LE AVASTIN INJ 0.05ML CONTAINING 1.25MG ON 25 th FEBRUARY	OD-small spectrum of NVI TEMPORALLY,LENS PSC+.OS-IRIS-NONVI,LENS-PSC+,polychromatic luster+.	OD-Glow-present,Media-clear,DISC-NORMAL,C:D-0.3,AV changes++FVM,TRD-NIL,MACULA-mild edema,FRDULL,PRPC marks+.OS-Glow-faint,Media-vitreous haemorrhage+,DISC-NOT SEEN,Bloodvessels -Details not seen,small FVM around ST arcade,MACULA-NOT SEEN,NASAL RETINA SEEN -FLAT,LASER MARKS SEEN NASALLY.	##########	LEFT EYE PROLIFERATIVE DIABETIC RETINOPATHY WITH CATARACT WITH VITREOUS HAEMORRHAGE	VITREOUS HAEMORRHAGE	17.3.2020	**PHACO+IOL+PPV+MP**	No	YES	No,AIR	No	MILD BLEEDING FROM FIBROUS MEMBRANE+.	UA-OD- OS- UA-PH-6/36	UA-OD-6/24(p) PH-6/12(P) OS-6/18(P) PH-NI WITH GLASSES-OD-6/12(P) OS-6/18(P)	OD-14mmhg OS-14 mmhg	OD-20 mmhg,OS-16mmhg	ON 20.3.2020 -anterior segment examination OF LE showed large epithelial defect inferiorly,DF++,SMALL HYPOPYON+,AC cells+,fibrin+,,so patient has been planned, for IVI ANTIBIOTICS VANCOMYCIN +CEFTAZIDIME,ON 21.3.2021-LIDS-SLIGHTLY SWOLLEN+,PUPIL-MEDICATED,AC-GRADE 3 CELLS,EXUDATE IS LESS IN AC NOW,CONTACT LENS ON., ON 17.4.2020-CORNEA-STRIATE KERATITIS +,AND CONTACT LENS REMOVED. OD-WITHIN NORMAL LIMITS,LENS-PSC+.	WITH IN NORMAL LIMITS OD-LENSPSC+,OS-PCIOL IN PLACE	OD-Media-Hazy,DISC-NORMAL,FOVEA-NORMAL,fe Haemorrhages in posterior pole,PRPC marks+,mild activity OS-Media-clear,DISC-NORMAL,FOVEA-FLAT,PRPC marks in the periphery outside arcade,few haemorrhages temporal to macula.	OD-Media-Hazy,DISC-NORMAL,FOVEA-NORMAL,fe Haemorrhages in posterior pole,PRPC marks+,mild activity+. OS-Media-clear,DISC-NORMAL,FOVEA-FLAT,PRPC marks in the periphery outside arcade,few haemorrhages temporal to macula.	RETINA-FLAT	No	No	No	No	No	No	No
21	Sikandar multani	47yrs	Male	5785132	1038814	9.2.2021	13.2.2021	Since 5 yrs	Galvus met 50/500 mg ,novonor 18u-0-12u	8.70%	Since 2 months	Telma-H	No	No	No	DOV in LE since 3 months	UA-OD-6/12(P) PH-6/9(P) OS-UA-6/60 PH-NI WITH GLASSES OD-6/9(P) OS-6/36	H/o laser done for both eyes -4times PRPC	No	OD-Within normal limits both eyes.mild lens changes in both eyes.	OD-Glow-present,media-clear,DISC-NORMAL,posterior pole Hage+,fully lasered. OS-Detached hyaloid,subretinal blood +,DISC-FAINTLY SEEN,Hard exudates temporal to macula present,PRPC marks+,FVM present nasal to disc,-1 QUADRANT,traction inferonasal to disc,**Inferior subhyaloid haemorrhage+.**	########	LEFT EYE PROLIFERATIVEDIABETIC RETINOPATHY WITH VITREOUS HAEMORRHAGE WITH LOCALIZED TRACTION	VITREOUS HAEMORRHAGE	11.2.2021	YES	YES	YES	SF6	No	BLEEDING+	UA-OD-6/9,PH-NI,OS CF3M,PH-NI	UA-OD-6/9,PH-NI OS 6/36 PH-NI	OD-16.7 mmhg OS-19.9 mmhg	OD-14.5 mmhg OS-19.9 mmhg	Within normal limits mild lens changes in both eyes.	Within normal limits mild lens changes in both eyes.	OD-Media-clear,DISC-NORMAL,PRPC marks+,MACULA-FLAT,hardexudates+.**OS-Media-hazy,DISC-faintly seen,dispersed vitreous haemorrhage +,RETINA-FLAT.**	OD-Media-clear,DISC-NORMAL,PRPC marks+,MACULA-FLAT,hardexudates+ temporal to macula+,Retinal haemorrhages+.OS-Media-clear,DISC-NORMAL,RETINA-FLAT,hardexudates temporal to macula+,few haemorrhages+,PRPC marks+.	RETINA-FLAT	No	**YES**	No	No	No	No	No
22	Kishore Mutagekar	50yrs	Male	5896134		5.3.2021	7.3.2021	Since 7yrs	Glimestar M1 0-0-1,Glimestar M2 1-0-0	7.40%	Since 6 months	Nebivolol 5 mg 1-0-0,Clinidipine 10 mg (LN BETA-5)	No	No	No	DOV in RE since 4-5 months	UA-OD-6/60 PH-NI OS-6/36 PH-NI	H/o Laser done for both eyes in november month and for RE 1 week gap	No	OD-Within normal limits OD-LENS-GREYISH,OS-PSC+,Anterior vitreous cells+.	OD-Glow -present,Faint view of retina,**inferiorly vitreous haemorrhage+,DISC-NVD+**,FVM on disc going a focal traction along ST arcade-2 QUADRANTS,Focal TRD along ST arcade+-2QUADRANTS,subhyaloid Hage+.OS-Glow-present,media-slightly hazy,DISC-NORMAL,PRPC marks+,	#########	RIGHT EYE PROLIFERATIVE DIABETIC RETINOPATHY WITH VITREOUS HAEMORRHAGE WITH TRD	VITREOUS HAEMORRHAGE +TRD	6.3.2021	YES	No	YES	C3F8	No	REPEATED BLEEDINGFROM THE MEMBRANE	UA-OD-6/18 PH-6/12(P) OS- 6/18(P) PH-NI	UA-OD-6/18 PH-6/12 OS-6/18 PH-NI	OD-22.4 mmhg OS-14.9 mmhg	OD- 20 mmhg ,OS-15.9 mmhg	Within normal limis LENS-GREYISH in both eyes	Within normal limis LENS-GREYISH in both eyes	**OD-Fovea-shows few haemorrhages around it ,small gas bubble+,RETINA-FLAT.**OS-DISC-NORMAL,some residual membrane inferiorly,RETINA-FLAT,sclerosed vessels present.	OD- Media-cleTINA-FLATar,DISC-NORMAL,FOVEA-shows few HAGES+ around it.RETINA-FLAT.OS-Media-clear,DISC-NORMAL,Multiple Hages temporal to macula+fovea-edema+prpc marks+,RETINA-FLAT	RETINA-FLAT	No	No	No	No	No	No	No
23	Basavanni Kupati	59yrs	Male	5942407		4.3.2021	8.3.2021	since 20yrs	T.Tenegliptin 20mg 0-1-0 ,Metformin hydrochloride 500 mg 1-0-1 since 2yrs.	7.70%	No	No	No	No	No	DOV in RE since2-3 months	UA-OD-6/60 PH--6/36 OS-6/36 PH-6/18	H/o Laser done only for RE PRPC 4 times	No	Within normal limits both eyes LENS-OD-mild cataract+,LENS-OS-PCIOL+.	OD-Glow-present,media-Hazy,**inferior vitreous haemorrhage+**,PRPC marks+,FVM nasal to disc,ST arcade,IT arcade-3QUADRANTS,Membrane on macula+,RETINA-FLAT.OS-Glow-present,Media-clear,DISC-NORMAL,Hardexudates present,PRPC marks+,RETINA-FLAT,MACULA-SHEEN+.	########	RIGHT EYE VITREOUS HAEMORRHAGE +NASAL TRACTIONAL RETINAL DETACHMENT+SECONDARY TO PDR WITH MACULOPATHY	VITREOUS HAEMORRHAGE	4.3.2021	YES	YES	YES	SF6	No	BLEEDINGFROM THE DISC AND INFERIOR TO POSTERIOR POLE.	UA-OD-6/24(P) PH-6/18(P) OS-6/24 PH-6/18	UA-OD-6/24(P) PH-6/18(P) OS-6/24 PH-6/9	OD-12.2 mmhg OS-13.4 mmhg	OD-14 mmhg OS-12.2 mmhg	Within normal limits,optic capture temporally,PCIOL in both eyes.	Within normal limits,optic capture temporally,PCIOL in both eyes.	**OD-Media-clear,DISC-NORMAL,Hard exudates temporal to fovea+,Nogas,PRPC marks+,RETINA-FLAT.**OS-Media-clear,DISC-NORMAL,fine hard exudates in macula,PRPC marks+,RETINA-FLAT.	OD- Media-clear,DISC-NORMAL,fovea -ring of hardexudates temporal to macula+,PRPC marks+.OS-Media-clear,DISC-NORMAL,nasally Hages+,Sclerosed vessels+,Hardexudates temporal to macula+,PRPC marks+.	RETINA-FLAT	No	No	No	No	No	No	No
24	Basavaraj Hanchinmani	51yrs	Male	5900300	1048447	5.4.2021	9.4.2021	Since 20yrs	GLIZID MU 500mg 1-0-1,on insulin lantus 0-0-14u	9.10%	Since 17yrs	TELMIKIND BETA 501-0-0,TAB.AMLOKIND 5MG 0-0-1,VILACT 500MG 0-1-0	No	No	No	DOV in RE Since 2.5 months	UA-OD-CF3M PH-NI OS-CF3M WITH+4 PH-NI	H/o laser done for RE 3 times	No	Within normal limits botheyes LENS-greyish in RE,OS-PCIOL+	OD-Glow -present,Media-clear,Thick FVM with TRD IT and ST- 2 QUADRANTS,**thick subhyaloid Hage+ inferiorly**,Posterior hyaloid detachment over macula and upper part attached to disc.OS-Glow-present,Media-clear,Preretinal haemorrhage+,Subretinal bands inferiorly,Large tear inferiorly,Superior RETINA-FLAT,MACULA-FLAT.	########	RIGHT EYE PROLIFERATIVE DIABETIC RETINOPATHY WITH POSTERIOR HYALOID DETACHMENT WITH TRD WITH SUBHYALOID HAEMORRHAGE	VITREOUS HAEMORRHAGE +TRD	8.4.2021	YES	No	YES	No	YES	**RETINAL TEAR IN INFEROTEMPORAL,SUPEROTEMPORAL AREAS**	UA-OD-6/60 WITH +5D PH-NI OS-CF1M PH-NI	UA-OD-6/60 WITH +5D PH-NI OS-CF1M PH-NI	OD-14.5 mmhg OS-13.1 mmhg	OD-15.5 mmhg OS-13.1 mmhg	Within normal limits,LENS-PCIOL+in both eyes	Within normal limits PCIOL+in both eyes	**OD-Media-clear,DISC-NORMAL,fovea-DULL,few haemorrhagesnear the fovea,PRPC marks+,inferotemporally Hages+,some fibrosis +inferotemporally,superiorly retina flat,ST RETINA-FLAT,NASALLYB RETINA-FLAT,silicone full**OS-INFERIOR retina-detached,subretinal band+,large hole inferiorly below the inferotemporal arcade.	OD- RETINA-FLAT,silicone full,some membrane inferiorly and inferotemporally.OS-inferior detachment+,superiorly retina -flat,PRPC marks+,silicone full	RETINA-FLAT	No	**YES**	No	No	No	No	YES
25	Sushma bonagale	60yrs	Female	5738736	1033260	29.12.2021	1.1.2021	Since 20yrs	Gliclazide and metformin Hydrochloride extended release tablets (DIAMICRON XR MEX 500 1-0-1),INSULIN GLARGINE 100 IU/ML 20-0-20U ,NOVOPEN 6U-6U-6U	10.70%	Since 3 yrs	Telmisartan chlorthalidone and metoprolol succinate tablets (MET XL 30 1-0-1)	No	No	No	DOV in RE since 6 months	UA-OD-6/36 PH-6/24(P) OS-CF1M PH-CF1.5M	H/o laser on 10/6/2020 botheyes.	LE AVASTIN inj on 22.6.2020	Within normal limits in both eyes OD-Cataract+,Lens-aphakia.	OD-Glow-present,Media-**Inferiorly vitreous haemorrhage present,**Hard exudates in centre,Detached posterior hyaloid on the posterior pole,Laser marks seen,Area of TRD just above disc,FVM along ST arcade and superiorly,-2 QUADRANTS.OS-Glow-present,Media-clear,DISC-NORMAL,Bloodvessels-AA+,Haemorrhage nasal to the disc,PRPC marks present,there is some membrane superior to the disc, MACULA-FLAT.	########	RIGHT EYE PROLIFERATIVE DIABETIC RETINOPATHY WITH VITREOUS HAEMORRHAGE +TRD	VITREOUS HAEMORRHAGE	31.12.2020	Phaco +IOL+ppv	No	YES	No	No	No	UA-OD-CF2.5M PH-6/60 OS-CF2.5M PH-6/60	UA-OD-6/24 PH-NI OS-CF2M PH-NI	OD-22.1 mmhg OS-18.7 mmhg	OD-17.2mmhg OS-12.7 mmhg	Within normal limits LENS-OD-ACIOL+,PI,OS-APHAKIA	Within normal limits LENS-OD-ACIOL+,PI,OS-APHAKIA	OD-Media-clear,DISC-NORMAL,Laser marks seen.OS-Media-clear,DISC-NORMAL,AA+,Lasermarks+.	**OD-Media-clear,DISC-NORMAL,RETINA-FLAT,PRPC marks+.**OS-media-clear ,DISC-NORMAL,Lasermarks+AA+,RETINA-FLAT.	RETINA-FLAT	No	No	No	No	No	No	No
26	Adivevva pagad	57 yrs	Female	5729979	1038569	8.2.2021	11.2.2021	Since 15 yrs	Glimeperide and Metformin hydrochloridetablets POSIMET G1 FORTE 1-0-1	12.80%	Since 8 yrs	Nifedipine sustained release tab.10mg	No	No	POSIGOLD ,Enteric coated aspirin,Rosuvastatin,clopidogrel capsules.	DOV in LE since 6 months	UA-OD-6/60 With+5D,PH-NI OS-CF1M PH-NI	Both eyes laser done 1 time	RE avastin inj on 16/6/2020	Within normal limits LENS-CATARACT + in both eyes.	OD-Glow-Present,Media-clear,DISC-NORMAL,Sclerosed vessels+,Silicone oil full,FVM present temporally,RETINA-FLAT.OS- Glow-present,Media-clear,Inferiorly partial PVD present,Thick membrane nasally and superotemporal arcade-2 QUADRANTS ,tractional detachment superotemporally-1 QUADRANT,Superior TRD	########	LEFT EYE TRD +RRD secondary to advanced PDR.	TRD	10.2.2021	Phaco fragmentation	No	YES	No	YES	**TEARS occurred nasally,inferiorly and temporally**	PT didn't folloup because of lockdown	UA-OD-6/60 OS-CF2M PH-NI	PT didn't folloup because of lockdown	OD-22.4 OS-20.4mmhg	PT didn't folloup because of lockdown	OD-Cornea-clear,AC-normal depth,No NVI,NS+2.OS-Cornea-clear,APHAKIA+.	PT didn't folloup because of lockdown	**OD-Media-clear,sclerosed superotemporal artery,membrane on macula,RETINA-FLAT,prpc marks+,silicone-full**OS-Media-clear,DISC-NORMAL,MACULA-FLAT,PRPC -MARKS+,RETINA-FLAT,SILICONE-FULL.	RETINA-FLAT	No	No	No	No	No	No	YES
27	Prakash Walvalkar	60yrs	Male	5672085	1008239	20.3.2020	22.3.2020	since 4yrs	GLIMITH MF-1MG,METFORMIN 500mg,Glimeperide 1mg	5.60%	since 3yrs	Torsemide,spironolactone tabletsCardivas LR 10 (carvedilol extended release tablets)	YES,2.5 yrs back stent has been placed	No	YES, since 2yrs on ECOSPRIN GOLD FORTE,CLOPIDOGERL AND ASPIRIN	DOV in LE since 2 months	UA-OD-6/6(P) PH-NI OS-HMCF PH-NI	H/o RE LASER done -2 times	No	Within normal limits-LENS-GREYISH in both eyes	OD-Glow-present,Media-clear,DISC-NORMAL,Haemorrhages present in all 4 QUADRANTS,Nasally PRPC marks+,MACULA-FLAT.OS-Media-**old dense vitreous haemorrhage,** No view of disc,c:d ratio,bloodvessels,RETINA.	USG done showed PVD+VITREOUS HAEMORRHAGE+TRD	LEFT EYE PVD+VITREOUS HAEMORRHAGE+TRD	VITREOUS HAEMORRHAGE+TRD	21.3.2021	ONLY PPV,NO MEMBRANE PEELING	No	YES	No	No	No	UA-OD-6/12(P),PH-6/6 OS-6/18 PH-6/12	UA-OD-6/12(P) PH-6/6 OS-6/12 PH-NI	OD-14 mmhg OS- 14 mmhg	OD-16 mmhg OS-14mmhg	Within normal limits LENS-MILD CATARCTOUS changes in both eyes	Within normal limits LENS-MILD CATARCTOUS changes in both eyes	OD-Glow-present,Media-clear,DISC-NORMAL,Haemorrhages present in all 4 QUADRANTS,Nasally PRPC marks+,MACULA-FLAT,OS-RETINA-FLAT,PRPC marks+,Few haemorrhages in macula.	OD-Glow-present,Media-clear,DISC-NORMAL,MACULA-shows microaneurysms+++, LASER MARKS+,MACULA-FLAT.**OS-Media-clear,DISC-NORMAL,few haemorrhages in macula,PRPC marks+,Superior to disc there is some residual membrane left.**	RETINA-FLAT	Superior to disc,there is some residual membrane	No	No	No	No	No	No
28	Mahadevi Housamani	55yrs	Female	5614471	1001559	11.2.2020	16.2.2020	Since 12yrs	TAB.METFORMIN 1-0-1,TAB.GLIBENCLIMIDE 1-0-1	7.40%	No	No	No	No	No	DOV in LE since 5-6 months	UA-OD-6/12 PH-6/6(P) OS-HMCF PH-NI	No	H/o LE AVASTIN INJECTION ON 11.2.2020	Within normal limits-LENS-GREYISH in both eyes	OD-Glow-present,Media-clear,DISC-NORMAL,C.D-0.2,HAGES+,FVM +superiorly-1QUADRANT,Inferior laser marks present.OS-Glow-present,Media-subhyaloid Hage+,FVM present superior to disc and surrounding area-3 QUADRANTS,TRD along ST arcade-1 QUADRANT,membrane is strongly attached causing posterior hyaloid detached all over except macula,**subhyaloid Hage present posterior.**	#############	LEFT EYE PDR WITH VITREOUS HAEMORRHAGE WITH TRD	VITREOUS HAEMORRHAGE +TRD	15.2.2020	YES+BIMANUAL TECHNIQUE	No	YES	No	YES	**Mild bleeding+,one tear superior to disc+**	UA-OD-6/12 PH-6/9 OS-CF1M PH-NI	UA-OD-6/12 PH-6/9 OS-CF2M	OD-18.8 mmhgOS-17.2 mmhg	OD-18 mmhg.OS-20mmhg	**OD-Cornea clear,No NVI,mild lens changes+.OS-Cornea-clear,1 bubble of silicone in A.C present,and patient was posted for silicone oil removal with reformation of anterior chamber on 13.3.2020**	OD-cornea-clear,No NVI,mild lens changes+,OS-cornea-clear,AC is normal,lens changes+.	OD-Glow-present,Media-clear,DISC-NORMAL,C.D-0.2,HAGES+,FVM +superiorly-1QUADRANT,Inferior laser marks present **OS-Siicone full,DISC-NORMAL,PRPC marks+,RETINA-FLAT.**	OD-FVM superior to superior arcade,RETINA-HAGES++,Laser marks inferiorly,incomplete PRPC.OS-Media-clear,DISC-NORMAL,MACULA-FLAT,laser marks seen,few HAGES seen,silicone full.	RETINA-FLAT	No	No	No	No	No	No	YES
29	Ravi gudadari	49yrs	Male	5869184	1026480	22.10.2020	30.10.2020	since 17yrs	Tab.Glycomet-GP1 500mg 1/2-0-1/2,Isophane insulin 40IU/ml 10u-0-0,Human actrapid 40IU/ml 10u-0-0	8.90%	No	No	CABG 1.5yr back,Metoprolol 1/2-0-1/2 ,betaloc 25,Rabeprazole,Ditor plus -10 1-0-0(Torsemide and spironolactone)	No	Clopilet A 75 0-1-0 ,Lipvas 20 0-0-1,Atorvastatin 20mg	DOV in LE since 15 days	UA-OD-6/36 PH--NI,OS- HMCF PL+VE PRACC	One sitting of laser done for RE on 19.10.2020	No	Within normal limits ,LENS-MILD CATARACT in both eyes.	OD-Media clear,There is FVM starting from the disc goes superiorly and nasally-3 QUADRANTS,Laser marks-inferiorly,There is FVM along inferior arcade with localized TRD-1 QUADRANT,few retinal Hages seen.OS-media-slightly Hazy,there is retinal deatchment along the ST arcade ,IT arcade and nasally-3QUADRANTS,Macula -off,there is **vitreous Hage inferiorly**,There is a hole nasal to disc,some part of temporal retina-flat,nasal to disc-some membrane is present -1 QUADRANT.in that hole we can see.	#######	LEFT EYE TRD+RRD+cataract	TRD+RRD	29.10.2020	BIMANUAL DISSECTION WITH SCHANDELIER LIGHT,**PHACO+PPV +MP**	No	YES	No	YES	**TEARS OCCURRED NASALLY AND INFEROTEMPORALLY**	UA-OD-CF 3M PH-6/60 OS- CF3M PH-6/60	UA-OD-CF2.5M PH-NI,OS-6/60 PH-6/36(p)	OD-11.4mmhg OS-7.9 mmhg	OD- 11.9 mmhg OS-14.9 mmhg	OD-Cornea clear,No NVI,cataract+,OS-AC-DEEP,PCIOL+.	OD-Cornea-clear,No NVI,Mild cataract,OS-cornea-clear,PCIOL+,PCO+.	OD-Media-Hazy,Tractional detachment, along superonasal,superotemporally,and along inferotemporal arcade,inferiorly laser marks seen,empty areas nasally.**OS-media-clear,DISC-NORMAL,sclerosed vessels nasally,MACULA-FLAT,RETINA-FLAT,Laser marks+,retina looks flat inferiorly,empty areas temporally.**	OD-Media-clear,DISC-slightly pale,RETINA-FLAT,some membrane ST arcade,fibrosis temporal to macula.OS- DISC-Slightly pale,MACULA-FLAT,peripheral fibrosis+,inferior fibrosis,PRPC marks+.	RETINA-FLAT	No	No	No	? Inferior periphery detachment	No	No	YES
30	Ravi gudadari	49yrs	Male	5869184	1030714	9.12.2020	11.12.2020	since 17yrs	Tab.Glycomet-GP1 500mg 1/2-0-1/2,Isophane insulin 40IU/ml 10u-0-0,Human actrapid 40IU/ml 10u-0-0	8.90%	No	No	CABG 1.5yr back,Metoprolol 1/2-0-1/2 ,betaloc 25,Rabeprazole,Ditor plus -10 1-0-0(Torsemide and spironolactone)	No	Clopilet A 75 0-1-0 ,Lipvas 20 0-0-1,Atorvastatin 20mg	DOV in RE since 1 month	UA-OD-6/24(p) PH-NI OS-6/60 PH- 6/36 with +4 DS	No	No	Within normal limits,Mild cataract in RE,PCIOLin LE.	OD-Media-clear,TRD along ST arcade,IT arcade,nasally,macula partially detached-4 QUADRANTS ,OS-Sclerosed vessels nasally,some fibrosis+,Macula flat,silicone oil full,RETINA-FLAT,some peripheral small detachment.	########	RIGHT EYE PDR WITH TRD	TRD	10.12.2020	YES	No	YES	No	YES	**MULTIPLE TEARS OCCURRED**	UA-OD--CF2.5M PH-NI,OS-6/60 PH-6/36(p) with +5D	UA-OD-CF2M PH-NI OS-6/60 With +5D PH NI.	OD- 11.9 mmhg OS-14.9 mmhg	OD-7.5 mmhg OS-11.5 mmhg	OD-Cornea-clear,No NVI,Mild cataract,OS-cornea-clear,PCIOL+,PCO+.	OD-Cornea clear,AC deep,PCIOL+.OS-cornea-clear,PCIOL+,PCO+.	**OD-Media-clear,DISC-slightly pale,RETINA-FLAT,some membrane ST arcade,fibrosis temporal to macula.** OS- DISC-Slightly pale,MACULA-FLAT,peripheral fibrosis+,inferior fibrosis,PRPC marks+.	OD-Sclerosed vessels+,Macula-flat,Hage above ST arcade,silicone full,laser marks+,RETINA-FLAT.OS-Media-clear,Membrane nasal to disc,MACULA-FLAT,RETINA-FLAT,Fibrosis inferiorly+.	RETNA-FLAT	No	No	**YES--RE PHACO+SILICONE OIL REMOVAL+MEMBRANE PEELING+RETINECTOMY+FAE+ENDOLASER+SILICONE OILINJECTION ON13.2.2021**	No	No	No	YES