Dissertation on

"AN OBSERVATIONAL STUDY TO DETERMINE THEASSOCIATION BETWEEN POSTERIOR VITREOUS DETACHMENT AND PROLIFERATIVE DIABETIC RETINOPATHY"

Submitted in partial fulfillment of requirements of

M.S. DEGREE

BRANCH –III (OPHTHALMOLOGY)

GOVT. RAJAJI HOSPITAL &

MADURAI MEDICAL COLLEGE

MADURAI

The Tamilnadu Dr.M.G.R. Medical University

CHENNAI, TAMILNADU

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CERTIFICATE

This is to certify that this dissertation entitled **"AN OBSERVATIONAL STUDY TO DETERMINE THE ASSOCIATION BETWEEN POSTERIOR VITREOUS DETACHMENT AND PROLIFERATIVE DIABETIC RETINOPATHY"** is the bonafide original work of Dr.HARSHINI G V, in partial fulfillment of the requirement for M.S.,(Branch III) Ophthalmology examination of the Tamilnadu Dr.M.G.R. Medical university to be held in May 2020.

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This is to certify that this dissertation work titled **"AN OBSERVATIONAL STUDY TO DETERMINE THE ASSOCIATION BETWEEN POSTERIOR VITREOUS DETACHMENT AND PROLIFERATIVE DIABETIC RETINOPATHY"** *of* the candidate **Dr. HARSHINI G V** with registration number 221713102, Post graduate Resident in Department of Ophthalmology, Madurai Medical College, Madurai, was done for the award of Master of Surgery Degree in the branch of Ophthalmology.

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This is to certify that this dissertation work titled **"AN OBSERVATIONAL STUDY TO DETERMINE THE ASSOCIATION BETWEEN POSTERIOR VITREOUS DETACHMENT AND PROLIFERATIVE DIABETIC RETINOPATHY"** of the candidate Dr. HARSHINI G V with registration number 221713102 was done for the award of Master of Surgery Degree in the branch of Ophthalmology. I personally verified www.urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contained from introduction to conclusion pages and result showed 13% plagiarism in the dissertation.

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I, **Dr. HARSHINI G V** hereby solemnly declare that, this dissertation titled **AN OBSERVATIONAL STUDY TO DETERMINE THE ASSOCIATION BETWEEN POSTERIOR VITREOUS DETACHMENT AND PROLIFERATIVE DIABETIC RETINOPATHY** was done by me.

I also declare that this bonafide work / a part of this work was not submitted by me / anyone else, for any award, for Degree / Diploma to any other University / Board either in India / abroad. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery Degree Branch -III (Ophthalmology) to be held in May 2020.

Place: Madurai (Dr.HARSHINI G V)

Date:

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VITREOUS

Vitreous chamber is filled with gel like vitreous body and occupies the largest portion of the globe. It is bounded anteriorly by posterior surface of the lens and the retro zonular portion of the posterior chamber. Peripherally and posteriorly, it is bounded by the pars plana of the ciliary body, the retina, and the optic disc. Anterior surface contains the patellar fossa in which the lens is present.

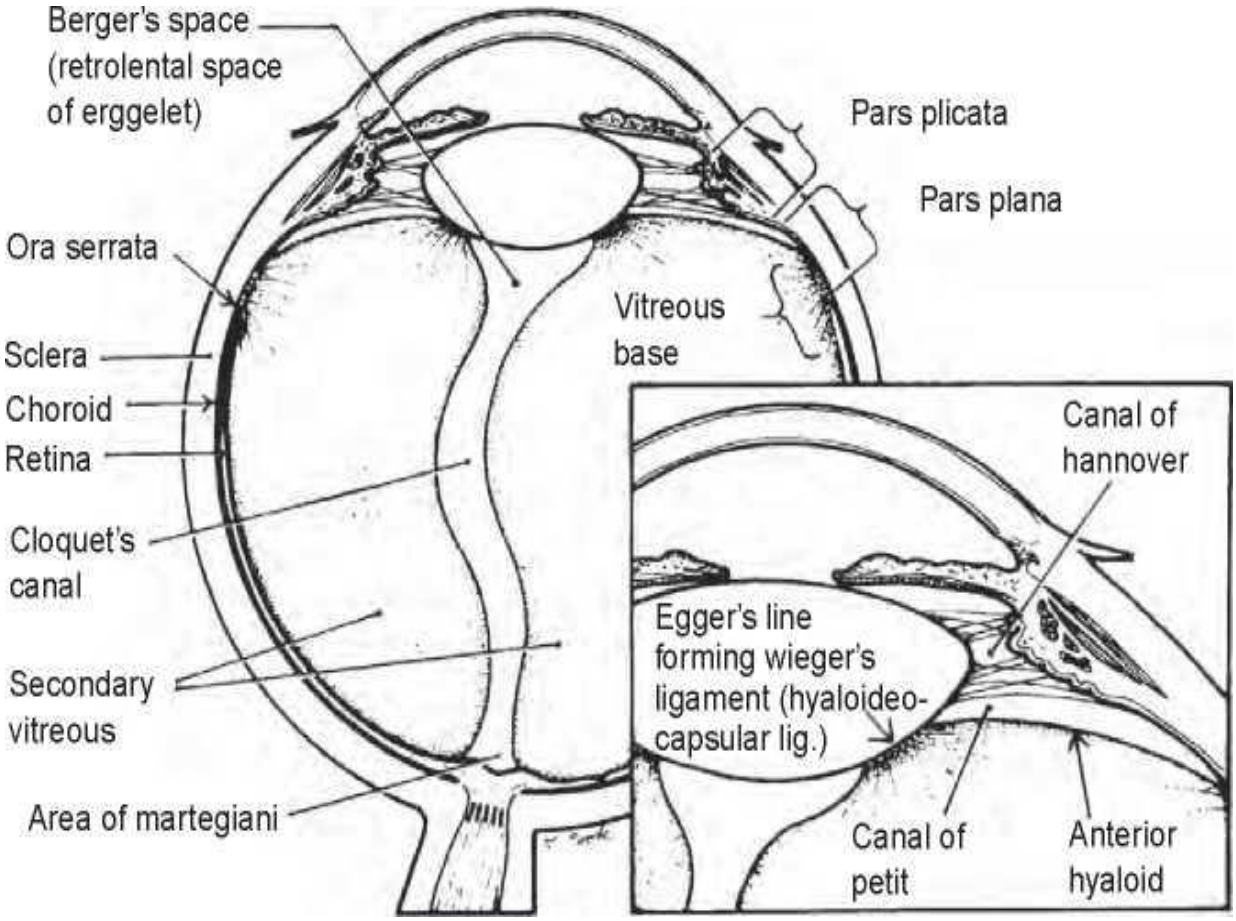
VITREAL ATTACHMENTS

Vitreous has several attachments to surrounding structures. The strongest is the vitreous base at the ora serrata. Other attachments are to the posterior lens, optic disc, macula, and retinal vessels.

Vitreous base, the most extensive adhesion, extends 1.5 to 2 mm anterior to the ora serrata, 1 to 3 mm posterior to it, and several millimetres into the vitreous.

The vitreal fibres that form the base are embedded firmly in the basement membrane of the non-pigmented epithelium of the ciliary body and the internal limiting membrane of the peripheral retina.

Hyaloideo capsular ligament of Wiegert otherwise called as **retrolental ligament,** forms an annular attachment 1 to 2 mm wide and 8 to 9 mm in diameter between the posterior surface of the lens and the anterior face of the vitreous. This is a firm attachment site in young , but the strength of the bond will diminish after third decade.



Ligament forms a potential space within the ring called as the **retrolental Space of Berger** which is present because the lens and vitreous are just juxta posed but are not attached. Peripapillary adhesion around the edge of the optic disc also diminishes with age.

The annular ring of attachment at the macula is 3 to 4 mm in diameter. The attachment of the vitreous to retinal blood vessels consists of fine strands that extend through the internal limiting membrane to branch and surround the larger retinal vessels.

These strands are responsible for the haemorrhages that occur when there is vitreous traction on the retina in case of posterior vitreous detachment. The nature of the attachment between the vitreous and the retinal internal limiting membrane throughout the rest of the retina remains unclear. The fibres from the posterior vitreous that attaches to the internal limiting membrane are uncertain in some other areas.

Vitreoretinal interface contains a molecular glue that links the outer part of the cortex and the inner part of the internal limiting membrane. This area contains extracellular matrix molecules, including laminin and fibronectin, that is having adhesive properties.

Vitreous Cortex

Vitreous cortex is the outer zone. It is 100 µm wide and composed of tightly packed collagen fibrils and some of which run parallel and some run perpendicular to the retinal surface.

Anterior cortex lies anterior to the base and has been adjacent to the ciliary body, posterior chamber, and lens.

Posterior cortex extends posterior to the base and is adjacent with the retina. It contains trans vitreal channels that appear as holes .

- prepapillary hole
- premacular hole
- prevascular fissures

- Prepapillary hole will be seen clinically when the posterior vitreous detaches from the retina.
- Premacular hole is less dense but is not an actual hole.
- Prevascular fissures provide the space by which fine fibres enter retina and surround retinal vessels.

Intermediate Zone

The **intermediate zone** contains fine fibres that are continuous and unbranched and run anteroposteriorly.

These fibres arise at the region of the vitreous base and insert into the posterior cortex.

The peripheral fibres parallel the cortex, whereas the more central fibres parallel Cloquet's canal. Membrane like condensations called as vitreous tracts may be differentiated as areas having differing fibre densities

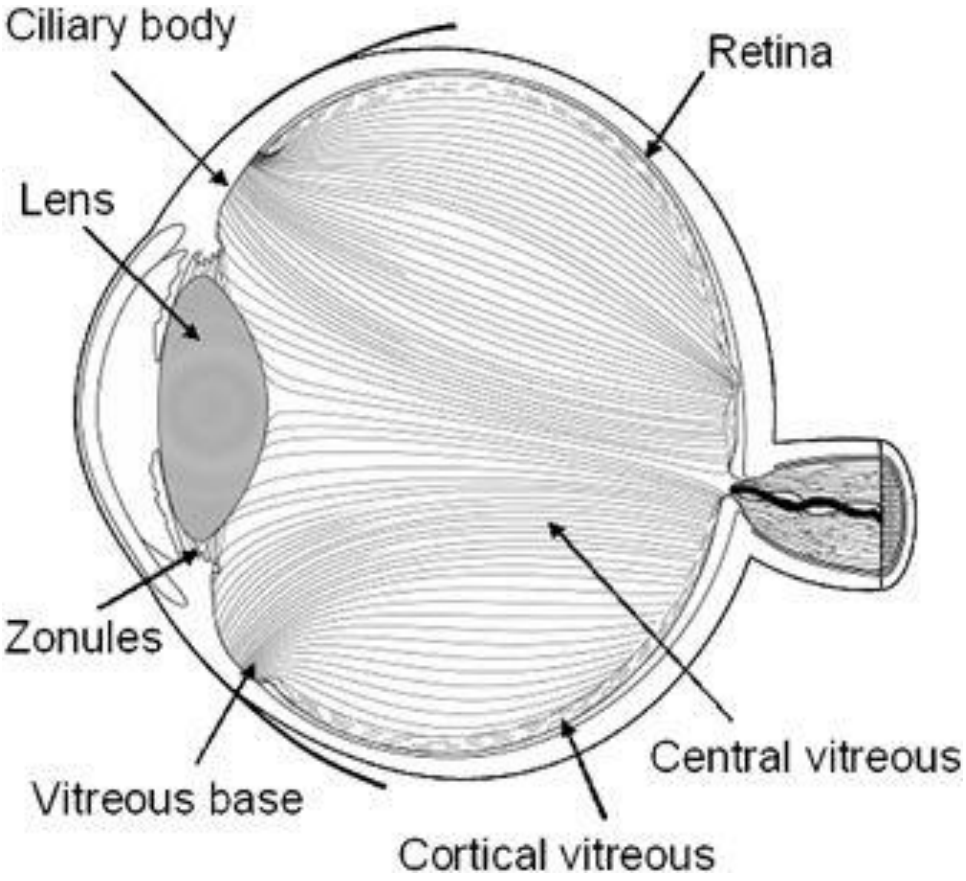
Cloquet's Canal

Cloquet's canal also called the **hyaloid channel** or the **retrolental tract** is located in the centre of the vitreous body.

It has an S shape with the centre facing downward, and is the former site of hyaloid artery system which was formed during embryologic development.

Cloquet's canal arises at the retrolental space. Its anterior face is approximately 4 to 5 mm in diameter.

It terminates at the **area of Martegiani** a funnel shaped space at the optic nerve head that extends forward into the vitreous to become continuous with the canal.



COMPOSITION OF VITREOUS

The transparent vitreous is a dilute solution of salts, soluble proteins, and hyaluronic acid contained within a meshwork of the insoluble protein, collagen.

Vitreous is 98.5% to 99.7% water and has been described as having connective tissue status and being an extracellular matrix.

Because of its high water content, study of the vitreous is difficult. Attempts at tissue fixation often have dehydrating effects that introduce artefacts.

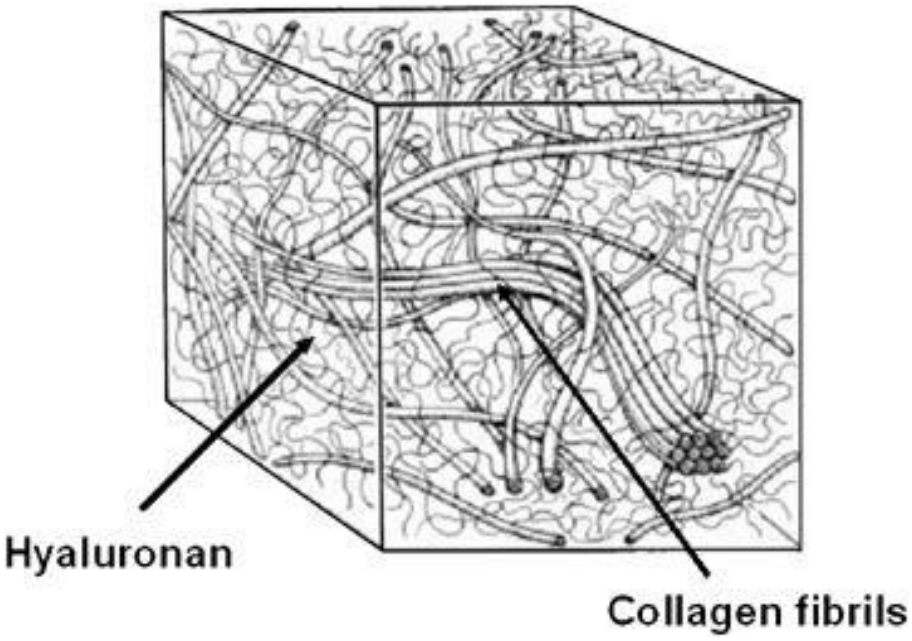
Recent investigations suggest that the epithelium of the pars plana has a significant role in the production and secretion of several connective tissue macromolecules of the vitreous body.

Collagen

The collagen content of the vitreous is highest in the vitreous base, next highest in the posterior cortex, next in the anterior cortex, and lowest in the centre.

A fine meshwork of uniform collagen fibrils, each 8 to 16 nm in diameter, is evident on electron microscopy and fills the vitreous body.

The individual fibrils cannot be seen with the slit lamp, but the pattern of variations in their density and regularity can be seen. The density of this collagen fibril network differs throughout the vitreous.



The insoluble proteins are present in the form of the fine fibres which are collagen in nature exhibit X ray diffraction pattern which will be degraded by collagenase.

Three different types of collagen type II, V, IX, XI assembled to form the fibrils

- Type II collagen - predominantly present around 90% arranged in staggered array pattern with lysine cross links.
- Type IX collagen – only around 10% located on the surface projects out antiparallel to type II collagen and it contains chondroitin sulphate chain.
- Type V and XI collagen – small amounts located close to the surface of the fibrils

Hyaluronic Acid

The second major vitreous component is the **hyaluronic acid(HA),** glycosaminoglycan, is a long unbranched linearly arranged molecule coiled into a twisted network forming sponge like spheroidal network.

This hydrophilic macromolecule is the mucopolysaccharide contains equal amount N-acetyl-glucosamine and glucuronic acid is located in specific sites within the collagen fibril network and is believed to maintain the wide spacing between fibrils.

The concentration of HA is highest in the posterior cortex and decreases centrally and anteriorly. This adds the property of viscosity to the elastic collagen

fibrils. HA intertwined with the collagen fibrils forms the water binding meshwork bind more than 50 times

HA stabilizes the network formed from **Hyalocytes**. Vitreous cells, or **hyalocytes,** are located in a single, widely spaced layer in the cortex near the vitreal surface and parallel to it.

Various functions have been attributed to these cells. It has been determined that these cells synthesize HA. Others have found evidence that hyalocytes synthesize glycoproteins for the collagen fibrils. Still others indicate that hyalocytes have phagocytic properties

Cells located in the vitreous base behave like the fibroblasts at the ora serrata and behind that it acts like the macrophages.

VITREOUS FUNCTION

The vitreous body provides physical support holding the retina in place next to the choroid, the blood supply for the outer retina. (Neural retina and choroid is only connected to each other at the disc and the ora serrata)

The vitreous is a storage area for metabolites for the retina and lens and provides an avenue for the movement of these substances within the eye. The vitreous, because of its viscoelastic properties, acts as a shock absorber protecting the fragile retinal tissue during rapid eye movements and strenuous physical activity.

The vitreous transmits and refracts light, aiding in focusing the rays on the retina. Minimal light scattering occurs in the vitreous because of its extremely low concentration of particles and the interfibrillar spacing ensured by the HA-collagen complex.

Ageing

The molecular basis of vitreous aging is not completely understood. Ageing process is gel liquefaction, is a common phenomenon noted in more than 60 percent of the eyes of persons between 80 to 89 years of age.

Between the ages of 45 and 50, a clinically detectable decline in the ratio of gel to liquid vitreous begins that continues through the tenth decade of life.

In vivo observations of vitreous morphology using ultrasonography reveal that these changes begin much earlier in life

During each decade of life, there is a further decrease in the proportion of gel to liquid. By 90 years of age, there is (on the average) more liquid volume to gel volume.

The transition from gel to liquefaction can be either abrupt or gradual, with a proportional decline in both collagen fibres and soluble vitreous found in the transitional regions.

The ageing vitreous shows rather rapid loss of type IX collagen, along with its chondroitin sulphate side chains. Although type IX collagen is a minor component of the insoluble matrix, it may serve an important role in protecting the surface of type II collagen from exposure.

It has been proposed that type II collagen may become less resistant to damage and more susceptible to dissolution following the loss of contiguous type IX collagen.

Despite the size of the cavity, the shell of residual vitreous remained intact, suggesting that size alone may not be a major factor in the development of posterior vitreous detachment.

Although the cause of age-related syneresis is uncertain, that photodegradation of hyaluronic acid contributes to the process.

This process may be mediated by light-induced, free-radical formation, which could damage vitreous collagen and hyaluronic acid over time.

PHYSIOLOGY OF VITREOUS

The vitreous was thought to merely passively interact and support surrounding tissues but a new understanding of the dynamic vitreous is developing. The cells in the cortex remain largely quiescent because factors present in the vitreous prevent cell migration and proliferation.

The interaction between HA and collagen fibrils contributes to the viscoelastic properties of the vitreous and influences the physical properties, i.e., the balance between gel and liquid, of the vitreous state.

Most of the water in the vitreous is bound in the widely-spaced network of collagen and HA.

A disruption in the HA- collagen complex can cause the collagen fibrils to aggregate into bundles, which may become large enough to be visible clinically, and reported by a patient as floaters.

While there is little metabolic activity within the vitreous and a slow degradation occurs with age, an intact vitreous gel may be quite important to ocular health; age-related degeneration of the vitreous gel and liquefaction accompanies several age-related ocular diseases, such as nuclear sclerotic cataract and neovascular diabetic retinopathy.

DETACHMENT OF THE POSTERIOR VITREOUS

Posterior vitreous detachment is defined as the separation of the cortical vitreous from the attachment of the internal limiting membrane from the retina either completely or partially with its attachment to the disc.

A variety of different conditions, from trauma to inflammation, will cause the separation of the vitreous from its posterior attachments to the surface of the retina and optic nerve.

The most common underlying cause in the general population is ageing and age related syneresis of the vitreous.

But can be occurred in conditions such as trauma, uveitis, cataract surgery, pan retinal photocoagulation.

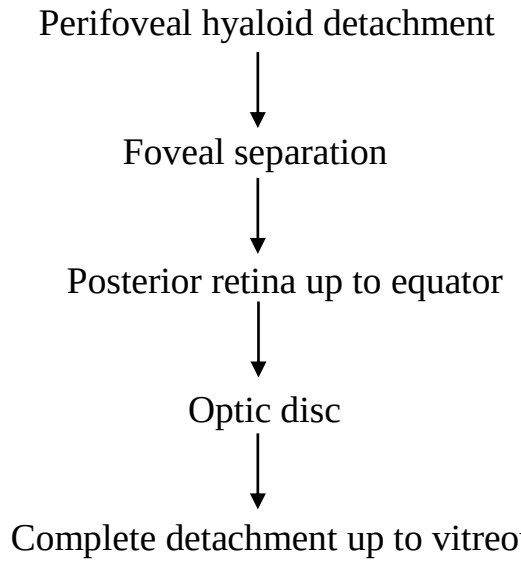
Like syneresis alone, spontaneous posterior vitreous detachment (PVD) is an age-related phenomenon whose fundamental biomechanisms still await. The prevalence of PVD increases both with advancing age and axial length of the eye.

As the aging occurs there is vitreous gel liquefaction to form the fluid filled cavities which is referred to as synchisis and followed by condensation of liquid referred to as syneresis.

The abrupt separation of the posterior vitreous face from the retina and optic nerve head likely occurs when a rush of liquefied vitreous pours through a rent in the cortical vitreous.

Much like a small hole, the watery vitreous rushes through the space dissecting the remaining posterior vitreous surface from the underlying retina and optic nerve.

It follows the following pattern .

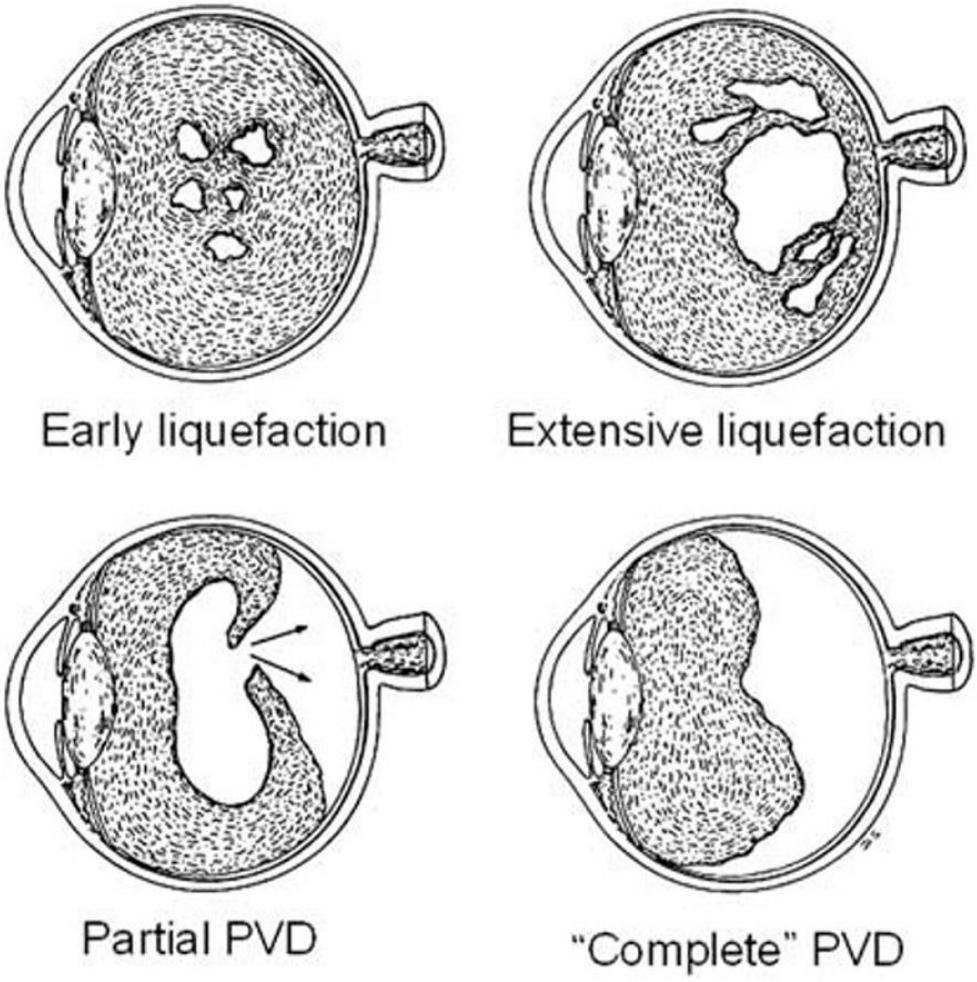


The force generated by the flow of fluid is likely great enough to break any vitreous attachments to the retina or to superficial blood vessels.

The process of separation appears to begin in the macula, where the concentration of insoluble and soluble vitreous components is the least.

The posterior wall of this pocket consisted of a thin layer of vitreous lying just anterior to the retina. Its anterior wall was the formed vitreous gel, which in some eyes mingled with lacunae of synergetic fluid.

A minority of spontaneous PVDs appear to present with the sudden onset of flashing lights and floaters. This discrepancy may be due to the fact that the process of vitreous separation from the retina probably occurs slowly in many patients where it goes unrecognized

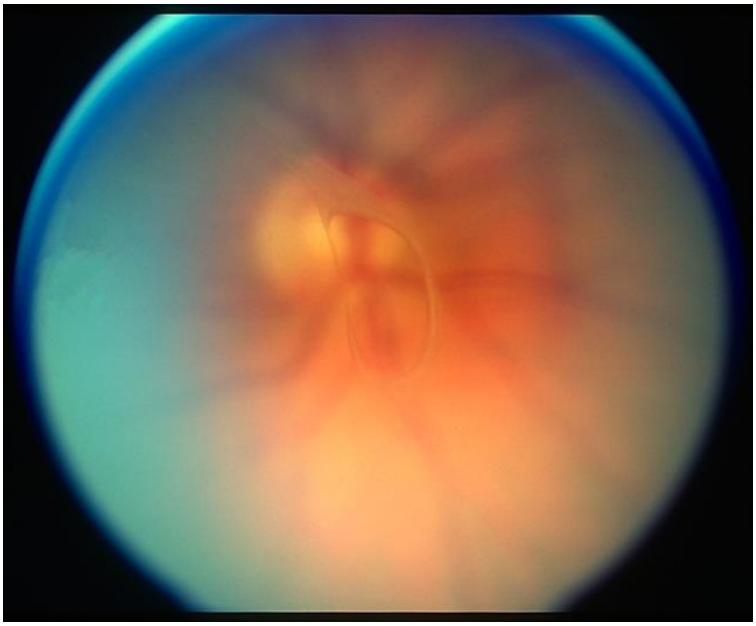


Patient usually is asymptomatic but there will be

Flashes of lights- photopsia on eye movements more in the dim illumination, seen in the temporal periphery based on the mechanism that the force of attachment on the optic disc and at the vitreoretinal adhesion.

Floaters due to the mobile vitreous opacities on bright background and may be also due to the vitreous blood

A Weiss ring is the detached posterior vitreous from the optic disc and seen by the patient as a circle or large single lesion but its presence does not tell that it is complete PVD



Visual acuity will be reduced if there is associated with vitreous haemorrhage which occurs in the proliferative diabetic retinopathy in which on detachment the new vessels will be torn.

CLINICAL FEATURES

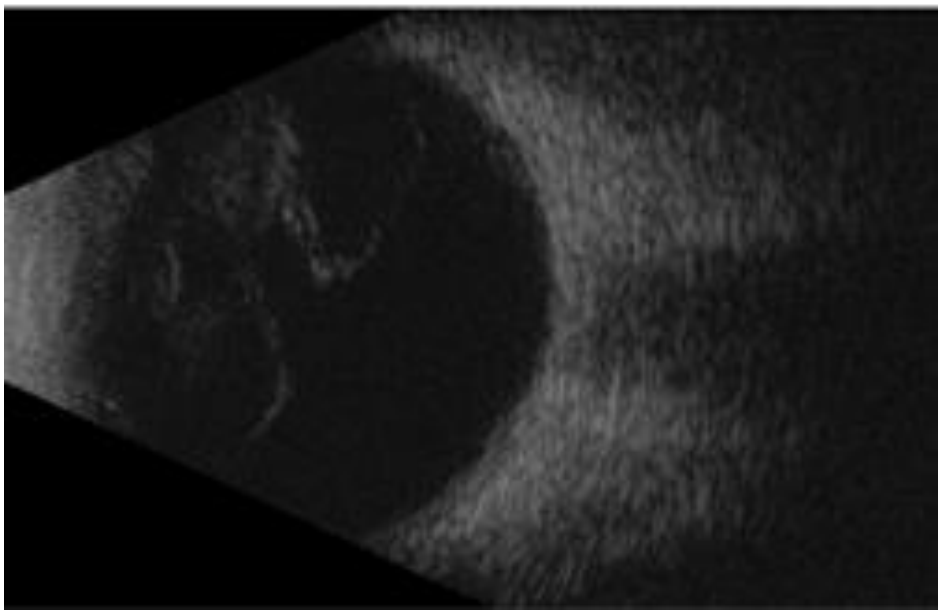
- Collapsed vitreous behind the lens
- Optically clear space between the detached posterior hyaloid phase and the retina
- Pathognomic sign is the Weiss ring

B SCAN

B scan is an important investigation to differentiate the grades of PVD. Based on that the clinical importance lies.

It is seen as an undulating membrane that moves freely and should move away from the optic disc in case of complete posterior vitreous detachment.

Its configuration of attachment to the macula or disc should be noted and it is very important in PDR for their prognostic value and also complete collapse following detachment can be easily identified.



- Shape of the detached membrane is fine smooth folded membrane and if it is associated with the neovascularisation of disc and elsewhere it will be seen as thick membrane
- Amplitude of the echo is less than 100%
- On reducing the gain, it disappears
- Dynamic ultrasonography the after movements are good
- On Doppler usually it is avascular but in case of fibrovascular proliferation it will have new vessels and attached membrane so they may have active vascularity

RETINA

The innermost coat of the eye is a neural layer, the retina, located between the choroid and the vitreous. It includes the macula, the area at the posterior pole used for sharpest acuity and colour vision.

The retina extends from the circular edge of the optic disc, where the nerve fibres exit the eye, to the ora serrata. It is continuous with the epithelial layers of the ciliary body; with which it shares embryologic origin.

The retina is derived from neural ectoderm and consists of an outer pigmented layer, derived from the outer layer of the optic cup, and the neural retina, derived from the inner layer of the optic cup.

The pigmented layer is tightly adherent to the choroid throughout, but

the neural retina is attached to the pigmented epithelium and thus to the choroid only in a peripapillary ring around the disc and at the ora serrata.

Although it contains millions of cell bodies and their processes, the neural retina has the appearance of a thin, transparent membrane. The retina is the site of transformation of light energy into a neural signal.

It contains the first three cells (photoreceptor, bipolar, and ganglion) in the visual pathway, the route by which visual information from the environment reaches the brain for interpretation.

Photoreceptor cells transform photons of light into a neural signal through the process of phototransduction, then transfer this signal to bipolar cells, which in turn synapse with ganglion cells, which transmit the signal from the eye. The “10-layered” arrangement of the retina is actually a remarkable organization of alternate groupings of the retinal neurons just described and their processes.

1. Retinal pigment epithelium
2. Photoreceptor cell layer
3. External limiting membrane
4. Outer nuclear layer
5. Outer plexiform layer
6. Inner nuclear layer
7. Inner plexiform layer
8. Ganglion cell layer
9. Nerve fibre layer

10. Internal limiting membrane

RETINAL PIGMENT EPITHELIUM

The **RPE** consists of a single layer of pigmented cells. There are 4 to 6 million RPE cells, and each cell interacts with 30 to 40 photoreceptors. There is little cell division in the layer.

EXTERNAL LIMITING MEMBRANE

The **external limiting membrane (ELM, outer limiting membrane)** is not a true membrane but is actually composed of zonula adherens junctions between photoreceptors and Müller cells at the level of the inner segments. On light microscopy, the so-called membrane appears as a series of dashes, resembling a fenestrated sheet through which processes of the rods and cones pass. This band of zonula adherens has the potential to act as a metabolic barrier restricting the passage of some large molecules.

OUTER NUCLEAR LAYER

The **outer nuclear layer (ONL)** contains the rod and cone cell bodies; the cone cell body and nucleus are larger than those of the rod. Cone outer fibres are very short, and therefore the cone nuclei lie in a single layer close to the external limiting membrane; cell bodies of the rods are arranged in several rows inner to the cone cell bodies. The ONL is 8 to 9 cells thick on the nasal edge of the optic disc and 4 rows thick at the temporal edge and is thickest in the fovea,where it contains approximately 10 layers of cone nuclei.

OUTER PLEXIFORM LAYER

The **outer plexiform layer (OPL** also **outer synaptic layer)** has a wide external band composed of inner fibres of rods and cones and a narrower inner band consisting of synapses between photoreceptor cells and from the inner nuclear layer. Rod spherules and cone pedicles synapse with bipolar cell dendrites and horizontal cell processes in the OPL. Many of these synapses consist of invaginations in the photoreceptor terminal; invaginations are deep in the spherule but more superficial in the pedicle.

INNER NUCLEAR LAYER

The **inner nuclear layer (INL)** consists of the cell bodies of horizontal cells, bipolar cells, amacrine cells, interplexiform neurons, Müller cells, and sometimes displaced ganglion cells. The nuclei of the horizontal cells are located next to the outer plexiform layer, where their processes synapse. The nuclei of the amacrine cells are located next to the inner plexiform layer, where their processes terminate.

INNER PLEXIFORM LAYER

The **inner plexiform layer (IPL** also **inner synaptic layer)** consists of synapses between the axons of first order neuron bipolar cells, dendrites of ganglion cells and the processes of integrative amacrine cells. The IPL contains the synapse between the second-order and third-order neuron in the visual pathway.

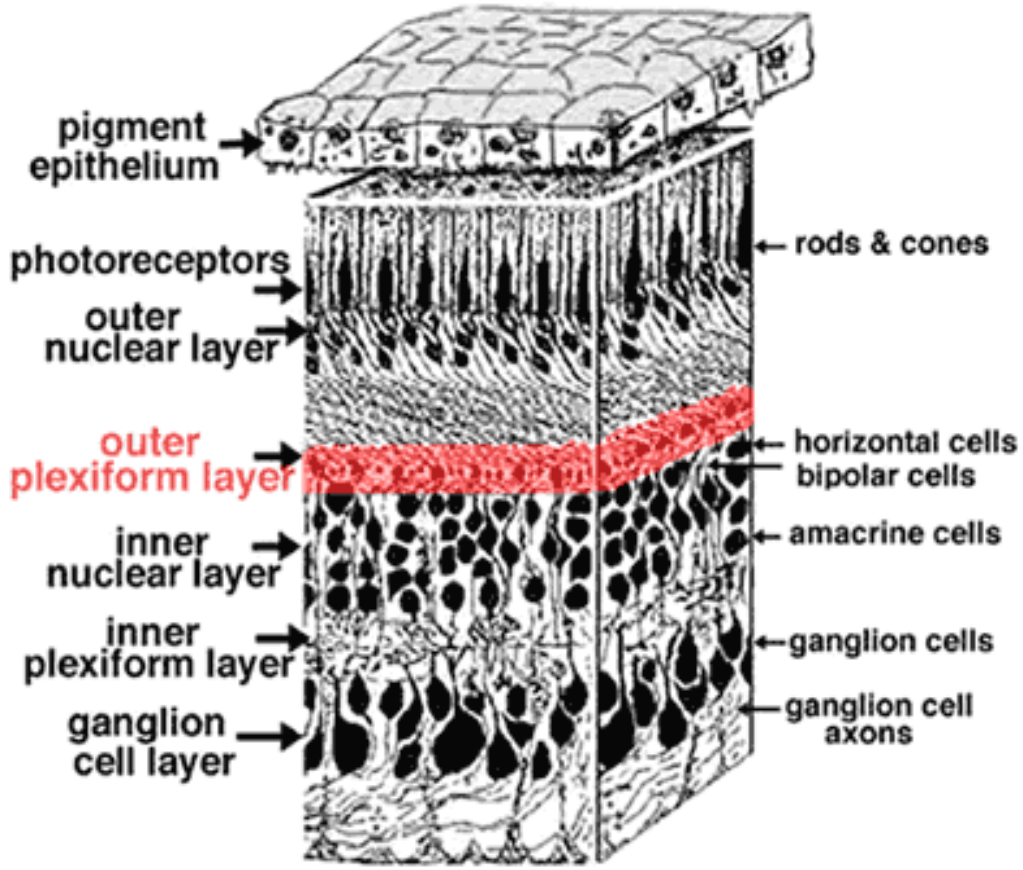
Synapses also occur between (1) amacrine processes and bipolar axons, (2) amacrine processes and ganglion cell bodies and dendrites, (3) amacrine cells, and (4) amacrine cells and interplexiform neurons

GANGLION CELL LAYER

The **ganglion cell layer** is generally a single cell thick except near the macula, where it might be 8 to 10 cells thick, and at the temporal side of the optic disc, where it is 2 cells thick. Although lying side by side, ganglion cells are separated from each other by glial processes of Müller cells. Displaced amacrine cells, which send their processes outward, may be found in the ganglion cell layer. Toward the ora serrata, the number of ganglion cells diminishes, and the nerve fibre layer thins.

INTERNAL LIMITING MEMBRANE

The **internal limiting membrane (inner limiting membrane)** forms the inner most boundary of the retina. The outer retinal surface of this membrane is uneven and is composed of extensive, expanded terminations of Müller cells often called footplates covered by a basement membrane. The inner or vitreal surface is smooth. The connection between this membrane and the vitreous is still under investigation and may actually occur at a biochemical level only in the periphery are vitreal fibres incorporated into the internal limiting membrane.



BLOOD-RETINAL BARRIER

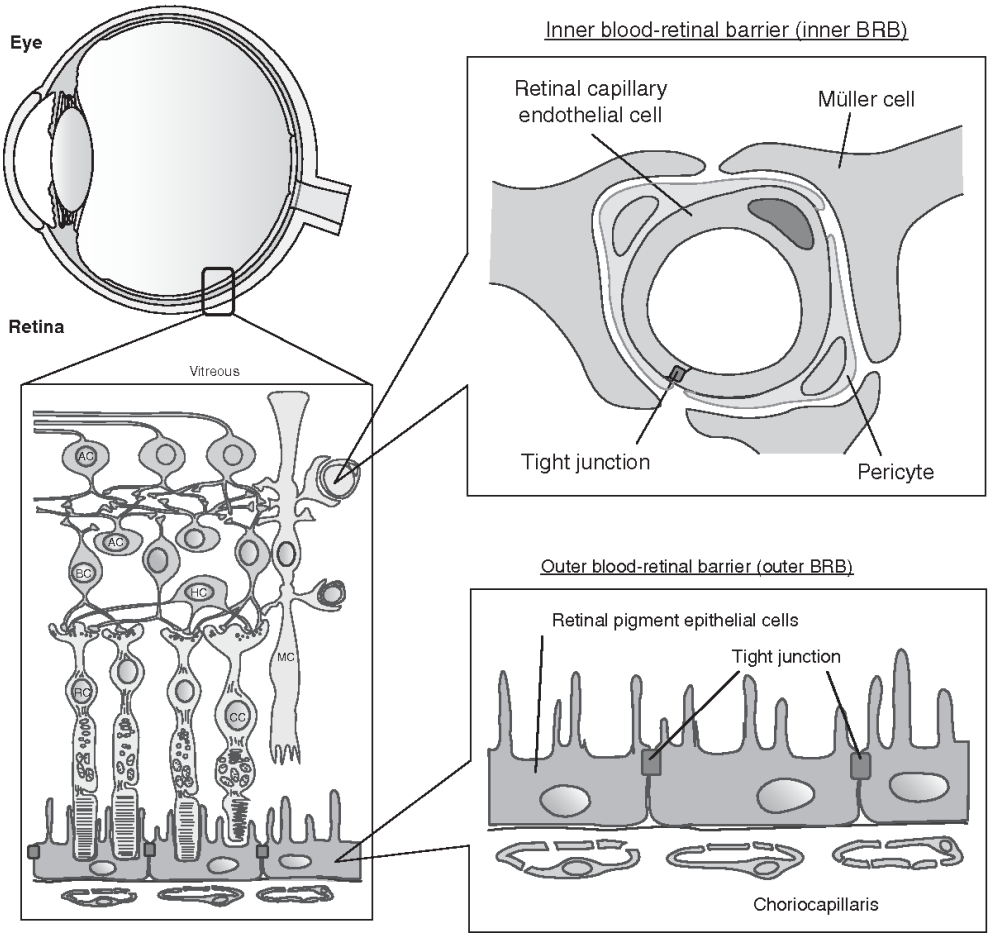
The **blood-retinal barrier** prevents components of blood plasma that might impede light from entering retinal tissue.

There are several factors to consider in the function of this barrier:

(1) the choriocapillaries is fenestrated allowing large molecules to exit into choroidal tissue; these molecules can usually pass through Bruch's membrane easily;

(2) the zonula occludens junctions joining the RPE cells prevent such molecules from moving into retinal tissue; and

(3) the retinal capillaries are not fenestrated and their endothelium contains zonula occludens that prevent large molecule exit from retinal vessels.



DIABETIC RETINOPATHY

Diabetes is the leading cause of blindness in the world in the age group of 30 -60 years. Earlier detection and adequate control of the diabetes will prevent further complications. So follow up of the patient is necessary for providing good vision.

PATHOGENESIS

Caused by structural changes of the vessels and rheological changes inside the vessels

STRUCTURAL ABNORMALITY

- Loss of pericytes
- Thickening of the basement membrane
- Endothelial cell loss
- Dysfunction of the endothelium

Increased glycocalyx

Increased adhesion of leucocytes due to increased PECAM

Structural changes result in the abnormal autoregulatory mechanism and leakage from capillaries into the extracellular space

RHEOLOGICAL CHANGES

Effect of hyperglycaemia on the plasma, RBC and platelets causes these changes

Increase in fibrinogen, alpha 2 globulins and decrease in serum albumin

↓

Decrease in fibrinolysis and viscosity is increased

Alteration of platelets and RBC

↓

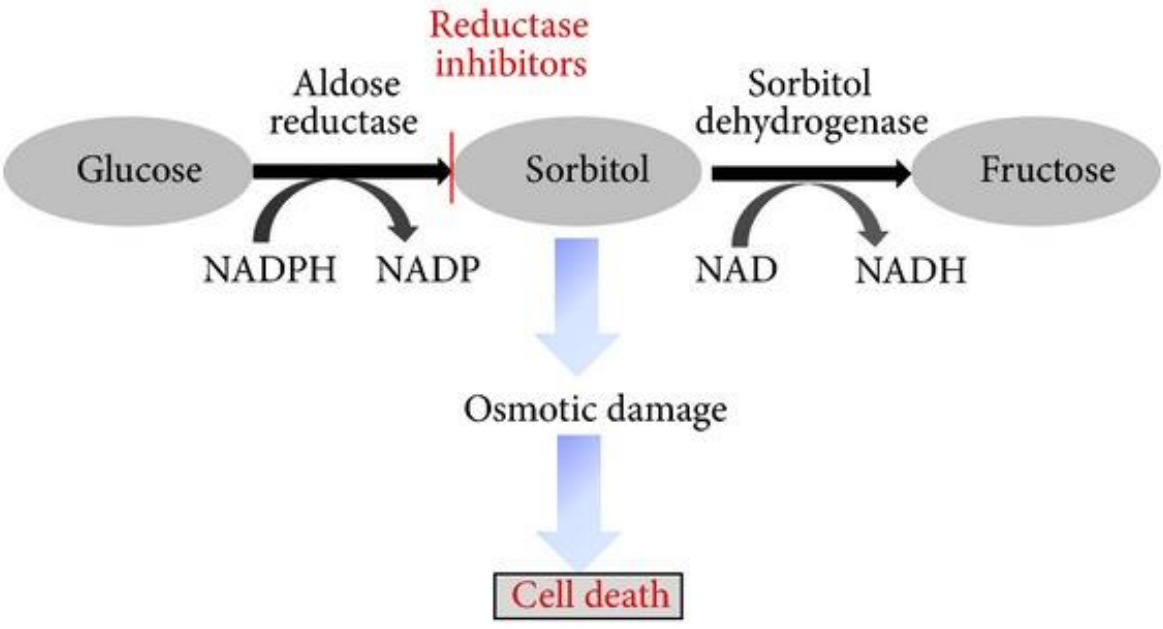
Tendency for aggregation and increased deformability

↓

Thrombus formation and occlusion of the capillary walls

BIOCHEMICAL CHANGES

POLYOL PATHWAY



ADVANCED GLYCATION END PRODUCTS

Hyperglycaemia

↓

Increased glycolysis and 3 diacyl glycerol

Advanced glycation end products are toxic to pericytes and endothelial cells

PROTEIN KINASE C

Increased 3 diacyl glycerol

↓

Increased protein kinase C activation

↓

Increased pro inflammatory mediators (cytokines, matrix metalloproteinase 2)

↓

Increased vascular permeability, endothelial dysfunction and

VEGF expression

These changes together causes the structural modification of the normal veins and leads to hypoxia and thereafter progression into the stages of the proliferative diabetic retinopathy in which the vision loss will be irreversible.

In diabetic patients in addition to the retinal changes vitreous changes also occurs and they have change in the collagen and extra celluar change in vitreous leads to vitreous detachment also.

CLINICAL FEATURES

- Retinal Microaneurysms
- Intraretinal hemorrhages
- Hard exudates
- Cotton wool spots
- Retinal edema
- Venous changes
- Intraretinal microvascular abnormalities (IRMAs)
- Flat neovascularization of disc
- Forward new disc vessels
- Preretinal new vessels elsewhere
- Fibrous band
- RPE appearance changes
- White blood vessels
- Tractional retinal detachment
- Vitreous hemorrhage
- Rubeosis iridis

RETINAL MICROANEURYSM

They are localized saccular outpouchings of the capillary wall, often caused by pericyte loss; continuous with the blood vessels.

They appear as red round intraretinal lesions of 10–120 μ in size, located in the inner nuclear layer of the retina.

Clinically they are indistinguishable from dot haemorrhages.

FFA reveals hyperfluorescence. They are saccular outpouchings of the capillary wall at the weak points due to loss of pericytes.

HARD EXUDATES

Caused by chronic localized retinal oedema leaking from the and appear at the junction of the normal and oedematous retina.

They are composed of lipoproteins and lipid filled macrophages and are located mainly in the outer plexiform layer of the retina.

FFA shows hypofluorescence.

HEMORRHAGES

These haemorrhages occur at the nerve fibre layer and they are flame shaped since they follow the architecture of the nerve fibre layer. They arise from superficial precapillary arterioles.

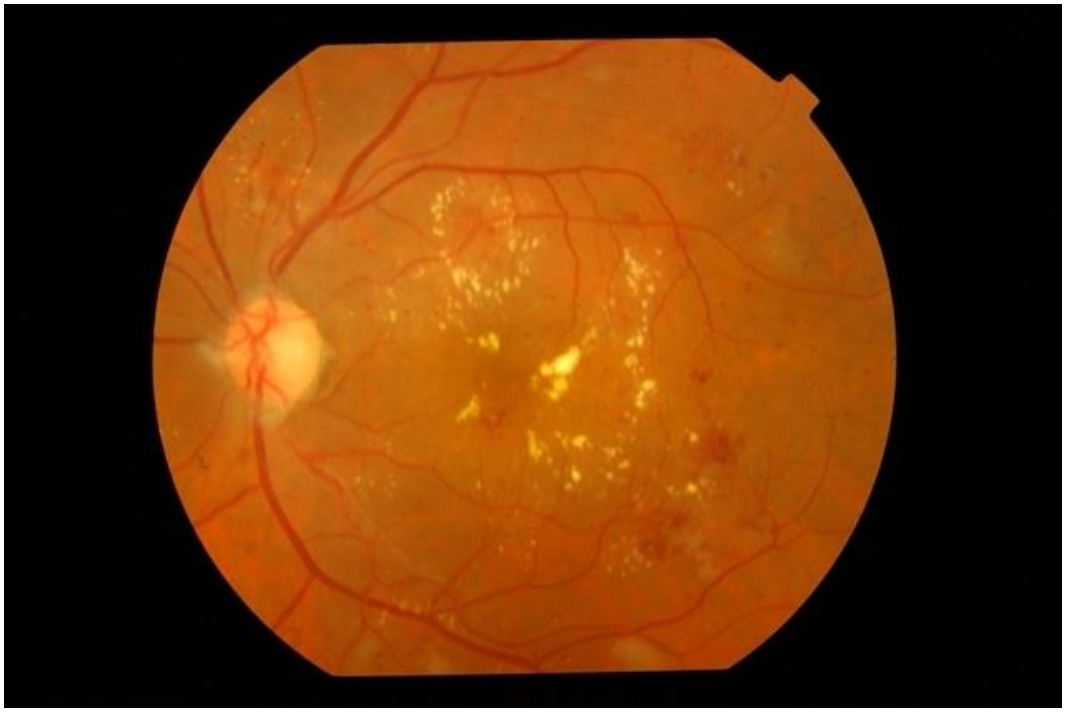
COTTON WOOL SPOTS

Cotton wool spots are due to the ischemic infarction of the nerve fibre layer. Because of ischemia, interruption of axoplasmic flow happens and build up transported material within axons occurs.

INTRARETINAL MICROVASCULAR ABNORMALITY

Intraretinal microvascular abnormality (IRMA) is frequently seen adjacent to capillary closure and they resemble focal areas of flat retinal new vessels clinically.

These are arteriovenous shunts that run from arterioles to venules. IRMA indicates severe NPDR and may herald the onset of the preproliferative stage of diabetic retinopathy.



EARLY STAGE OF NEW VESSELS

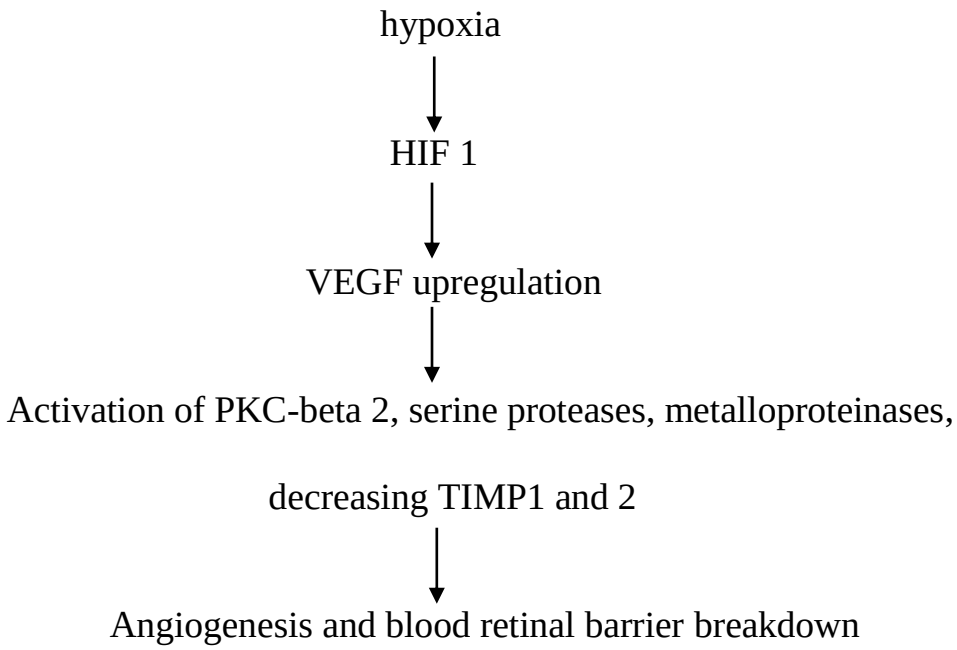
After the closure of the retinal capillaries there is ischemia of the retina which leads to the VEGF expression and causes proliferation of the endothelium and therefore new vessels formation occurs.

VEGF factors not only acts on the retina but due to the capillary permeability and diffuses into vitreous and goes to the other parts of retina causing neovascularisation elsewhere, optic disc causing neovascularisation of the optic disc and through the vitreous even it goes to the anterior chamber and causing iris neovascularisation which leads to secondary open angle and sometimes angle closure neovascular glaucoma.

Risk for the development of the proliferative diabetic retinopathy will be more in eyes with some clinical features if their presence is there the eye will go for proliferative state.

- Cotton wool spots,
- Venous beading,
- Intra retinal microvascular anomalies,
- Dot blot retinal haemorrhages and MA in 4 quadrants more than 20 in each quadrant.

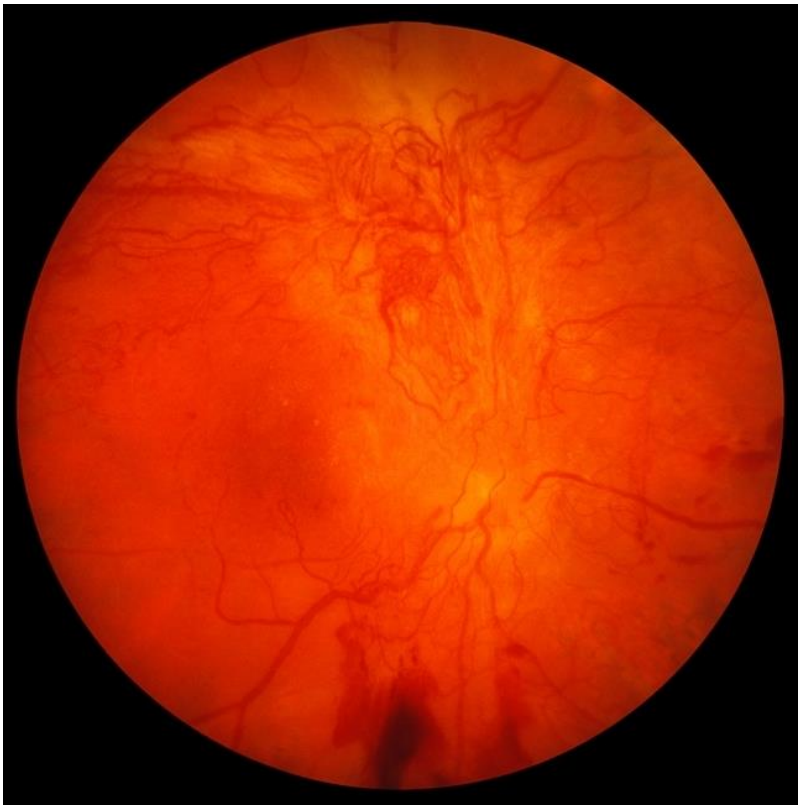
VEGF plays a major role in the new vessels formation. Mechanism behind this is well understood for the further treatment of proliferative diabetic retinopathy



Patients with the very severe NPDR stage will go for proliferative stage within few years if there is inadequate blood glucose control and once they reach the proliferative stage the lesions such as cotton wool spots disappears and haemorrhages and IRMA tend to disappear as there is complete closure of the capillaries and small vascular branches appears as sclerosed vessel giving the appearance of the featureless retina

Usually the new vessels start from the area of 5 DD from within the disc area and neovascularisation of elsewhere will be present outside this area. Careful examination should be done to pick up the neovascularisation of elsewhere so indirect ophthalmoscopy along with stereographic fundus photography should be done. In the early stages it is difficult to differentiate between the IRMA and new vessels without any complication in the fundus. Careful examination of the calibre, their crossing over the major veins and arteries should be noted. Additional finding an be get from fundus fluorescene angiography in which

IRMA will not leak and only the tip leaks in the late stage but there will be early hyperfluorescence and leakage present in the new vessels.



NEW VESSELS FORMATION

At the earlier stage the new vessels are difficult to appreciate and are barely seen but if they are present their size is smaller than the retinal vein as like the one which is branching from.

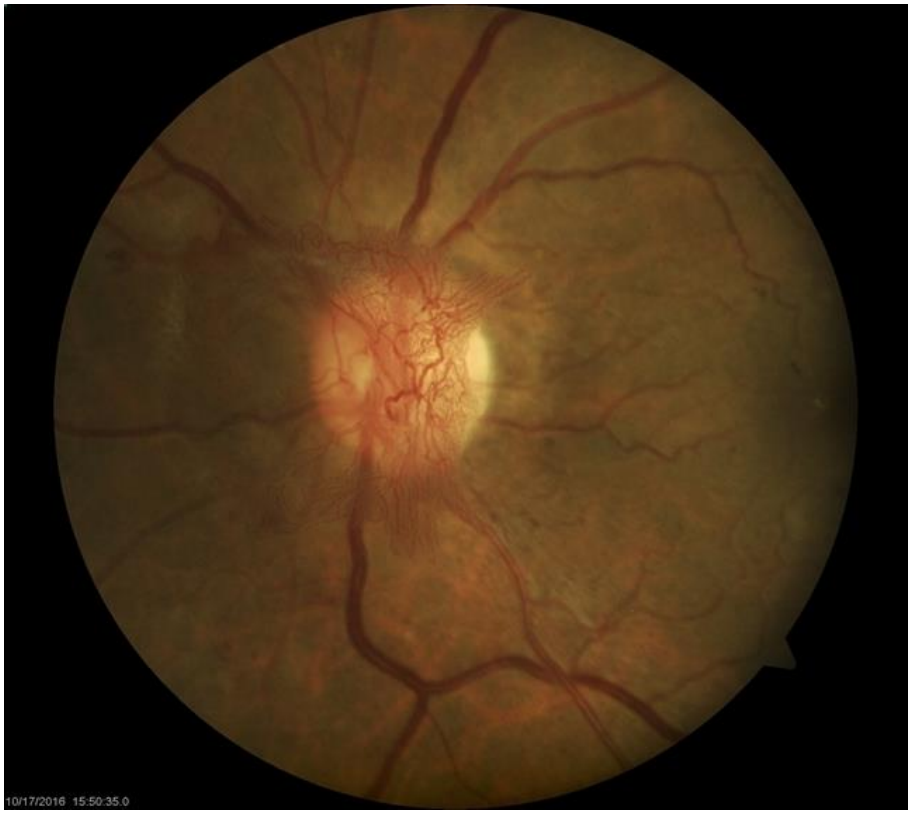
New vessels are always clustered and they usually form the spokes pattern.

New vessels grow like bush with the clumped centre and fan like surrounding the new vessel like flower pattern.

New vessels usually cross the major retinal veins and arteries and flows to the veins. Always they are not having any definite shape. Supero temporal vein is most commonly involved than others.

New vessels sometimes grow for more distance in the retina without any irregular pattern and they will look like regular one but they will cross the major vessels and easily identified by the fundus fluorescence angiography.

New vessels most commonly originate over the disc and are often associated with the retinal oedema, from mild to moderate grade in and around the disc during their growing stage0. This appearance gives the same picture of diabetic papillopathy.



But in diabetic papillopathy the vessels are dilated and are present intraretinally around the disc and does not leak on fluorescein angiography.

In diabetic papillopathy the disc oedema and the vessels in the disc regress but in diabetic retinopathy this will not occur

Rate of growth of new vessels has been always variable. In minority of patients, vessels may show little change over many days to months, but in some patients it shows the most rapid and flourishing pattern which is seen within 1 or 3 weeks.

First in the evolution of new vessels they will be bare without any adjacent changes, but after sometimes white fibrous tissue usually becomes visible over or adjacent to them and on histology it consists of fibroblast and gliosis.

New vessels undergo phase of proliferation and persists for sometimes and then may or may not enter the regression phase either fully or incompletely.

New vessels undergo regression with their first finding of reduction in their size and count at the interior of the vessel. Complete fibrous tissue or incompletely they take the place of the new vessels undergoing the regression.

In the path of above mechanism in addition to that, the peripheral vessels will reduce in their size but they also increase in their size and involve the surrounding area and spread further.

Sometimes, regressing new vessels may be associated with sheathing. The literal meaning of sheathing is nothing but it represents the opacification and

thickening of the vessel walls which enlarges till blood column disappears and they appear as the opaque vessels.

Sometimes there is preferential vessels present which supplies the area adjacent to it so the nearby vessels can regress and disappear. At the edges of the partial regressed new vessels emerges the new fresh new vessels and at the different sites of retina they will be presenting with different stages in the same eye.

At the early stages of the evolution of the fibrous proliferations they tend to be translucent without any associated features and always they are undetectable and underestimated.

On further progression with the increasing growth there will be contraction of the fibrous proliferation and may be separated from the retina and become more prominent.

If the vitreous does not contracts and there is no fibrous tissue proliferation along with new vessels, the forms new vessels will not proliferate and they pass into the quiescent stage with no complications and any vision loss.

Consecutive decrease in calibre of retinal vessels and intraretinal lesion takes place once the diabetic retinopathy enters the burned out stage. Sometimes the new vessels completely regress without any evidence of their presence.

POSTERIOR VITREOUS DETACHMENT AND CONSECUTIVE CONTRACTION WITH FIBROUS TISSUE PROLIFERATION

On biomicroscopic examination of the retina the new vessels appear to be elevated and on over the retina before the occurrence of the posterior vitreous detachment.

If the new vessels are examined now they are seen as to be raised from the surrounding retina but the vitreous present near to the new vessel is perceived as having no changes and the new vessels and the retina are opposed to each other and the raised appearance of the new vessel will be sometimes seen as the retina is higher than the surrounding area.

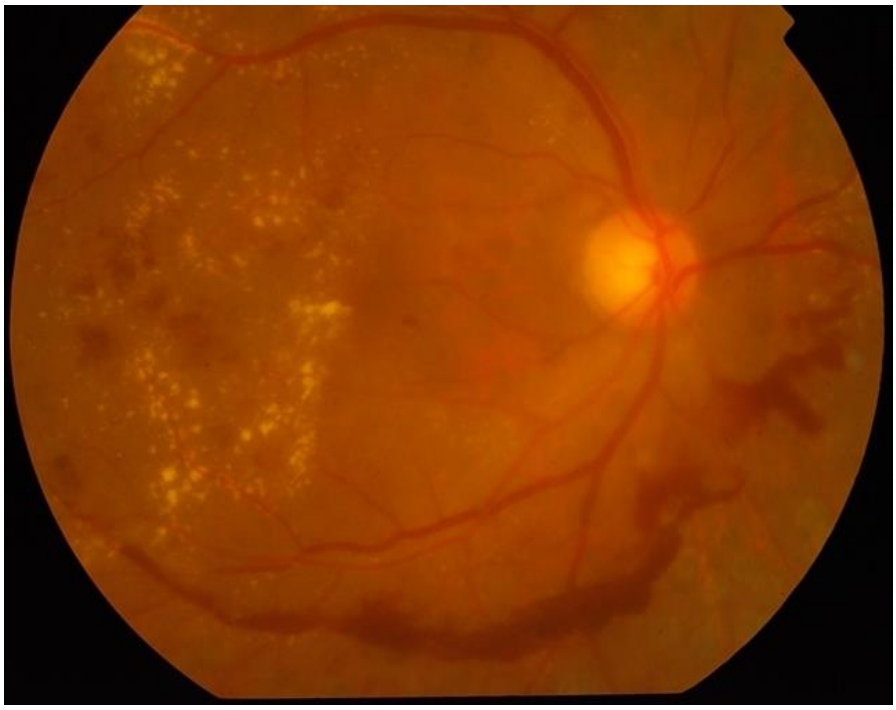
The new vessels are strongly adherent to the retina even though the centre of the new vessel appear to be raised from the surface shows the new vessel surface is slightly curved and dome shaped. new vessels in this area are also anchored to the surface of the posterior vitreous.

This adhesion becomes more prominent when posterior vitreous detachment occurs adjacent to the new vessel, pulling its edge forward. If vitreous detachment occurs around the patch of new vessel, all its edges become more elevated than its centre.

There will not be any symptoms to the patient and new vessels will be silent till the posterior vitreous detaches.

Initially there will be only streak shaped haemorrhages in the posterior vitreous at the edges of the new vessels and they collected in the gravity force

within the detached area of vitreous giving the boat shaped haemorrhage and does not affect the vision of the patient. If the vitreous haemorrhage has been identified it is usually associated with that area of posterior vitreous detachment but there is vitreous attachment present near to it.



When there is localised posterior vitreous detachment it will be flat and very close to the retina without any elevation but if the posterior vitreous detachment is more extensive the surface moves forward and give the appearance of curved contour most commonly parallel to the retina about 0.5 to 2 DD anterior to it.

If the new vessels present, there is vitreoretinal adhesion present at the site. After the posterior vitreous detachment, the new vessels are pulled forward wherever there is vitreoretinal adhesion present.

Floaters and strands of the vitreous are present in the centre of the vitreous in the medullary area while the haemorrhages and old red blood cells are present in between the detached posterior vitreous and the retina.

The principal force acting on the posterior vitreous pulls the surface forward by forward vector force resulting from contraction of the surface and the fibrovascular proliferations grows along it.

Posterior vitreous will be seen as different contour in different surfaces near the new vessels it is condensed and is identified without any difficulty because the fibrous tissue has proliferated around it and appears opacified.

In adjacent areas away from the new vessel sometimes there is thickening of retina which is visible ophthalmoscopically.

In the areas of new vessels and the adjacent to the new vessel they become more evident, with some areas having more crowded and some areas there is sparse arrangement giving the sieve like appearance that is not the actual holes it is considered as an alternate layer of fibrous tissue.

Posterior vitreous detachment usually present and starts near the posterior pole and the most common location is the super temporal vessels temporal to the macula above and below the disc.

Posterior vitreous detachment at the disc will not take place usually as the vitreous gets adhered to the disc because of the presence of the fibrous tissue proliferation. Vitreous detachment is not smoothly progressive process.

Whenever the advancing edge of the posterior vitreous detachment meets the active or regressed new vessels it abruptly stops. But inspire of the stop if contraction proceeds the new vessel will be pulled forward with the underlying retina sometimes and detachment spreads away and occurs beyond it.

Sometimes posterior vitreous detachment may be stopped at the areas of retina in the periphery due to adhesion of the retina with the posterior vitreous which is not easily detectable where no new vessels are present.

Attachment of the new vessels with the vitreous which is undergoing detachment exerts traction on the new vessel causes the recurrent vitreous haemorrhage coinciding with extension of vitreous detachment.

Vitreous haemorrhage that is present between the retina and the posterior vitreous surface will resolve within 3 months, retaining its red colour until it gets absorbed. Haemorrhage in the centre that is in the medullary vitreous will not have a red appearance it becomes opacified and appears as white vitreous floaters until it gets absorbed.

Haemorrhage that seeped into the medullary vitreous will resolve very slowly and takes more months to year and often does not resolve completely and within that time new haemorrhage can occur without giving the time for resolving except in one condition that PDR should to the burned out stage.

Based on the shape and the extent of the spread of the vitreous haemorrhage we can identify the extent of the posterior vitreous detachment at

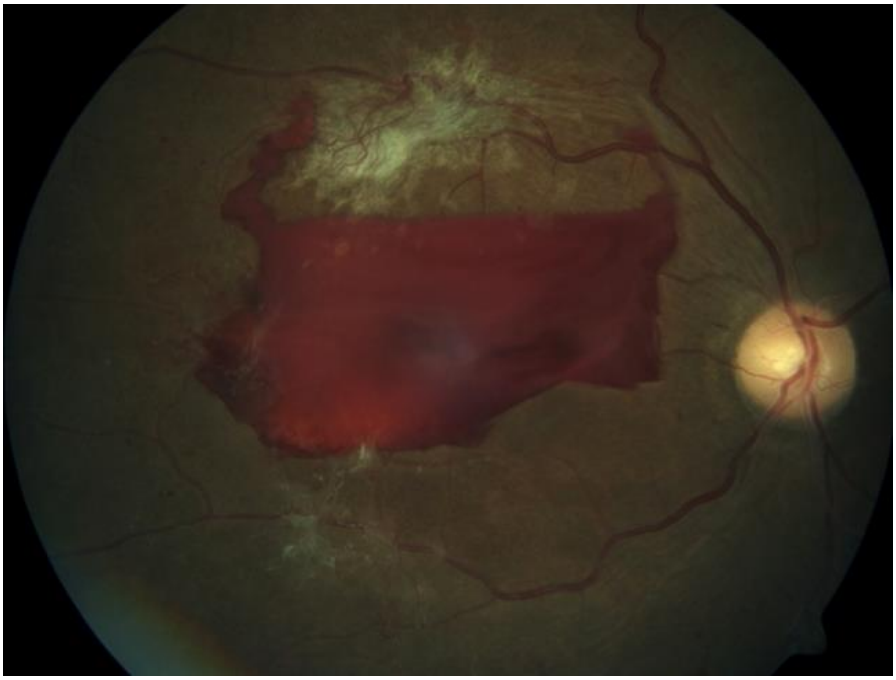
the area of haemorrhage it is detached and the adjacent area is devoid of the posterior vitreous detachment.

In areas of vitreous detachment, the underlying retinal details couldn’t be made out due to the fresh blood and it can be easily identified with the surrounding clear area where the vitreous remain attached.

If the vitreous haemorrhage is present in the superior part of the retina with the detached posterior vitreous superiorly it will be seen as thin line of the vitreous haemorrhage from that we can come to a conclusion that the posterior vitreous detachment is present only in the superior quadrant.

if the vitreous haemorrhage is present in the macular region it has been postulated that there is partial posterior vitreous detachment with the presence of the macular attachment and so the delineation is seen at the macula.

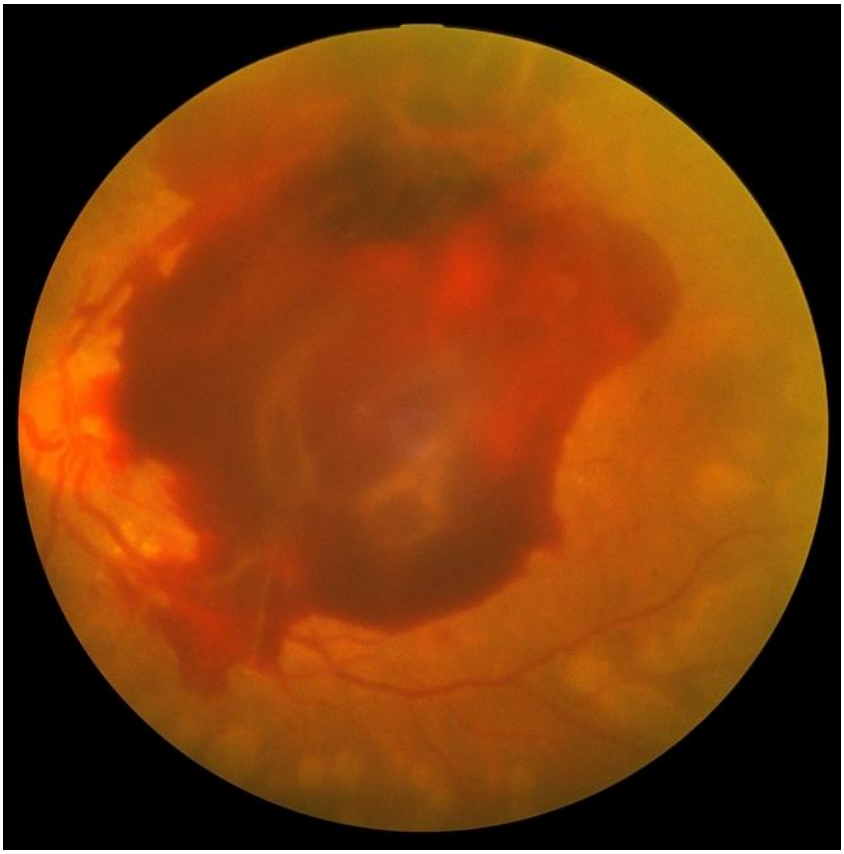
If the vitreous haemorrhage collects at the bottom in shape of boat, there will be partial posterior vitreous detachment but it has extended up to the inferior quadrant and there may be attachment around the boat shape.



Posterior vitreous detachment if occurs complete there will be shifting of the vitreous haemorrhage depending on the position of the head and on stationary there is wide spreading of the haemorrhage full of the inferior quadrant and can move into any part of the retina inferiorly.

If posterior vitreous has not been seen via biomicroscopy we can delineate the extent of the posterior vitreous with the help of the gravitation of the vitreous haemorrhage such as line, boat shaped, spreading type.

Sometimes the posterior vitreous detachment occurs in the small well define area like a sieve with well-defined borders with the size of doubling of the disc size.



The liquefied vitreous gel will come out through the defect and spreads between the posterior vitreous surface and the retina and will be present over the retina.

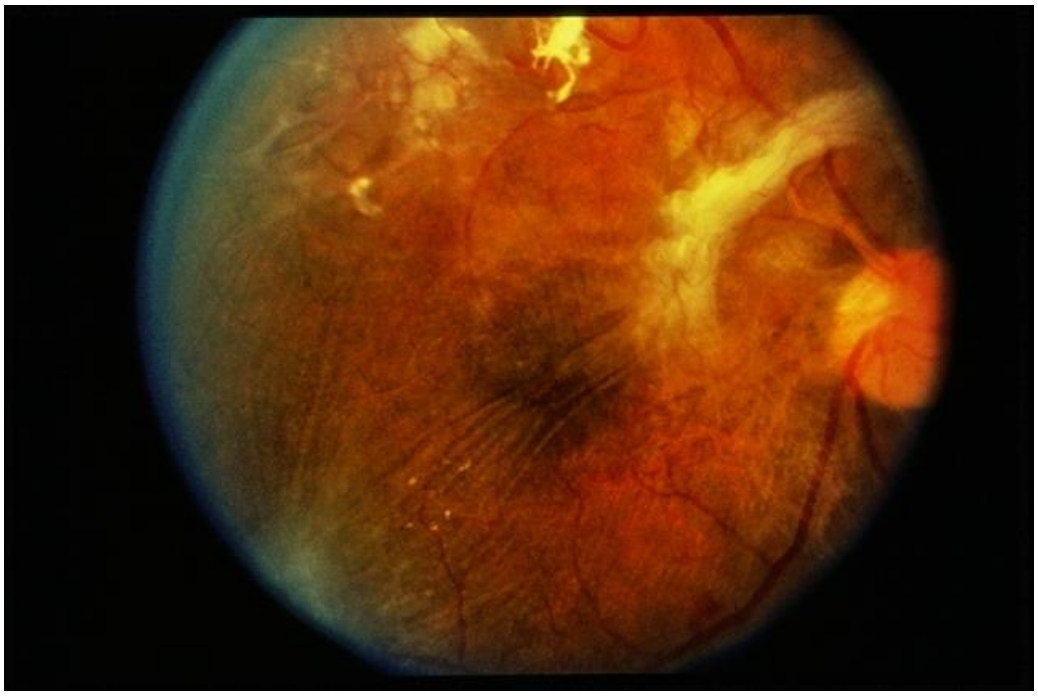
If fresh blood is present in the posterior fluid vitreous in such cases, it can be made to flow across the macula by positioning the patient's head, indicating that the vitreous is indeed detached from the retina throughout the posterior pole.

EFFECT OF FIBROUS BANDS ON RETINA

After the contraction of the fibrovascular proliferation sheet there occurs distortion of the retinal surface and the macula may be dragged and displacement occurs.

More concentrated pigmented area of retinal pigment epithelium with the macula will be dragged towards the side which the fibrovascular tissue contracted and pulling the retina towards it but in other areas it will be normal.

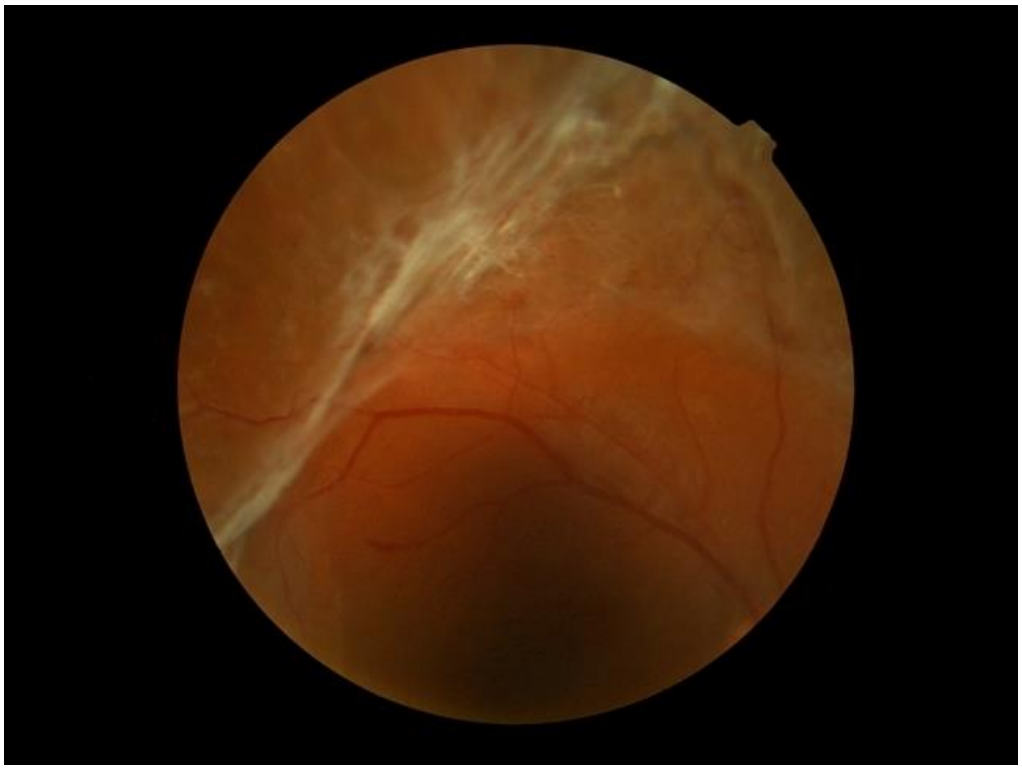
As the most common site for the neovascularisation is the disc fibrovascular proliferation is most commonly present in and around the disc so the tractional force will drag the macula and retina nasally and somewhat vertically.



Following posterior vitreous detachment in around the new vessels and the fibrous tissue attachment to the vitreous and retina there is traction on the retina and it causes tractional retinal detachment. Sometimes there is only detachment of the new vessels only and it gets forcibly teared from the retina following posterior vitreous detachment and vitreous haemorrhage occurs.

Sometimes the retinal detachment is only localised to the small area of vitreous detachment near the base delineated by the pigmentary changes.in case of tractional retinal detachment there will be finding of concave configuration along with the fibrous tissue proliferation with vitreous haemorrhage present.

But sometimes in addition to the tractional retinal detachment due to the traction in the retina there occurs the rhegmatogenous retinal detachment which will be small and involving the entire thickness giving the appearance of convex configuration.



Retinal detachment will be based on the area of the new vessel and the fibrous band surrounding the new vessel and also based on duration and

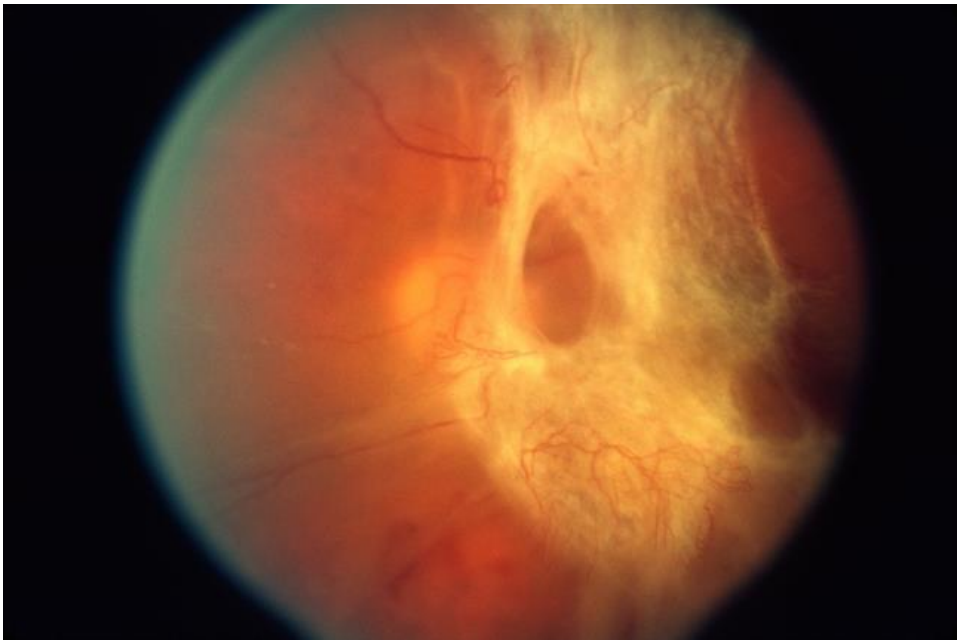
depending on the grading of the posterior vitreous detachment and where the new vessels will be presenting on the retina along with the configuration of the fibrous band around it.

More clumped new vessels along with the thick fibrous band proliferation will lead to the dense vitreous and retinal interface attachments and further posterior vitreous detachment which cause the severe retinal detachment of both tractional and rhegmatogenous types.

If the new vessels have only sparse fibrous tissue proliferation it will have only less vitreous retinal interface adhesion so after the posterior vitreous detachment there is only low prevalence of retinal detachment.

Sometimes there is only end to end attachment of the vitreous to the new vessels in their long course without any attachment in their full length at that time contraction causes the traction on the retina over full distance of that vessel.

But if the new vessels are present over the disc due to their presence only over the disc posterior vitreous can easily detach from the surrounding retina so that there will be no retinal detachment as there is no vitreo retinal attachment. But there will be repeated episodes of vitreous haemorrhage will be there.



Regressive diabetic retinopathy

When vitreous detaches fully from the retina without any attachment except from the areas of the new vessels where vitreo retinal adhesion is present proliferative diabetic retinopathy as the new vessels still holds it, enters the involutional stage

Vitreous haemorrhages will decrease in frequency and severity completely stops, but it takes many months for the vitreous to be clear from the vitreous haemorrhage.

In the regression phase if prior retinal detachment was there and if it had involved the retina without involving the macula vision will be good in that patient.

Even though sometimes it has not involved the macula but constant traction in other areas of retina leads to the macular oedema responsible for the vision loss.

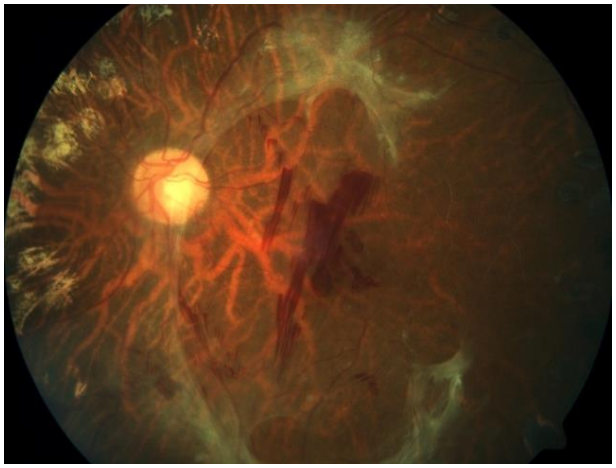
If retinal detachment had occurred over the entire retina even though the regression will not help there will be severe vision loss.

But sometimes there will be reattachment of retina occurs and vision will not improve if it was long standing and involved the macula

The size of the new vessels will be reduced in this phase which is the characteristic one. dilated veins will come to normal size or will be reduced more along with the sheathing of vessels.

Vessels will be reduced in size, area and branches appearing as opaque sclerosed vessel without any blood flow but sometimes the retinal haemorrhages and the microaneurysms are still persistent.

Fibrous tissue adjacent to the new vessels will become thinner and more transparent and the underlying retina will be seen clearly. If there is vision loss at this stage it leads to the contribution of severe retinal ischemia.



REVIEW OF LITERATURE

Prevalence of posterior vitreous detachment in the population with type II diabetes mellitus and its effect on diabetic retinopathy: Sankara Nethralaya Diabetic Retinopathy Epidemiology and Molecular Genetic Study SN-DREAMS report no. 23

Laxmi Gella**.**Rajiv Raman**.**Tarun Sharma**.**Vaitheeswaran Kulothungan

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Japanese Ophthalmological Society 2012

Abstract

Objective -To report the prevalence of posterior vitreous detachment (PVD), and predisposing factors to PVD and their effect on diabetic retinopathy.

Study design- Population-based study.

Methods The study included subjects with type II diabetes mellitus enrolled from a cross-sectional study. Participants underwent a biochemical examination, and a comprehensive ocular examination which included stereo fundus photography. Diabetic retinopathy was graded by use of Klein's classification and diabetic maculopathy was graded by use of the Early Treatment Diabetic Retinopathy Study (ETDRS) criteria. The status of the posterior vitreous was assessed by use of B-scan ultrasonography.

Results The prevalence of PVD was 63.3 %. The risk factors for PVD included age, gender, sight-threatening diabetic retinopathy, and axial length. It was

observed that incomplete PVD could lead to sight-threatening diabetic retinopathy.

Conclusion We report the prevalence and risk factors of PVD in subjects with diabetes mellitus. Uncompleted is a major risk factor for sight-threatening diabetic retinopathy.

Classification of posterior vitreous detachment

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Abstract: Diagnosing a posterior vitreous detachment (PVD) is important for predicting the prognosis and determining the indication for vitreoretinal surgery in many vitreoretinal diseases. This article presents both classifications of a PVD by slit-lamp biomicroscopy and of a shallow PVD by optical coherence tomography (OCT). By biomicroscopy, the vitreous condition is deter- mined based on the presence or absence of a PVD. The PVD then is classified as either a complete posterior vitreous detachment (C-PVD) or a partial posterior vitreous detachment (P-PVD). A C-PVD is further divided into a C-PVD with collapse and a C-PVD without collapse, while a P-PVD is divided into a P-PVD with shrinkage of the posterior hyaloid membrane (P-PVD with shrinkage) and a P-PVD without shrinkage of the posterior hyaloid membrane (P-PVD without

shrinkage). A P-PVD without shrinkage has a subtype characterized by vitreous gel attachment through the premacular hole in a posterior hyaloid membrane to the macula (P-PVD without shrinkage [M]). By OCT, a shallow PVD is classified as the absence of a shallow PVD or as a shallow PVD. A shallow PVD is then subclassified as a shallow PVD without shrinkage of the posterior vitreous cortex, a shallow PVD with shrinkage of the posterior vitreous cortex, and a peripheral shallow PVD. A shallow PVD without shrinkage of the posterior vitreous cortex has two subtypes: an age-related shallow PVD and a perifoveal PVD associated with a macular hole.

Modifying factors related to asymmetric diabetic retinopathy

Abstract

Purpose To identify the modifying factors related to the development of proliferative diabetic retinopathy (PDR).

Methods Thirty-eight eyes of 19 non-insulin- dependent diabetes mellitus (NIDDM) patients with maintained asymmetric PDR were retrospectively reviewed.

Results Five patients with ipsilateral carotid stenosis 90% had PDR. Four patients with high myopia over 6 diopters and 4 patients with optic atrophy and at least a quadrant defect in the visual field had non-proliferative diabetic retinopathy. Of 6 patients with unilateral asteroid hyalosis, 5 had no posterior vitreous detachment (PVD) and PDR.

Conclusion Two factors reached statistical significance as factors modifying PDR: carotid occlusive disease and PVD. Optic atrophy and high myopia showed trends of being a protective factor.

AIMS AND OBJECTIVES

The aim of this study is to observe the association between posterior vitreous detachment and proliferative diabetic retinopathy.

STUDY DESIGN: Non randomized, cross sectional (prevalence) study

INTENDED SAMPLE SIZE: 50 patients

MATERIALS AND METHODS:

This study includes type 2 diabetic patients with DR who were visiting outpatient department of GRH, Madurai for diabetic retinopathy screening between February 2019 and September 2019.

STUDY PERIOD: 8 months (February 2019 to September 2019).

SELECTION OF SUBJECTS:

A total of 50 type 2 diabetic patients with DR of age 40 -70 years visiting OPD of ophthalmology GRH, Madurai who were within the following inclusion and exclusion criteria.

THE INCLUSION CRITERIA:

1.Type 2 diabetic patients with DR.

2.Age between 40-70 years.

THE EXCLUSION CRITERIA:

1.Comorbid conditions such as hypertension, blood dyscrasia, collagen vascular disorder, sarcoidosis.

2.Extremes of refractive error and high axial length.

3.Ocular comorbidities such as glaucoma, uveitis.

4.Previous retinal photocoagulation and intravitreal injections.

5.Previous h/o trauma, intraocular surgeries, electroconvulsive therapy.

6.Carotid occlusive disease excluded by physical examination.

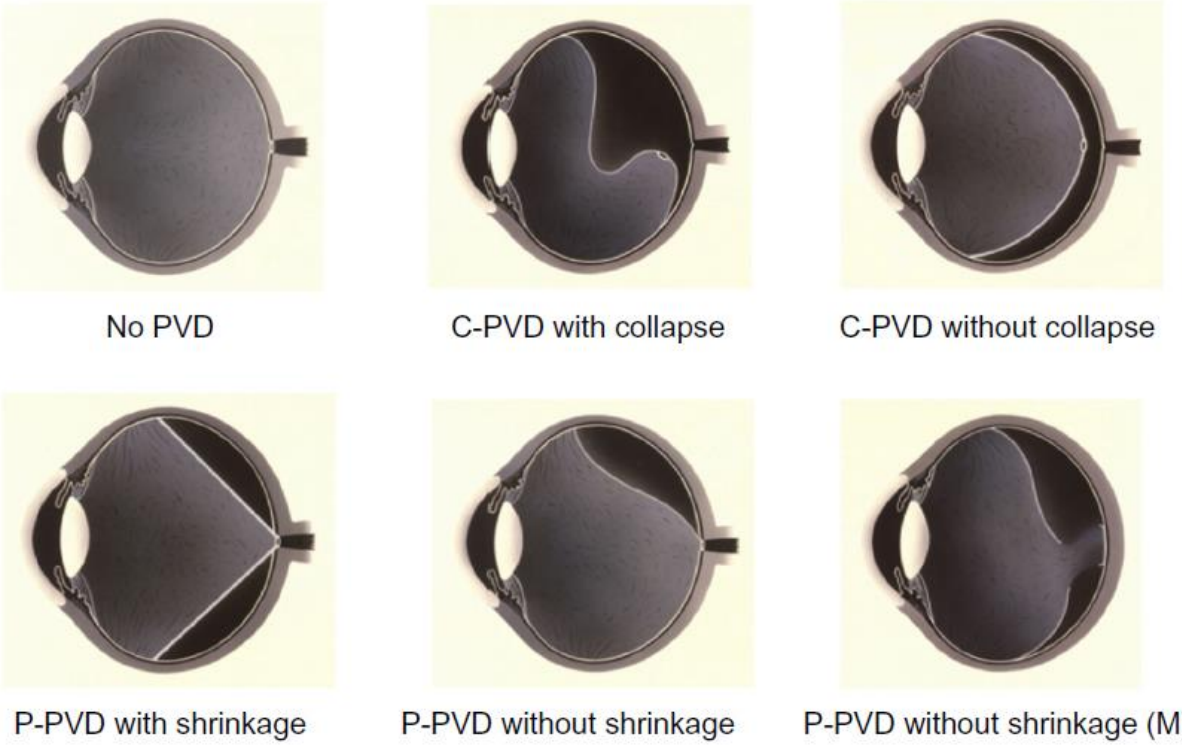
7.Patients in whom fundus examination and FFA could not be done.

METHODOLOGY:

- Patients satisfying inclusion criteria selected
- Getting informed consent from the patient after explaining the procedure
- Visual acuity recorded by Snellen chart
- Anterior segment examination by slit lamp biomicroscopy to rule out rubeosis
- Intraocular pressure measured by Goldmann applanation tonometry
- Posterior segment examined with +90 d lens, indirect ophthalmoscopy
- Fundus photography taken with canon fundus photography
- Classification of posterior vitreous detachment done by + 90 D lens and confirmed by brightness mode ultrasonography.
- Classification of diabetic retinopathy by **ETDRS Revised modified Airlie House diabetic retinopathy classification**
- Classification of posterior vitreous detachment done by the **KAKEHASHI AND ASSOCIATES CLASSIFICATION OF POSTERIOR VITREOUS DETACHMENT**

C-PVD		P-PVD		
C-PVD with Collapse	C-PVD without collapse	P-PVD with shrinkage	P-PVD without shrinkage	
			Attachment to Macula (M)	No attachment to Macula

C-PVD (complete posterior vitreous detachment; P-PVD (partial posterior vitreous detachment).



ETDRS Revised modified Airlie House diabetic retinopathy classification

ETDRS level	**ETDRS severity**	**ETDRS definition**
10	No retinopathy	Diabetic retinopathy absent
20	Very mild NPDR	Microaneurysms only
35	Mild NPDR	Hard exudates, cotton-wool spots, and/or mild retinal Hemorrhages
43	Moderate NPDR	43A: retinal hemorrhages moderate (>photograph 1A) in 4 quadrant or severe (≥ photograph 2A) in 1 quadrant 43B: mild IRMA (<photograph 8A) in 1 to 3 quadrants
47	Moderate NPDR	47A: both level 43 characteristics 47B: mild IRMA in 4 quadrants 47C: severe retinal hemorrhage in two to three quadrants 47D: venous beading in one quadrant"

53A-D	Severe NPDR	53A: ≥2 level 47 characteristics 53B: severe retinal hemorrhages in 4 53C: moderate to severe IRMA (≥ photograph 8A) in at least 1 quadrant 53D: venous beading in at least 2 quadrants"
53E	Very severe NPDR	≥2 level 53A-D characteristics
61	Mild PDR	NVE <0.5 disk area in 1 or more quadrants
65	Moderate PDR	65A: NVE≥0.5 disk area in 1 or more quadrants 65B: NVD<photograph 10A (0.25-0.33 disk area)
71 and 75	High-risk PDR	NVD ≥ photograph 10A, or NVD < photograph 10A or NVE ≥ 0.5 disk area plus VH or PRH, or VH or PRH obscuring ≥ 1 disk area
81 and 85	Advanced PDR	Fundus partially obscured by VH and either new vessels ungradable or retina detached at the center of the macula

OBSERVATION AND ANALYSIS

TABLE 1: AGE DISTRIBUTION OF THE STUDY POPULATION

AGE	No. of cases	Percentage
<50	3	6
51 - 60	19	38
>60	28	56
Total	50	100

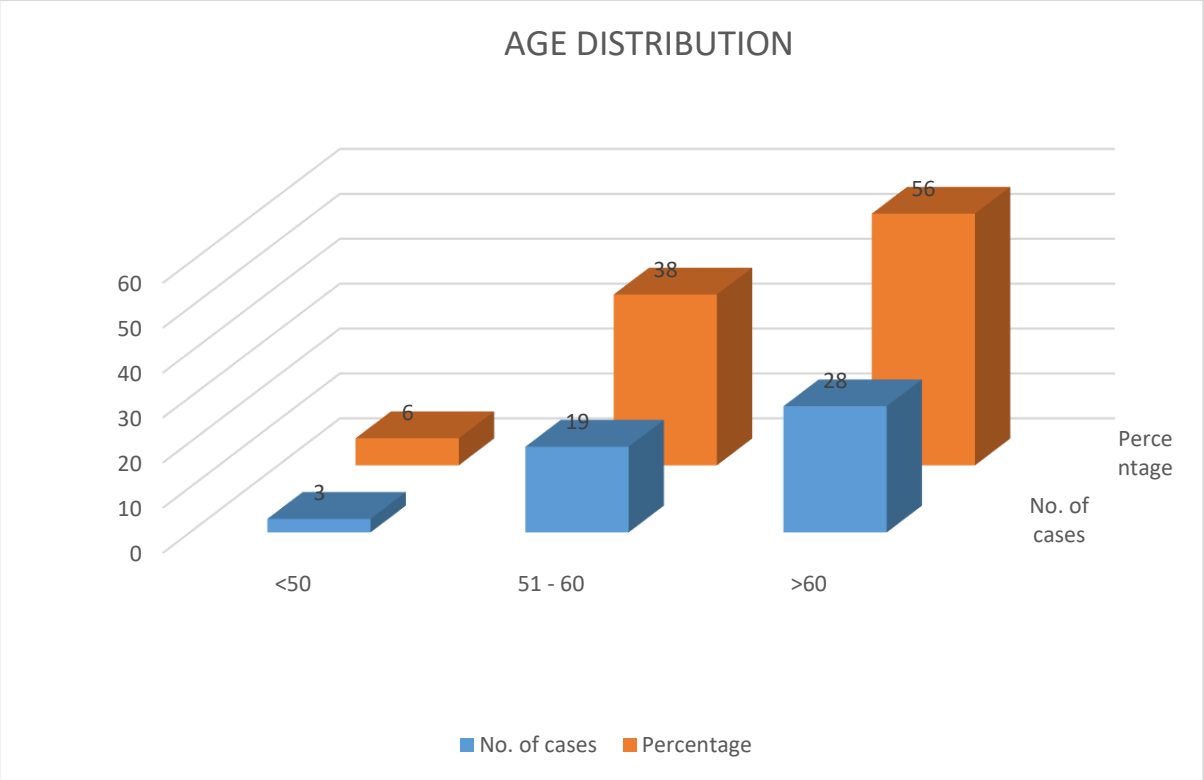


TABLE 1 and GRAPH show that in our study the age distribution of patients was within years with majority falling between 60-70 years.

TABLE 2: DISTRIBUTION OF THE STUDY POPULATION BASED ON SEX

SEX	No. of cases	Percentage
MALE	31	62
FEMALE	19	38
Total	50	100

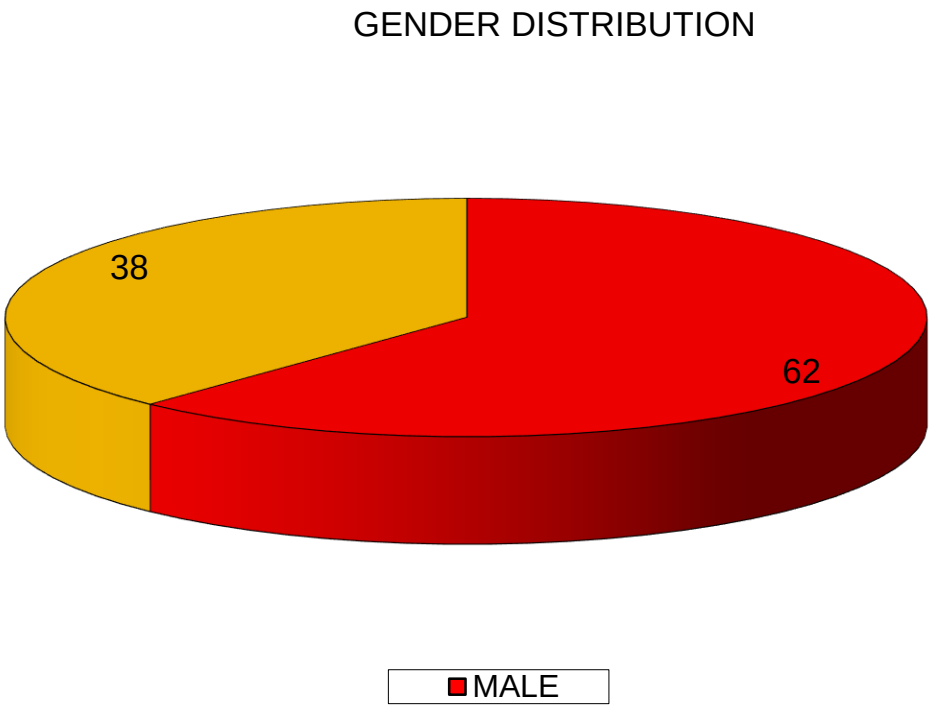


TABLE 2 and GRAPH show that 62% of the population affected are male among 50 persons

TABLE 3: DURATION OF DIABETES MELLITUS

DURATION OF DM	No. of cases	Percentage
<10	24	48
>10	26	52
Total	50	100

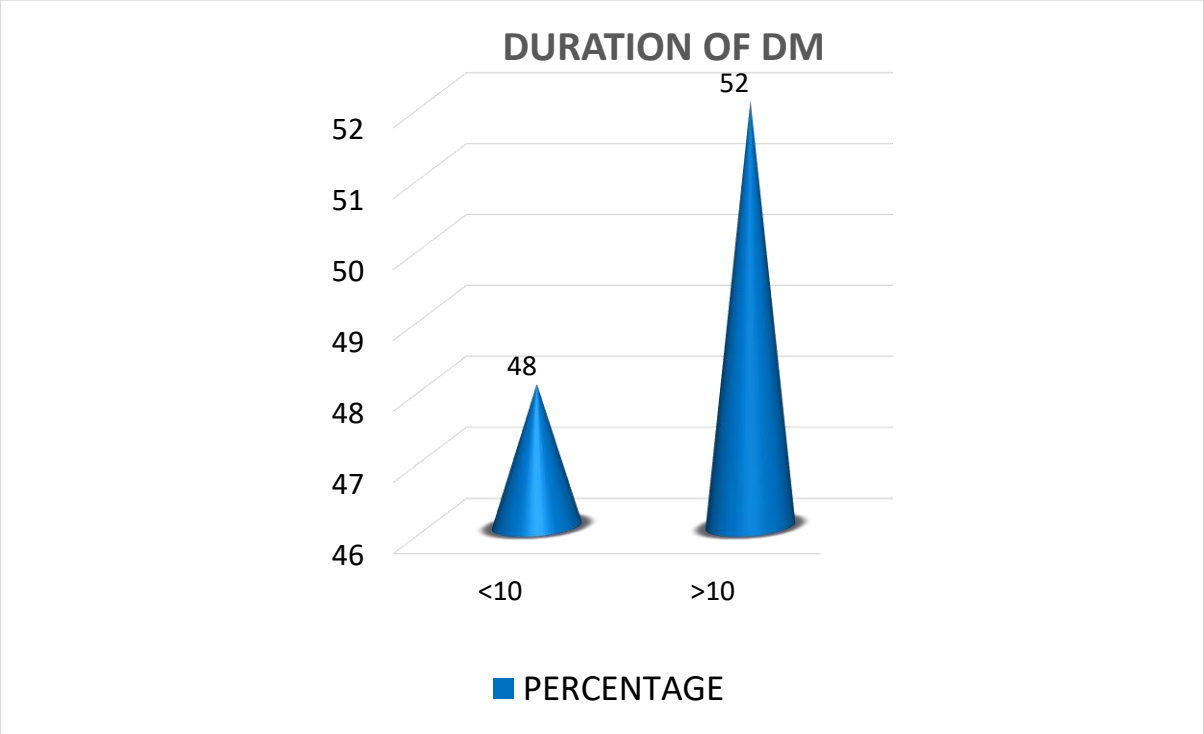


TABLE 3 and GRAPH shows that some preponderance towards the duration greater than 10 years

TABLE 4: VISUAL ACUITY OF RIGHT EYE

VISUAL ACUITY		
RIGHT EYE	No. of cases	Percentage
1/60	3	6
2/60	2	4
3/60	3	6
4/60	5	10
5/60	8	16
6/60	17	34
6/36	4	8
6/24	1	2
HM	7	14
Total	50	100

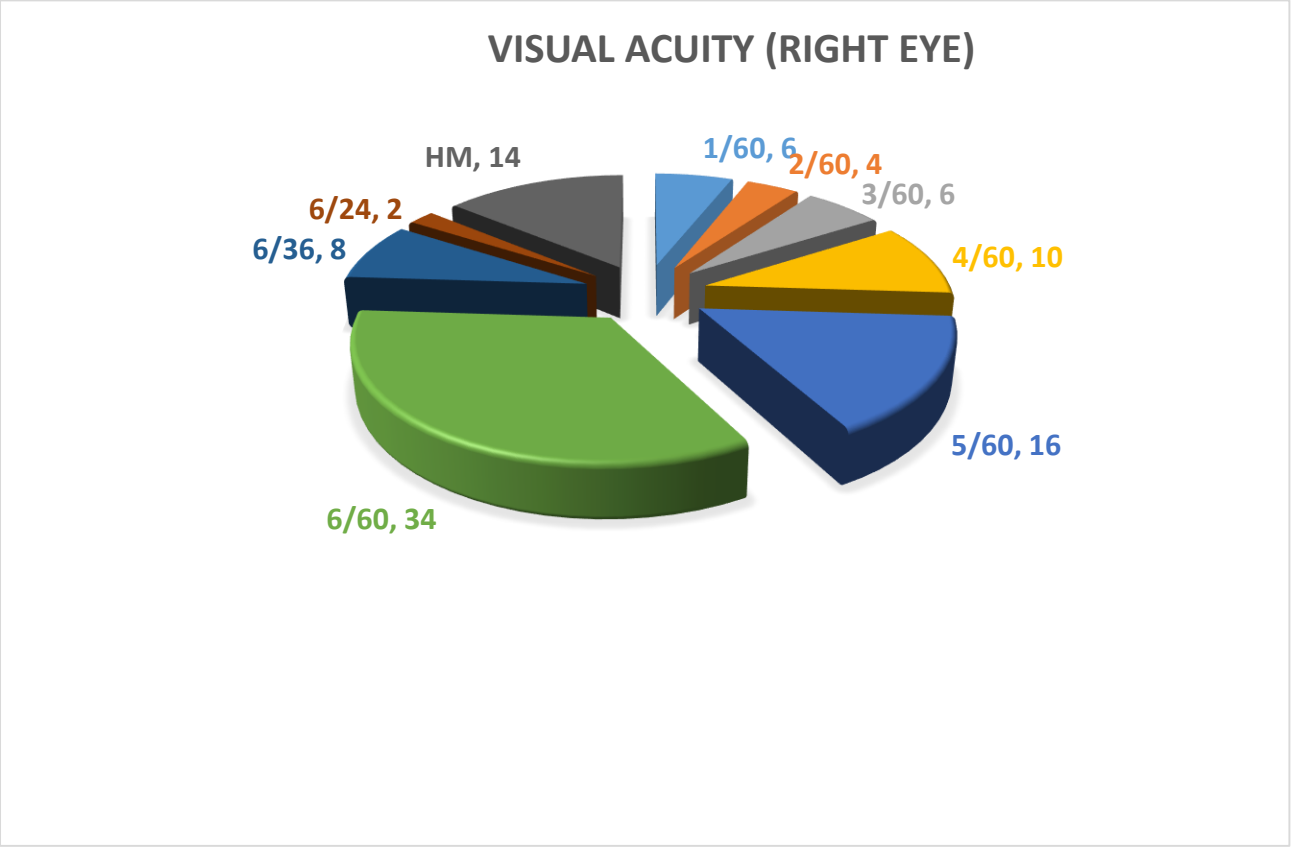


TABLE 4 and GRAPH shows 10 % only had better vision in right eye.

TABLE 5: VISUAL ACUITY OF LEFT EYE

VISUAL ACUITY		
LEFT EYE	No. of cases	Percentage
1/60	3	6
2/60	4	8
3/60	7	14
4/60	4	8
5/60	6	12
6/60	16	32
6/36	4	8
HM	4	8
PL +	2	4
Total	50	100

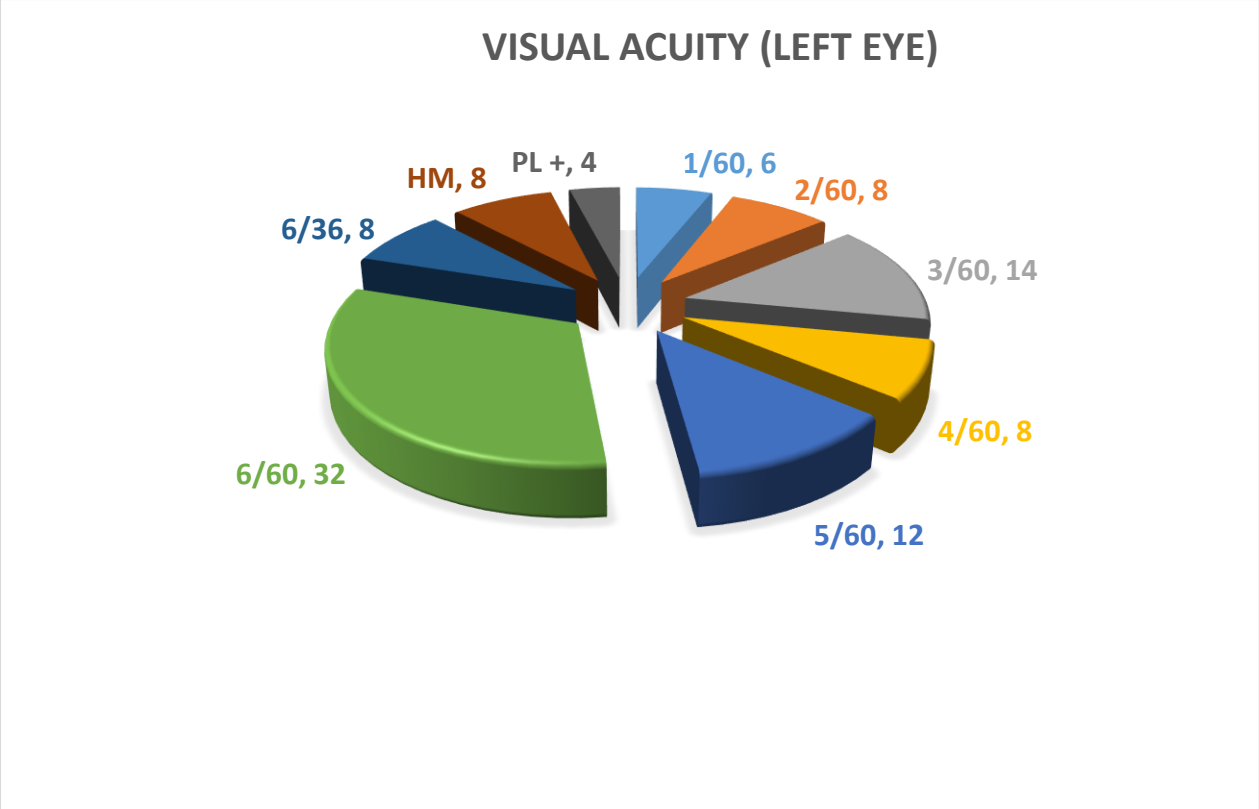


TABLE 5 and GRAPH showed only 8% had better vision in left eye

TABLE 6: GRADE OF DIABETIC RETINOPATHY OF RIGHT EYE

GRADE OF DR RIGHT EYE	No. of cases	Percentage
ADED	4	8
HIGH RISK PDR	15	30
MILD PDR	18	36
MODERATE PDR	13	26
Total	50	100

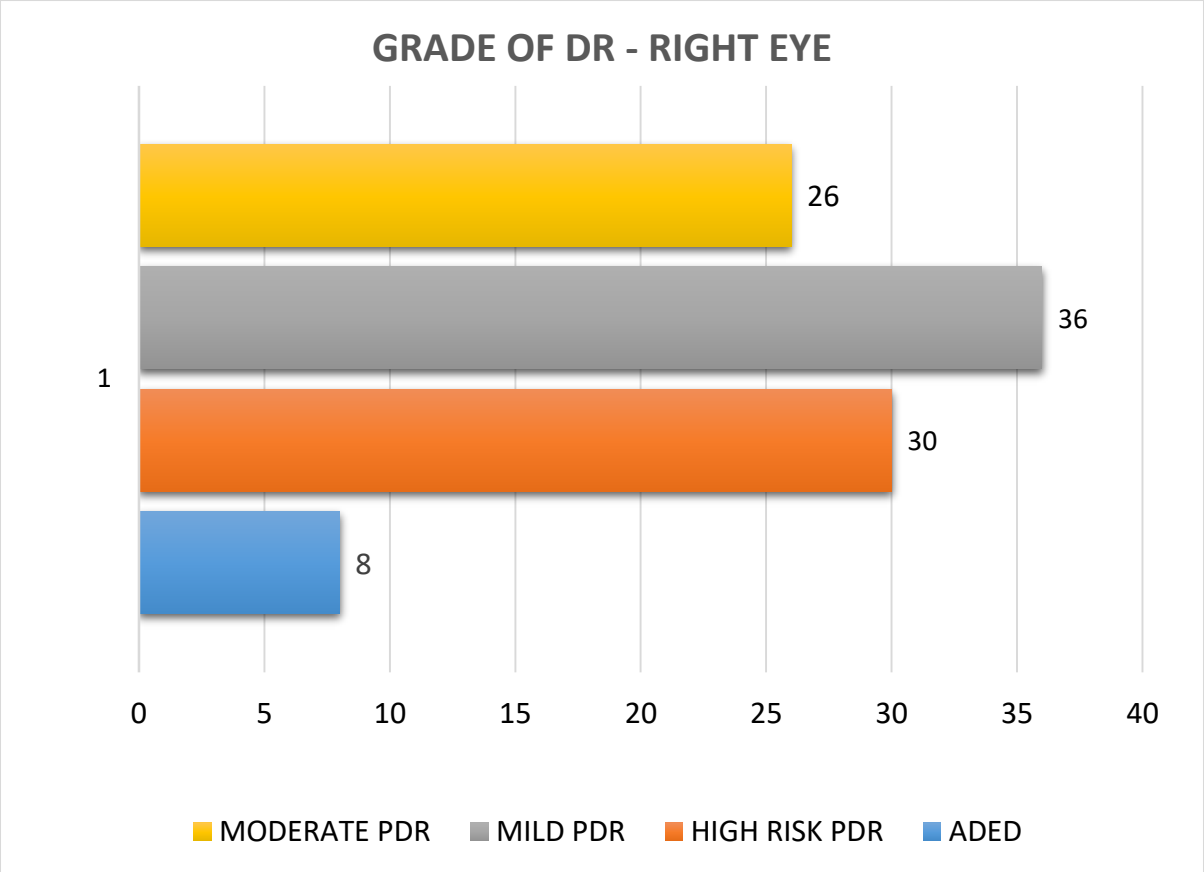


TABLE 6 and GRAPH shows the grading 38% having higher grades of diabetic retinopathy

TABLE 7: GRADE OF DIABETIC RETINOPATHY OF LEFT EYE

GRADE OF DR LEFT EYE	No. of cases	Percentage
ADED	3	6
HIGH RISK PDR	17	34
MILD PDR	13	26
MODERATE PDR	15	30
VERY SEVERE NPDR	2	4
Total	50	100

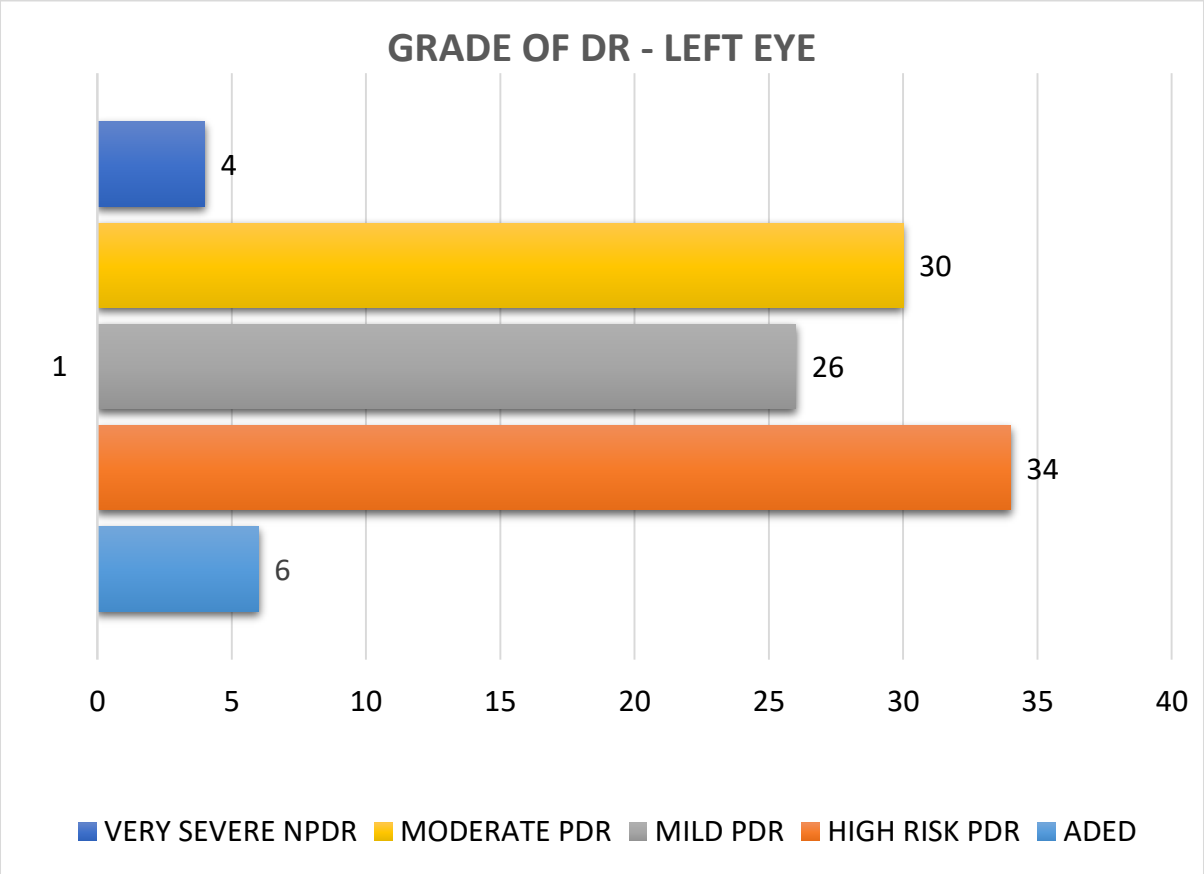


TABLE 7 and GRAPH shows the grading 40% having higher grades of diabetic retinopathy

TABLE 8: GRADE OF PVD OF RIGHT EYE

GRADE OF PVD	RIGHT EYE		
GRADE OF PVD	C-PVD	P-PVD	**Total**
PRESENT	24	6	30
ABSENT	20	0	20
Total	44	6	50

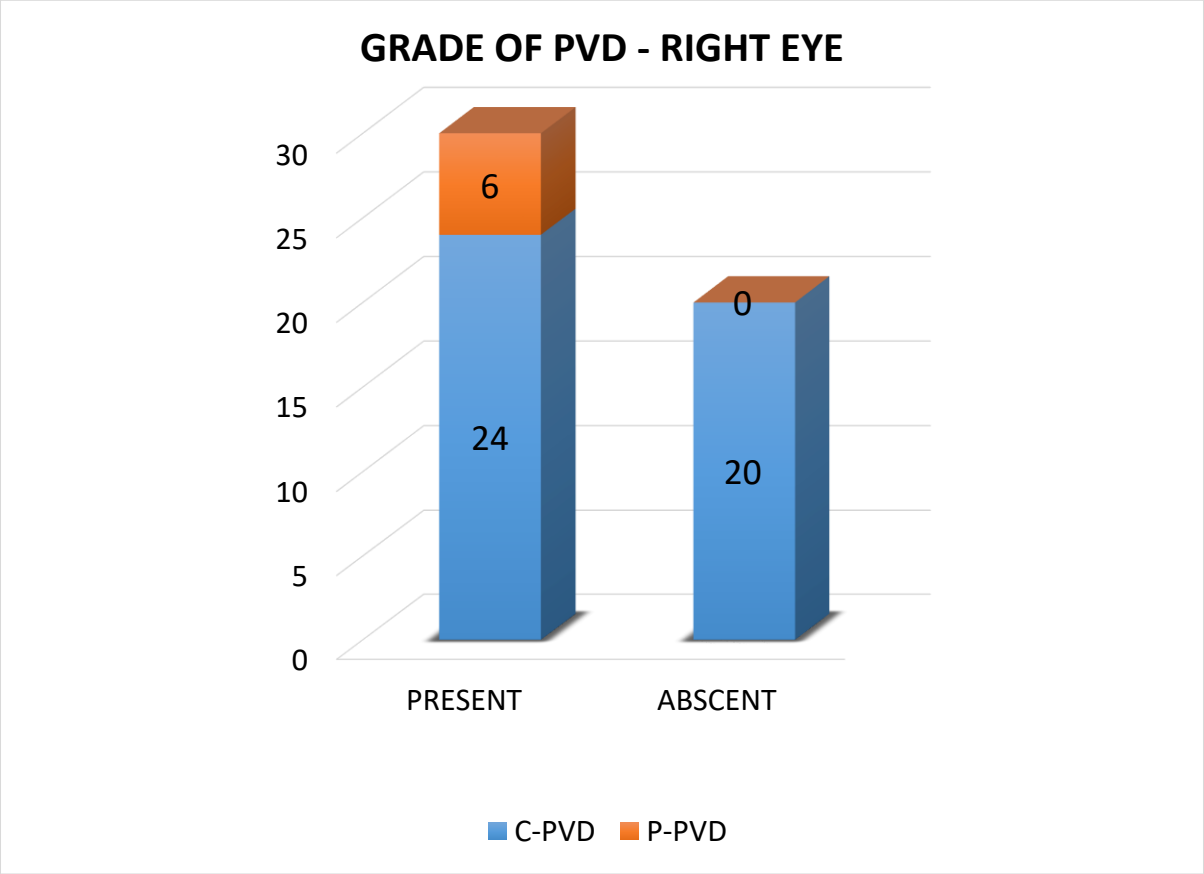


TABLE 8 and GRAPH shows that out of all 60% had PVD in that C-PVD is present 48%having high prevalence

TABLE 9: GRADE OF PVD OF LEFT EYE

GRADE OF PVD	LEFT EYE		
GRADE OF PVD	C-PVD	P-PVD	**Total**
PRESENT	20	5	25
ABSENT	25	0	25
Total	45	5	50

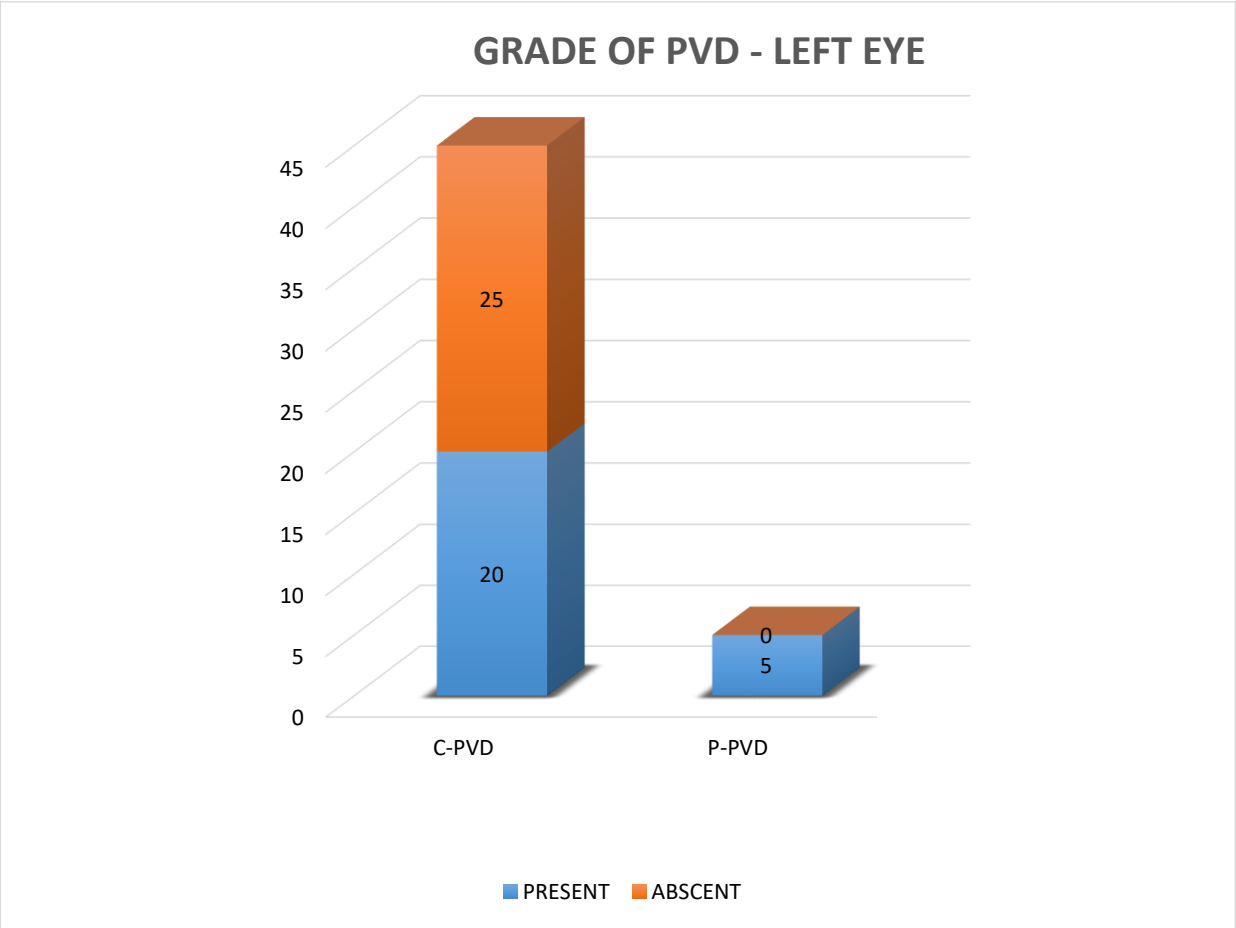


TABLE 9 and GRAPH shows that out of all 50% had PVD in that C-PVD is present in 40% having high prevalence

TABLE 10: OVERALL GRADE OF PVD

OVER ALL GRADE OF PVD			
	C-PVD	P-PVD	**Total**
PRESENT	44	11	**55**
ABSENT	45	0	**45**
Total	89	11	**100**

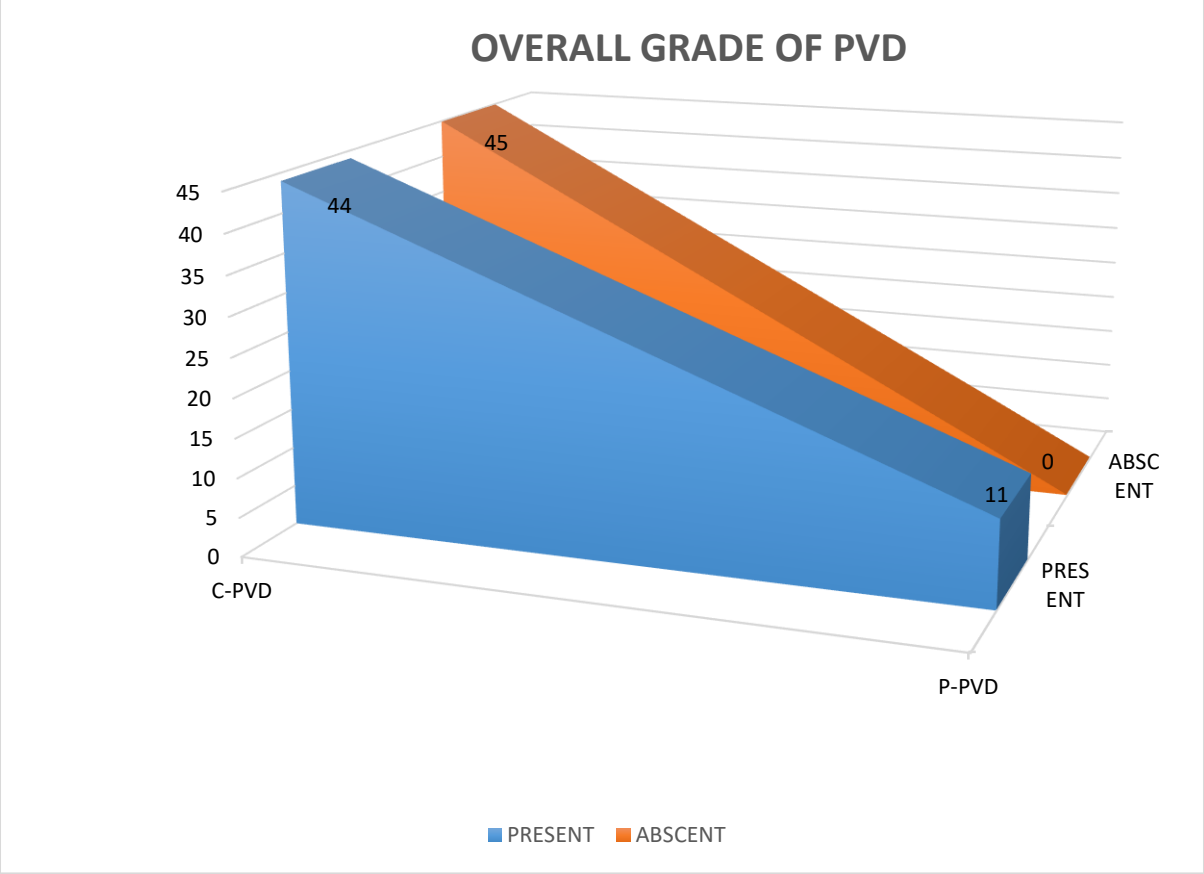


TABLE 10 and GRAPH shows that out of all 55% had PVD in that C-PVD is present in 44% having high prevalence

TABLE 11: PREVALENCE OF PVD VS AGE

	AGE	
GRADE OF PVD	Mean	SD
C-PVD (44)	60.12	5.01
P-PVD (11)	61.64	4.03

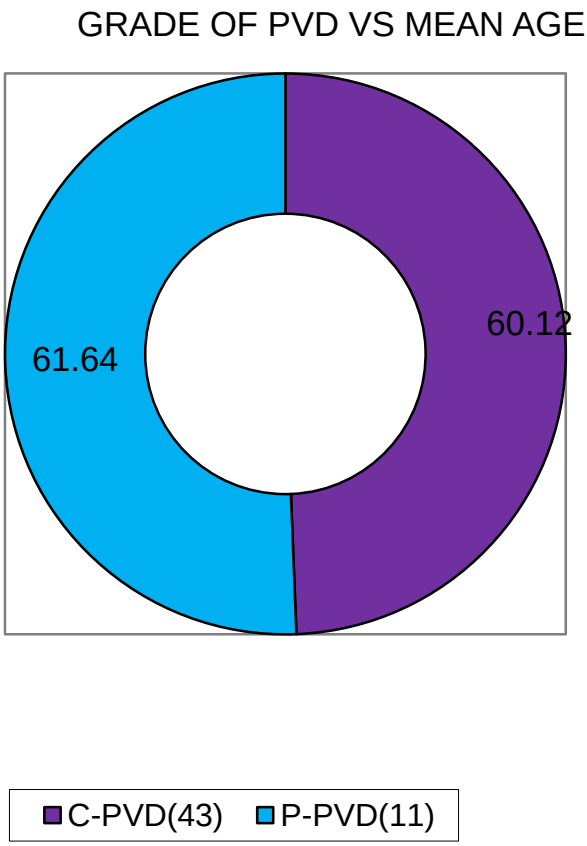


TABLE 11 and GRAPH shows that the mean age is 60 years for the prevalence of PVD

TABLE 12: GRADE OF PVD VS GENDER

	SEX		
GRADE OF PVD	MALE	FEMALE	Total
C-PVD (44)	25	19	44
P-PVD (11)	7	4	11
Total	32	23	55

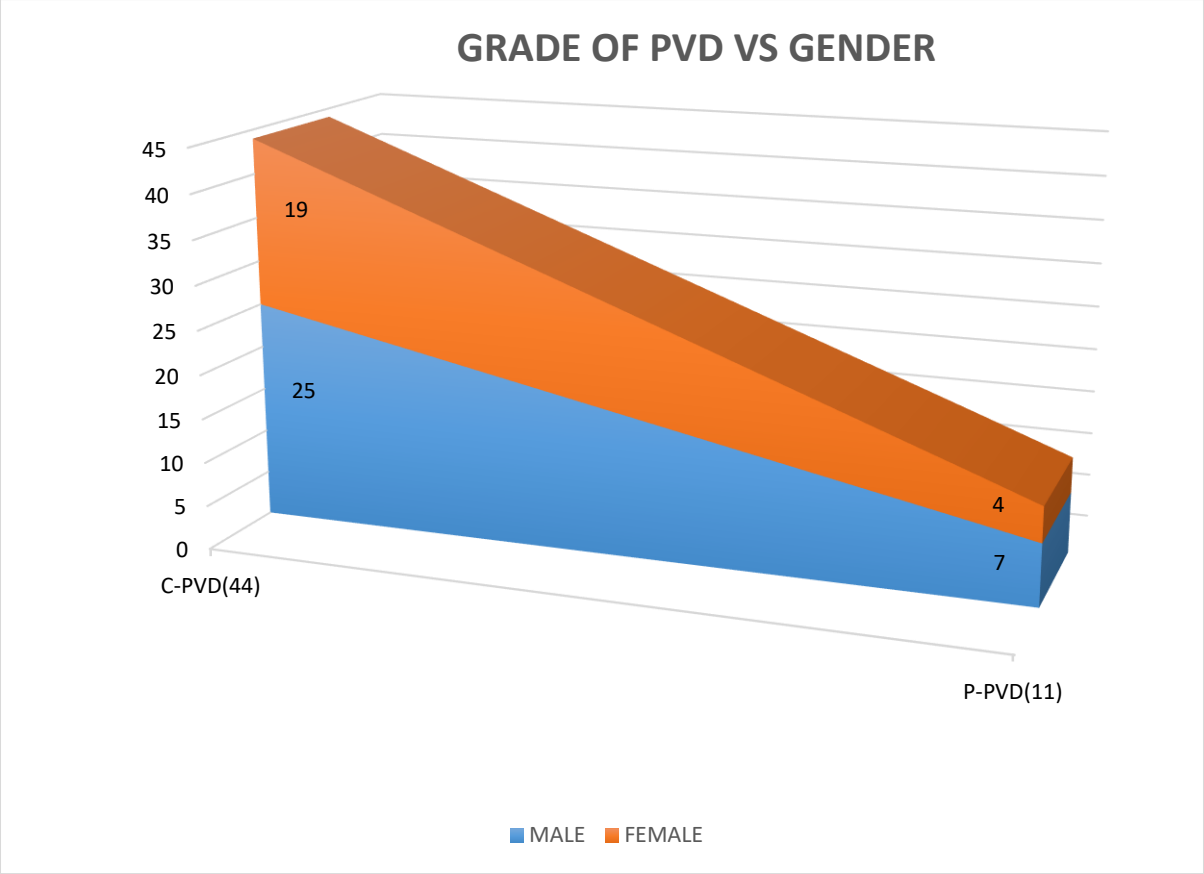
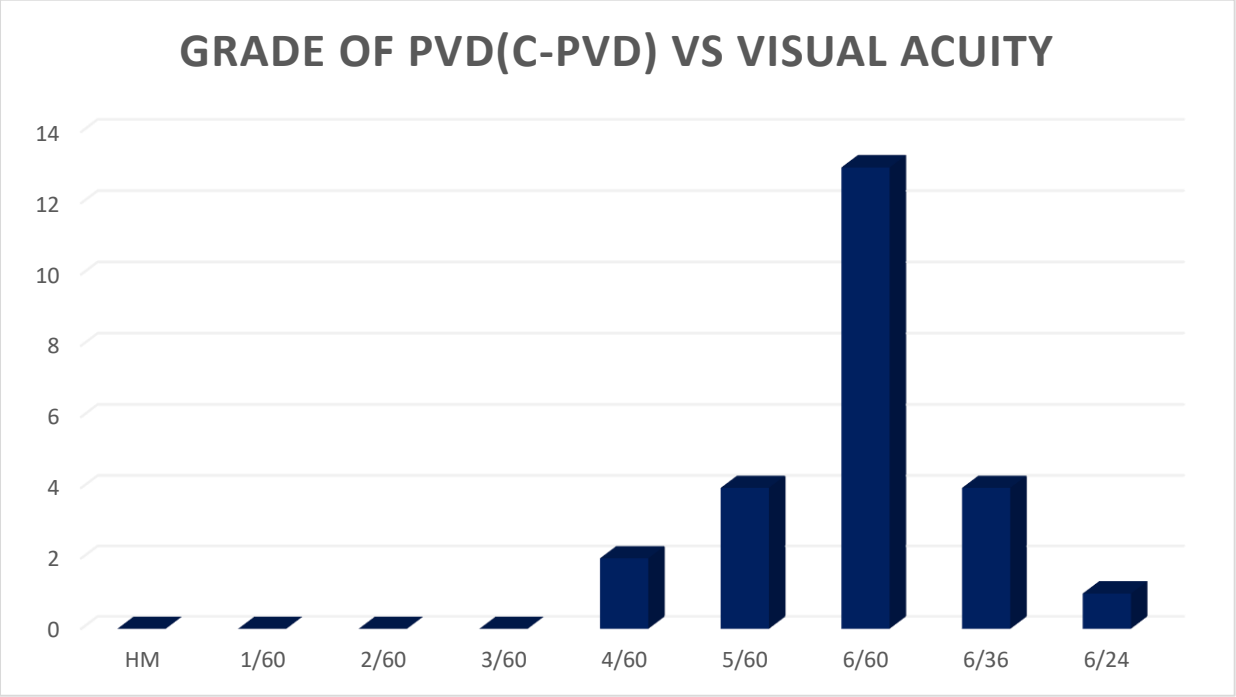


TABLE 12 and GRAPH shows that male gender have 58% of PVD

TABLE 13: VISUAL ACUITY IN COMPARISON WITH PVD OF RIGHT EYE

VISUAL ACUITY	RIGHT EYE	
	C-PVD (24)	P-PVD (6)
HM	0	3
1/60	0	1
2/60	0	0
3/60	0	0
4/60	2	1
5/60	4	1
6/60	13	0
6/36	4	0
6/24	1	0
Total	24	6



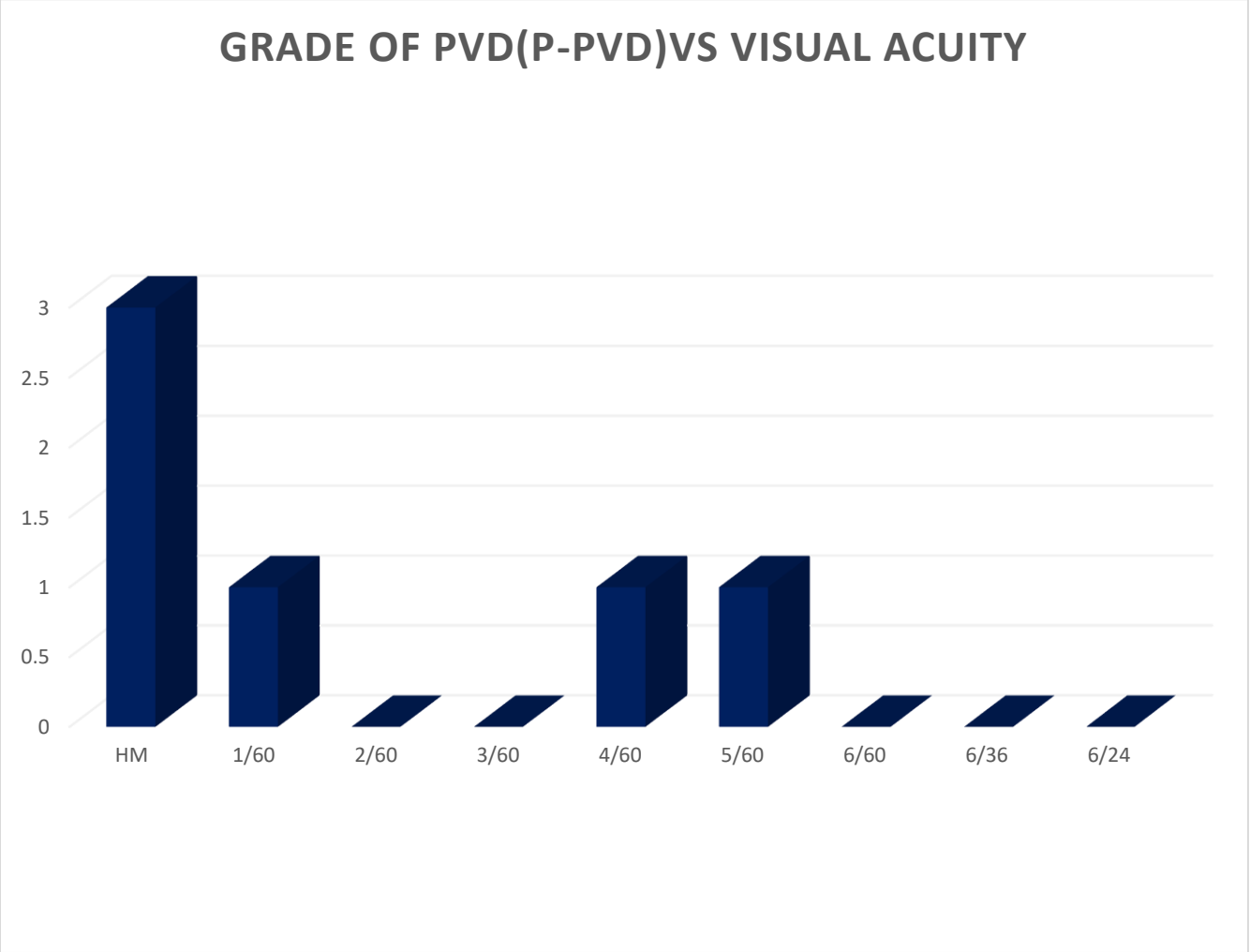
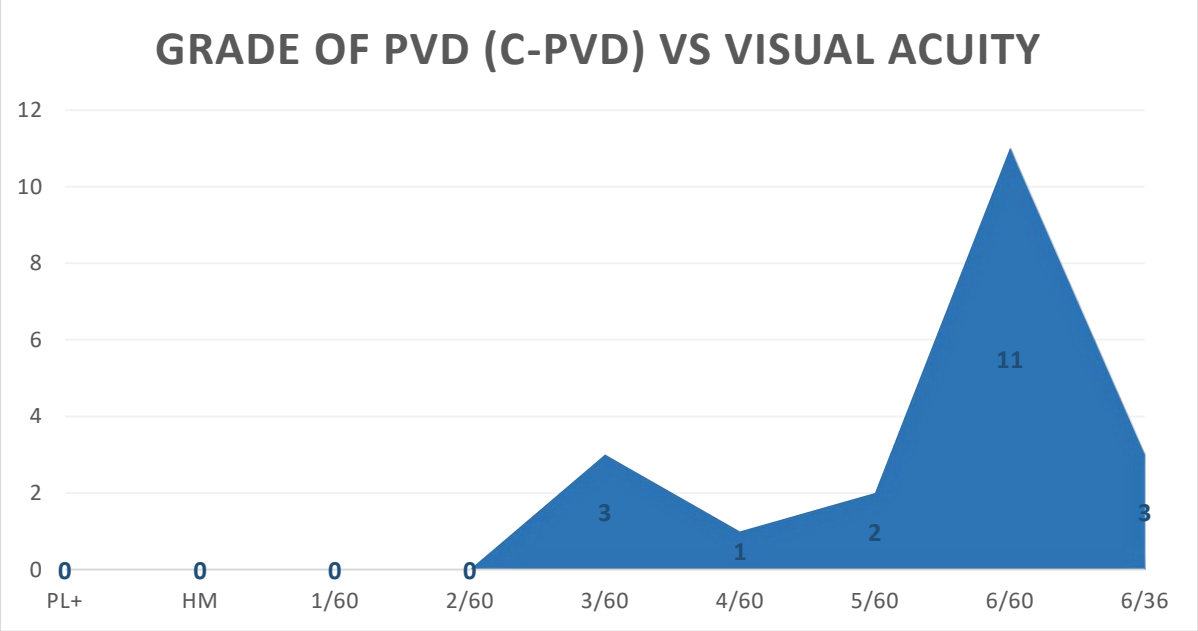


TABLE 13 and GRAPH shows that vision is better in patients having complete PVD totally 60 % had PVD in that 48% had complete PVD out of that 75% had better vision with the P value of 0.002 which is significant.

TABLE 14: VISUAL ACUITY IN COMPARISON WITH PVD OF LEFT EYE

VISUAL ACUITY	LEFT EYE	
	C-PVD (20)	P-PVD (5)
PL+	0	0
HM	0	0
1/60	0	1
2/60	0	1
3/60	3	1
4/60	1	1
5/60	2	1
6/60	11	0
6/36	3	0
Total	20	5



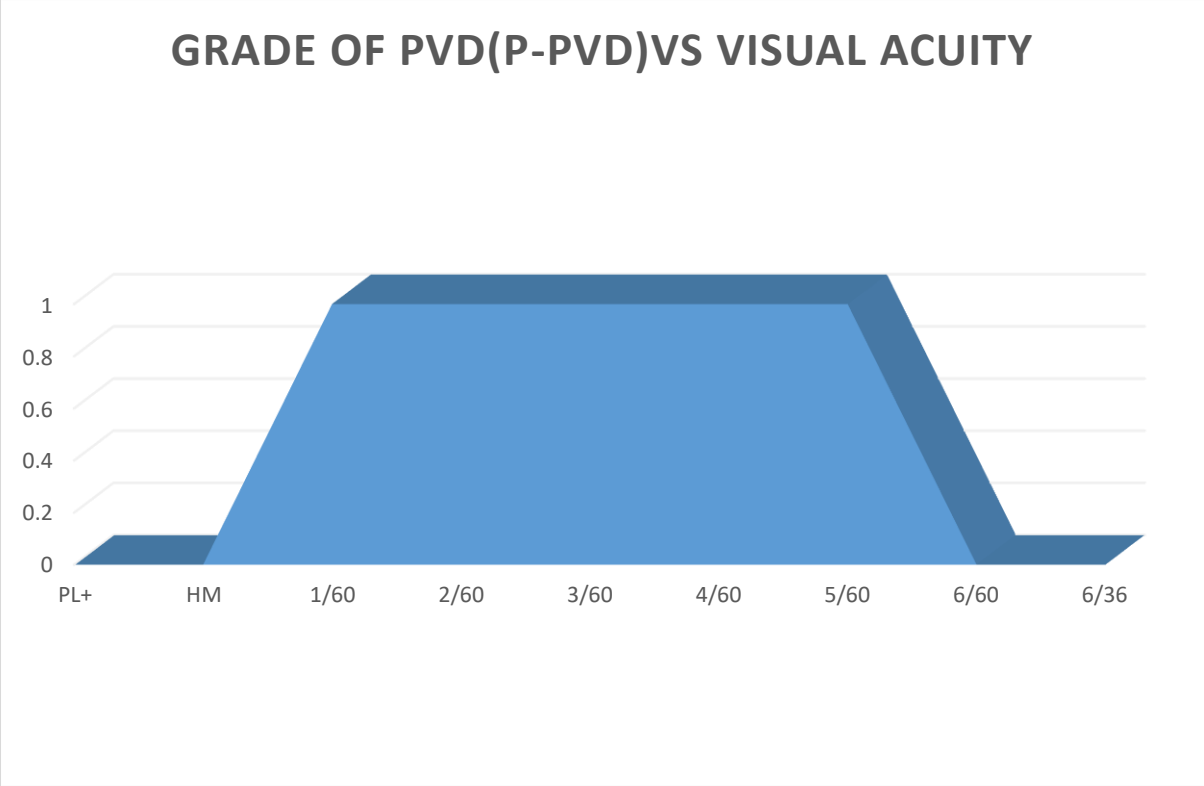


TABLE 14 and GRAPH shows that vision is better in patients having complete PVD totally 50% had PVD in that 40% had complete PVD out of that 70 % had better vision with p value of 0.043 which is significant.

TABLE 15: DR COMPARISON WITH PVD OF RIGHT EYE

GRADE OF DR	RIGHT EYE	
	C-PVD (24)	P-PVD (6)
ADED	0	1
HIGH RISK PDR	0	4
MILD PDR	15	0
MODERATE PDR	9	1
Total	24	6

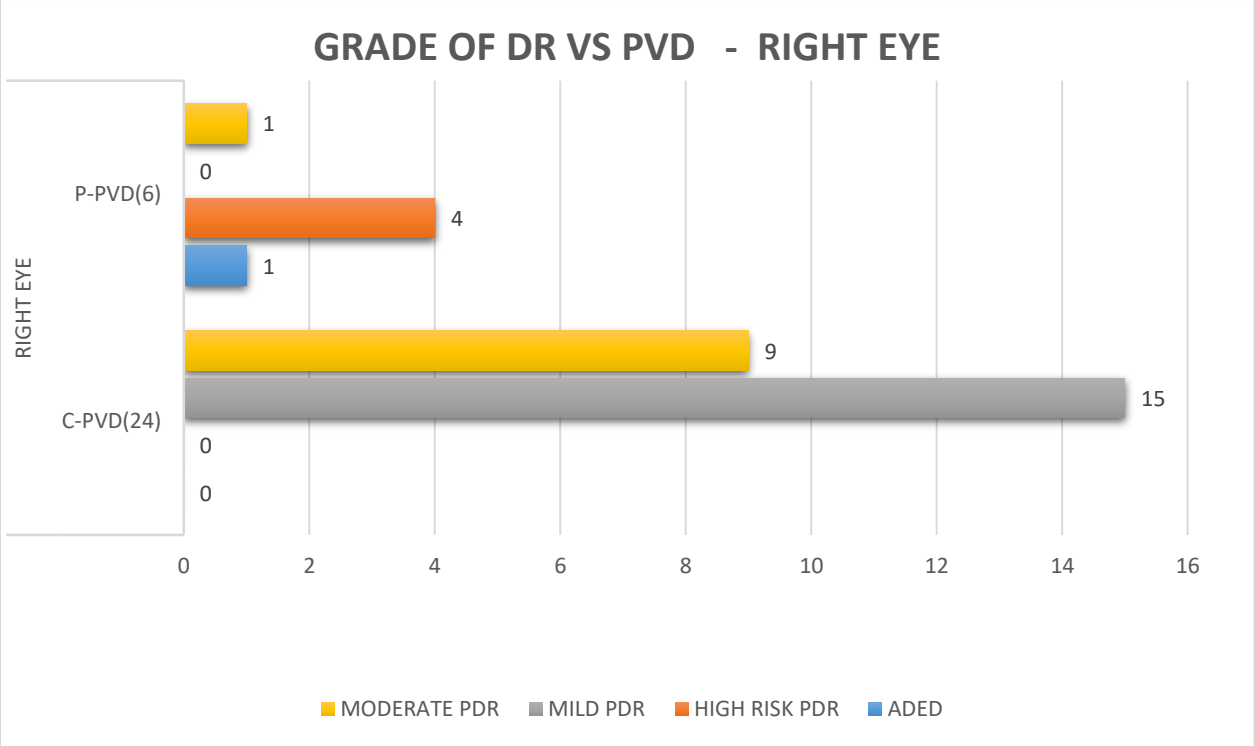


TABLE 15 and GRAPH showing the comparison between the DR grading and PVD out of 50 patients with PDR 48% had complete PVD in which the grading is mild to moderate PDR with P value of <0.001 which is significant in addition it had correlation with P-PVD grading that had grading of high PDR.

TABLE 16: DR COMPARISON WITH PVD OF LEFT EYE

GRADE OF DR	LEFT EYE	
	C-PVD (20)	P-PVD (5)
ADED	0	0
HIGH RISK PDR	0	5
MILD PDR	9	0
MODERATE PDR	8	0
VERY SEVERE NPDR	2	0

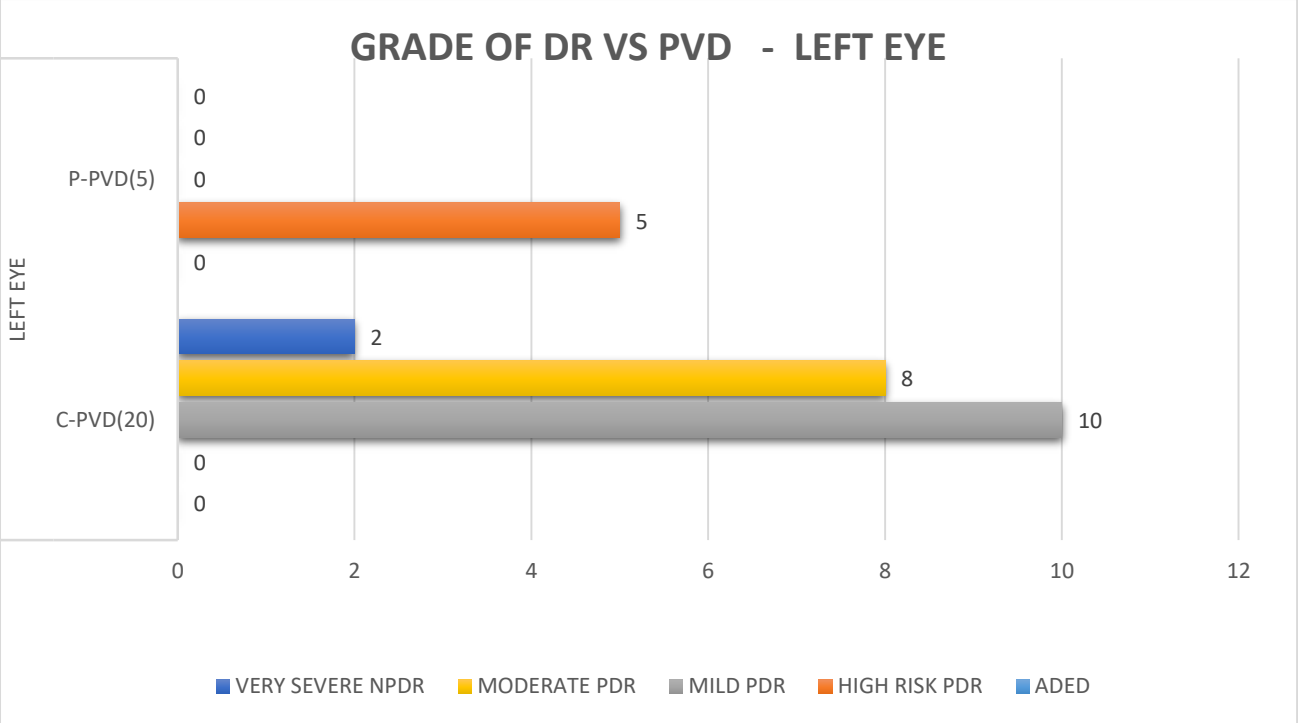


TABLE 16 and GRAPH showing the comparison between the DR grading and PVD out of 50 patients with PDR 40% had complete PVD in which the grading is mild to moderate PDR with P value of <0.001 which is significant in addition it had correlation with P-PVD grading that had grading of high PDR.

TABLE 17: COMPARISON OF COMPLICATIONS WITH PVD OF RIGHT EYE

COMPLICATIONS	RIGHT EYE	
	C-PVD (0)	P-PVD (5)
VH	0	1
TB	0	2
TRD	0	1
VH WITH TRD	0	1

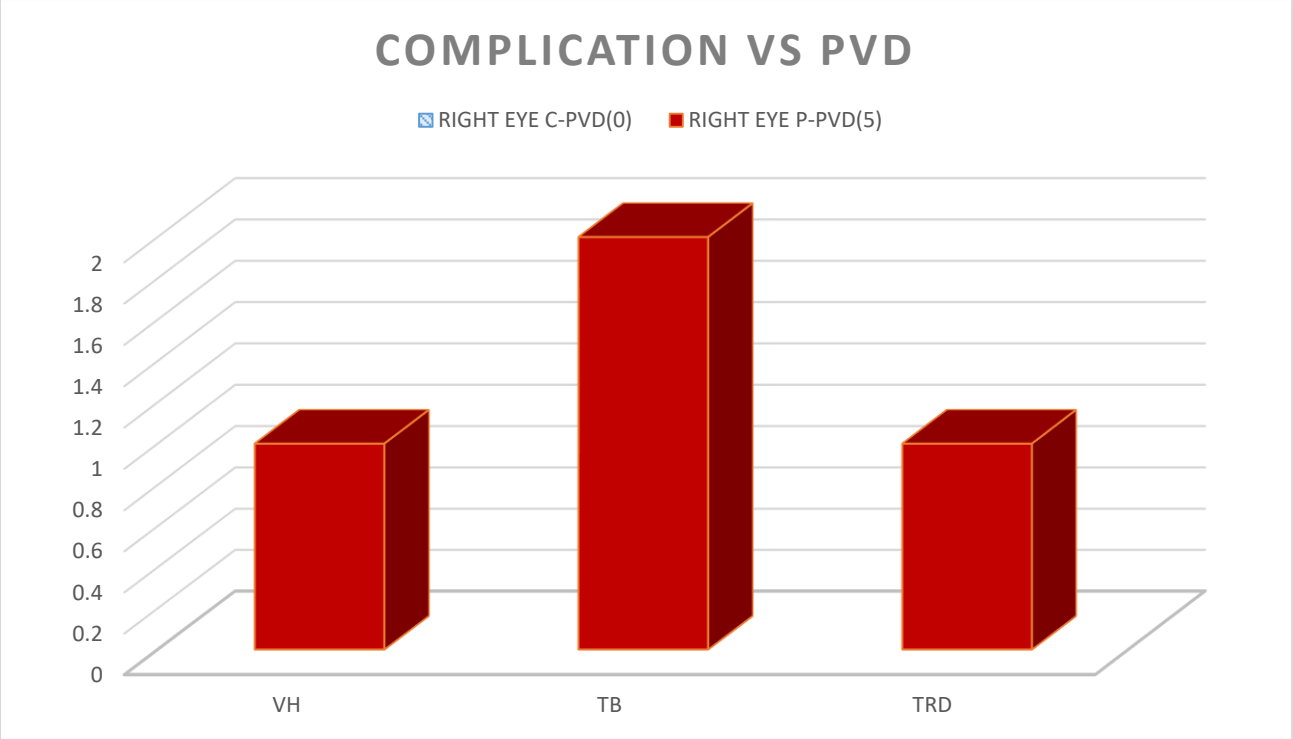


TABLE 17 and GRAPH showing the comparison between the grading of PVD and complications in which there is positive correlation between the P-PVD with complications had been arrived in addition and C-PVD shows negative correlation

TABLE 18: COMPARISON OF COMPLICATIONS WITH PVD OF LEFT EYE

COMPLICATIONS	**LEFT EYE**	
	C-PVD (0)	P-PVD (5)
VH	0	3
TB	0	1
TRD	0	1

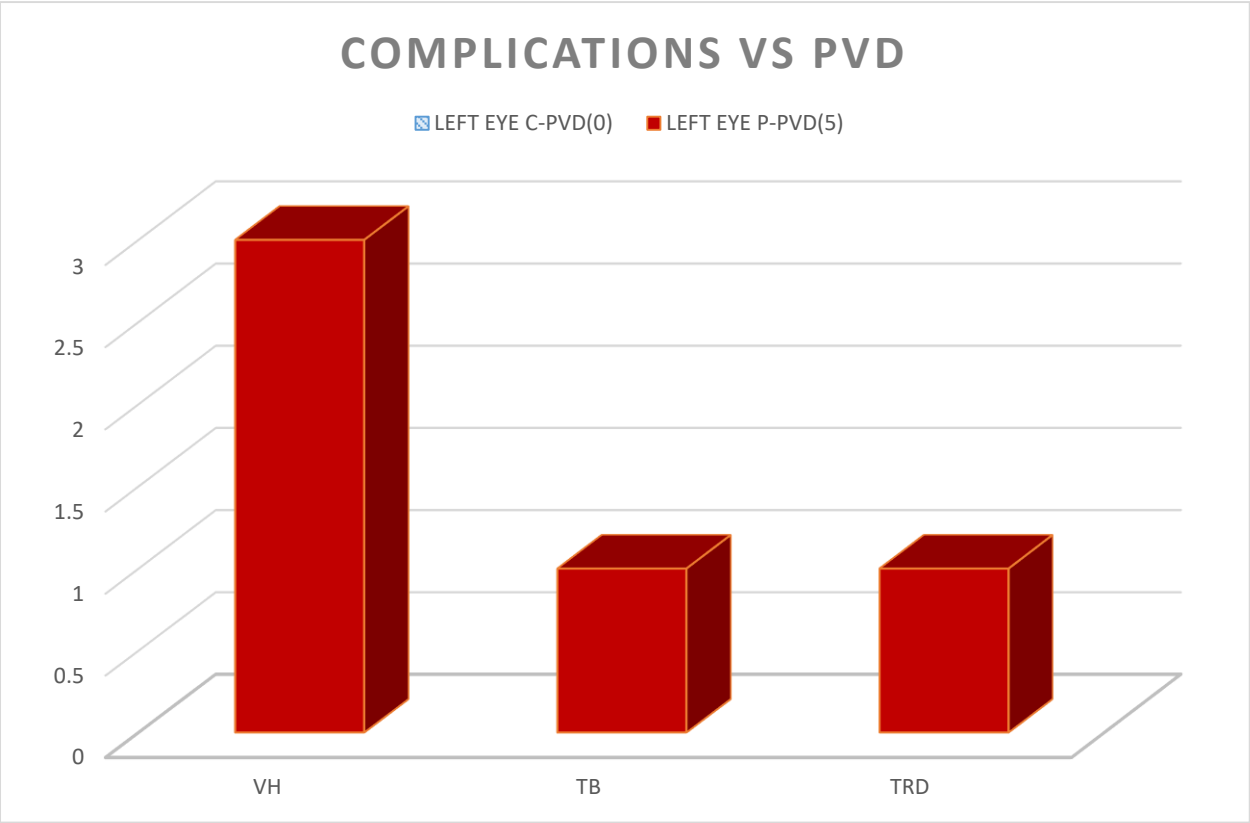


TABLE 18 and GRAPH showing the comparison between the grading of PVD and complications in which there is positive correlation between the P-PVD with complications had been arrived in addition and C-PVD shows negative correlation

All the collected information is recorded in the master chart. Data analysis was done with the help of computer using SPSS 16 software. Using this software percentages, means, standard deviation and P values were calculated through student ‘t’ test for raw data and chi square test for consolidated data to test the significance of difference between the variables. A p value less than 0.05 is taken to denote significant relationship.

DISCUSSION

As diabetic retinopathy is the most common cause of blindness it should be regularly followed up for the progression.

Patients having the borderline severe non proliferative disease should be carefully monitored and early pick up of proliferative diabetic retinopathy disease will prevent permanent visual loss.

While assessing the progression some of the factors acts as a protective factor against the proliferative disease in which PVD is one of the important factor.

As vitreous attachment is the scaffold for the new blood vessel to grow and cause traction leads to complications such as vitreous haemorrhage, tractional retinal detachment, neovascular glaucoma.

Complete PVD is the protective factor for complications of PDR but there is high risk of PDR in incomplete PVD as there is availability of scaffold for new vessels to proliferate.

So in this study PVD and its association with complications of PDR had been elicited which gave results for assessing further progression. In our study, we had taken one fifty patients with proliferative diabetic retinopathy classified based on EDTRS classification.

In our study we had studied the age group from 40 to 70 years in that we had arrived the correlation of age and PVD and found out that the mean age for

PVD was 60 years. there was a positive correlation between posterior vitreous detachment grading with the severity of diabetic retinopathy and their complications.

With the summary of age group of mean age of 60 years 62% of the population affected are male having higher grades of diabetic retinopathy of 54% and 70% in right and left eye,55% had posterior vitreous detachment out of that complete posterior vitreous detachment is 44%.

Comparing visual acuity with the posterior vitreous detachment in right eye 60% had PVD in that 48% had complete PVD out of that 75% had better vision with the significant P value of 0.002 in left eye 50% had PVD in that 40 % had complete PVD out of that 70% had better vision with significant P value of 0.043.

Purpose of the study by comparing the grading of posterior vitreous detachment and diabetic retinopathy we had arrived that 50 patients with PDR 48% had complete PVD in right eye and 40% in left eye in which the grading is mild to moderate grade of diabetic retinopathy with the significant P value of <0.001 high positive correlation.

In addition to above findings we had arrived that there was negative correlation between complete posterior vitreous detachment and the complications and positive correlation with partial posterior vitreous detachment

and also there is additional finding that the grade of diabetic retinopathy was higher in partial posterior vitreous detachment.

Our study gave the correlation between the complete posterior detachment with the vision, grading of diabetic retinopathy and complications of proliferative diabetic retinopathy.

CONCLUSION

From our study we had arrived at a positive correlation that patients having complete posterior vitreous detachment had less complications of proliferative diabetic retinopathy had better vision than patients without having posterior vitreous detachment and partial vitreous detachment.

LIMITATIONS

As this is only cross sectional study there is no follow up of the very severe non proliferative diabetic patients who will go for proliferative phase in which complete posterior vitreous detachment really plays a role in prevention of the complications and further progression into proliferative phase.

Limited sample size and.

Duration of this study is only 8 months for this correlation we need an extended study to prove the exact mechanism.

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ANNEXURE II-ABBREVIATIONS USED IN THIS STUDY

DR- DIABETIC RETINOPATHY

DD- DISC DIAMETER/DISC DIOPTER

NVG- NEOVASCULAR GLAUCOMA

FFA- FUNDUS FLUORESCEIN ANGIOGRAPHY

MA- MICRO ANEURYSMS

BDR- BACKGROUND DIABETIC RETINOPATHY

NVD- NEOVASCULARISATION OF DISC

NVE- NEOVASCULARISATION ELSEWHERE

CSME- CLINICALLY SIGNIFICANT MACULAR EDEMA

IRMA- INTRA RETINAL MICROVASCULAR ANOMALIES

NPDR-NON PROLIFERATIVE DIABETIC RETINOPATHY

PDR- PROLIFERATIVE DIABETIC RETINOPATHY

VH- VITREOUS HEMORRHAGE

ETDRS- EARLY TREATMENT OF DIABETIC RETINOPATHY STUDY

PVD -POSTERIOR VITREOUS DETACHMENT

C-PVD- COMPLETE VITREOUS DETACHMENT

P-PVD-PARTIAL VITREOUS DETACHMENT

B-SCAN-BRIGHTNESS MODE ULTRASONOGRAPHY

M-MACULA

STUDY NO	AGE	SEX	DURATION OF DM	VA		GRADE OF DR		COMPLICATIONS	GRADE OF PVD			
									RIGHT EYE		LEFT EYE	
				RE	LE	RIGHT EYE	LEFT EYE		C-PVD	P-PVD	C-PVD	P-PVD
1	60	M	12	6/60	6/60	MODERATE PDR	MODERATE PDR		PRESENT		PRESENT	
2	54	M	7	4/60	6/60	MILD PDR	MILD PDR		PRESENT		PRESENT	
3	63	F	15	HM	3/60	HIGH RISK PDR	MILD PDR	RE-VH		PRESENT	PRESENT	
4	61	F	20	5/60	PL +	MODERATE PDR	ADED	LE-TRD	PRESENT		ABSCENT	
5	62	M	9	6/60	6/60	MILD PDR	MILD PDR		PRESENT		PRESENT	
6	59	M	12	6/36	6/60	MILD PDR	MILD PDR		PRESENT		ABSCENT	
7	61	M	18	HM	HM	HIGH RISK PDR	HIGH RISK PDR	BE-VH	ABSCENT		ABSCENT	
8	54	M	20	6/60	5/60	HIGH RISK PDR	MODERATE PDR		ABSCENT		ABSCENT	
9	57	M	8	4/60	6/36	HIGH RISK PDR	MILD PDR	RE-TB		PRESENT	PRESENT	
10	48	F	6	6/60	6/36	MILD PDR	MILD PDR		ABSCENT		ABSCENT	
11	67	M	18	HM	4/60	ADED	HIGH RISK PDR	RE-TB,LE-VH	ABSCENT			PRESENT
12	59	F	8	6/36	6/60	MILD PDR	MILD PDR		PRESENT		PRESENT	
13	62	F	12	6/60	3/60	MILD PDR	MODERATE PDR		PRESENT		ABSCENT	
14	65	M	10	HM	1/60	ADED	HIGH RISK PDR	BE-VH RE-TRD		PRESENT	ABSCENT	
15	69	M	15	6/60	6/60	MILD PDR	MILD PDR		PRESENT		ABSCENT	
16	65	M	11	5/60	4/60	HIGH RISK PDR	MILD PDR		ABSCENT		PRESENT	
17	67	M	9	2/60	4/60	HIGH RISK PDR	MODERATE PDR	RE-VH	ABSCENT		ABSCENT	
18	55	F	7	3/60	6/36	MILD PDR	VERY SEVERE NPDR		ABSCENT		PRESENT	
19	64	F	12	6/24	6/60	MILD PDR	MODERATE PDR		PRESENT		ABSCENT	
20	57	F	8	6/60	5/60	MODERATE PDR	HIGH RISK PDR	LE-VH	PRESENT			PRESENT
21	60	M	10	5/60	3/60	MODERATE PDR	HIGH RISK PDR	LE-TB		PRESENT	ABSCENT	
22	58	M	9	HM	6/60	HIGH RISK PDR	MILD PDR		ABSCENT		PRESENT	
23	55	F	6	5/60	3/60	MILD PDR	VERY SEVERE NPDR		ABSCENT		PRESENT	
24	61	M	10	6/60	6/36	MILD PDR	MILD PDR		PRESENT		PRESENT	
25	63	F	11	6/36	3/60	MILD PDR	HIGH RISK PDR	LE-VH	PRESENT		ABSCENT	
26	67	M	15	5/60	2/60	MODERATE PDR	ADED	LE-VH,TB	PRESENT		ABSCENT	
27	65	F	13	6/60	6/60	MODERATE PDR	MILD PDR		ABSCENT		PRESENT	

STUDY NO	AGE	SEX	DURATION OF DM	VA		GRADE OF DR		COMPLICATIONS	GRADE OF PVD			
									RIGHT EYE		LEFT EYE	
				RE	LE	RIGHT EYE	LEFT EYE		C-PVD	P-PVD	C-PVD	P-PVD
28	57	F	8	6/60	HM	MODERATE PDR	HIGH RISK PDR	LE-VH	ABSCENT		ABSCENT	
29	54	M	10	3/60	5/60	HIGH RISK PDR	HIGH RISK PDR	BE-VH	ABSCENT		ABSCENT	
30	57	M	10	5/60	6/60	MODERATE PDR	MODERATE PDR		PRESENT		PRESENT	
31	55	M	7	4/60	6/60	HIGH RISK PDR	MODERATE PDR	RE-TB	ABSCENT		ABSCENT	
32	59	M	10	5/60	2/60	MODERATE PDR	HIGH RISK PDR	LE-VH	ABSCENT			PRESENT
33	64	F	14	6/60	5/60	MODERATE PDR	MODERATE PDR		PRESENT		ABSCENT	
34	62	M	16	4/60	3/60	HIGH RISK PDR	HIGH RISK PDR	BE-VH	ABSCENT			PRESENT
35	63	M	14	6/36	6/60	MILD PDR	MODERATE PDR		PRESENT		ABSCENT	
36	66	M	16	HM	1/60	ADED	HIGH RISK PDR	RE-TRD LE-VH	ABSCENT		ABSCENT	
37	50	F	7	4/60	3/60	MILD PDR	MILD PDR		PRESENT		PRESENT	
38	69	M	17	1/60	6/60	HIGH RISK PDR	MODERATE PDR	RE-VH	ABSCENT		PRESENT	
39	58	M	9	6/60	HM	MILD PDR	HIGH RISK PDR	LE-TB	PRESENT		ABSCENT	
40	59	F	10	5/60	5/60	MODERATE PDR	MODERATE PDR		PRESENT		PRESENT	
41	58	M	11	HM	6/60	HIGH RISK PDR	MODERATE PDR	RE-TB		PRESENT	PRESENT	
42	62	M	10	6/60	2/60	MILD PDR	HIGH RISK PDR	LE-VH	PRESENT		ABSCENT	
43	67	M	15	1/60	6/60	ADED	MODERATE PDR	RE-TRD	ABSCENT		PRESENT	
44	49	M	8	6/60	6/60	MILD PDR	MODERATE PDR		PRESENT		PRESENT	
45	69	F	18	2/60	1/60	HIGH RISK PDR	HIGH RISK PDR	BE-VH	ABSCENT			PRESENT
46	67	F	12	6/60	HM	MODERATE PDR	HIGH RISK PDR	LE-VH	PRESENT		ABSCENT	
47	63	F	10	6/60	2/60	MILD PDR	HIGH RISK PDR	LE-VH	PRESENT		ABSCENT	
48	61	F	11	1/60	5/60	HIGH RISK PDR	MODERATE PDR	RE-TRD		PRESENT	PRESENT	
49	63	M	15	3/60	PL +	HIGH RISK PDR	ADED	BE-VH,RE-TRD	ABSCENT		ABSCENT	
50	65	M	14	6/60	4/60	MODERATE PDR	HIGH RISK PDR	LE-TB	PRESENT		ABSCENT	

M-MALE,F-FEMALE,DM-DIABETES MELLITUS,HM-HAND MOVEMENTS,PL-PERCEPTION OF LIGHT,PDR-PROLIFERATIVE DIABETIC RETINOPATHY,ADED-ADVANCED DIABETIC EYE DISEASE,RE-RIGHT EYE,LE-LEFT EYE,VH-VITREOUS HEMORRHAGE,TB-TRACTIONAL BAND,TRD-TRACTIONAL RETINAL DETACHMENT,RD-RETINAL DETACHMENT,PVD-POSTERIOR VITREOUS DETACHMENT,C-PVD COMLETE POSTERIOR VITREOUS DETACHMENT,P-PVD-PARTIAL POSTERIOR VITREOUS DETACHMENT

ANNEXURE IV - PROFORMA

STUDY NO: OP NO:

NAME : DATE :

AGE : PH NO:

GENDER : M ☐ F ☐

DIET : V ☐ NV ☐

SYSTEMIC ILLNESS:

DM ☐ BA ☐ TB ☐ EPILEPSY ☐ CAD ☐

OTHERS :

CLINICAL DATA:

DURATION OF DM : ☐ YRS

BP : PULSE: IOP : OD ☐ ☐

VISION

EYE	OD	OS
UCVA		
CVA		

ASTEROID HYALOSIS : OD ☐ OS ☐

GRADING OF DR

EYE	GRADE
OD	
OS	

GRADIND OF PVD

EYE	GRADE
OD	
OS	

RETINOSCOPIC REFRACTION :

OD	OS

ANNEXURE V- CONSENT FORM IN REGIONAL LANGUAGE (TAMIL)

ஆராய்ச்சி ஒப்புதல் படிவம்

தேதி:

ஆராய்ச்சி தலைப்பு:

பெயர்:

வயது: உள்நோயாளி எண்:

பால்: ஆராய்ச்சி சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விபரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்துகொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகிறேன், மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்த நேரமும் பின்வாங்கலாம் என்பதையும், அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும், நான் புரிந்து கொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன் முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ என்னுடைய பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டார்கள் என்பதையும் அறிந்து கொண்டேன்.

எனது நோயின் தன்மை மற்றும் பின்விளைவுகளையும் முழுமையாக புரிந்துகொண்டேன். இந்த ஆராய்ச்சியில் எனது நோயின் மூலக்கூறு மற்றும் தன்மையை மட்டுமே ஆராய்வார்கள் என்பதை அறிந்து கொண்டேன்.

இதனால் என் வைத்திய முறைகளில் எந்த மாற்றமும் பார்வைத்திறனில் எந்தவித பாதிப்பும் ஏற்படாது என்பதையும் தெரிந்துகொண்டேன். எனக்கு விளக்கப்பட்ட விஷயங்களை முழுமையாக புரிந்துகொண்டு இந்த ஆராய்ச்சியில் பங்குகொள்ள என் முழு மனதுடன் ஒப்புக்கொள்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம்

பங்கேற்பாளர் கையொப்பம்

தேதி:

Ref.No.1142/E1/5/2019

Madurai Medical College,
Madurai – 20. Dated : 11.02.2019

MINUTES OF ETHICAL COMMITTEE MEETING

Date & Day : 11.02.2019
Time : 10.00 AM to 12.30 Noon
Venue : GRH Auditorium
Government Rajaji Hospital, Madurai

The Institutional Ethical Committee Meeting was presided by the Prof. Dr.K.Vanitha, MD., DCH.,Dean /convenor & Prof Dr. V Nagaraajan MD MNAMS DM (Neuro) DSc.,(Neurosciences), DSc (Hons), Chairman.

The following members of the IEC attended the meeting.

1. Dr.M.Shanthi, Prof. of Pharmacology,
   Madurai Medical College, Madurai — Member Secretary
2. Dr.PSL.Saravanan, Prof. of Physiology
   Vice Principal i/c, Madurai Medical College — Member
3. Dr.P.Raja,MD., Prof. of Urology
   Medical Superintendent i/c, — Member
4. Dr.V.T.Premkumar, MD., (General Medicine)
   Professor and Head of General Medicine — Member
5. Dr.S.R.Dhamotharan, M.S.,
   Professor and HOD of Surgery i/c, Govt. Rajaji Hospital — Member
6. Dr.Sharmila Thilagavathi, MD., Professor of Pathology,
   Madurai Medical College, Madurai — Member
7. Thiru P.K.M.Chellaiah, Business man — Member

Total No. of candidates presented their Research Protocal to Committee : 20 Nos.

After perfect discussion of the subjects, Approval was given by the members

Projects approved	:	4
Pending Approval	:	15
Represent	:	1
Absentees	:	Nil

Member Secretary	Chairman	Dean/Convenor
Ethical Committee	Ethical Committee	Ethical committee

Minutes of the Research Protocal by the committee

Sl. No	Name of the Candidate	Title of Study	comments
1	Dr.S.Sumithra Assistant Professor of DVL Madurai Medical College, Madurai sumibala15@yahoo.co.in 9865846226‘	Undiagnosed musculoskeletal disorders in psoriasis	Pending approval Modify title as incidence / prevalence of musculoskeletal disorders in psoriasis - send mail
2	Dr.Mohan Raj. V PG in MD., Paediatrics Madurai Medical College & Govt. Rajaji Hospital, Madurai mohanraj1704@gmail.com 9865160865	Serum ferritin as an early indicator for severity of dengue	Pending approval WHO guidelines regarding the elevated ferritin to be highlighted Correlate ferritin with severity of infection Message to be added to send mail
3	Dr.J.S.Rasiga Thivya PG in MS., Ophthalmology Madurai Medical College & Govt. Rajaji Hospital, Madurai ramuso3o7@gmail.com 9994595526	A study to analyse the association between timing of surfactant administration and retinopathy of prematurity (ROP)	Pending approval 1. Specify the surfactant used 2. Indication, contraindications, side effects of the surfactant Compare prophylactic verses therapeutic – send mail for approval
4	Dr.Karthik pandian.T PG in MD., General Medicine Madurai Medical College & Govt. Rajaji Hospital, Madurai karthi0493@gmail.com 9994278762	Severity and prognosis of acute organophosphorus pesticide poisoning as indicated by C-reactive Protein	Pending approval 1. Cut off value for C-reactive Protein to be specified 2. CRP to be measure at the time of admission before starting treatment 3. Include scoring for severity - send mail for approval

5	Dr.S.Naveen PG in Mch., (Cardio thoracic surgery) Madurai Medical College & Govt. Rajaji Hospital, Madurai naveensrinivasan89@gmail.com 9944980898	Post conditioning the human heart with adenosine : study of its effects in open Heart surgery	Pending approval 1.Use approved dose of adenosine 2.Send a copy of consent form
6	Dr.P.Jayadurga devi PG in MS., Ophthalmology Madurai Medical College & Govt. Rajaji Hospital, Madurai drdurgambbs@gmail.com 9655849042	An observational study to study the pattern of optic nerve head filling in fundus flourescein angiography in hypertensive patients with normal and crowded optic disc	Pending approval Mention the novelty Include grading, statistical analysis
7	Mr.Abhishekkumar Ramasamy, 210 Vaigai Colony, Anna Nagar, Madurai.	Wound Regeneration analysis with polymer and proteins	Approved
8	Dr.B.Karthikeyan, PG in Mch., (Cardio vascular thoraci surgery) Madurai Medical College & Govt. Rajaji Hospital, Madurai karthikeyanctsmbbs@gmail.com 9003156763	Lactate level during cardiopulmonary bypass and immediate post operative period as a predictor of postoperative outcomes in patients undergoing open heart surgery	Pending approval Restrict your aim and include statistic- send mail for approval
9	Dr.Harshini G.V. PG in MS., Ophthalmology Madurai Medical College & Govt. Rajaji Hospital, Madurai harshinipankaj1992@gmail.com 9159936093	An observational study to determine association between posterior vitreous detachment and proliferative diabetic retinopathy	Approved
10	Dr.Ranjithkanna .M PG in MD., General Medicine Madurai Medical College & Govt. Rajaji Hospital, Madurai drranjithkanna@gmail.com 8825716195	Usefulness of glycated albumin as a biomarker for glucose control and prognostic factor in chronic kidney disease patients on hemodialysis (CKD –G5D)	Pending approval Add statistics highlight Modify and send mail for approval

11	Dr.N.J.Niroshini, PG in MD., Paediatrics Madurai Medical College & Govt. Rajaji Hospital, Madurai niroshini17@gmail.com 9790894017	Correlation of proteinuria and urine Protein / Creatinine Ratio with disease severity in pediatric dengue fever.	Approved
12	Dr.Muthu .A PG in MD., General Medicine Madurai Medical College & Govt. Rajaji Hospital, Madurai muthustan@gmail.com 9626228847	A cross sectional study of correlation between poor lung function and early markers of atherosclerosis (microalbuminuria , carotid, intima medial thickness and ankle –brachial index)	Pending approval Include younger age group with restricted lung pathology. Modify and send
13	Dr.R.Giridharan, PG in MS., General Surgery Madurai Medical College & Govt. Rajaji Hospital, Madurai rgridharan@gmail.com 9840128109	Evaluation of the use of subcuteneous drains to prevent wound complications in emergency abdominal surgeries.	Pending approval Include statistical analysis to be send mail
14	Dr.N.Murugan, PG in MS., Otorhinolaryngology Madurai Medical College & Govt. Rajaji Hospital, Madurai murugansn87@gmail.com 9787494549	A study on outcome ossiculoplasty in surgeries of CSOM with ossicular erosion	Pending approval Certificate regarding Bio Medical norms of the prosthesis used – send mail for approval
15	Dr.R.Pushpavani, PG in MD., DVL Madurai Medical College & Govt. Rajaji Hospital, Madurai pushpavaniravi@gmail.com 9789339503	A study on prevalence of complicated and uncomplicated vulvovaginal candidiasis among women attending STD clinic at Govt. Rajaji Hospital, Madurai	Pending approval Include diabetic patients Species correlation to be added modify and send

16	Dr.N.Santhanam, PG in MD., General Medicine Madurai Medical College & Govt. Rajaji Hospital, Madurai dr.santhanam07@gmail.com 9944497529	MRI appearance and clinical correlative findings in assessing the recovery of patients from subarachnoid hemorrhage	Represent
17	Dr.C. Arunraj PG in MS., , Otorhinolaryngology Madurai Medical College & Govt. Rajaji Hospital, Madurai arunraj2588@gmail.com 7598250001	Study of Post operative outcome in open septorhinoplasty	Pending approval Include scoring of olfaction before and after surgery -
18	Saranya PG in MD., DVL Madurai Medical College & Govt. Rajaji Hospital, Madurai saran8390@gmail.com 9566963228	A study on cutaneous adverse reactions to imatinib	Pending approval
19	Dr.Jananipriya PG in MS., Obstetrics & Gynaecology, Madurai Medical College & Govt. Rajaji Hospital, Madurai jansanghtmjs@gmail.com 9940881649	A prospective study of programmed labour protocol in nonsevere preeclampsia	Pending approval Define low risk Modify and send
20	Dr.T.Amulprathap PG in MD., DVL Madurai Medical College & Govt.Rajaji Hospital, Madurai amulprathap@yahoo.com 9940852058	Comparative study of methotrexate and apremilast in the treatment of moderate to severe plaque psoriasis	Approved

Member Secretary
Ethical Committee

Chairman
Ethical Committee

DEAN/CONVENOR
Ethical Committee

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