Dissertation on

A PROSPECTIVE AND RETROSPECTIVE STUDY ON VITREOUS HAEMORRHAGE AND MANAGEMENT IN A TERTIARY EYE CARE CENTRE

Submitted in partial fulfillment of requirements of

M.S. OPHTHALMOLOGY

BRANCH - III

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI- 600 003

CERTIFICATE

This is to certify that this dissertion entitled **"A prospective and retrospective study on vitreous haemorrhage and management in a Tertiary Eye Care Centre"** is a bonafide record of the research work done by Dr. S. Bharathi rajan, post graduate in Regional institute of ophthalmology and Govt. Ophthalmic Hospital, madras Medical College and Govt. General Hospital, Chennai-03 in partial fulfillment of the regulation laid by the Tamilnadu Dr. M.G.R Medical university for the award of M.S. Ophthamology Branch III, under my guidance and supervision during the academic years 2015 – 2018.

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My deepest gratitude goes to my alma mater, Regional institute of ophthalmology and Govt. Ophthalmic Hospital, Chennai, from where I the learnt the science of ophthalmology.

INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

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Dear Dr.Bharathi Rajan.S,

The Institutional Ethics Committee has considered your request and approved your study titled **"A PROSPECTIVE AND RETROSPECTIVE STUDY ON VITREOUS HAEMORRHAGE AND MANAGEMENT IN A TERTIARY EYE CARE CENTRE" - NO.07022017 (II)**

The following members of Ethics Committee were present in the meeting hold on **21.02.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
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We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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DECLARATIONS BY THE CANDIDATE

In hereby declare that this dissertation entitled **"A prospective and retrospective study on vitreous haemorrhage and management in a Tertiary Eye Care Centre"** is a bonafide and genuine research work carried out by me. This is submitted to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the awards of M.S. degree.

Place : Chennai Dr. S. BHARATHI RAJAN

Date :

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INTRODUCTION

Vitreous hemorrhage is defined as the presence of extravasated blood in the vitreous cavity. The source of the blood may be due to various etiology and pathologic mechanism. The anatomical, physiological and biochemical properties of the vitreous affects the blood catabolism in a different way from other tissues so the vitreous hemorrhage differs from other tissues of the body.

Hence it is appropriate to discuss the text in the following format prior to the discussion of the vitreous hemorrhage:

- Anatomy of the vitreous cavity
- Physiology of the vitreous
- Morphology and composition of the vitreous
- Physiochemical properties of the vitreous

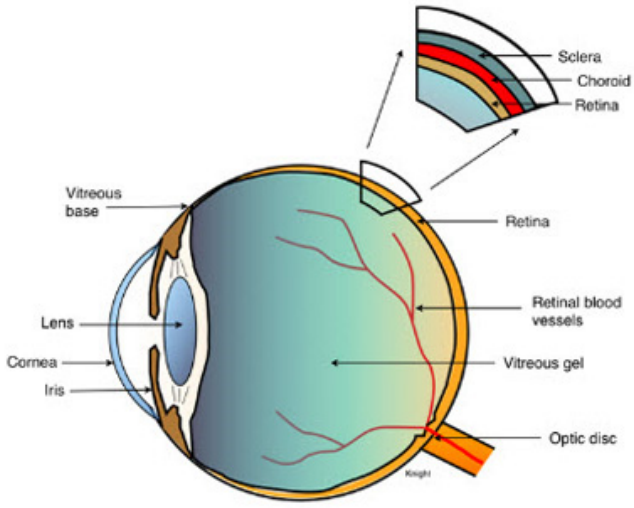
ANATOMY OF THE VITREOUS CAVITY

The vitreous cavity is bounded anteriorly by the lens and the ciliary body. Posteriorly by the retina. Its volume is 4cc about 2/3 volume of the globe. It is a gel like substance made up of 99% of water. Vitreous base extends 2mm anteriorly to pars plana and 4mm posterior to ora serrata. The outer or the cortical layer of the vitreous contains a small number of cells termed as halocytes. The concentration of the halocytes is more

around the vitreous base. They have the phagocytic activity and produce hyaluronic acid.

A delicate meshwork of collagen fiber is present along with the hyaluronic acid and gives the vitreous its rigidity and viscosity. This collagen meshwork is less dense in the centre than in the periphery. It is most dense at the vitreous base. The strongest attachment is at the pars plana and the retina periphery. The vitreous is firmly attached to the peri papillary region and less firmly to the posterior surface of the lens and the macula. This is called hyaloidcapsular ligament of weigert.

The vitreous around the blood vessels is irregularly arranged. The anterior hyaloid membrane is formed by the anterior condensation of the peripheral vitreous .the vitreous condensation posterior to the vitreous base produces the posterior hyaloid membrane. The vitreous fibrils are arranged parallel to the retina elsewhere except at the vitreous base where it is attached perpendicular to the retinal surface.



VITREOUS ATTACHMENTS

1. Optic Disc
2. Fovea
3. Retinal Vessels
4. Vitreous Base

EMBRYOLOGY

During the first month of gestation the primary vitreous contains the mesodermally derived hyaloid vascular system and the ectodermally derived fibrillar meshwork present between the lens and the retina.

By the second month of gestation the collagen and the hyaluronic acid develops forming the secondary vitreous called the adult vitreous. The vitreous cavity starts filling by secondary vitreous the 14^{th} week of gestation and gets completed by 7^{th} month of gestation. The cloquet's canal which is nothing but the remnant of the primary vitreous extends from the posterior surface of the lens to the optic disc in a S shaped pattern.

VITREOUS BARRIERS

Vitreoretinal barrier is the term used describe that the vitreous constituents cannot equilibriate with the blood and with surrounding fluids. The mechanism involved are

1. Tight junctional complexes inhibiting the transport of high molecular weight components at the level of non pigment epithelium of ciliary body, pigment epithelium of retina and the retinal vascular endothelium.
2. At the above sites the active transport also plays the role.
3. Physical blockage of large molecules at the basal lamina of the vitreoretinal junction.
4. The collagen fibrillary network has a specific physiochemical property which also plays a major role in the transport to effectively block or retard the movement of the cells.

PHYSIOLOGY OF THE VITREOUS

The vitreous provides an optically negative space occupying the posterior 2/3 of the globe for the light rays to travel to the retina focused by the cornea and the lens.

The hyalocytes and the fibrocytes are specific cellular element present in the cortical layer lying in the collagen network. The major morphological development is over before the birth but the biochemical properties are gained till reaching the adulthood. The vitreous differs from aqeous by its physical and biochemical properties.

MORPHOLOGY AND COMPOSTION

The vitreous is almost a fully hydrated gel with 95% of water content. It consists of cortical layer of cells and collagen. The hyalocytes are concentrated more in the narrow zone adjacent to the retina and the ciliary body. The fibrocytes are located next to the optic disc. These cells are the centre for the metabolism. The hyalocytes have a phagocytic function and are the source of extracellular hyaluronic acid.The components of vitreous body is divided into

1. Macromolecular constituents
2. Low macromolecular constituents.

Macromolecular constituents

The various macromolecular constituents are:

A. Collagen

It is an insoluble protein obtained from the vitreous by high speed centrifugation or by filtration. It is present more near the vitreous base and in the cortical areas adjacent to the retina.

It has certain specific features of typical collagen

1. It is soluble in boiling water.
2. May be hydrolysed to form gel
3. Can be degraded by collagenase.

4. Has typical x-ray diffraction pattern.

It has certain atypical features like

1. Those insoluble collagen fibrils are thinner.
2. They do not show the usual banding pattern of collagen.
3. Its has a slightly higher glycoprotein and nitrogen content.
4. Can be degraded by proteolytic enzymes.

B. Soluble proteins

The soluble proteins are disproportionate amount of acidic glycoproteins ,when compared to aqueous or serum. Its concentration is more in the cortical area adjacent to the retina.

C. Hyaluronic acid

It is also known as glycos aminoglycans. It is a linearly arranged polymer consisting of glucoronic acid and N-acetyl glycosamine. The hyaluronic acid is a negatively charged molecule and its coils upon itself. It produces a large sponge like spheroidal configuration that can bind more than fifty times its weight in water.

Low molecular weight components

The concentration of the various component is similar to that of aqueous or serum with minor differences, which are due to the various metabolic or transport activities of the vitreous and other ocular tissues.

A. Water

The turnover of vitreous water is extremely high, half of that present is replaced every 10 to 15 minutes.

B. Sodium

As sodium is distributed passively , its concentration in vitreous is same as that of other intraocular extracellular fluids.

C. Potassium

The active transport of potassium from the anterior surface of the lens and passive diffusion from the posterior surface into the vitreous leads to the increased potassium concentration in the vitreous.

D. Bicarbonate

Due to the active metabolism of the surrounding tissues its level is low.

E. Glucose

It diffuses from the ciliary body and the retina into the vitreous cavity.

F. Amino acid

Except glutamic acid all other amino acids are at lower concentration this is because of the uptake and usage by the cortical hyalocytes.

G. Ascorbic acid

It is much higher concentration in the vitreous than in the blood due to the active transport of ascorbic acid by ciliary epithelium from the blood. with copper and oxygen it forms a polymerization system that plays a role in vitreous hyaluronic acid turnover.

PHYSICOCHEMICAL PROPERTIES

They are responsible for the following properties

1. Rigidity, viscosity and elasticity of the vitreous gel.
2. vitreous transparency
3. volume change properties
4. vitreous cell distribution in the cortical layers.

The physicochemical properties are

- **Frictional interaction**

The dual network consisting of collagen fibres and hyaluronic acid filling the spaces between the fibres forms a stable system that can resist

disorganizing forces like centrifugation. The ineraction between the two provides the stable structure.

- **Vitreous expansion and contraction**

Normal vitreous contains sodium ions and sodium chloride molecules that interlies the negatively charged hyaluronic acid. If positively charged molecules introduced the gel will contract and collapse.

If sodium chloride molecules removed the entire gel will expand because the hyaluronic acid molecules are free to repel one another.

- **Excluded volume concept**

The vitreous is able to restrict the diffusion of cellular element because of the collagen fibrils and large sized hyaluronic acid.

- **Molecular sieve effect**

The cortical vitreous can even retard the flow of low molecular weight component the positively charged molecule are trapped by the electrostatic field of poly anionic chains of hyaluronic acid.

VITREOUS HEMORRHAGE

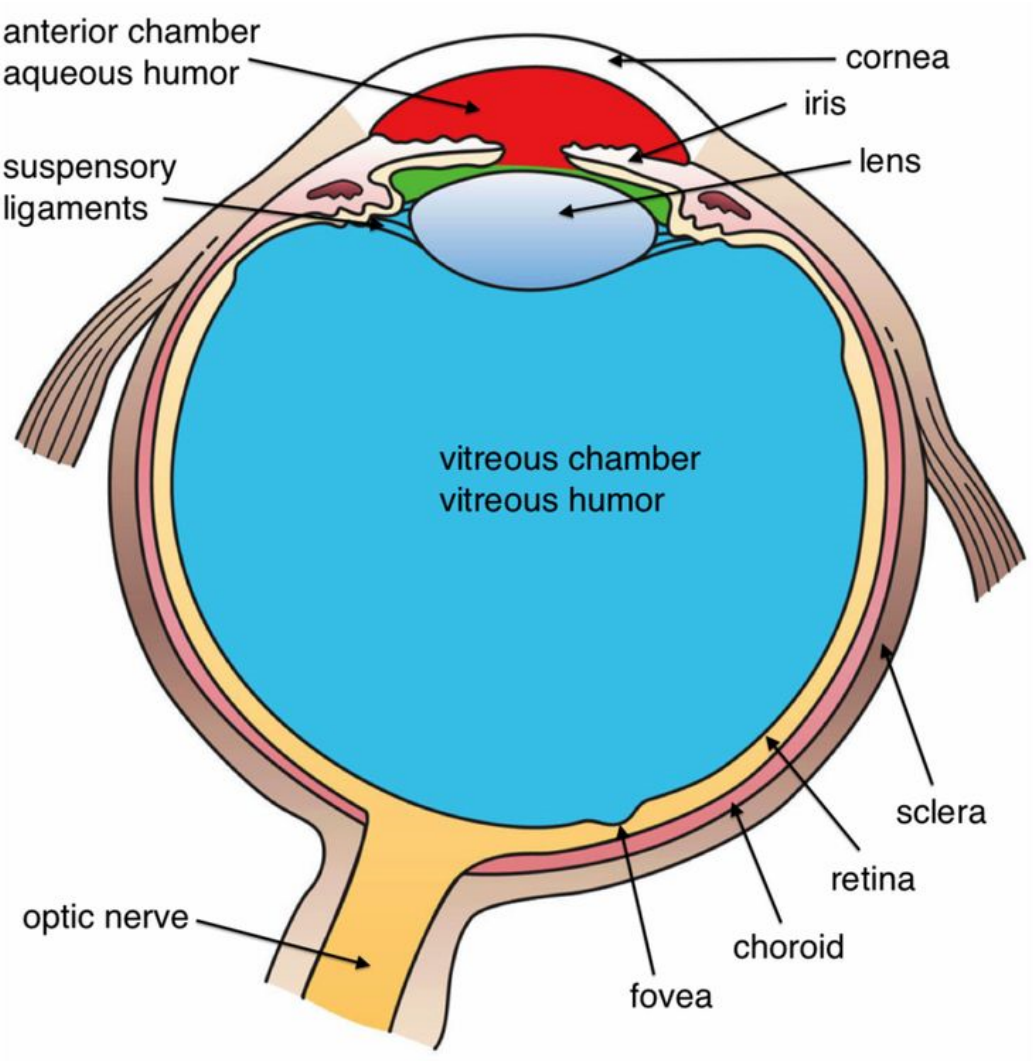
Historical review

Helmholtz invent the ophthalmoscope in 1850 but it was not used for many years Gullstrand invent the first crude version of slit lamp in 1910 until early part of century the studies about vitreous hemorrhage were done with little clinical correlation.

The first experimental study of vitreous hemorrhage were performed more than 100 years ago by Probsting and Von hippel. Those authors described findings after injection of blood into the vitreous of rabbits. Sub choroidal hemorrhage, one of the earliest conditions reported to associated with vitreous hemorrhage ,was first described by Litten in 1881 and named after Terson in 1900.

Before the advent of vitrectomy there were no treatment for non resolving vitreous hemorrhage. In 1967, Duke Elder described that proliferative diabetic retinopathy and vitreous hemorrhage are untreatable and not preventable.

This dramatically changed with the introduction of xenon and argon laser photocoagulation. In 1962 Kasner, used open sky vitrectomy. Machemer performed first human pars plana vitrectomy on april 20,1970.



Vitreous Humour Anatomy

DEFINITION

Vitreous hemorrhage is defined as the presence of extravasated blood within the space outlined by internal limiting membrane of retina posteriorly and laterally, Non pigmented epithelium of ciliary body laterally and the lens zonular fibres and posterior lens capsule anteriorly.

Site:

1. within formed vitreous
2. Bergers space [retrolental space of Erggelet]
3. Canal of petit
4. Cloquets canal
5. Bursapremacularis
6. Retro hyaloid or sub hyaloid space

INCIDENCE

7 cases/lakh population each year.

ETIOLOGY

1. Proliferative diabetic retinopathy
2. Ocular trauma
3. Eales disease
4. Vitreous detachment with or without retinal tear, retinal detachment
5. Vitreous hemorrhage secondary to retinal vein occlusion.
6. Proliferative sickle cell retinopathy
7. Retinalmacroaneurysm
8. Age related macular degeneration
9. Subarachnoid hemorrhage

Inflammatory causes

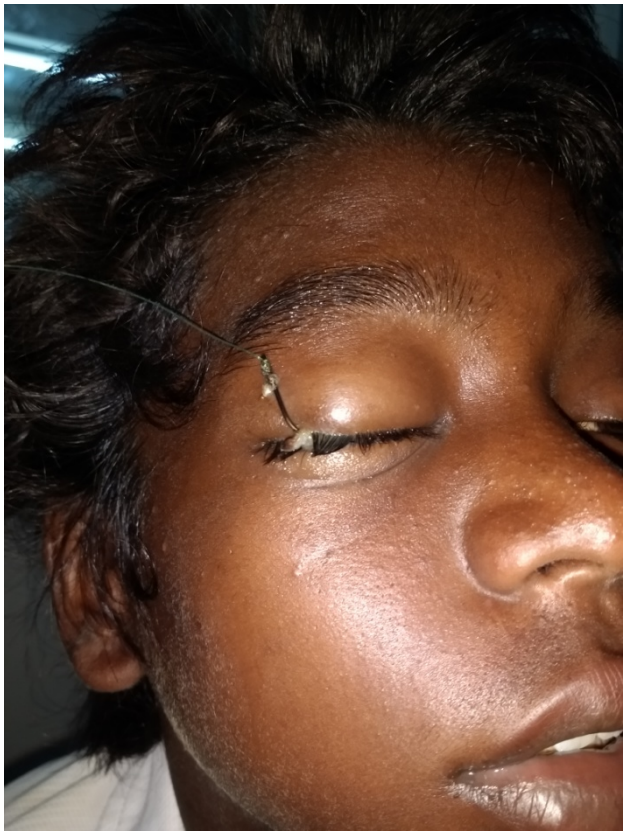
1. Bechets disease
2. Retinal vasculitis
3. Parsplanitis
4. Syphilitic retinitis
5. Systemic lupus erythematosus

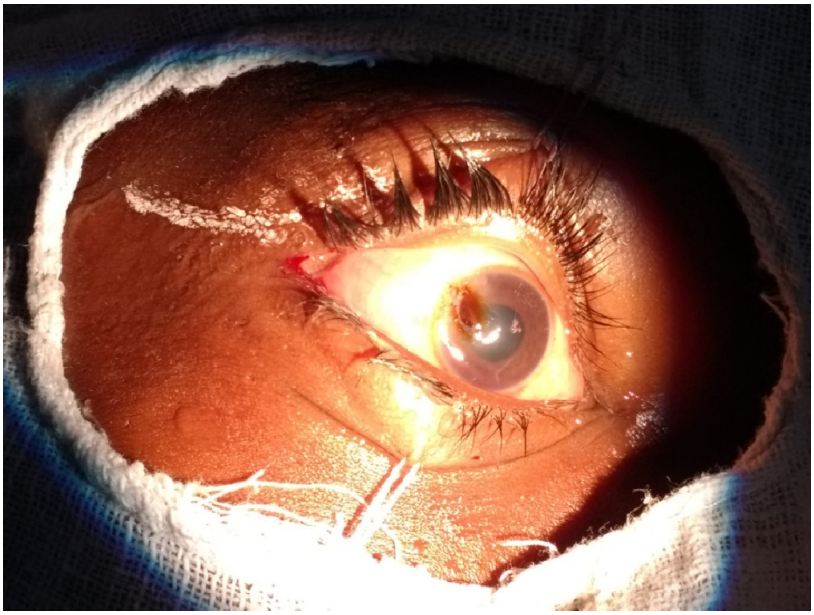
Vascular causes

1. Coats disease
2. Retinal vein rupture
3. Hypertensive retinopathy
4. Venous stasis retinopathy
5. Sarcoid posterior uveitis
6. Retinopathy of prematurity

Blunt Trauma with Vitreous Haemorrhage

Penetrating injury with Vitreous Haemorrhage





Blood disorder

1. Anemia
2. Thrombocytopenia
3. Idiopathic thrombocytopenic purpura
4. Thrombotic thrombocytopenic purpura
5. Disseminated intravascular coagulation
6. Hemophilia
7. Leukemia
8. Anti coagulant therapy

Tumor

1. Choroidal malignant melanoma
2. Retinoblastoma
3. Cavernous hemangioma of optic disc
4. Retinalangioma

Other causes

1. adverse effect of retinal laser photocoagulation .
2. After cataract surgery
3. After trabeculectomy
4. After secondary iol implantation, posterior chamber implantation
5. Penetrating keratoplasty

6. Bulbous perforation during retrobulbar, peribulbar injections surgeries
7. Alteration of iris vessels by prolapsed pciol
8. pseudo tumour cerebri
9. Valsalva retinopathy
10. chest compression
11. physical exertion
12. Retinitis pigmentosa
13. Tearing of retinal pigment epithelium
14. Topical pilocarpine therapy

CLINICAL FEATURES

Symptoms

1. Visual haze
2. Floaters
3. Smoke signals
4. Photophobia
5. Perception of shadows
6. Flashes of light
7. Defective/loss of vision

Signs

Visual Acuity

Visual acuity is determined by the location and density of the hemorrhage.

Ophthalmic examination

Blood is found within vitreous gel/anterior hyaloid retrohyaloid space.

Blood within berger's space settles down and forms crescent shaped pool with hyaloideo capsular ligament since inferior border. Hemorrhage into canal of petit has crescent shaped superior border. Blood in cloquet's canal outline its inferior border. Blood in retrohyaloid space collect as meniscus at inferior retrohyaloid. Intravitreal hemorrhage shows no characteristic border and rapidly clots.

B SCAN

Determination of location and density of vitreous hemorrhage , location and extent of traction membrane and retinal detachment and virtreo retinal relationship.

CT SCAN AND MRI

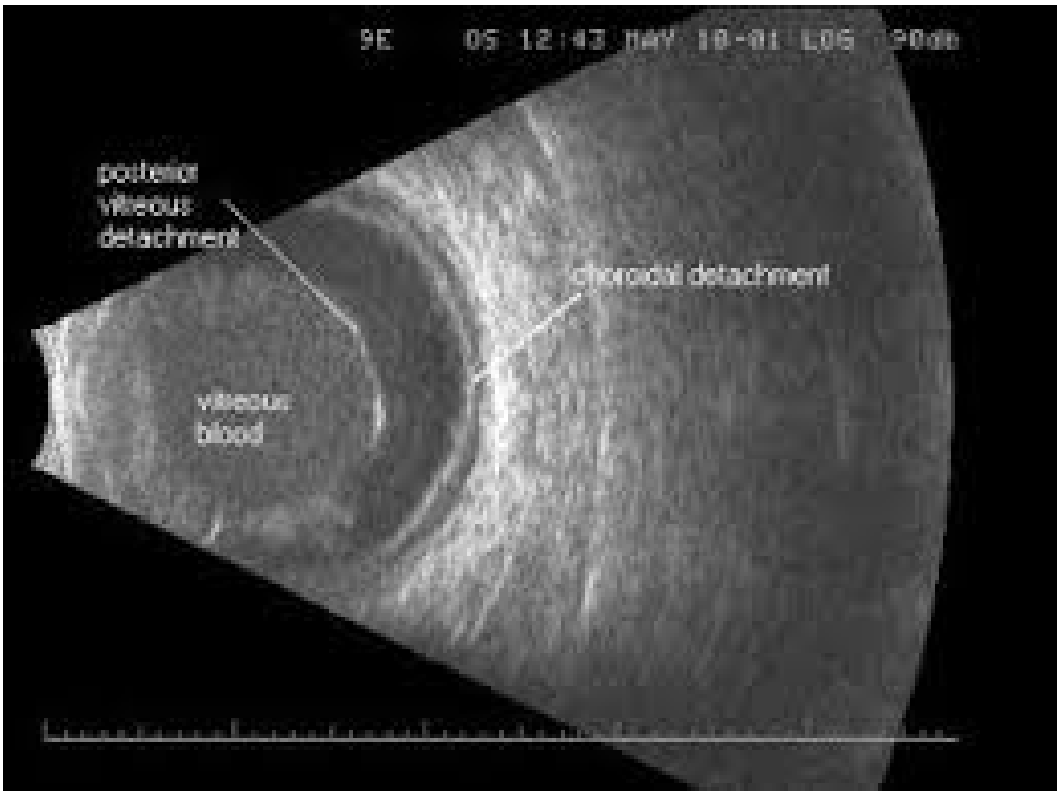
Hemorrhages in other location such as brain to vitreous. Pathological mechanism

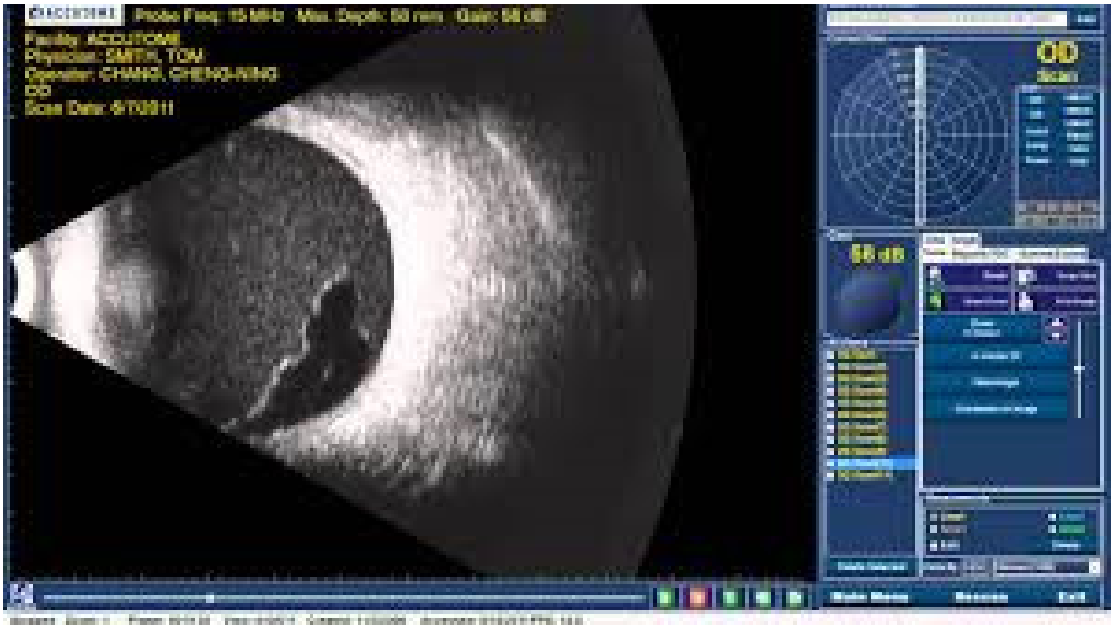
Bleeding from diseased retinal vessels/abnormal new vessels:

Retinal ischemia results in new vessels by production of angio genic factors.

- Proliferative diabetic retinopathy
- Proliferative sickle cell retinopathy
- Retinopathy of prematurity
- Rupture of normal retinal vessels:
- Retinal break or posterior vitreous detachment as in case of trauma.
- Extension of hemorrhage through retina from other sources.
- Subretinal bleeding with secondary break through into vitreous cavity as in ARMD and choroidal melanoma.

B-Scan Ultrasonography Shows Dense Vitreous haemorrhage





PATHOPHYSIOLOGY OF BLOOD CATABOLISM IN VITREOUS:

It differs from hemorrhage into other tissue outside eye.

- Rapid clot formation with short borders
- Slow lysis of fibrin.
- Extracellular lysis of red blood cells.
- Persistence of intact red blood cells for months
- Lack of early PMN response

RAPID CLOT FORMATION:

Bleeding into vitreous gel results in rapid formation of clot with short borders although tissue thromboplastin activity is low.

Vitreous collagen facilitates platelet aggregation and enhancement of clotting via intrinsic clotting process and membrane formation.

Passive diffusion of red cells and fibrin is inhibited because of lattice work of vitreous collagen fibres with large hyaluronic acid molecules with large hyaluronic acid molecules in inter spaces.

Thromboplastin activity in vitreous is 5.5*,fibrinolytic activity in vitreous is 0”

SLOW LYSIS OF FIBRIN

Lysis of fibrin in formed vitreous is very slow because of low level of vitreal tissue fibrinolytic activity on lack of pmn response.

EXTRACELLULAR LYSIS OF RED BLOOD CELL:

Erythrocytes in vitreous undergo hemolysis either by extracellular release of lysosomal enzymes by macrophages or auto hemolysis.

PERSISTANCE OF INTACT RED BLODD CELLS FOR MONTHS:

Red cells possess metabolic requirements to adjust special conditions in vitreous and fail to elicit macrophage reaction.

LACK OF EARLY POLYMORPHONUCLEAR LEUCOCYTE RESPONSE:

This results in persistence of fibrin this in turn leads to lack of fibrin degradation products a stimulator of PMN. This cellular response leads to phagocytosis of erythrocytic debris than whole erythrocytes.

NATURAL COURSE OF VITREOUS HEMORRHAGE:

Depends on the site of vitreous hemorrhage.

1. hemorrhages into spaces outside the formed vitreous:

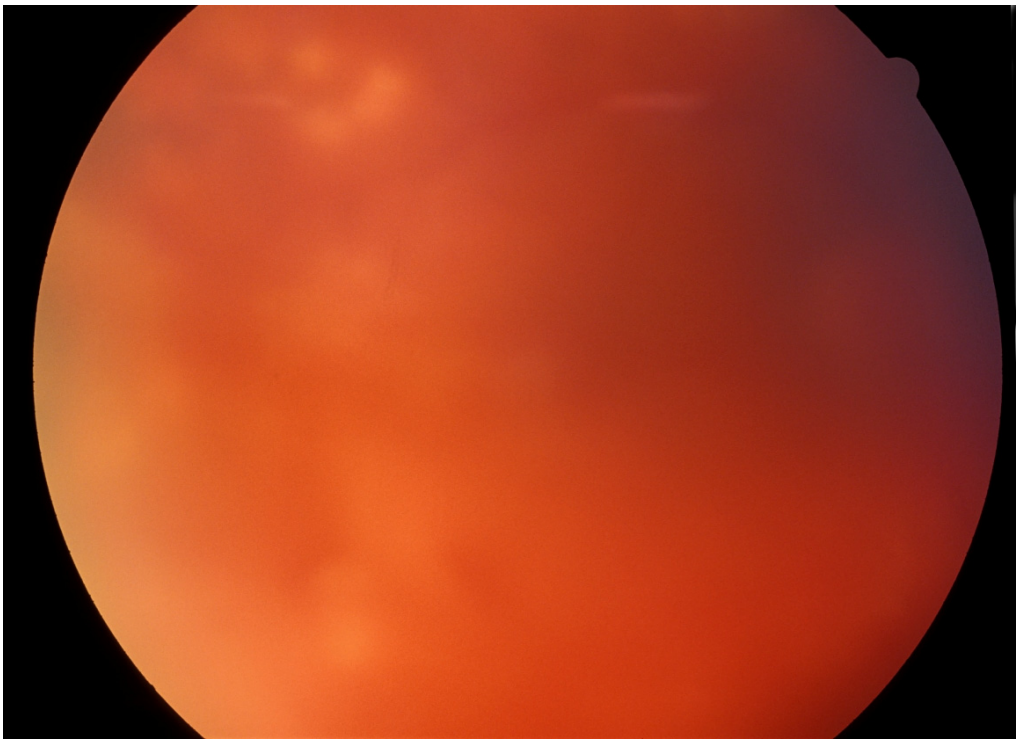
It gets absorbed readily in cases of sub or retro hyaloid hemorrhage resorption of hemorrhage leaves only few deposits of fibrin on posterior surface of detached vitreous this is described assilk like veils.

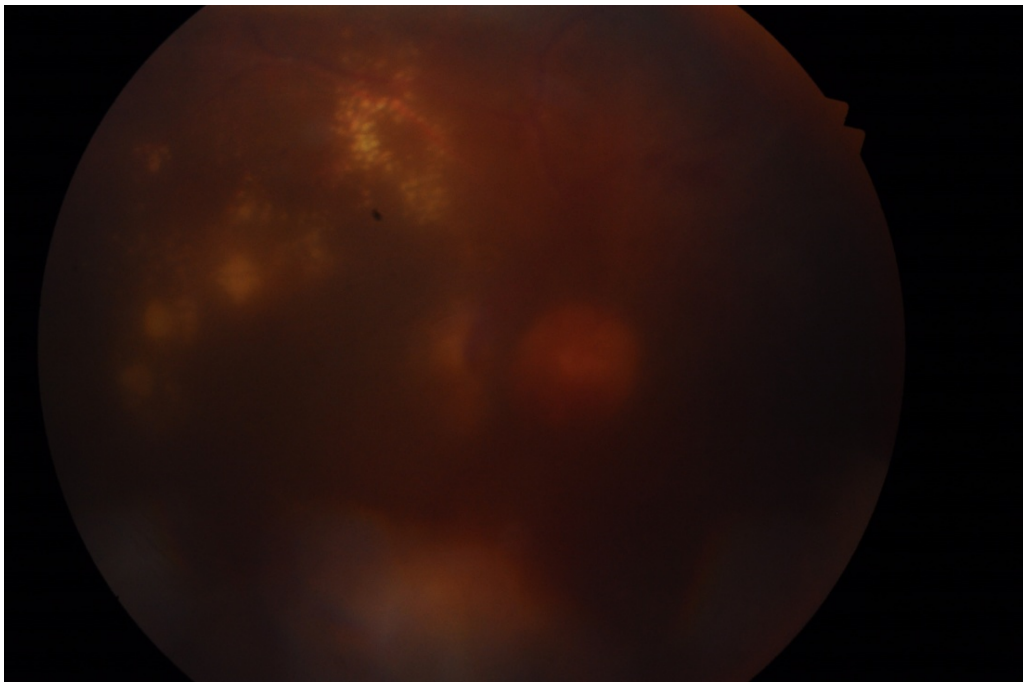
2. Hemorrhage into the formed vitreous gel:

It clears very slowly with time constant 1percent per day. Intra vitreal hemorrhage is followed by rapid clotting mediated by contact of platelets with intra vitreal collagen with slow fibrinolysis.

Fibrinolysis of clotted blood diffusion of erythrocytes out of blood clot, extracellular hemolysis limits resorbtion of vitreous hemorrhage. clotted blood in vitreous gel remains in lamellar fashion until vitreous liquefies and the blood sinks to bottom of vitreous cavity where it is absorbed. Accumulation of red blood cells and red cell debris suspended in and mixed with vitreous collagen can present as ‘ochre membrane’.

Iindirect ophthalmoscope showing dense vitreous haemorrhage





SYNCHISIS SCINTILLANS:

Muticoloured glittering particles settles in lower part of vitreous cavity due to gravity but can be thrown up by eye movements to form shower of iridescence this occurs in long standing vitreous hemorrhage. It represents cholesterol crystals as a result of break down of red blood cells.

VITTREOUS CYLINDERS

It is secondary to vitreous hemorrhage. Tubular structures develop from detached vitreous lamella that condenses to form cylinder structures.

PROGNOSIS:

It depends on site, density, recurrence of vitreous hemorrhage

1. avulsion of vessel associated retinal tear or posterior vitreous detachment has good prognosis since there is no tendency of recurrent bleeding.
2. Proliferative diabetic retinopathy has worst prognosis due to recurrent/persistent vitreous hemorrhage.
3. Retinal vein occlusion.

   Central vein occlusion has worst prognosis

   Branch retinal vein occlusion has best prognosis.

   Hemi retinal vein occlusion has prognosis in between two

4. Proliferative sickle cell retinopathy shows better prognosis as it affects peripheral vessels of retina.
5. vitreous hemorrhage secondary to ARMD has poor prognosis.

VITREOUS HEMORRHAGE IN BLUNT TRAUMA

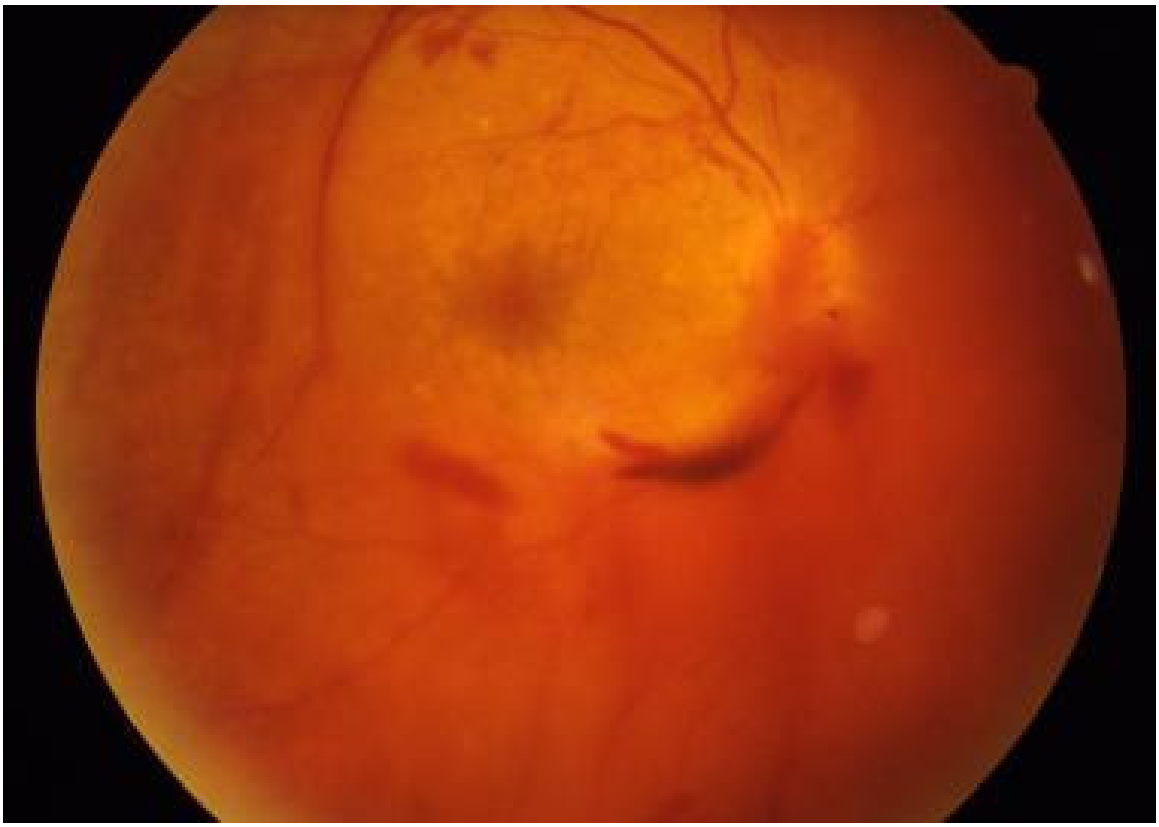
BLUNT TRAUMA:

The objects causing the blunt trauma do not penetrate the eye but cause rupture of the eye wall. It can have number of sequelae :

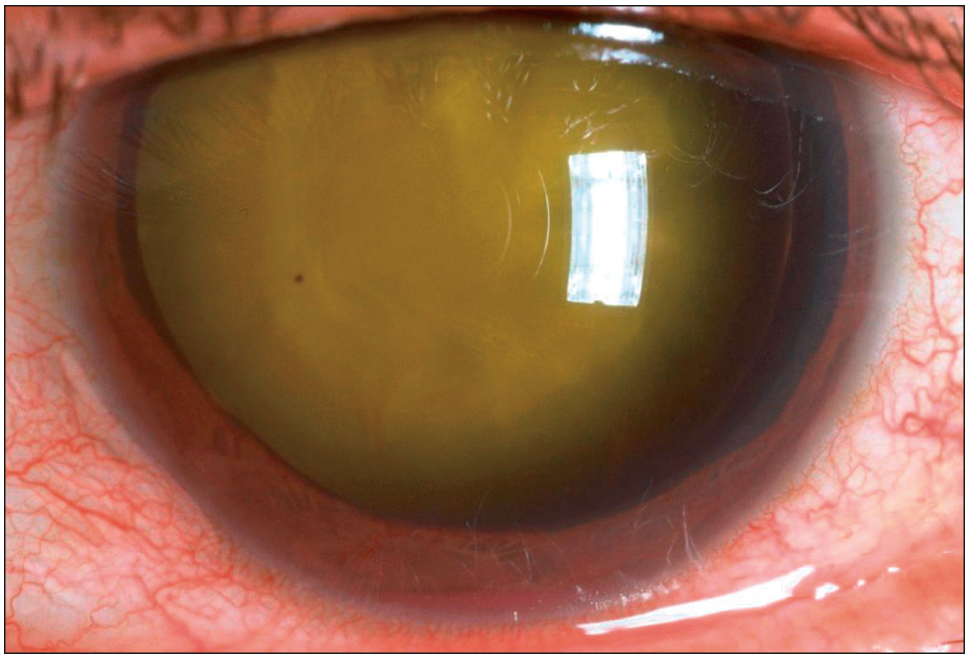
1. angle recession
2. hemorrhege into the anterior chamber or the vitreous
3. retinal tears or the detachment
4. subluxated or the dislocated lens
5. commotio retinae
6. choroidal rupture
7. macular hole
8. avulsed optic nerve
9. scleral rupture

A complete ophthalmic examination is required for a blunt trauma because an eye without hyphaema or iritis can have a retinal tear or choroidal rupture or blowout fracture.

Choroidal Detachment Following Blunt Trauma



Slit lamp examination shows yellowish Hue in case of Vitreous Haemorrhage



VITREOUS HEMORRHAGE

Vitreous hemorrhage results from the damage to the blood vessels of iris, ciliary body ,retina ,choroid. A search for the cause of the hemorrhage should be undertaken as sometimes a hemorrhage which is located at time of presentation can become diffuse later. So an initial thorough IDO examination is required. If posterior segment could not be seen because of vitreous hemorrhage ultrasound examination is necessary. Retinal or choroidal detachment or tear and posterior vitreous detachment can be identified. Echographic sign of occult scleral rupture include vitreous strands that feed into the rupture site.

More often bed rest with elevation of patients head is sufficient to allow the blood to settle down for detailed ophthalmoscopic examination. If the source of the hemorrhage is still not identified regular follow up with ultrasound examination is indicated until the hemorrhage clears. Macular hole, choroidal rupture, traumatic maculopathy, retinal detachment or their injuries can limit the visual outcome.

COMMOTIO RETINAE:

The term commotion retinae refers to damage to the outer retinal layers caused by the shock waves that traverse from the site of impact following blunt trauma. Ophthalmoscopic examination reveals the sheen like retinal whitening that appears some hours after the injury. The retinal

whitening is mostly found in the posterior pole but also in the periphery as well. Mechanism for retinal opacification includes extracellular edema, glial swelling and photoreceptor disruption. With involvement of the fovea cherry red spot appears as the cells involved in the whitening are not present in the foveola.

Commotio retinae in the posterior pole is called the berlins edema that may reduce the visual acuity to as low as 20/200. The prognosis of the vision is good as the condition clears in 3-4weeks.in some cases visual prognosis is limited by the presence of macular pigment epitheliopathy, choroidal rupture or macular hole formation. No effective treatment is known.

COMPLICATIONS:

1. siderosis bulbi and retinal damage

sub retinal hemorrhage irreversibly damage photoreceptor cells this leads to cell lysis with release of iron and hemoglobin leading to retinal degeneration.

2. Proliferative retinopathy:

It is caused by phagocyte mediated stimulation of retinal glial cells vascular endothelial cells and retinal pigment epithelial cells. Once pre retinal proliferation are established recurrent bleeding may cause vitreous

collapse with traction on newly formed vessels and subsequent recurrent hemorrhage.

3. Glaucoma:

It is rare complication.

A. Ghost cell glaucoma:

Following vitreous hemorrhage fresh RBCs degenerate into ghost cell within one to three weeks and obstruct trabecular meshwork causing secondary open glaucoma.

B. Hemosiderotic glaucoama

hemosiderotic glaucoama is a late onset glaucoma following a trauma that causes intraocular hemorrhage with iron deposition and damage to the trabecular meshwork. This is an extremely rare glaucoma and is chronic, does not have ghost cells in the anterior chamber. Usually occurs many years following trauma. In contrast to the ghost cell glaucoma which occurs within months or weeks following trauma.

C. Hemolytic glaucoma

Hemolytic glaucoma is a type of secondary glaucoma occurs following vitreous hemorrhage due to the obstruction of the trabecular mesh work by red blood cell debris and the macrophages.

MANAGEMENT

Principles of management:

1. To arrest bleeding from vessels
2. To accelerate absorption of formed blood
3. In non clearing vitreous hemorrhage surgical intervention

TYPES

1. Conservative
2. Surgical

CONSERVATIVE MANAGEMENT:

In case of retro hyaloid hemorrhage blood resorption occurs rapidly. Normally if eye movements are restricted blood in space gravitates to bottom and collects as meniscus at inferior vitreoretinal demarcation hence management includes binocular patching an position the patient vertically.

PHOTOCOAGULATION

Principle is to arrest the bleeding and prevent the recurrence. It is indicated in proliferative diabetic retinopathy,BRVO, sickle cell retinopathy.

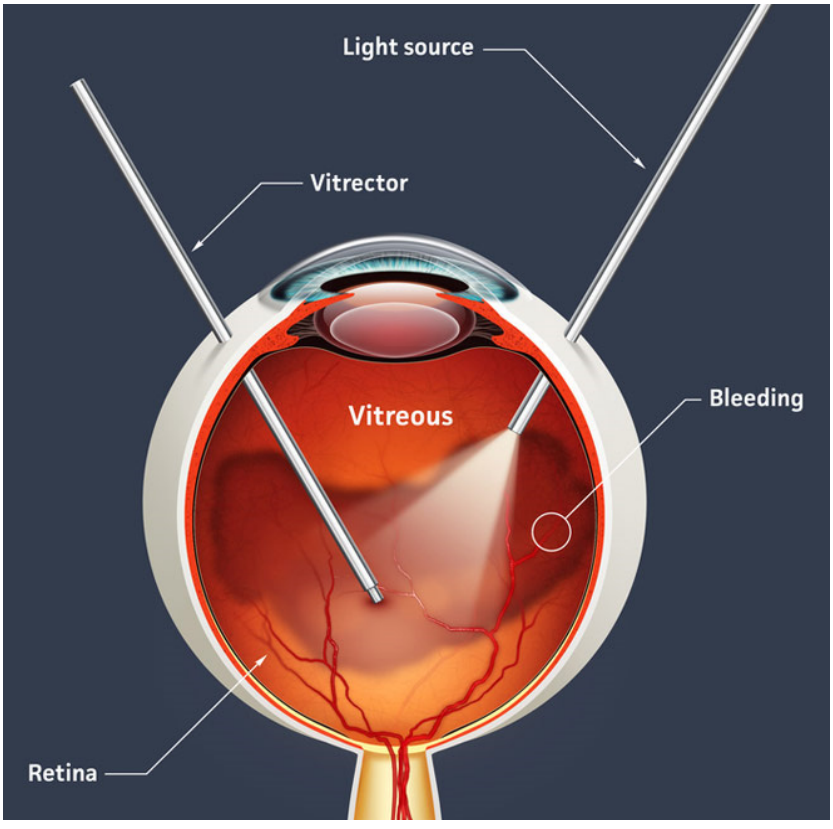
CRYOTHERAPY:

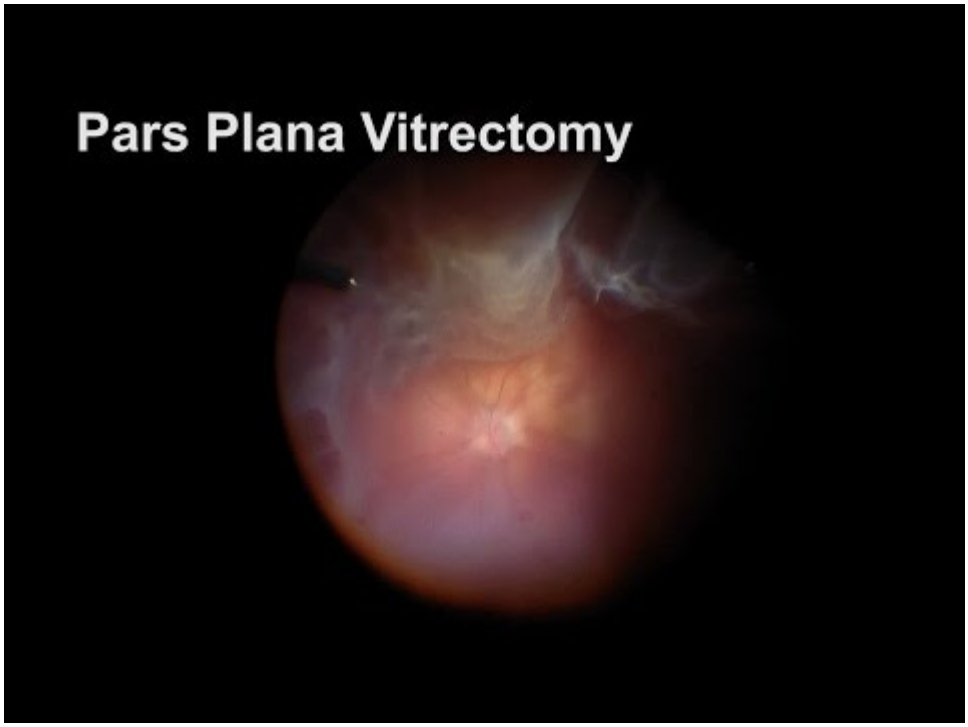
In cryotherapy there is break down of blood retinal barrier followed by increasing passage of plasminogen activators from choroid and retina .this causes blood clot lysis and resorption. It is indicated only when pars plana vitrectomy is not available in non resolving vitreous hemorrhage.

POSTERIOR HYALOIDOTOMY:

In case of hemorrhage located between internal limiting membrane and retina posterior hyaloidotomy using nd-yag laser disrupt internal limiting membrane and release blood in vitreous cavity this prevents macular degeneration.

3 Port Pars Plana Vitrectomy





SURGICAL MAMNAGEMENT:

Pars plana vitrectomy

Pars plana vitrectomy is a vitreo retinal surgical technique typically used for removing the vitreous opacities, removing the vitreo retinal traction and restoring the normal anatomical relationship between vitreous and retina. It requires the placement of 3 ports at 3-4mm from the surgical limbus. One port is made for the infusion of balanced solution to maintain the required intraocular pressure. Other port is for endo illumination to view the posterior segment. Other port is for manipulate, dissect or remove intraocular objects ,ocular tissues, fluids.

Vitrectomy is performed with an operating microscope using contact or non contact lenses. Direct and indirect visualization is possible.

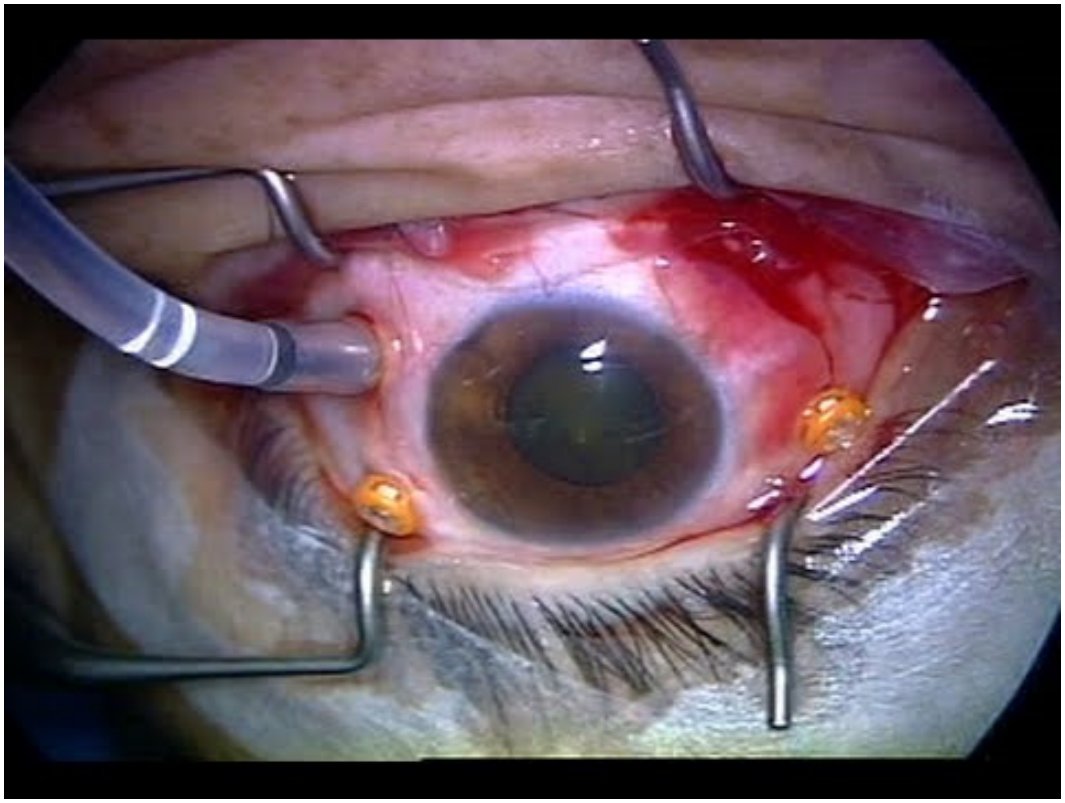
In indirect visualization there is advantage of wider angle visualization ,better view through medium opacities, miotic pupil. The direct visualization provides better stereopsis and enhanced magnification at expense of smaller field.

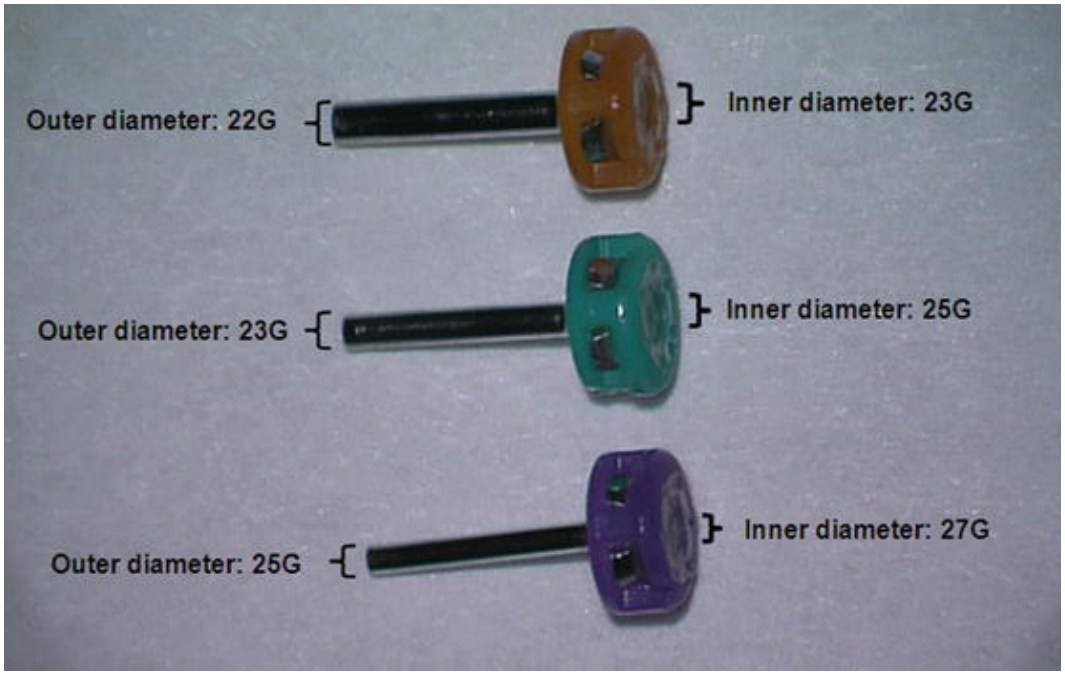
Advanced instrumentation includes high speed cutters, endo laser probe, micro-pic, retinalscissors, fragmentome. Visualization aids includes indocyanin green for viewing internal limiting membrane and triamcinolone suspension for identifying the cortical vitreous. Tamponade of the retina is achieved using air, gas, silicone oil as substitutes. Commonly used gases includes sulfur hexafluoride[S3F6] and perflouropropane[C3F8].

They have a half life of 1-3weeks at an isovolumetric concentration. Perfluorocarbon liquids are heavier than water and can be used for temporary stabilization of retina during the dissection and draining of fluid from subretinal space for repair of retinal detachment.

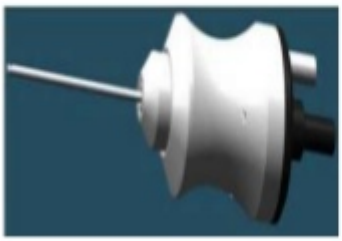
A more recent advance is the development of smaller gauge trans conjunctival technique vitrectomy technique. Uses the 23,25 gauge instead of older 20 gauge sclerotomy. Entry cites into the vitreous cavity is made using 23 or 25 gauge so that suturing can be avoided and avoids the conjunctival cut down procedure and the need for cautery. Potential advantages are the shortened operative time, better post operative comfort

and faster visual recovery. But there are disadvantages including postoperative hypotony, endophthalmitis and retinal tears.

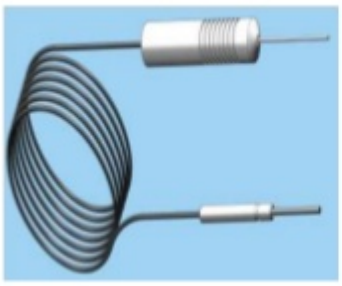




23G vitrectomy probes –



23G single use vitrectomy probe



23G standard fiber optic
23G wide-angle fiber optic

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INDICATIONS OF VITRECTOMY FOR VITREOUS HEMORRHAGE:

Vitrectomy is indicated after 6weeks to 3months of failure of spontaneous clearance of vitreous hemorrhage. Possible indications for prompt intervention is the bilateral hemorrhage , associated retinal tear

and retinal detachment. During the interval period of observation serial ultrasonography is done to monitor anatomical condition of retina in case of absence of retinal visualization. If surgery is indicated it involves removal of vitreous hemorrhage, and release of hyaloid from fronds of retinal neovascular ingrowth.

COMPLICATIONS OF PARS PLANA VIRECTOMY

1. long term risk of open angle glaucoma
2. intra operative or post operative retinal break
3. intraoperative or postoperative retinal detachment
4. intraoperative cataract
5. postoperative vitreous hemorrhage
6. postoperative massive fibrin exudation
7. postoperative anterior segment neo vascularization.
8. post operative nuclear sclerotic cataract.

COMPLICATIONS ASSOCIATED WITH SILICONE OIL:

- Glaucoma
- Band keratopathy

GENERAL COMPLICATIONS:

- Endophthalmitis.
- Sympathetic ophthalmia.

- Recurrent erosions.

Other indications: eales disease, bechets disease, uveitis, retinal tears, leukemia, trauma.

AIM OF THE STUDY

To analyse various etiological incidence of vireous hemorrhage.

MATERIALS AND METHODS

Prospective study was carried out on 176 patients diagnosed vitreous hemorrhage irrespective of the age, sex after history taking routine clinical examination direct ophthalmoscopy, indirect ophthalmoscopy, slit lamp bio microscopy, A scan, B scan, clinical findings of other eye, systemic evaluation and laboratory investigations.

History taking includes history of symptoms like flashes of light, smoke signal, photophobia, perception of shadows and cob webs , visual haze , defective vision ans loss of vision etc.

History of trauma

History of ocular disorders like proliferative retinopathy, inflammatory occlusive retinal vascular disorders, retinal tear, posterior vitreous detachment etc.

History of infections

History if systemic illness like diabetes mellitus , hypertension, blood disorder, connective tissue disorder.

- History of ocular tumours
- History of surgical procedures

CLINICAL EXAMINATION

Anterior segment examination by oblique illumination and slit lamp examination.

Visual acuity is determined by the location and density of vitreous hemorrhage.

Tension is raised in ghost cell hemolytic and hemosiderrotic glaucoma. fundus examination by direct ophthalmoscopy, indirect ophthalmoscopy , slit lamp bio microscopy includes three mirror examination with + 90D LENS

Fundus fluorescein angiography- vascular status of choroid and retina.

A scan and B scan ultra sonography determines the location and density of vitreous hemorrhage and extent of tractional membranes and retinal detachment which predict visual outcome after vitreous surgery.

General and systemic evaluation

To rule out hypertension diabetes and infective disease like tuberculosis, syphilis, connective tissue disorder etc.

Laboratory investigations include peripheral smear, blood sugar, serum cholesterol, serum proteins, VDRL, mantoux , anti nuclear antibodies , rheumatoid factors .

X RAY skull , CT scan, MRI to rule out increased intra cranial tension, hemorrhages.

RESULTS

The results of the study conducted at RIOGOH during the period from Feb'17 to Sept'17.

The incidence of the various etiology of vitreous hemorrhage

Total number of cases 100

Age incidence between 10 to 70 years

Sex incidence Male – 67 Female – 33

DIAGNOSIS	AGE (YRS)	SEX		LATERALITY		WITHOUT PVD	WITH PVD	RT	RD	TOTAL	%
		M	F	UNI	BI						
PDR	35 – 70	26	9	22	13	8	21	-	6	35	35
OCULAR TRAUMA	22 – 52	16	10	26	0	10	16	5	5	26	26
EALE'S DISEASE	22 – 58	8	3	6	5	8	3	-	-	11	11
SPONTANEOUS PVD	62 – 70	7	1	8	0	-	3	4	1	8	8
RETINAL VEIN OCCLUSION	45 – 63	3	1	4	0	3	1	-	-	4	4
PARS PLANITIS	34 – 46	1	1	2	0	2	-	-	-	2	2
RETINAL VASCULITIS	44 – 58	0	2	2	0	2	-	-	-	2	2
SENILE PVD	44 – 78	1	1	2	0	0	2	-	-	2	2
POST OP COMPLICATION (ECCE/PCIOL)	65 – 68	1	1	2	0	2	-	-	-	2	2
UNKNOWN	23 – 54	3	5	8	0	8	-	-	-	8	8

PDR- Proliferative diabetic retinopathy

PVD –Posterior vitreous detachment

RT –Retinal tear

RD- Retinal detachment

ANALYSIS

Causes of vitreous hemorrhage in different studies ANALYSIS AND DISCUSSION

CAUSES OF VITROUS HEMORRHAGE IN DIFFERENT STUDIES

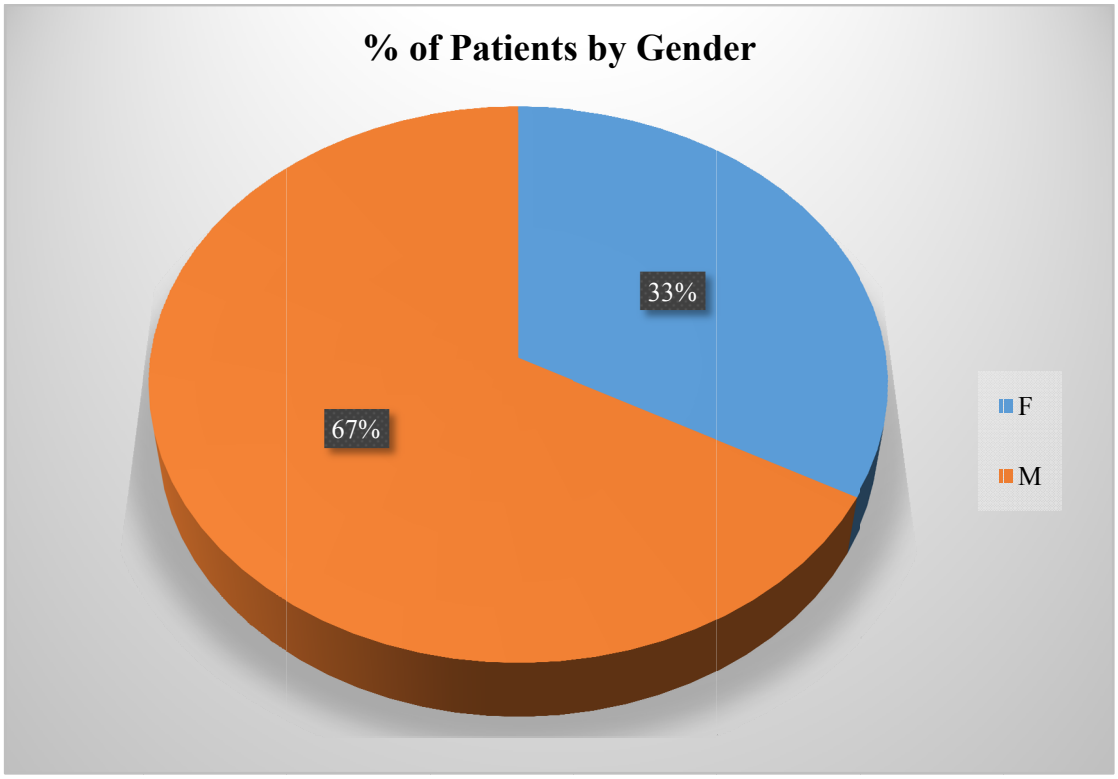
AUTHOR AND REFERENCE	Winslow and Taylor	Butner and Mecpherson	Dana et al	Lindgrend et al	RIOGOH
Year	1980	1982	1993	1995	2017
Mean age(years)	N.A	49	48	65	50
Region	DALLAS	LAS VEGAS	ILLIONOIS	SWEDEN	CHENNAI
Patients	100	653	198	94	100
DIAGNOSIS					
PDR	31.5%	34.1%	34.7%	19.1%	35
TRAUMA	-	-	18.8%	-	26
EALES DISEASE	-	-	-	-	11
RVO	12%	13.0%	7.9%	16.0%	4
OTHERS	6.9%	7.2%	8.9%	6.4%	14
UNKNOWN	7.6%	2.1%	2.0%	4.3%	8

The above previous six studies show proliferative diabetic retinopathy, spontaneous posterior vitreous detachment with or without retinal tear,with retinal detachment or without retinal detachment and ocular trauma are the three most common causes of vitreous hemorrhage and accounts for 59.8% to 88.9% of all causes.

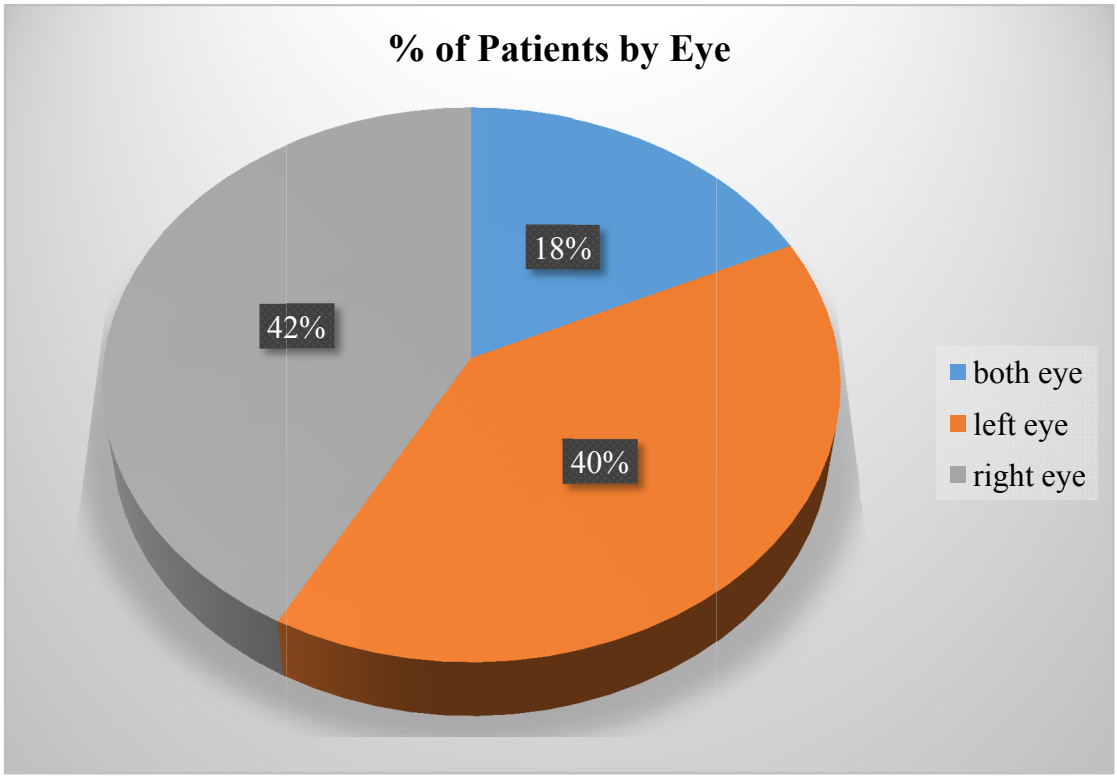
The study conducted at RIOGOH ,Chennai during the period from February 2017 to September 2017 revealed proliferative diabetic retinopathy, ocular trauma, Eale’s disease and senile posterior vitreous

detachment with or without retinal tear are the four most common causes of vitreous hemorrhage and accounts for 74% (74 out of 100) of all cases. Vitreous hemorrhage secondary to retinal vein occlusion, pars planitis, spontaneous PVD, retinal vasculitis, post operative complications, unknown causes accounts for 26% (26 out of100) of all cases.

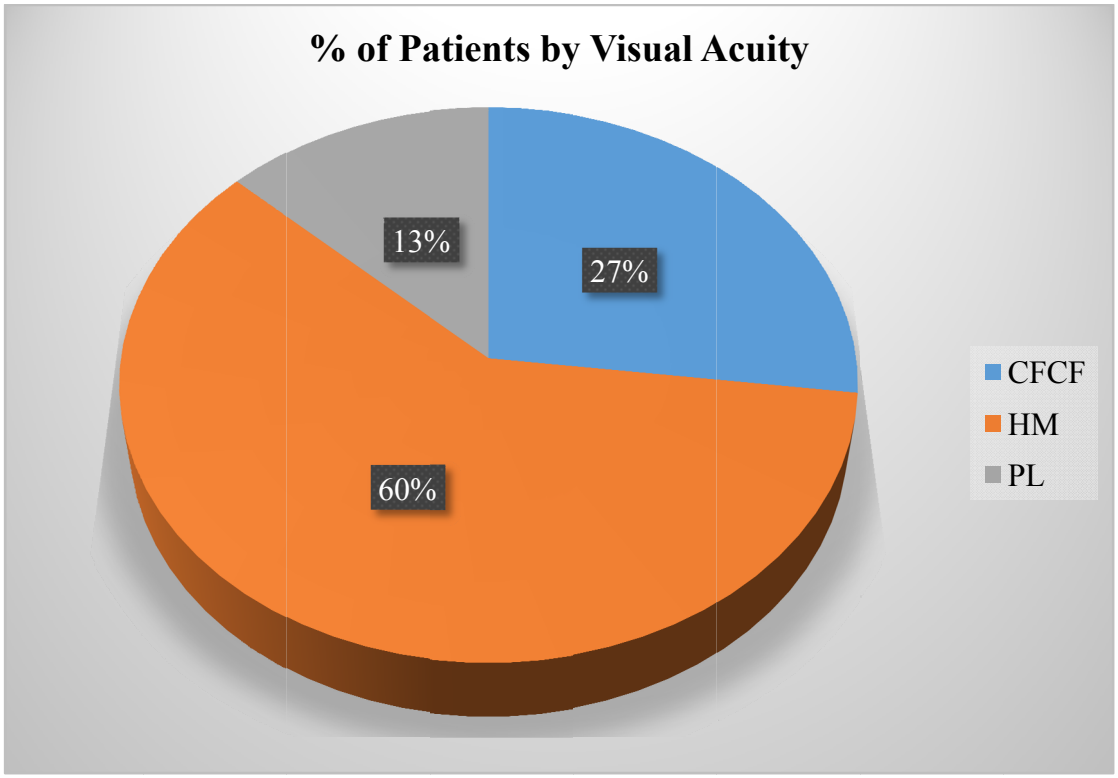
Gender	% of Patients
F	33%
M	67%
Grand Total	**100%**



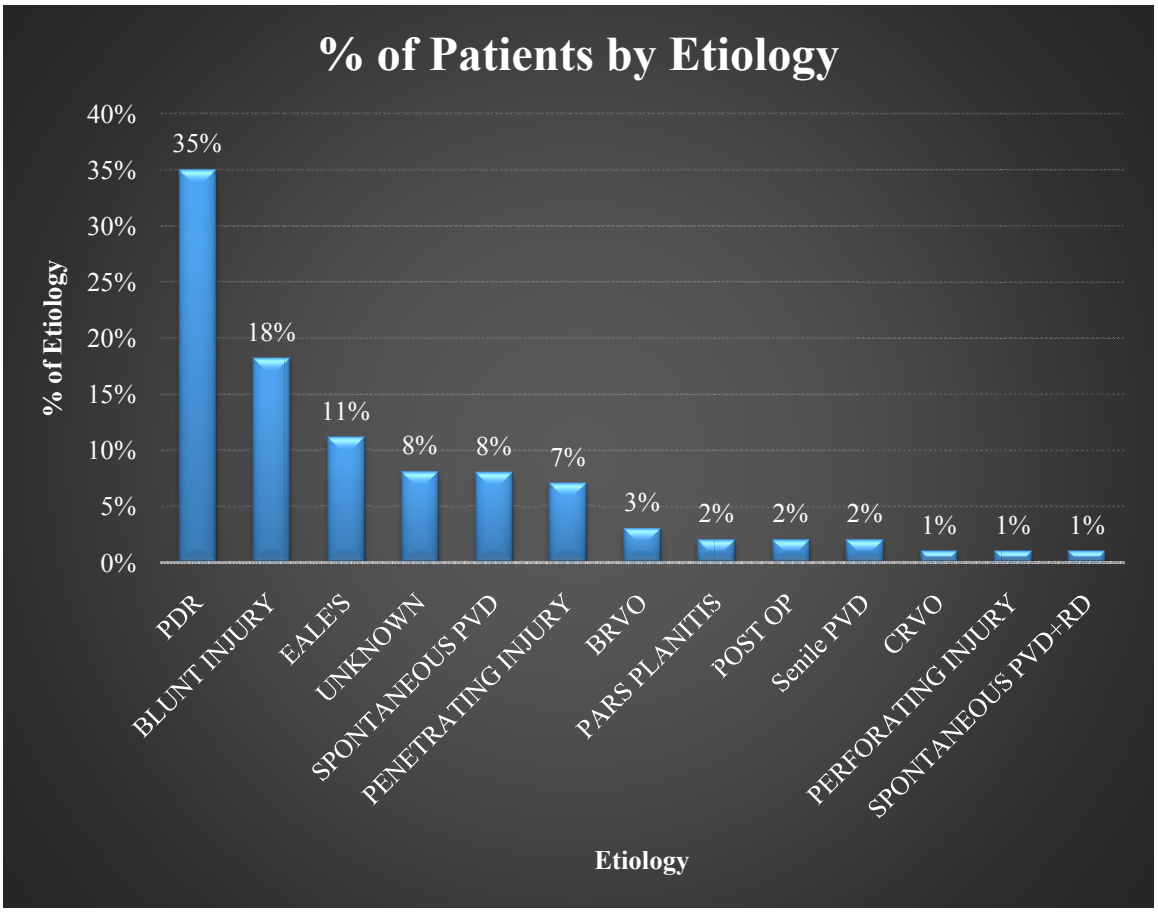
EYE	% of Patients
both eye	18%
left eye	40%
right eye	42%
Grand Total	**100%**



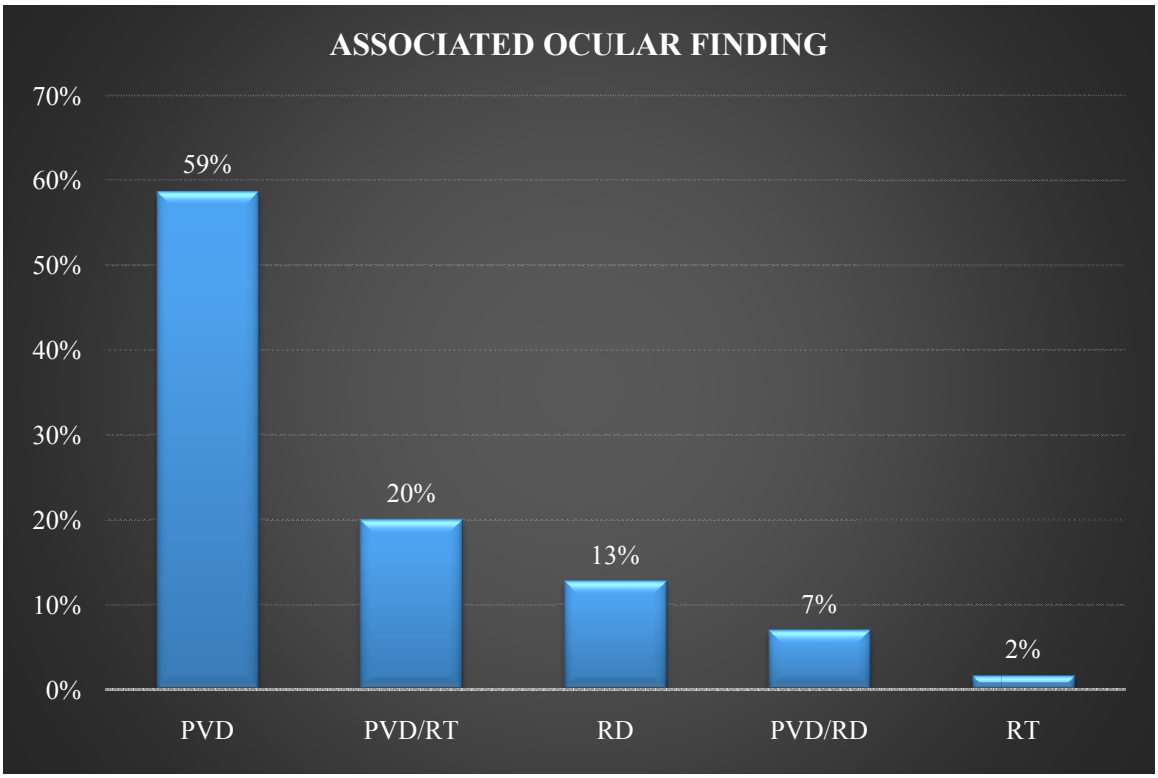
V/A at Presentation	% of Patients
CFCF	27%
HM	60%
PL	13%
Grand Total	**100%**

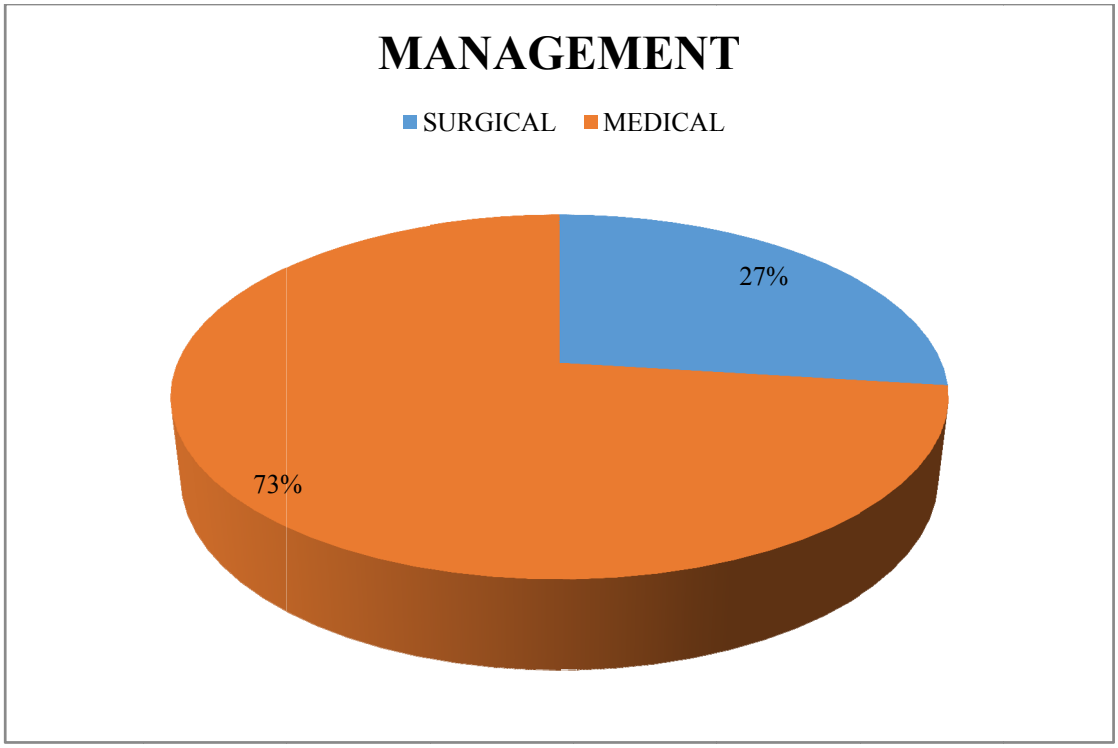


Incidence of various etiology of vitreous haemorrhage in our study

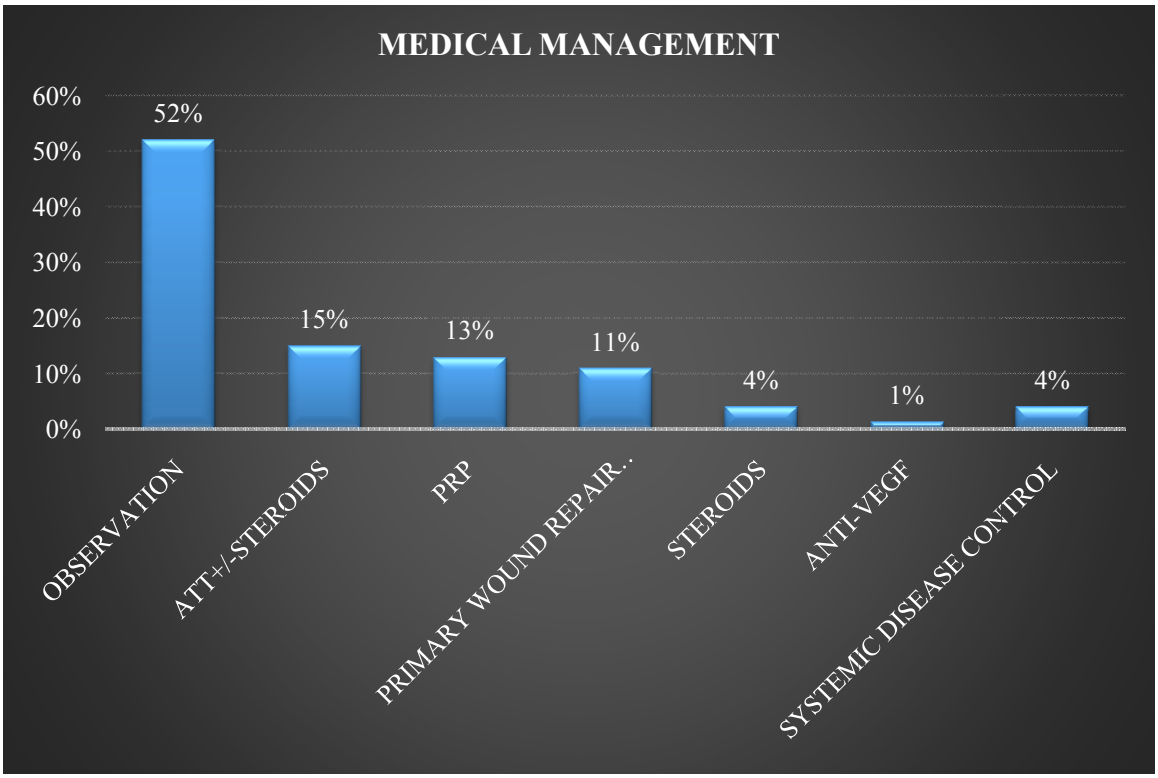


Associated ocular findings with Vitreous haemorrhage

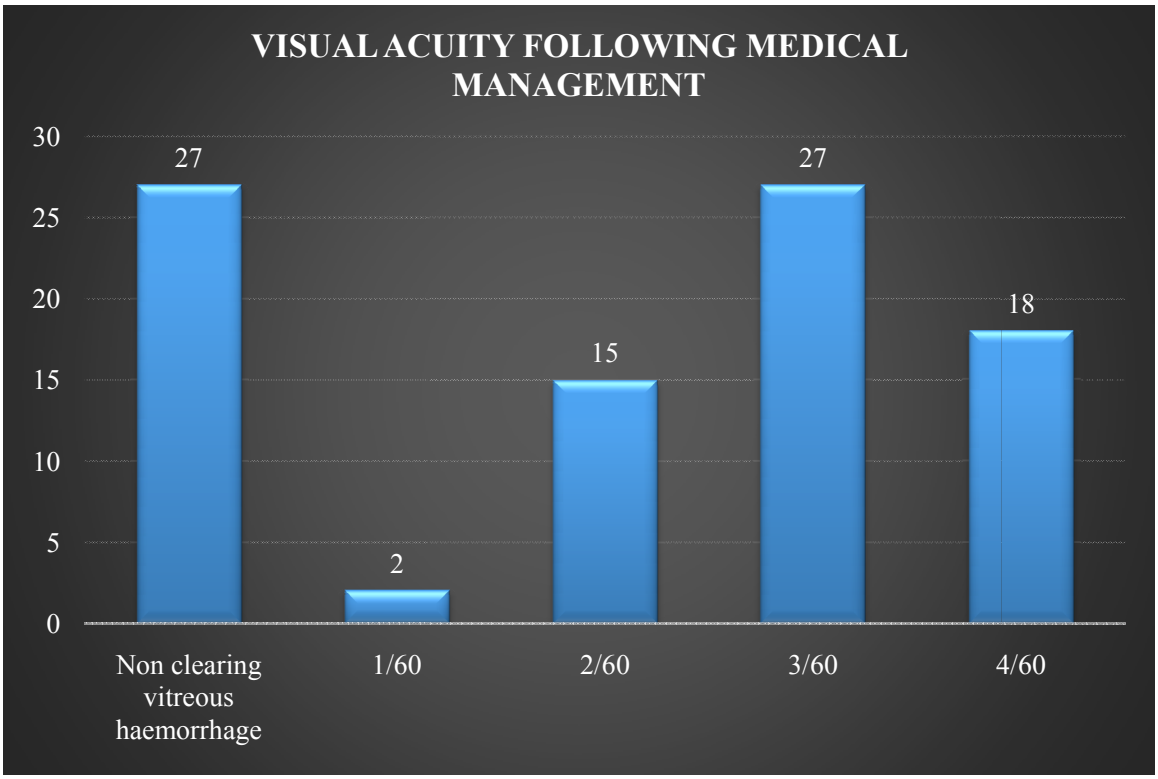


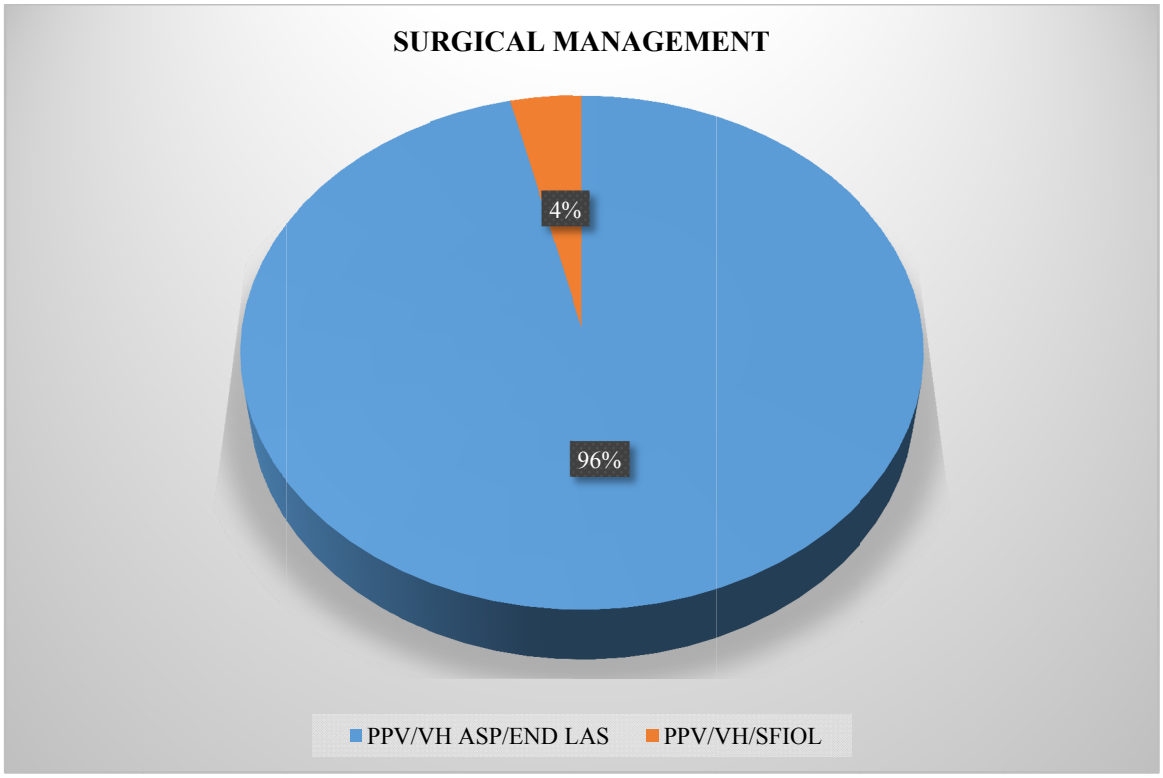


Cases treated with medical management

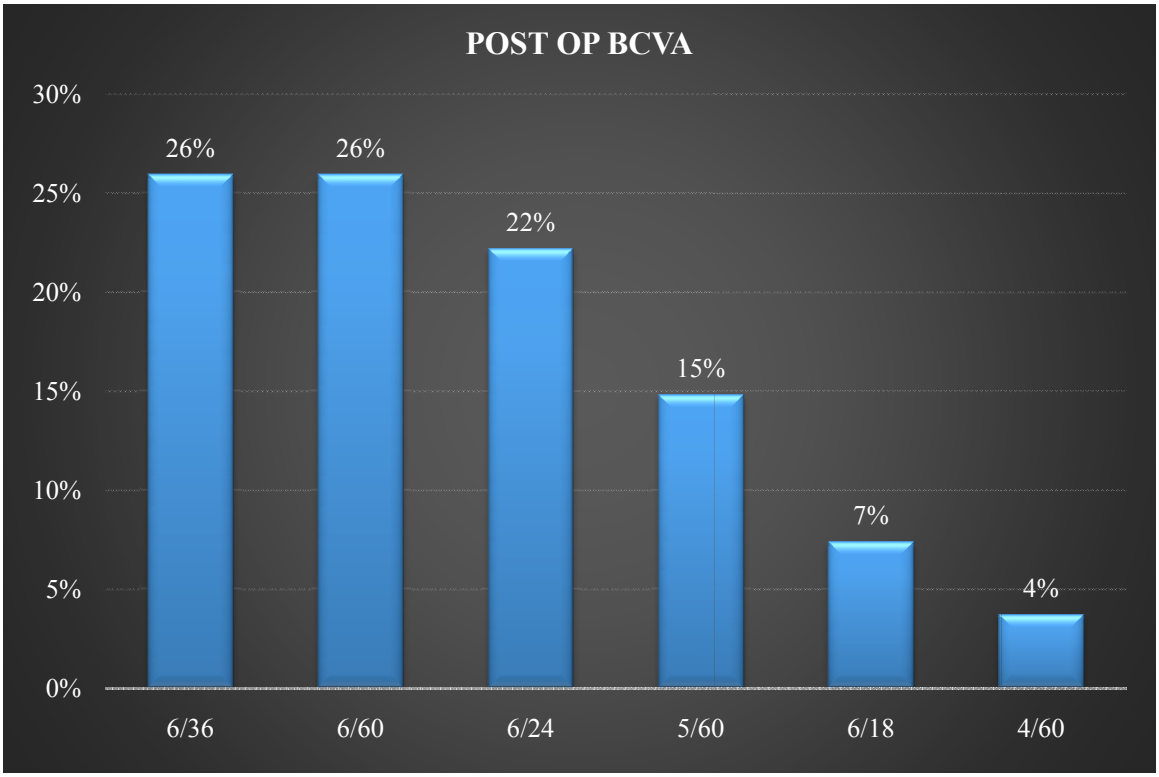


Visual Acuity Following Medical Management





Post operative vision following Pars Plana Vitrectomy



The five most common causes of vitreous hemorrhage are

1. Proliferative diabetic retinopathy

It is the most common cause of vitreous hemorrhage accounts for 35%(35 out of 100)the age incidence varying between 35 years to 70 years. Among them type 1 diabetes mellitus accounts for 20%(7 out of 35 cases) and remaining 80%(28 out of 35cases) accounts for type 2 diabetes mellitus,62% (22 out of 35 cases) of patients presented with unilateral incidence and 38% (13 out of 35 cases) of patients presented with bilateral incidence.

The study shows incidence of vitreous hemorrhage in proliferative diabetic retinopathy, associated with other ocular pathology are as follows

Vitreous hemorrhage without PVD 22% (8 out of 35 cases)

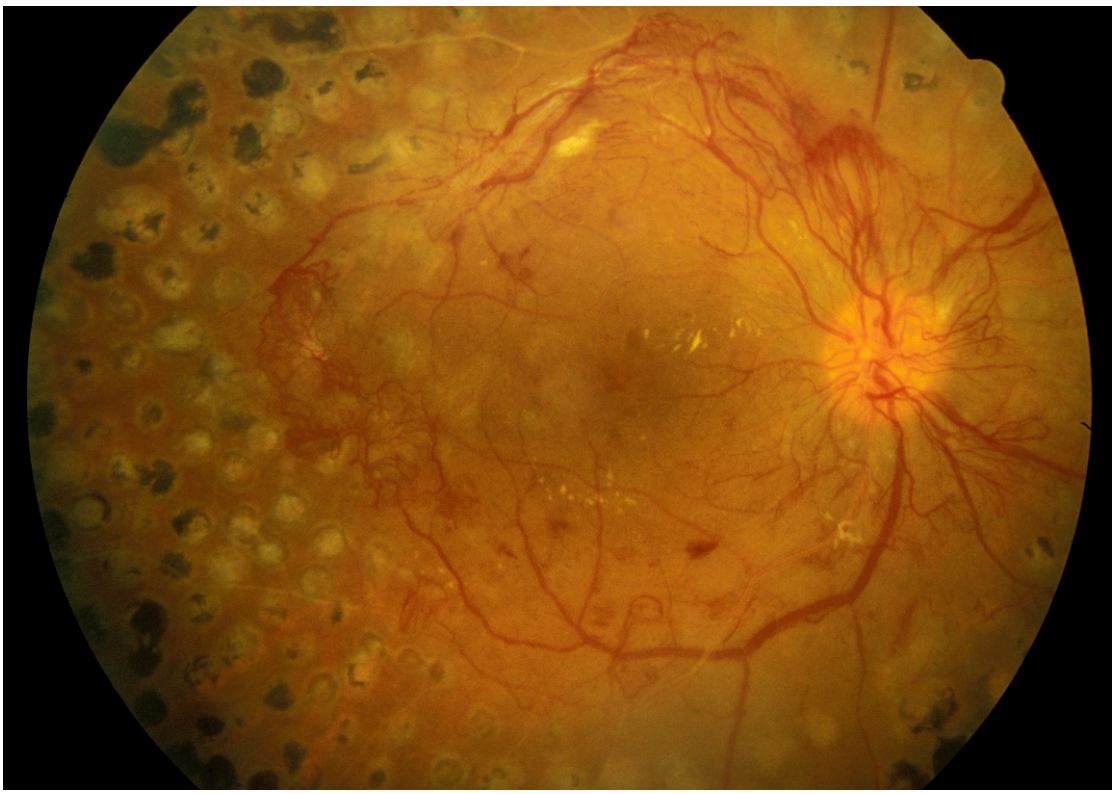
Vitreous hemorrhage with PVD 60%(21 out of 35 cases)

Vitreous hemorrhage with RD 18%(6 out of 35 cases)

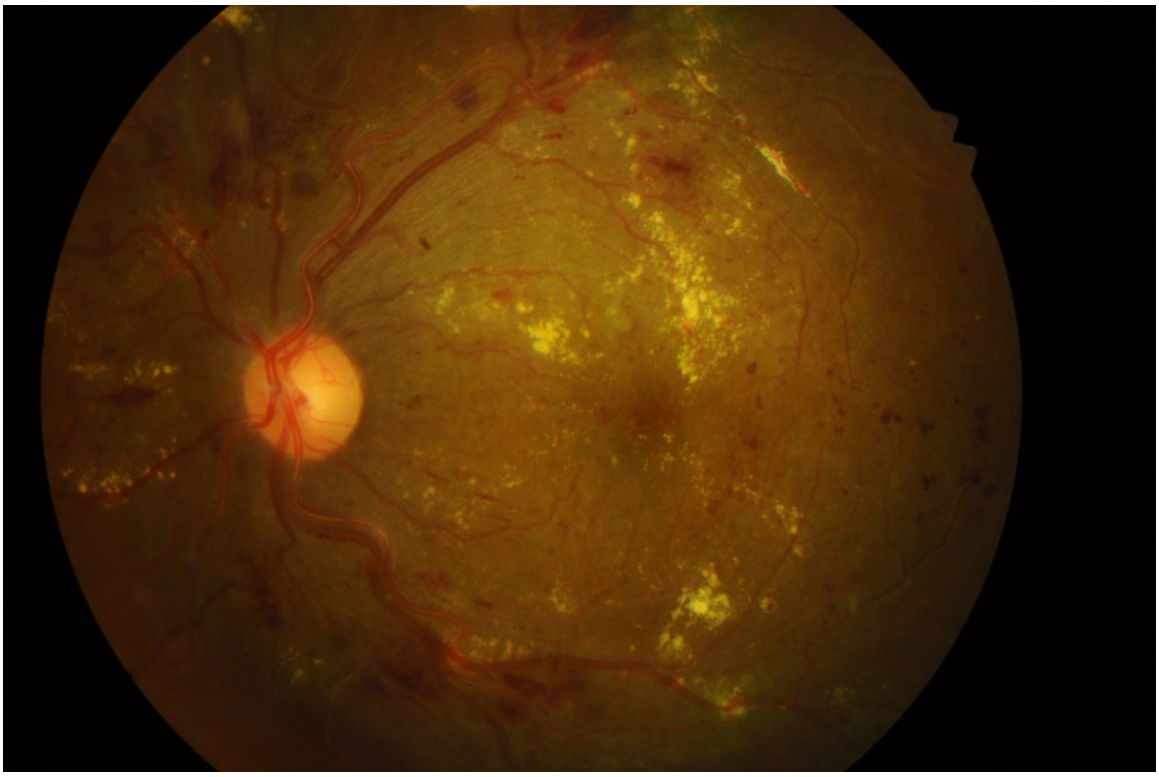
The proliferative diabetic retinopathy was the most common cause of vitreous hemorrhage in two to six available retrospective epidemiologic studies (Morse et al in the year 1974 at Philadelphia(200), Winslow and Taylor in the year 1980 at Dallas(100 cases), Lean and Gregor in the year 1980 at London (317cases) , Butner and Mecpherson in the year 1982 at Lasvegas, (653 cases),Dana et al in the year 1993 at

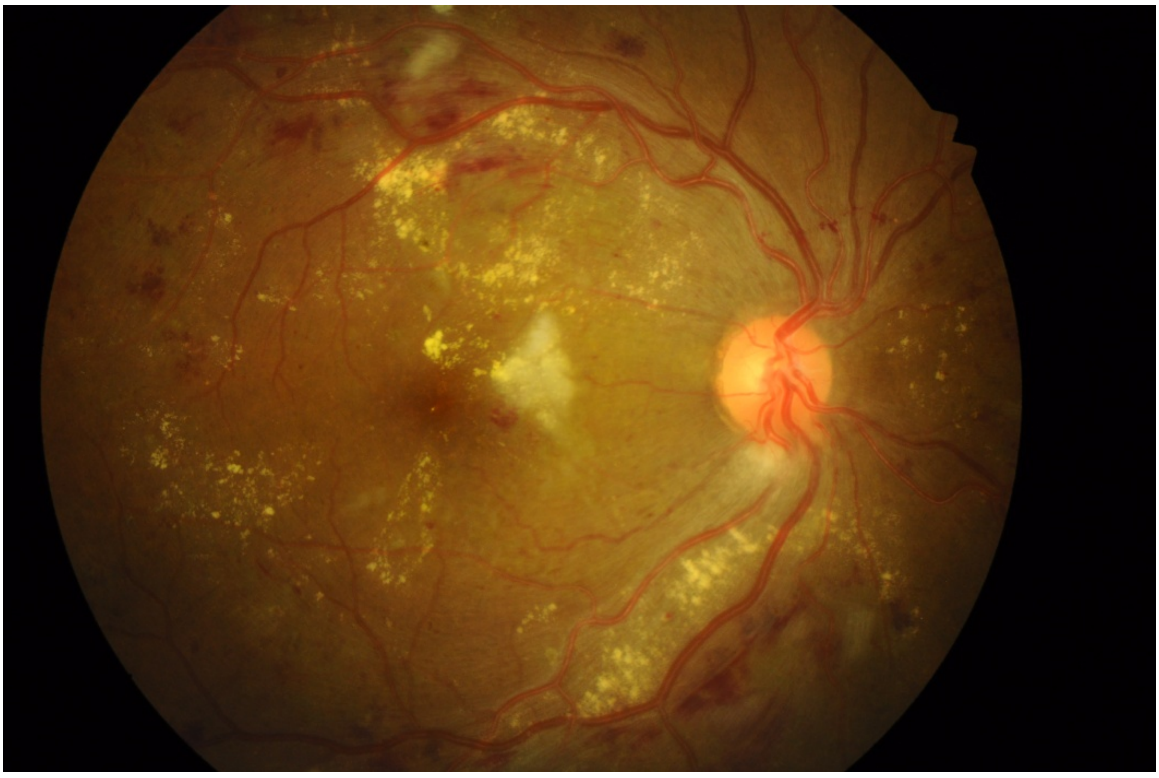
Illinois (198 cases),Lingrend et al in the year 1995 at Sweden (94 cases) of the patients witth incidence of vitreous hemorrhage, mean age varying between 48-65years and accounts for 32%of cases. Among them patients with type 1 diabetes mellitus accounts for 89% and type 2 diabetes mellitus accounts for64%.

Proliferative Diabetic Retinopathy with Post Panretinal Photocoagulation



Proliferative Diabetic Retinopathy





2. Ocular trauma:

It is the second most common cause of vitreous hemorrhage accounts for 26%(26 out of 100 cases) of all cases, the age incidence varying between 22-52years, sex incidence of male 65%(16 out of 26 cases), female- 35%(10 out of 26 cases). The incidence of various types of ocular trauma associated with vitreous hemorrhage are as follows:

Blunt injury 69%(18 out of 26 cases)

Penetrating injury 27%(7 out of 26 cases)

Perforating injury 4%(1 out of 26 cases)

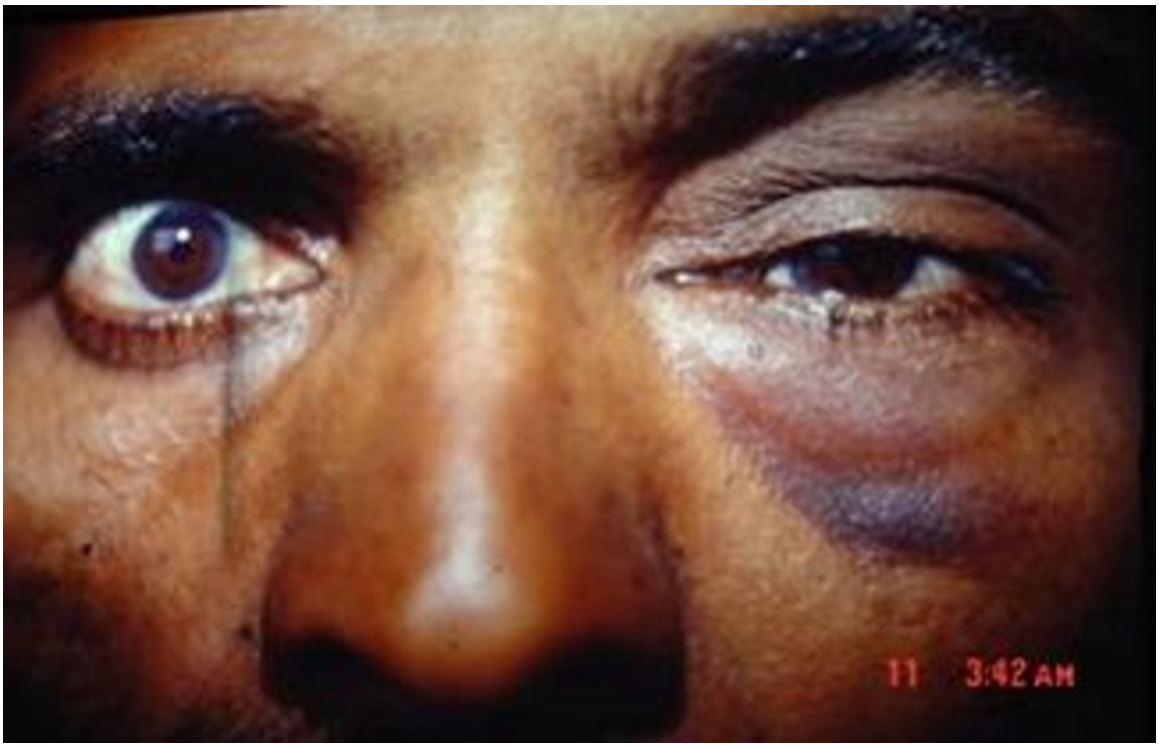
The study shows the incidence of hemorrhage in ocular trauma with vitreous associated ocular pathology are as follows:

TYPE OF OCULAR INJURY	WITHOUT PVD	WITH PVD	RETINAL TEAR		RETINAL DETACHMENT	
			WITH PVD	WITHOUT PVD	WITH PVD	WITHOUT PVD
BLUNT INJURY (18)	2	9	5	0	2	0
PENETRATING INJURY(7)	0	0	3	1	2	1
PEFORATING INJURY (1)	0	0	1	0	0	0

The incidence of ocular trauma in vitreous hemorrhage was included in two series (Lean and Gregor in the year 1980 at London(12%

out of 317cases). Dana et al in the year 1993 at Illinois (18.8% of 198 cases) of six available retrospective studies and represented the second most common cause of vitreous hemorrhage, wuth age incidence of under 40 years and male showed higher incidence than female.

Blunt Trauma



3. Eale's disease

It is the most common cause of vitreous hemorrhage accounts for 11% of all cases, the age incidence between 22-58 years. 73%(8 out of 11 cases) are males and 27%(3 out of 11 cases) are females, 55%(6 out of 11 cases) of patients presented with unilateral incidence and 45%(5 out of 11 cases)of patients presented with bilateral incidence.

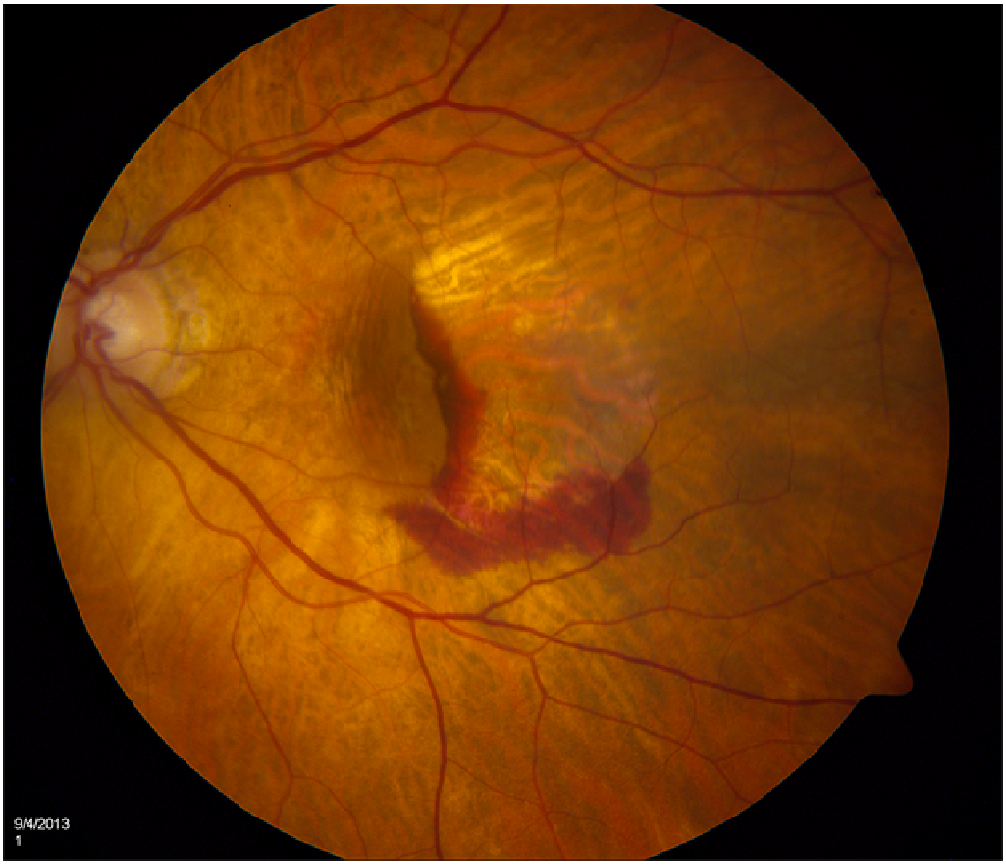
The incidence of vitreous hemorrhage in Eale's disease associated with other ocular pathology are as follows

Vitreous hemorrhage without PVD 73%(8 out of 11 cases)

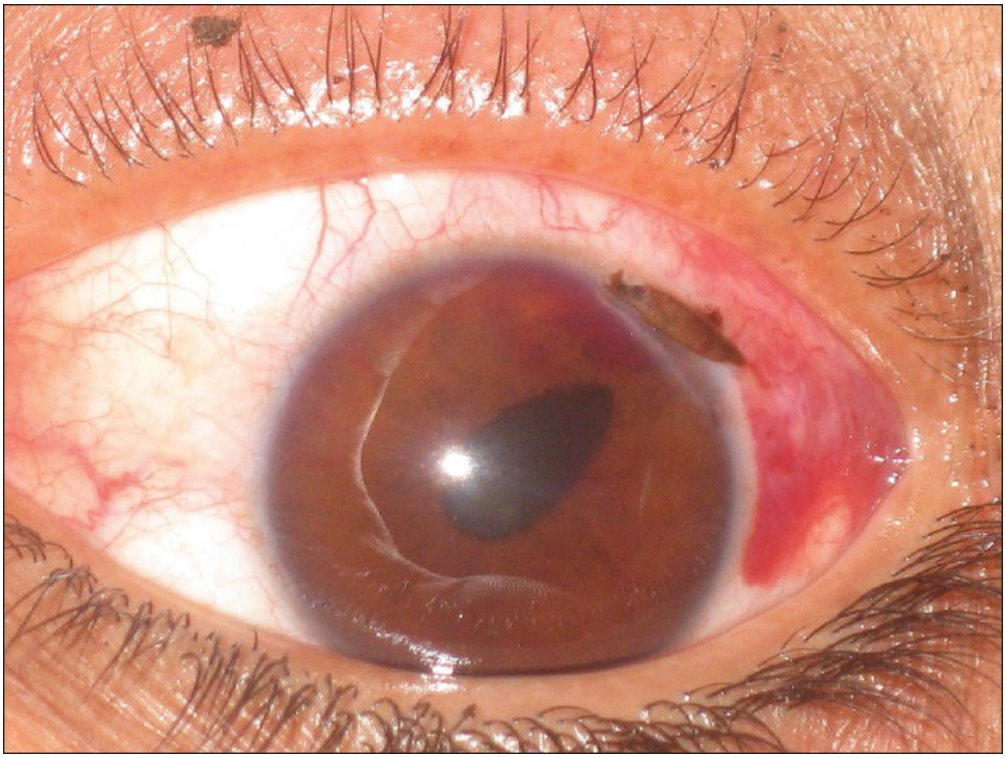
Vitreous hemorrhage with PVD 27%(3 out of 11 cases)

The previous available six retrospective studies (Morse et al in the year 1974 at Philadelphia(200 cases), Winslow and Taylor in the year 1980 at Dallas(100cases), Lean and Gregor in the year 1980 at London(317 cases), Butner and Mecpherson in the year 1982 at Lasvegas,(653 cases), dana et al in the year 1993 at Illinois (198cases), Lindgrend et al in the year 1995 at Sweden(94 cases) if incidence of vitreous hemorrhage showed no incidence of Eale's disease as a cause of vitreous hemorrhage. The previus six studies conducted in western countries where the incidence of Eale's disease is very low.

Retinal Tear with Vitreous Haemorrhage



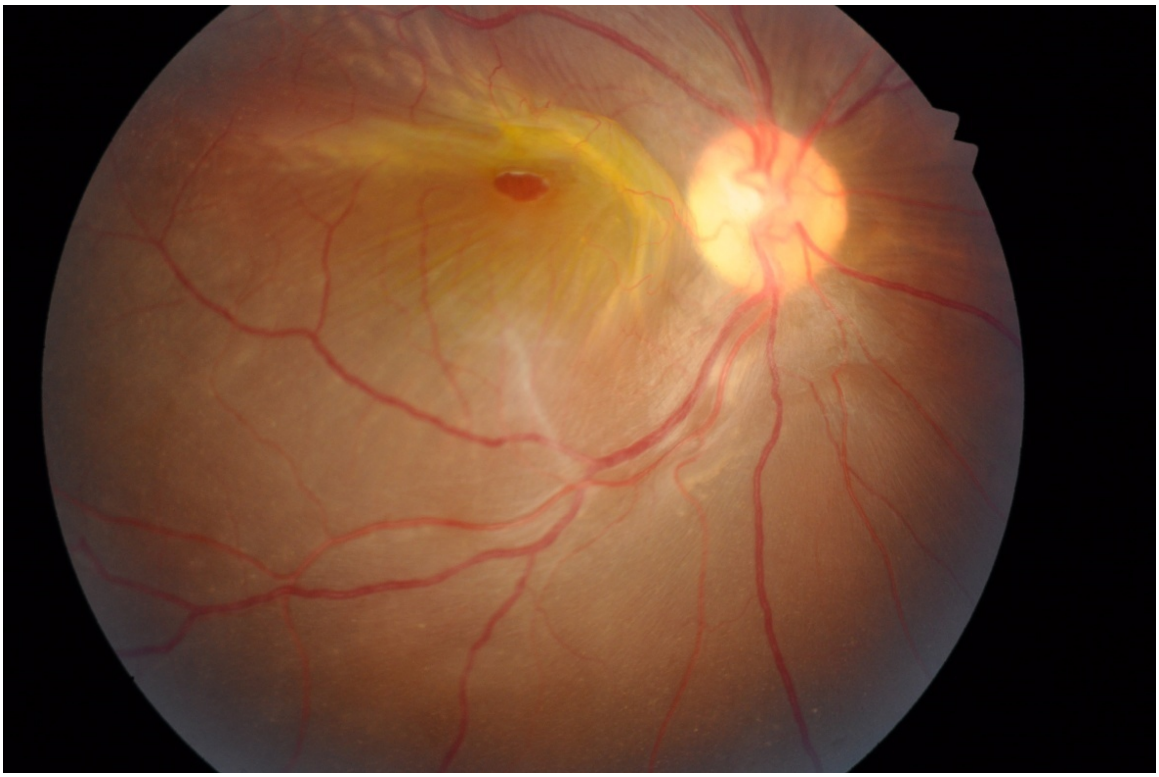
Perforating Injury with Vitreous Haemorrhage

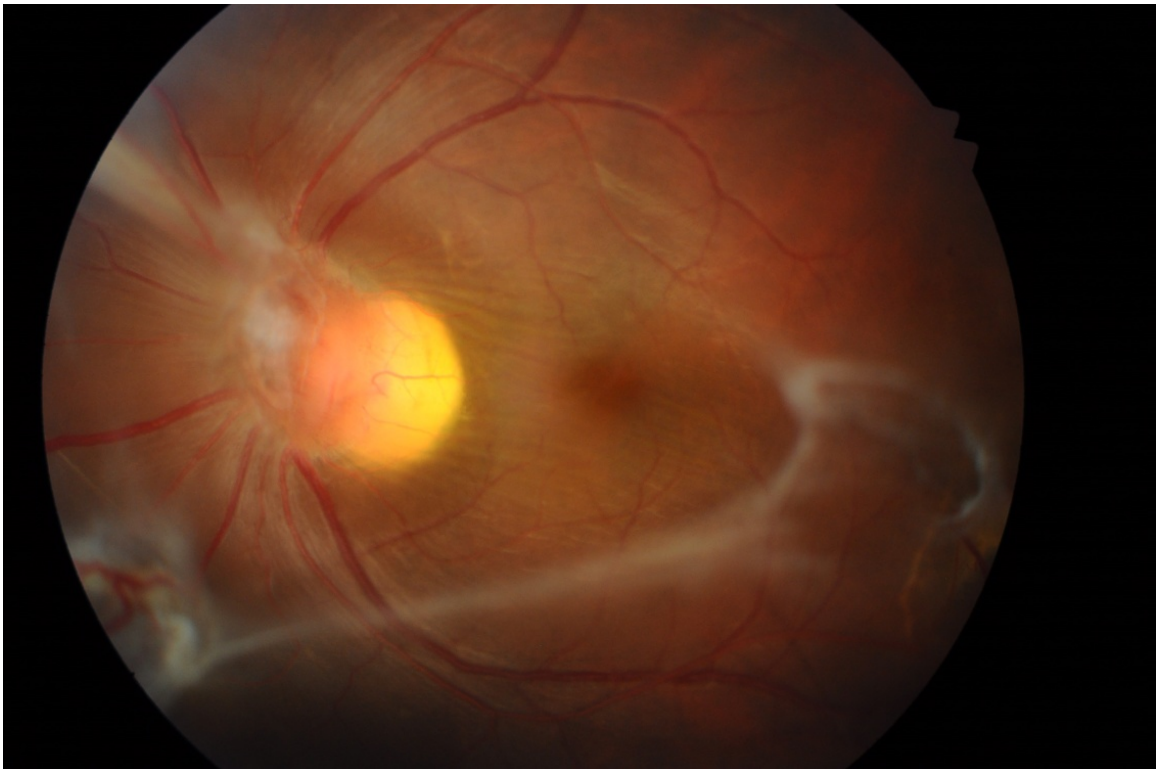


Eale's disease(Eale's disease by Tara Prasad Das, Jyothirmay Biswas, Atul Kumar, P.N.Nagpal, P.A.Namperumal samy,Bijayanandha Patnaik and H.K.Tewari, I J O volume 42 no.1 March 1994) has been reported from the united kingodom, the United States, and Canada in later half of 19^{th} and early 20^{th} century. But for unknown reasons, it is now rare in more developed countries and is more commonly reported from the Indian sib continent. The reported incidence in India is 1 in 200 to 250 ophthalmic patients. Eventually between the peak age of incidence between 20 to 30years, between 70 to 80 years of patients develop bilateral involvement though the extent of retinal in volvement may not be to the same extent.

Duke elder noted 90% bilateral involvement and others have noted only 50% of involvement.

Eales Disease





4. Spontaneous posterior vitreous detachment:

It is the fourth most common cause of vitreous hemorrhage accounts for 8%(8 out of 100 cases). The age of incidence varying between 62 years to 70 years. 88% (7 out of 8 cases) are found to be males and 12%(1 out of 8 cases)are found to be females. The incidence of vitreous hemorrhage in spontaneous PVD associated with other pathology are as follows

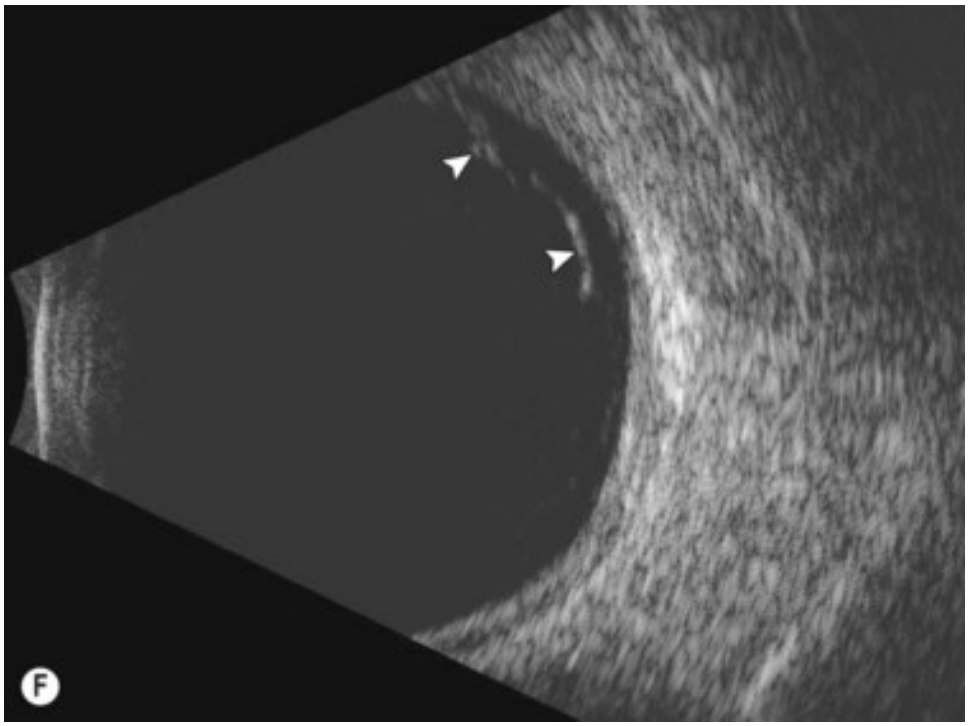
Vitreous hemorrhage with PVD 38% (3 out of 8 cases)

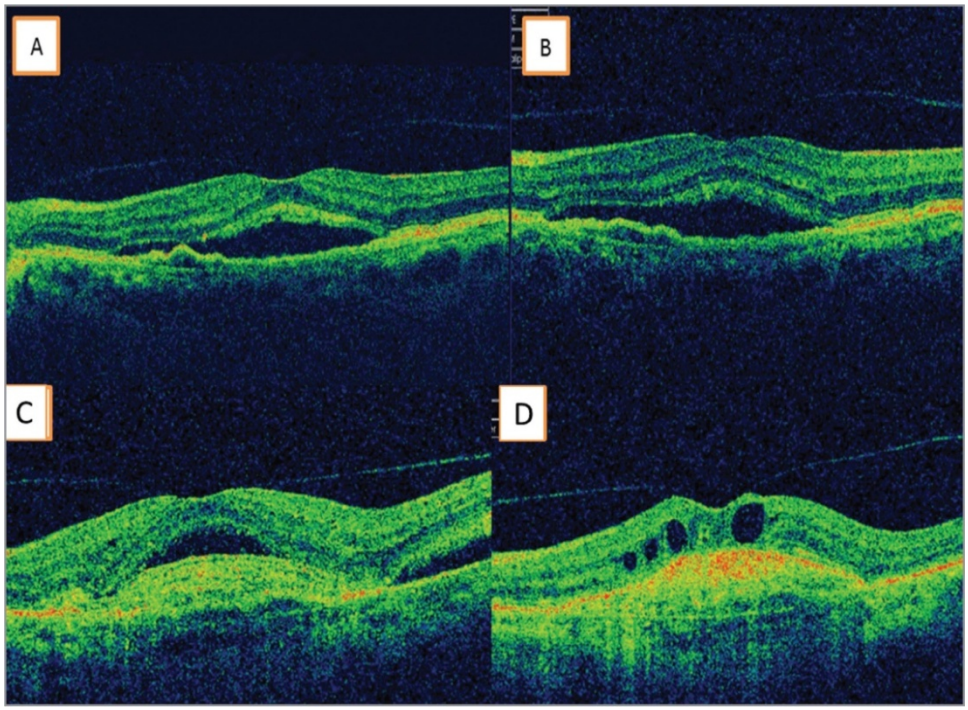
Vitreous hemorrhage with retinal tear 50%(4 out of 8 cases)

Vitreous hemorrhage with RD 12%(1 out of 8cases)

The previous available six retrospective studies(Morse et al in the year 1974 at Philadelphia(200 cases), Winslow and Taylor in the year 1980 at Dallas (100 cases), Lean and Gregor in the year 1980 at London (317 cases), Dana et al in the year 1993 at Illinois (198 cases), Lindgrend et al in the year 1995 at Sweden(94 cases) of the incidence of vitreous hemorrhage shows spontaneous posterior vitreous detachment with or without retinal tear are found to be the first most common cause of vitreous hemorrhage in four studies and accounts for 38% of all cases.

B-Scan ultra sonography Shows Posterior Vitreous Detachment





Optical Coherence Tomography shows **incomplete** Posterior Vitreous Detachment

5. Retinal vein occlusion :

It is the fifth most common cause of vitreous hemorrhage accounts 4%(4 out of 100 cases) for all cases, the age incidence between 45 years to 63 years.75%(3 out of 4 cases) are males and 25%(1 out of 4 cases) are females . All patients studied were found to be hypertensive. The branch retinal vein occlusion accounts for75%(3 out of 4 cases). The central retinal vein occlusion accounts for 25%(1 out of 4 cases). the incidence of vitreous hemorrhage in retinal vein occlusion associated with ocular pathology are as follows:

Vitreous hemorrhage with PVD 25%(1 out of 4 cases)

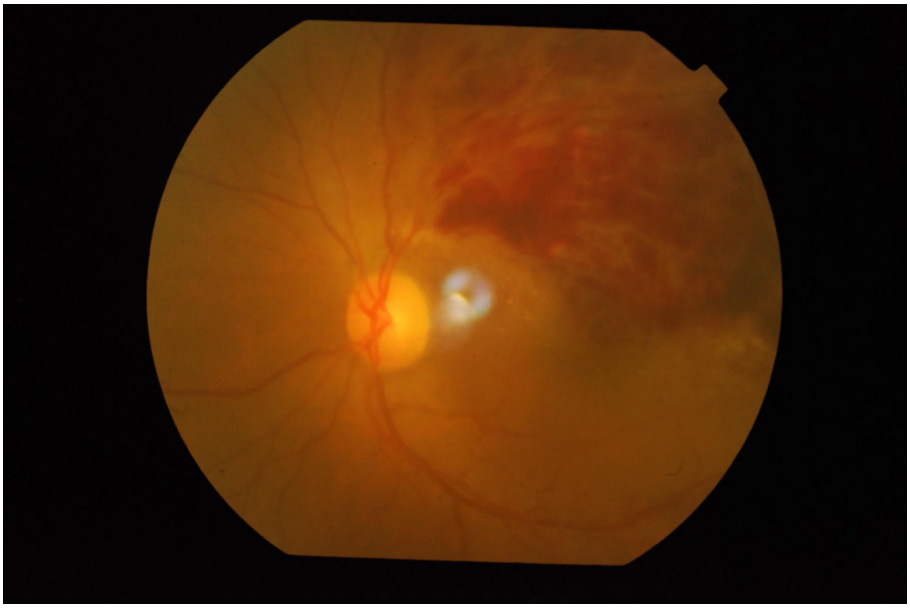
Vitreous hemorrhage without PVD 75%(3 out of 4 cases)

In the past six retrospective studies (Morse et al in the year 1974 at Philadelphia (200 cases), Winslow and Taylor in the year 1980 at Dallas(100 cases), Lean and Gregor in the year 1980 at London (317 cases), Dana et al in the year 1993 at Illianos (198 cases), Butner and Mecpherson in the year 1982at Lasvegas,(653 cases), Lindgrend et al in the year 1995 at Sweden (94 cases) of the incidence of vitreous hemorrhage it was found to be third or fourth single most common cause of vitreous hemorrhage, accounts for 11% of all cases, the mean age of incidence was 64 years and 88% were associated with hypertension.

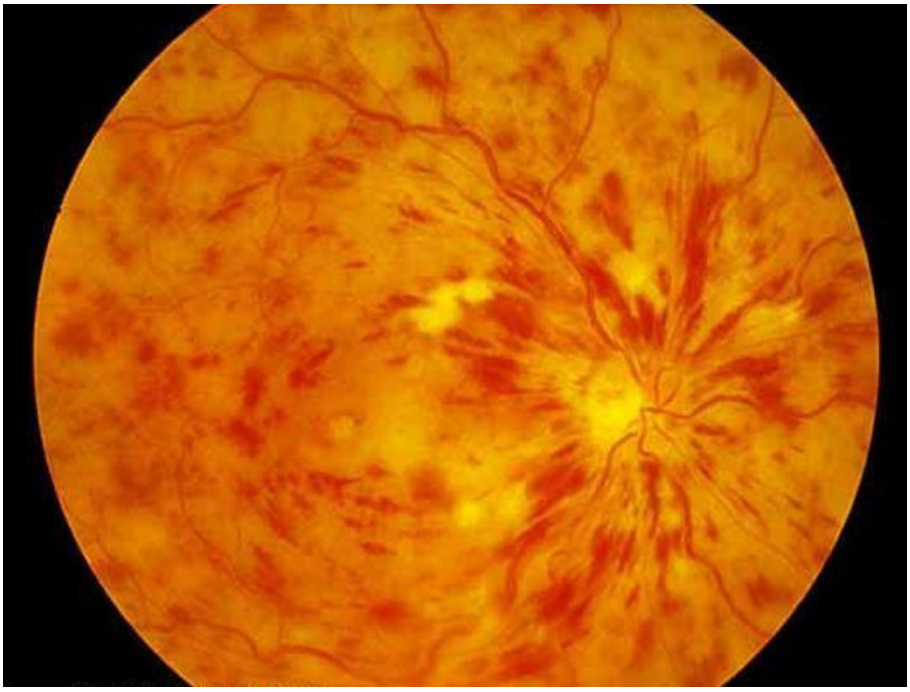
Other causes of vitreous hemorrhage:

The pars planitis, retinal vasculitis, Senile PVD, following post-operative complications, unknown causes accounts for 16%(16 out of 100) of all cases.

Supero Temporal Branch Retinal Vein Occlusion



Central Retinal Vein Occlusion



SUMMARY

The prospective study carried out on 100 patients who were all diagnosed as vitreous hemorrhage to analyse the various etiological incidence. The various etiologies of vitreus hemorrhage in this study are the following: proliferative diabetic retinopathy, ocular trauma, Eale's disease, spontaneous PVD, Retinal vein occlusion, pars planitis, retinal vasculitis, senile PVD, post operative complication, and unknown.

The total incidence of various etiologies wth respect to age, sex, involvement of the eye and associated ocular and systemic pathology were studied and recorded as follows:

Total number of cases 100

Age incidence between 22 to 78 Years

Sex incidence Male 67 and Female 33

DIAGNOSIS	AGE (YRS)	SEX M	SEX F	LATERALITY UNI	LATERALITY BI	WITHOUT PVD	WITH PVD	RT	RD	TOTAL	%
PDR	35 – 70	26	9	22	13	8	21	-	6	35	35
OCULAR TRAUMA	22 – 52	16	10	26	0	10	16	5	5	26	26
EALE'S DISEASE	22 – 58	8	3	6	5	8	3	-	-	11	11
SPONTANEOUS PVD	62 – 70	7	1	8	0	-	3	4	1	8	8
RETINAL VEIN OCCLUSION	45 – 63	3	1	4	0	3	1	-	-	4	4
PARS PLANITIS	34 – 46	1	1	2	0	2	-	-	-	2	2
RETINAL VASCULITIS	44 – 58	0	2	2	0	2	-	-	-	2	2
SENILE PVD	44 – 78	1	1	2	0	0	2	-	-	2	2
POST OP COMPLICATION (ECCE/PCIOL)	65 - 68	1	1	2	0	2	-	-	-	2	2
UNKNOWN	23 – 54	3	5	8	0	8	-	-	-	8	8

PROFORMA FOR THE STUDY OF VARIOUS ETIOLOGICAL INCIDENCE OF VITREOUS HAEMORRHAGE

1. **NAME :**
2. **AGE/SEX :**
3. **IP NO :**
4. **ADDRESS:**
5. **OCCUPATION:**
6. **CHIEF COMPLAINTS:**

A.OCULAR

	RE	**LE**
Duration		
a. Defective Vision (Distant/Near)		
b. Total loss of vision		
c. Floaters		
d. Flashes		
e. Pain		
f. Redness		

Flashes of light :

Smoke signals :

Photophobia :

Perceptions of shadows and cobwebs :

Visual haze:

Others :

B.SYSTEMIC-

- **Diabetes Mellitus.**
- **Hyper tension.**
- **Systemic Lupus Erythematosis.**
- **Rhenumatoid Arthiritis.**
- **Poly Arteritis Nodosa.**
- **Dermatomyositis.**
- **Bechet's Disease.**
- **Blood Disorders.**

Thermbocytopenia

Leukemia

Hemophilia

DIC

Anemia

- **Protein C Deficiency**
- **Anticoagulant Therapy**

7. HISTORY:

H/O – TRAUMA

Penetrating Ocular Injury.

Blunt Injury.

Perforating injury.

Chest injury.

H/O – INFECTION

BACTERIAL

- **Tuberculosis**
- **Sysphilis**
- **Others**

VIRAL

- **Cytomegalo virus**
- **Human Immuno Deficiency**
- **Others**

PROTOZOAL INFESTATION

- **Toxocara**
- **Toxoplasma Gondi**
- **Hydatid**
- **Cysticercosis**
- **Microfilaria**

H/O – OCULAR DISORDER

- **Eale's Disease.**
- **Posterior virteous detachment.**
- **Retinal tear.**

- **Retinal detachment**
- **Parsplanitis**
- **Retinal vasculitis**
- **Coat's Disease**
- **Central Retinal Artery Occlusion**
- **Branch Retinal Artery Occlusion**
- **Diabetic Retinopathy**
- **Hypertension Retinopathy**
- **Retinal Branch Artery Malformation**
- **Retinal vein repture**
- **Central retinal vein occlusion**
- **Branch retinal vein occlusion**
- **Persistant Hyaloid Artery**
- **Prematurity Retinopathy**
- **Arterio venous communication of retina**

H/O – TUMOUR

- **Retinal angioma**
- **Choroidal malignant melanoma**
- **Melanocytoma of the optic disc**
- **Retinoblastoma**
- **Retinal astrocytic hamartoma**

- **Carvernous haemangioma of the optic disc**

Duration:

8. FAMILY HISTORY:

9. PERSONAL HISTORY:

Smoking/ Alcoholism/ Vegetarian/ Non Vegetarian

10. TREATMENT HISTORY :

	Medications	**Surgery**

11. EXAMINATION:	**RE**	**LE**
Visual Acuity		
Extra Ocular Examination		
Tension		
Lids		
Conjunctiva		

Cornea

Iris

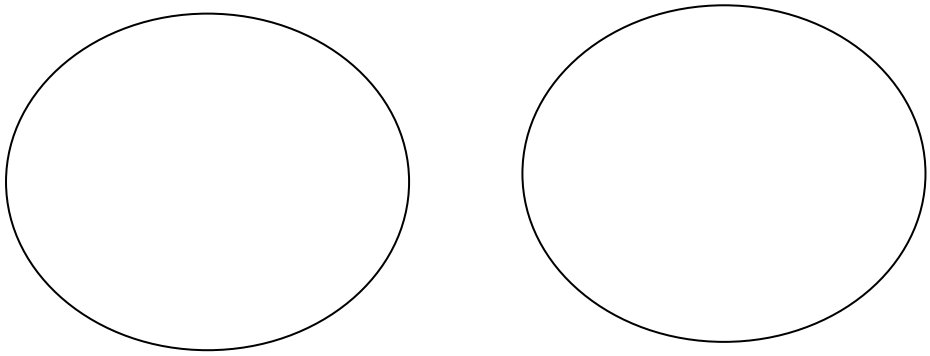
Anterior Chamber

Pupil

Lens

Vitreous

12. FUNDUS EXAMINATION:



RE **LE**

DIRECT OPHTHALMOSCOPE :

INDIRECT OPHTHALMOSCOPE :

SLIT LAMP EXAMINATION by the +90D lens.

13.CLINICAL DIAGNOSIS :

14.INVESTIGATIONS :

a. Blood investigations:Total WBC count/Differential count/Erythrocyte sedimentation rate
b. Investigations for collagen vascular disorders: CRP, Serum Creatinine, Rheumatoid factor, Antinuclear antibodies.
c. Blood sugar/Hba1c/BP
d. PT
e. aPTT

15. B SCAN:

16.TREATMENT:

17.FOLLOW UP:

CONCLUSION

The various etiological incidence of vitreous hemorrhage depends on the study population and vary with mean age and sex of the patient and region where the study is performed. The study concludes that

Proliferative diabetic retinopathy is the most common etiology because the incidence of the Diabetes mellitus is higher among the present population.

Ocular trauma is the second most common cause of vitreous hemorrhage because the frequency of ocular injury is remarkably prevalent in our region and one of the leading causes of non congenital blindness in younger individual.

Eale's disease is the third most common cause of vitreous hemorrhage because the incidence of Eale's disease is 1 in 250 of ophthalmic patients of Indian subcontinent.

Spontaneous PVD is the fourth most common cause of vitreous hemorrhage.

Retinal vein occlusion is fifth most common cause of vitreous hemorrhage.

Other causes includes parsplanitis ,retinal vasculitis ,senile PVD, post operative complications and unknown causes.

In our study we managed the cases medically and surgically.medical management included head end elevation, bed rest , control of systemic factors, pan retinal photocoagulation, and observation for three months.however it was noticed that these patients did not improve sidnificantly , and 27 %of the patients ,managed medically had persistent non clearing vitreous haemorrhage during the 3 rd visit. .these cases were taken up for surgical management in the form of 3 port 23 guage PPV with vitreous haemorrhage aspiration± endo laser . The patients who underwent surgical management had a significant improvement in visual acuity during the first week of post-op follow up. Hence it was concluded that surgical management had better outcome in cases of non clearing vitreous haemorrhage.

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MASTER CHART

S.No	Name	Age	Sex	Eye	V/A	Diagnosis	Etiology	Associated Ocular pathology	Associated Systemic diseases	Medical	Visual Acuity	Surgery	POST OP VA[BCVA]
1	BALAN	54	M	right eye	HM	Vitreous He	UNKNOWN			observation	2/60		
2	SHANMUGAM	61	M	right eye	HM	Vitreous He	PDR	PVD	Type II		HM (non clearing)	PPV/VH ASP/END LAS	6/18
3	ARUN	24	M	left eye	HM	Vitreous He	EALE'S DIS		TB	ATT/STER	2/60		
4	RAMESH	31	M	right eye	HM	Vitreous He	BLT INJ	PVD		observation	3/60		
5	VICTOR	33	M	left eye	HM	Vitreous He	BLT INJ			observation	3/60		
6	VANITHA	24	F	both eye	HM	Vitreous He	EALE'S DIS		TB	ATT/STER	2/60		
7	AROKIYAMMAL	65	F	right eye	HM	Vitreous He	PDR	RD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/24
8	SORNAMBAL	62	F	right eye	HM	Vitreous He	PDR		Type II	PRP	4/60		
9	MENAKA	48	F	right eye	HM	Vitreous He	BLT INJ	PVD		observation	3/60		
10	RAVI	60	M	left eye	HM	Vitreous He	PDR	PVD	Type II		CFCF(Non clearing)	PPV/VH ASP/END LAS	6/36
11	RAJAN	55	M	left eye	HM	Vitreous He	PDR	PVD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/36
12	SELVARAJ	49	M	both eye	PL	Vitreous He	PDR	PVD	Type I		HM(non clearing)	PPV/VH ASP/END LAS	6/24
13	NESAMANI	48	M	right eye	CF CF	Vitreous He	PDR	RD	Type I		CFCF(non clearing)	PPV/VH ASP/END LAS	6/24
14	SAROJA	58	F	right eye	HM	Vitreous He	PDR	PVD	Type II		CFCF(non clearing)	PPV/VH ASP/END LAS	6/36
15	DEVAKI	63	F	left eye	HM	Vitreous He	PDR	PVD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/60

S.No	Name	Age	Sex	Eye	V/A	Diagnosis	Etiology	Associated Ocular pathology	Associated Systemic diseases	Medical	Visual Acuity	Surgery	POST OP VA[BCVA]
16	PETER	35	M	left eye	HM	Vitreous He	PDR	RD	Type I		HM(non clearing)	PPV/VH ASP/END LAS	5/60
17	ABDUL	66	M	left eye	HM	Vitreous He	PDR	PVD	Type II		CFCF(non clearing)	PPV/VH ASP/END LAS	6/60
18	ANITHA	22	F	right eye	CF CF	Vitreous He	EALE'S DIS		TB	ATT/STER	2/60		
19	GEETHA	26	F	right eye	CF CF	Vitreous He	EALE'S DIS		TB	ATT/STER	3/60		
20	VIMALA	68	F	left eye	CF CF	Vitreous He	SPONT PVD	PVD/RT		observation	4/60		
21	KUMAR	40	M	right eye	PL	Vitreous He	BLT INJ	PVD		observation	4/60		
22	RAGAVAN	54	M	right eye	HM	Vitreous He	PDR		Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/60
23	AMUDHAM	58	M	both eye	HM	Vitreous He	EALE'S DIS	PVD	TB	ATT/STER	3/60		
24	RAJALAKSHMI	57	F	left eye	HM	Vitreous He	PDR		Type II	PRP	4/60		
25	GANAPATHY	49	M	left eye	HM	Vitreous He	BLT INJ	PVD		observation	2/60		
26	VELAN	60	M	both eye	HM	Vitreous He	PDR	PVD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/36
27	SAMBANDAM	62	M	right eye	HM	Vitreous He	PDR	PVD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/24
28	JAYARAM	66	M	right eye	HM	Vitreous He	SPONT PVD	PVD/RT		observation	4/60		
29	MANNAR	63	M	left eye	HM	Vitreous He	PDR	PVD	Type II		CFCF(non clearing)	PPV/VH ASP/END LAS	6/60
30	SUNDARI	46	F	right eye	CF CF	Vitreous He	UNKNOWN			observation	4/60		

S.No	Name	Age	Sex	Eye	V/A	Diagnosis	Etiology	Associated Ocular pathology	Associated Systemic diseases	Medical	Visual Acuity	Surgery	POST OP VA[BCVA]
31	MANOJ	25	M	right eye	CF CF	Vitreous He	BLT INJ	PVD			CFCF(non clearing)	PPV/VH/SFIOL	5/60
32	PERUMAL	53	M	both eye	PL	Vitreous He	EALE'S DIS	PVD	TB	ATT/STER	2/60		
33	KOUSALYA	29	F	right eye	CF CF	Vitreous He	UNKNOWN			observation	4/60		
34	PALANI	46	M	left eye	HM	Vitreous He	BLT INJ	PVD/RT		observation	3/60		
35	PANDIAN	62	M	right eye	HM	Vitreous He	SPONT PVD	PVD/RT		observation	3/60		
36	SUKUMARAN	54	M	left eye	HM	Vitreous He	PDR	PVD	Type I	observation	CFCF(non clearing)	PPV/VH ASP/END LAS	4/60
37	RAMARAJAN	36	M	right eye	HM	Vitreous He	BLT INJ			observation	4/60		
38	KRISHNAKUMAR	38	M	right eye	HM	Vitreous He	BLT INJ	PVD/RT		observation	4/60		
39	JOTHI	27	F	left eye	CF CF	Vitreous He	UNKNOWN			obervation	3/60		
40	VALI	26	F	left eye	CF CF	Vitreous He	BLT INJ	PVD		observation	5/60		
41	SUBBIAH	64	M	both eye	HM	Vitreous He	PDR		Type II	PRP	5/60		
42	POORNIMA	23	F	right eye	CF CF	Vitreous He	UNKNOWN			observation	3/60		
43	GANDHIRAJ	65	M	left eye	HM	Vitreous He	PDR	PVD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/18
44	THANGAVEL	48	M	right eye	HM	Vitreous He	BLT INJ	PVD/RT		observation	4/60		
45	ANAND BABU	50	M	right eye	HM	Vitreous He	PNT INJ	RT/PVD		Wound repair/observation repair/observe	4/60		

S.No	Name	Age	Sex	Eye	V/A	Diagnosis	Etiology	Associated Ocular pathology	Associated Systemic diseases	Medical	Visual Acuity	Surgery	POST OP VA[BCVA]
46	MALAR VIZHI	52	F	left eye	HM	Vitreous He	PER INJ	PVD/RT		Wound repair/observe	4/60		
47	NALA PERUMAL	59	M	both eye	HM	Vitreous He	PDR	PVD	Type I	PRP	5/60		
48	LOGANATHAN	63	M	left eye	HM	Vitreous He	PDR	PVD	Type II	PRP	5/60		
49	KAMARAJ	60	M	right eye	HM	Vitreous He	PDR		Type II	PRP	5/60		
50	RADHA	22	F	left eye	CF CF	Vitreous He	PNT INJ	PVD/RT		Wound repair/observe	2/60		
51	RAMASWAMY	49	M	left eye	PL	Vitreous He	BLT INJ	PVD/RD		observation	4/60		
52	KATHIRAVAN	46	M	right eye	PL	Vitreous He	PNT INJ	RD		Wound repair/observe	2/60		
53	SIVAKAMI	33	F	right eye	CF CF	Vitreous He	PNT INJ	RT/PVD		Wound repair/observe	2/60		
54	SRINIVASAN	35	M	right eye	CF CF	Vitreous He	BLT INJ	PVD		observation	3/60		
55	DURAIRAJ	65	M	left eye	PL	Vitreous He	SPONT PVD	PVD		observation	4/60		
56	MADHAVAN	68	M	left eye	CF CF	Vitreous He	SPONT PVD	PVD/RT		observation	4/60		
57	UNNAMALAI	60	F	left eye	HM	Vitreous He	PDR	PVD	Type II		CFCF(non clearing)	PPV/VH ASP/END LAS	6/24
58	MUHAMADIN	60	M	both eye	HM	Vitreous He	PDR	RD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/36
59	SUDHA	78	M	left eye	CF CF	Vitreous He	Senile PVD	PVD		observation	2/60		
60	SANKARI	45	F	right eye	PL	Vitreous He	BRVO		HT	systemic control	5/60		

S.No	Name	Age	Sex	Eye	V/A	Diagnosis	Etiology	Associated Ocular pathology	Associated Systemic diseases	Medical	Visual Acuity	Surgery	POST OP VA[BCVA]
61	KAMESH	34	M	left eye	CF CF	Vitreous He	PARS PLANITIS			STEROIDS	4/60		
62	PANKAJAM	62	F	both eye	HM	Vitreous He	PDR	RD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/60
63	NAGESHWARI	60	F	right eye	HM	Vitreous He	PDR		Type II		HM(non clearing)	PPV/VH ASP/END LAS	5/60
64	VASANTHI	32	M	left eye	HM	Vitreous He	EALE'S DIS	PVD	TB	ATT/STER	4/60		
65	INDRA	31	F	left eye	CF CF	Vitreous He	UNKNOWN			observation	3/60		
66	BABU	28	M	left eye	CF CF	Vitreous He	EALE'S DIS		TB	ATT/STER	3/60		
67	SRIDHAR	62	M	left eye	HM	Vitreous He	SPONT PVD	PVD		observation	3/60		
68	VIMALA	35	F	right eye	CF CF	Vitreous He	PNT INJ	RT		Wound rep/observation	2/60		
69	ANBARASI	46	F	right eye	HM	Vitreous He	PARS PLANITIS			STEROIDS	3/60		
70	UNNAMALAI	70	F	both eye	HM	Vitreous He	PDR	PVD	Type II		CFCF(non clearing)	PPV/VH ASP/END LAS	6/36
71	UDAYAN	59	M	right eye	PL	Vitreous He	CRVO	PVD	HT		1/60		
72	SELVAM	48	M	left eye	PL	Vitreous He	BLT INJ	PVD/RD		observation	1/60		
73	HARIRAM	57	M	right eye	PL	Vitreous He	BRVO		HT	Systemic control	5/60		
74	ANBARASI	58	F	right eye	PL	Vitreous He	RETINAL VAS			STEROIDS	4/60		
75	KUMARAESAN	46	M	left eye	HM	Vitreous He	BLT INJ	PVD		observa	3/60		

S.No	Name	Age	Sex	Eye	V/A	Diagnosis	Etiology	Associated Ocular pathology	Associated Systemic diseases	Medical	Visual Acuity	Surgery	POST OP VA[BCVA]
76	MUNNIYAPPAN	60	M	both eye	HM	Vitreous He	PDR		Type I	PRP	5/60		
77	KOVALAN	63	M	right eye	HM	Vitreous He	BRVO		HT	Systemic control/Observe	5/60		
78	KAVITHA DEVI	65	F	right eye	HM	Vitreous He	POST OP			ECCE	2/60		
79	GOURILATHA	44	F	left eye	HM	Vitreous He	PNT INJ	PVD/RD		Wound repair/observation repair/observation	3/60		
80	THAMARAI SELVI	44	F	left eye	HM	Vitreous He	RETINAL VAS			STEROIDS	3/60		
81	KARUPASAMY	44	M	both eye	HM	Vitreous He	PDR	PVD	Type II		CFCF(non clearing)	PPV/VH ASP/END LAS	6/60
82	MASILAMANI	68	M	right eye	HM	Vitreous He	POST OP			anti-VEGF	2/60		
83	THANIGAI	44	M	right eye	HM	Vitreous He	UNKNOWN			observation	3/60		
84	JAYAMANI	44	M	both eye	HM	Vitreous He	PDR	PVD	Type II		HM(non clearing0	PPV/VH ASP/END LAS	5/60
85	PONNUSAMY	44	M	right eye	HM	Vitreous He	UNKNOWN			observation	3/60		
86	GOPAL	64	M	left eye	HM	Vitreous He	SPON PVD/RD	PVD		observation	3/60		
87	SUMITHA	44	F	left eye	CF CF	Vitreous He	Senile PVD	PVD		observation	3/60		
88	FREDRICK	44	M	right eye	CF CF	Vitreous He	BLT INJ	PVD/RT		observation	3/60		
89	BALASUNDARAM	44	M	both eye	HM	Vitreous He	PDR	PVD	Type II	PRP	5/60		

S.No	Name	Age	Sex	Eye	V/A	Diagnosis	Etiology	Associated Ocular pathology	Associated Systemic diseases	Medical	Visual Acuity	Surgery	POST OP VA[BCVA]
90	YAMUNA	44	F	left eye	CF CF	Vitreous He	PNT INJ	PVD/RD		Wound rep/observation	2/60		
91	PRIYA	44	F	left eye	CF CF	Vitreous He	BLT INJ	PVD		observation	2/60		
92	MUTHAIAH	44	M	both eye	HM	Vitreous He	PDR	PVD	Type II	PRP	5/60		
93	NANDHU	44	M	both eye	CF CF	Vitreous He	EALE'S DIS		TB	ATT/STER	3/60		
94	KANDAN	44	M	left eye	HM	Vitreous He	PDR		Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/24
95	KANNAPAN	44	M	left eye	CF CF	Vitreous He	BLT INJ	PVD/RT		observation	3/60	-	
96	VEDHA	70	M	right eye	HM	Vitreous He	SPONT PVD	RD		observation	3/60	-	
97	RAJAPPA	44	M	left eye	PL	Vitreous He	PDR	RD	Type II		HM(non clearing)	PPV/VH ASP/END LAS	6/60
98	VIJAY	44	M	both eye	CF CF	Vitreous He	EALE'S DIS		TB	ATT/STER	3/60	-	
99	VARADARAJAN	44	M	both eye	PL	Vitreous He	PDR	PVD	Type I		CFCF(non clearing)	PPV/VH ASP/END LAS	6/36
100	SIVA	44	M	right eye	CF CF	Vitreous He	EALE'S DIS		TB	ATT/STER	3/60	-	

KEY TO MASTER CHART

VH	Vitreous hemorrhage
PPV/VH ASAP/END LAS	Pars plana vitrectomy with vitreous haemorrhage aspiration with endolaser
ATT	Anti tubercular treatment
STER	Steroids
PRP	Pan retinal photocoagulation
BRVO	Branch retinal vein occlusion
CRVO	Central retinal vein occlusion
BLT INJ	Blunt injury
PER INJ	Perforating injury
PNT INJ	Penetrating injury
SPONT PVD	Spontaneous posterior vitreous detachment
PVD	Posterior vitreous detachment
RT	Retinal tear
RD	Retinal detachment
Anti-VEGF	Anti vascular endothelial growth factor

LIST OF SURGERIES

S.No	Name	Age	Sex	Diagosis	Etiology	Surgery
1	SHANMUGAM	61	M	Vitreous He	PDR	PPV/VH ASP/END LAS
2	AROKIYAMMAL	65	F	Vitreous He	PDR	PPV/VH ASP/END LAS
3	RAVI	60	M	Vitreous He	PDR	PPV/VH ASP/END LAS
4	RAJAN	55	M	Vitreous He	PDR	PPV/VH ASP/END LAS
5	SELVARAJ	49	M	Vitreous He	PDR	PPV/VH ASP/END LAS
6	NESAMANI	48	M	Vitreous He	PDR	PPV/VH ASP/END LAS
7	SAROJA	58	F	Vitreous He	PDR	PPV/VH ASP/END LAS
8	DEVAKI	63	F	Vitreous He	PDR	PPV/VH ASP/END LAS
9	PETER	35	M	Vitreous He	PDR	PPV/VH ASP/END LAS
10	ABDUL	66	M	Vitreous He	PDR	PPV/VH ASP/END LAS
11	RAGAVAN	54	M	Vitreous He	PDR	PPV/VH ASP/END LAS
12	VELAN	60	M	Vitreous He	PDR	PPV/VH ASP/END LAS
13	SAMBANDAM	62	M	Vitreous He	PDR	PPV/VH ASP/END LAS
14	MANNAR	63	M	Vitreous He	PDR	PPV/VH ASP/END LAS
15	MANOJ	25	M	Vitreous He	BLT INJ	PPV/VH/SFIOL
16	SUKUMARAN	54	M	Vitreous He	PDR	PPV/VH ASP/END LAS
17	GANDHIRAJ	65	M	Vitreous He	PDR	PPV/VH ASP/END LAS
18	UNNAMALAI	60	F	Vitreous He	PDR	PPV/VH ASP/END LAS
19	MUHAMADIN	60	M	Vitreous He	PDR	PPV/VH ASP/END LAS
20	PANKAJAM	62	F	Vitreous He	PDR	PPV/VH ASP/END LAS
21	NAGESHWARI	60	F	Vitreous He	PDR	PPV/VH ASP/END LAS
22	UNNAMALAI	70	F	Vitreous He	PDR	PPV/VH ASP/END LAS
23	KARUPASAMY	44	M	Vitreous He	PDR	PPV/VH ASP/END LAS
24	JAYAMANI	44	M	Vitreous He	PDR	PPV/VH ASP/END LAS
25	KANDAN	44	M	Vitreous He	PDR	PPV/VH ASP/END LAS
26	RAJAPPA	44	M	Vitreous He	PDR	PPV/VH ASP/END LAS
27	VARADARAJAN	44	M	Vitreous He	PDR	PPV/VH ASP/END LAS