CLINICAL APPROACH TO VITREOUS HEMORRHAGE

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DEFINATION

Vitreous hemorrhage is defined as the presence of extravasated blood within the space outlined by the internal limiting membrane of the retina posteriorly and laterally, the non-pigmented epithelium of the ciliary body antero-laterally and the lens zonular fibers and posterior lens capsule anteriorly.[8]

Symptoms

Patients present with sudden painless diminution of vision, floaters, photopsia, and the perception of shadow and cobwebs.

Signs

1. **Slit lamp examination:** - Red blood cells can be appreciated when the light beam is focused posterior to the lens. Mild afferent pupillary defect may be present.
2. **Ophthalmoscopy:-** In severe vitreous hemorrhage, the red fundus reflex may be absent, and there may be no fundus view. In mild vitreous hemorrhage, blood obscures part of the retina and retinal vessels.

Chronic vitreous hemorrhage has a yellow ochre appearance secondary to the break down of hemoglobin. Depending on the etiology there may be other abnormalities.

TYPES OF VITREOUS HAEMORRHAGE

a) Intravitreal Haemorrhage

Blood present within vitreous cavity readily clots along the vitreous fibres, looks red in colour and from the site of haemorrhage it forms finger like intravitreal projections. The blood remains stationary and along with the globe movements, it shows slight movement. The intravitreal blood tends to accumulate in the dependent part of the globe due to gravity. Blood in the vitreous gel itself promotes vitreous degeneration, liquefaction and cavity formation.[9]

b) Subvitreal haemorrhage

Also called as Subhyaloid or Preretinal haemorrhage. Blood present between the posterior face of the vitreous and internal limiting membrane of the neural retina. Blood in this location remains unclotted and shifts along with change of posture. It is typically described as boat shaped haemorrhage with a straight superior horizontal configuration and a semicircular inferior border. This type of haemorrhage is commonly seen in diabetics.[9]

c) **Breakthrough vitreous haemorrhage**

Haemorrhage occurring under retina following trauma or from subretinal neovascular membrane, may break through retina into vitreous cavity, called Breakthrough vitreous haemorrhage.[9]

PATHOGENESIS OF VITREOUS HAEMORRHAGE

1. Retinal vascular disorders that cause retinal ischemia.

Mechanism :9 Retinal ischemia

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Release of angiogenic factors like VEGF,
Insulin like growth factors and Basic fibroblast growth factor

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New vessels grow from disc and retina

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Blood vessels bleed easily due to vitreous traction.

e.g. Proliferative Diabetic Retinopathy, Ischemic retinal vein occlusion, Eale's disease, Familial exudative vitreoretinopathy and Proliferative sickle cell retinopathy.[1]

2. Retinal vascular abnormality not associated with retinal ischemia

Eg.Rupture of retinal arterioles, macroaneurysm associated with systemic hypertension or haemorrhage from retinal angioma.

3. Rupture of normal retinal vessel

Occurs due to traction exerted on normal retinal vessels during posterior vitreous detachment that may be spontaneous or caused by blunt trauma or in retinal tears and retinoschisis. It can also occur in Terson's syndrome, Valsalva retinopathy, haematological disorders anaemia, leukemia/ coagulation disturbances and vascular occlusions.[1]

4. Breakthrough Bleeding

Breakthrough of subretinal haemorrhage dissecting through the retina without an associated retinal detachment occurs following subretinal haemorrhage from choroidal neovascualr membrane.Other important causes are choroidal malignant melanoma, retinal vascular occlusion and idiopathic polypoidal choroidal vasculopathy.[1]

ETIOLOGY

Children:

1. Trauma
   a. Birth Trauma
   b. Shaken Baby Syndrome
   c. Traumatic Child abuse
2. Retinopathy of Prematurity
3. Retinal Break
4 Retinal Detachment
5. Retinoblastoma
6. Congenital Retinoschisis
7. Pars Planitis
8. PHPV
9. Terson's Syndrome

Adult:

A. Bleeding from diseased retinal vessels or abnormal new vessels
   a. Diabetic Retinopathy
   b. Eales' Disease
   c. Acute Severe Vasculitis
   d. Acute Central Retinal Vein Occlusion
   e. Sickle Cell Retinopathy
   f. Retinal Capillary Angioma
   g. Hypertensive Retinopathy
   h. Radiation Retinopathy
   i. Macroaneurysm

B. Rupture of Normal Retinal vessels.
   a. Retinal Breaks
   b. Retinal Detachment
   c. Posterior Vitreous Detachment
   d. Trauma

C. Extension of hemorrhage from other sources
   a. Age related macular degeneration with CNVM
   b. Intraocular Tumour - Choroidal Melanoma

D. Miscellaneous
   a. Terson's Syndrome
   b. Bleeding

EXAMINATION AND INVESTIGATION

1. **History:** History of ocular or systemic disease especially diabetes, hypertension, trauma, retinal break or detachment in the other eye and any family history of detachment.

2. **Slit lamp examination:** Complete ocular examination with slit lamp including check for neovascularization of iris, intraocular pressure measurement and a dilated fundus examination of both eyes using indirect ophthalmoscope. Contralateral eye examination is often diagnostic e.g. diabetes, age related macular degeneration, Eale's disease, retinopathy of prematurity. Presence of peripheral retinal lesions in the other eye should alert one to possibility of retinal break. In cases of spontaneous vitreous hemorrhage without obvious vascular disease it is important to do an indirect ophthalmoscopic examination with scleral depression. Retinal breaks are commonly located superiorly in

cases of dense vitreous hemorrhage. In traumatic vitreous hemorrhage (especially open globe injuries) scleral depression is avoided for the first 3-4 weeks.

3. **Ultrasonography :** When there is no fundal glow a B-scan Ultrasound helps in a diagnosis of vitreous hemorrhage as well as in detection of any associated posterior vitreous detachment, retinal detachment (traction or rhegmatogenous), intraocular tumor, retinal break (if large), scleral rupture . In fresh, mild hemorrhage, dots and short lines are displayed on B-Scan, and a chain of low amplitude spikes is found on A-Scan. The more dense the hemorrhage, the more opacities are seen on B-Scan and the higher is the reflectivity on A-scan. If the blood organizes, larger interfaces are found which have higher reflectivity on A-Scan and may be confused with retinal detachment. Kinetic echography shows undulating after movements on B-Scan which are to be differentiated from the less mobile retinal and choroidal detachment.[10]

4. **Fluorescein angiography** may aid in defining the etiology although the quality of the angiogram may depend on the density of the hemorrhage. Angiography is especially useful in diagnosis of proliferative retinopathies, wherein abnormal new vessels show leakage of the dye into the vitreous cavity, and in age related macular degeneration, where there is subretinal leak. Fluorescein angiography of the contra lateral eye is also an important diagnostic aid.

DIFFERENTIAL DIAGNOSIS

1. **Vitritis :** The onset is rarely as sudden as in vitreous hemorrhage. History is very important. Pain or redness at onset can alert one to vitritis. History of any surgical intervention is important, the possibility of trivial undiagnosed trauma should be specifically asked for and explored. Detailed history of systemic illnesses must be taken if vitritis is suspected. There may also be antecedent signs of anterior or posterior uveitis. Pars planitis should be looked for. On slit lamp examination there may be presence of vitreous cells in the anterior vitreous. If unexplained Vitritis' is seen, masquerade syndromes like intra-ocular lymphoma should be ruled out.[10]

2. **Retinal Detachment:** may occur without a vitreous hemorrhage, yet the symptoms may be identical, the fundus view may be difficult in a fresh retinal detachment with dense vitreous hemorrhage. However, the retina can usually be viewed with indirect ophthalmoscopy by an experienced observer. Ultrasonography is indicated in case of doubt.

3. Very advanced **'brown'** or **'black' cataract** can sometimes give an impression of vitreous hemorrhage on indirect ophthalmoscopy. The ability to see some retinal detail despite the advanced cataract can often help to differentiate the two conditions. If a doubt persists a combined A & B Scan is helpful.

NATURAL HISTORY AND PROGNOSIS

The natural history and prognosis of vitreous hemorrhage depends on the underlying disease. In general, patients with diseases that have no tendency for recurrent bleeding, such as avulsion of a vessel associated with a retinal tear or posterior vitreous detachment have good prognosis for resolution of the vitreous hemorrhage and restoration of vision. Clearance of blood from the vitreous is a slow process, with a time constant in the order of 1% per day. Hemorrhage in the vitreous gel remains suspended in a lamellar fashion until the vitreous liquifies and the blood sinks to the bottom of the vitreous cavity where it is absorbed.

Patients with diabetic retinopathy as the underlying disease process have a relatively poorer prognosis for spontaneous clearing of vitreous hemorrhages and restoration of vision especially alter recurrent bleeds. Among patients with vitreous hemorrhage secondary to retinal vein obstruction, branch vein occlusion patients have the best visual prognosis, hemi-central vein occlusion patient inter-mediate and central retinal vein occlusion worst. Natural history of vitreous hemorrhage secondary to Eale's Disease and retinal arterial macro aneurysms is generally good while that following age related macular degeneration and sickle cells retinopathy is poor.

Visual recovery following trauma is unpredictable and depends on the nature and extent of injury.

Uniqueness of Vitreous Haemorrhage

Rapid clot formation, slow lysis of fibrin, extra cellular lysis of red blood cells persistence of intact red blood cells for month and lack of early, polymorphoneuclear response[2] are the unique features of VH.

The vitreous haemorrhage does not always clear spontaneously in patients with diabetic retinopathy. Sometimes long standing vitreous haemorrhage with the accumulated red cells and red cells debris suspended in and mixed with vitreous collagen can clinically present as an ochre membrane.[2]

Complications due to non-clearing of Vitreous Haemorrhage

Haemosiderosis bulbi, retinal damage glial and fibrovascualr proliferation and glaucoma (ghost cell / haemolytic) .

MANAGEMENT

Once the diagnosis of vitreous hemorrhage has been made, the patient must be referred to an ophthalmologist with special interest in vitreo-retinal disease and well-versed with indirect ophthalmoscopy. Unlike retinal detachment, vitreous hemorrhage is usually not a surgical emergency. However, prompt examination especially for a first time bleed is important as surgical intervention may be required if there is retinal detachment. If other causes like AMD with CNVM, intra-ocular tumour are suspected these must be specifically investigated for and treated promptly. In most other cases one can safely wait for a few weeks for the hemorrhage to clear up spontaneously. The patient must be reassured and the plan of management explained. The treatment modalities are:

A. Conservative Management: Bed rest with head end of the bed elevated. Eye movements maintain RBC in the vitreous cavity in diffuse suspension. If the eye movements are diminished, the blood gravitates to the bottom of the space. Sometimes bilateral patching is advised as this accelerates the settling of the blood cells. Settling of blood enables visualization of the superior retina for examination and treatment. If dense vitreous hemorrhage persists, and the etiology remains unknown, the patient is followed with a B-scan ultrasound every 1-3 weeks to rule out a retinal detachment.

Eliminate asprin, nonsteroidal anti-inflammatory drugs and other anticlotting agents unless they are medically necessary. No topical or systemic medication is needed in this situation as none is of proven benefit. Oral Ascorbic Acid (Vit. C) may be given for faster clearance (though not clinically proven) as there is more liquificaiton and loss of gel structures in eye with exogenous Vit. C.[3]

Checklist for preoperative evaluation of vitreous haemorrhage

- Recent visual acuity
- Relative afferent papillary defect
- IOP
- Rubeosis iridis, angle new vessels.
- Lenticular opacities
- Lacrimal sac and adenexal evaluation
- USG : B Scan, A Scan, kinetic echography
- Examination of fellow eye.

B. Photocoagulation or cryotherapy: Laser photocoagulation or cryotherapy is used to seal retinal breaks. Photocoagulation is done as soon as media is clear enough to permit it. Indirect ophthalmoscopic laser delivery especially with 810-nm diode laser is very useful when it may not be possible to achieve adequate laser burns with the green laser. As these retinal breaks are associated with vitreous traction they should be surrounded completely by 3-4 rows of confluent laser burns. Patents must be informed that if retinal detachment occurs before firm chorio-retinal adhesion is formed, surgery in the form of scleral buckling or vitrectomy may be required.

If there are large retinal tears and the media is not clear enough for adequate prophylactic treatment the option of early surgery has to be seriously considered. In the case of vascular retinopathies it is usually safer

to wait for longer periods for the hemorrhage to clear before starting laser photocoagulation. The other eye must have a fundus fluorescein angiography to detect proliferative vascular disease and receive immediate laser photocoagulation if new vessels are present.

Some surgeons advocate peripheral retinal cryotherapy for non-resolving vitreous hemorrhage. Peripheral retinal cryotherapy accelerates the resorption rate of vitreous hemorrhage by causing a breakdown of the blood retinal barriers and an increase in tissue plasminogen activator and has been used as a treatment modality for non-resolving vitreous hemorrhage. However this should be used with caution in cases where organisation and contraction of the vitreous gel can precipitate a traction retinal detachment. This is especially true in cases where there is no PVD or very limited PVD.

C. Posterior Hyaloidotomy: Hemorrhage located - between the internal limiting membrane and the retina may cause permanent macular changes before spontaneous resolution occurs. In few cases posterior hyaloidotomy may be performed using a Nd-YAG laser which disrupts the internal limiting membrane and releases blood cells into the vitreous cavity.

D. Pars Plana Vitrectomy: Pars plana vitreous surgery has revolutionised the management of vitreous hemorrhage, dramatically improving the prognosis in a number of cases and enabling quicker visual rehabilitation in may others. Vitrectomy or surgical removal of blood is usually performed as:

Urgent Surgery

1. Vitreous hemorrhage accompanied by RD
2. Neovascular AMD with vitreous hemorrhage and subretinal hemorrhage.
3. Certain cases of trauma.

Early surgery :

Vitreous hemorrhage secondary to vascular retinopathies:

1. Bilateral hemorrhage.
2. Associated with early iris neovascularization
3. Associated with hemolytic "ghost cell" glaucoma
4. With severe progressive fibrovascular proliferation
5. Dense premacular hemorrhage.

Late/Elective Surgery :

Chronic vitreous hemorrhage with no traction on retina or other associated problems, with good vision in the other eye may be observed almost indefinitely if the patient is not handicapped or for medical reasons, is not a good candidate for surgery.

Management in Specific conditions

Proliferative Diabetic Retinopathy (63% of cases of Vitreous Haemorrhage)

Diabetic Retinopathy Study (DRS) has shown that treatment by pan retinal photocoagulation reduced the risk of visual loss of 60% when high risk characteristics are noted.[4] DRS showed that once there is visually disabling VH early virectomy was advantageous only in Type I DM, but it should not be interpreted that we must wait for few months in all patients with Type-2 DM.[4]

Retinal Vein Occlusion (Incidence 7%)

VH is more common in BRVO than CRVO, as in CRVO anterior segment neovascularisation is more common than posterior segment neovascularisation.

So, in vitreous hemorrhage is also common.

First patient is observed and followed up for 2-4months. If VH clears then laser photocoagulation is given.

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If VH does not clear

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Vitrectomy with Sector Photocoagulation.

Eale's Disease

62% of patients with Eale's disease present VH. Early vitrectomy has been advocated with 87% of eyes showing improvement in VA.[5]

Macroaneurysms

Macroaneurysm due to systemic hypertension usually resolve spontaneously or may require laser treatment occasionally.

ARMD

Haemorrhage results from extravasations of the subretinal

blood from a bleeding choroidal neovascularisation. In these patients haemorrhage usually resolves spontaneously. If vitrecotmy is planned it should be for the better seeing eye, to restore the peripheral vision.

Vitreous Hemorrhage in Children

Most common cause is trauma (exclude shaken baby syndrome) followed by retinoblastoma and leukemia. Early surgery may prove beneficial in these eyes as delay can lead to both amblyopia and anisometropia.[6]

Vitreous Haemorrhage in vitrectomised eyes[9]

- **Early :** 1st week following surgery
- **Late :** Within 1-6 months

Early	Late
Release of erythrocytes trapped in vitreous base or results from intra-operative inadequate homeostasis	➢ Rapidly growing neovascular tissue particularly on the remnants of posterior cortical vitreous attached to pre-existing neovascular tissues. ➢ Iris neovascularisation ➢ Fibrovascualr ingrowth at the internal aspects of the sclerotomy sites.

TREATMENT

Intra-operative endolaser photocoagulation or post-operative panretinal laser photocoagulation promotes regression of neovascularisation. Anterior fibrovascualr proliferation causing haemorrhage in the vitreous cavity is treated by indirect laser photocoagulation or by retinal cryopexy of the 360° peripheral retina extending upto ora.

Haemorrhage in vitrectomised eyes clears spontaneously and in about 30% cases repeated haemorrhage occurs. Non-clearing haemorrhage in vitrectomised eye following fluid gas exchange may require revitrectomy.

Intra-operative Vitreous haemorrhage

Such bleeding clears spontaneously on vitreous wash or following temporary raising of IOP. Otherwise endodiathermy or liquid perflurocarbon is used to arrest bleeding.

Rehabilitation and Upcoming Trends

Patients with macular degeneration and diabetic maculopathy and optic nerve pathology have poor vision prognosis. They can be rehabilitated with low vision aids.

The upcoming modalities are **pharmacological vitreolysis** with hyaluronidase and autologous plasmin enzyme which may reduce the frequency of vitrectomy or facilitate as adjuvants in easing vitreous surgery.[7]

SUMMARY

Vitreous hemorrhage is a common presenting sign in patients with a vitreo-retinal disorder. It is alarming to the patient as it produces dramatic visual symptoms and often marked visual loss. Though, most of the time, vitreous hemorrhage is not a surgical emergency the patient should be examined and investigated very carefully to detect those who do require early intervention. Appropriate management ensures good prognosis in most cases of vitreous hemorrhage.

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