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VITREOMACULAR INTERFACE ANOMALIES: A REVIEW

Ophthalmology

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KEYWORDS

Vitreous is a clear gel that occupies the posterior segment of the eye which is made up of 98% water and 2% macromolecules (1, 2). The outer cortex of vitreous which is made up of dense collagen (Type II) is firmly attached to internal limiting membrane (Type IV collagen). Vitreous is strongly attached at vitreous base, optic disc and to some extend on macula. As the age advances, gel undergoes liquefaction and attachment between vitreous and internal limiting membrane (ILM) weakens and it gets completely separated which is called posterior vitreous detachment(PVD), which usually begins in the perifoveal macula(3,4). PVD occurs in two phases- liquefaction followed by separation. The completion of vitreopapillary separation often characterized by the Weiss ring, is usually an acute and symptomatic event. Inadequate or incomplete vitreoretinal interface separation result in anomalous PVD with vitreomacular interface (VMI) anomalies. Anomalous PVD is defined as partial vitreous detachment with persistent attachment in the macular region featuring an anomalous strength of adhesion to one or more structures in the posterior pole, resulting in tractional deformation of retinal tissue (5).
With the evolution of Optical Coherence Tomography visualization and understanding of the vitreo-retinal interface has improved (6). OCT is central to diagnosis and increased the likelihood of detecting vitreomacular interface anomalies. Fully detached vitreous is difficult to detect by OCT, clinical examination and ultrasound are useful tools to diagnose PVD (Fig 1).

Posterior Vitreous Detachment (PVD) and anomalous PVD:
Outer cortex of vitreous is made up of type II collagen which is strongly attached to internal Limiting membrane (type IV Collagen). With the advancing age vitreous gel liquefies, a process called synchysis and collapses which is known as syneresis. As the age advances vitreous detaches from retina, a process known as Posterior vitreous Detachment(PVD) (Fig 2). Though some people experience some floaters, generally PVD is asymptomatic. If the traction on optic disc (vitreo papillary adhesion) persists, it can lead to neovascularization of optic disc (NVD), which may cause vitreous haemorrhage and loss of vision(6) especially in diabetics. Traction near vitreous base cause retinal breaks and retinal detachment. When remnants of perifoveal vitreous cortex get attached on to macula after detached from surroundings, Focal VMA develops(7), which is usually asymptomatic. If traction is enough to cause disturbance in macular architecture VMA can lead to VMT, which is always pathologic and symptomatic.

Definition and Classification of Vitreomacular Adhesion(VMA) and vitreomacular Traction(VMT):

Vitreomacular adhesion denotes residual strong adhesion between vitreous and macula when PVD is incomplete whereas Vitreomacular Traction (VMT) is defined as structural abnormality associated with loss of vision. Symptoms associated with VMT are blurring of vision, metamorphopsia and difficulty in reading.

Vitreomacular Adhesion: focal adhesion of the vitreous face within macular region.

Vitreomacular Traction: VMA causing focal tractional distortion of macula greater than or equal to the Simpson standard.

Vitreo macular traction syndrome: VMT associated with loss of visual function

Symptomatic VMA: VMT syndrome, macular hole or cases where normal or abnormal VMA coexist with macular diseases, with loss of vision.

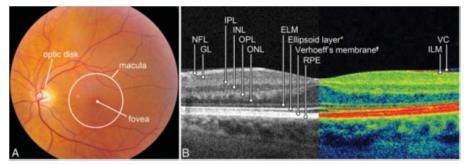


Fig 1. Fundus photograph and OCT scan of normal normal macula. * formerly known as inner segment – outer segment junction. †interdigitation between photo receptors outer segment and RPE.

Diagram showing relationship between PVD, VMA and VMT.

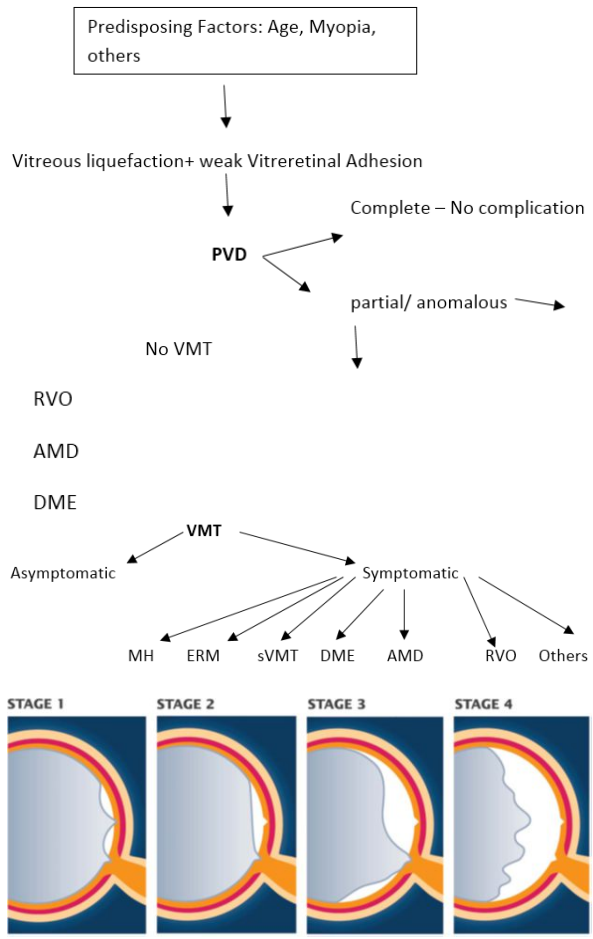
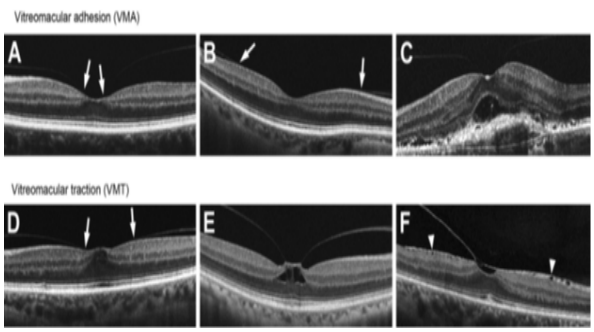


Fig 2 .Stages of PVD.Johnson and Sebag(4,5). Stage 1: Perifoveal detachment. Vitreous attached to fovea, optic disc and mid –peripheral retina. Stage 2: Vitreous attached to optic disc and mid periphery. Stage 3 PVD: Vitreous detached from fovea, mid- peripheral retina and attached only at optic disc. Stage 4: Complete PVD (courtesy Ref :1)

International Vitreomacular Traction Study (IVTS) Group has developed a simple, evidence based clinically applicable classification system to identify, monitor and manage vitreomacular interface disorders. This is based on multiple OCT B-line scans images and classified by the size of attachment or lesion and presence of retinal or vitreoretinal conditions (8).

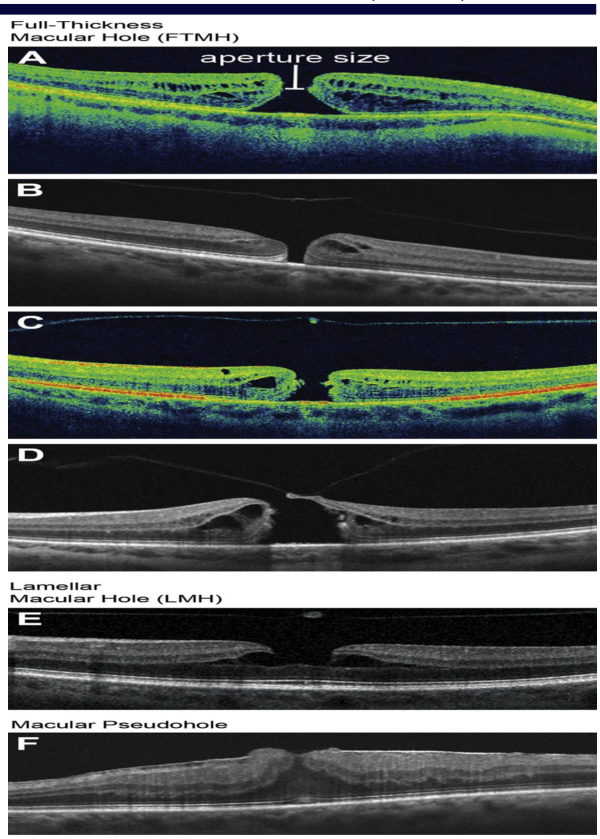
Optical Coherence Tomography – Based Definition and classification of Vitreomacular Adhesion:

Anatomical State	IVTS classification system
VMA	**Definition** Evidence of perifoveal vitreous detachment from retinal surface vitreous cortex attached to macula within a radius of 3mm no change in foveal anatomy **Classification:** by extend of attachment area-**focal** <1500µm, **Broad** >1500µm: **Isolated** absence of concurrent retinal condition, **Concurrent:** associated with retinal anomaly
VMT	**Definition:** Evidence of perifoveal vitreous detachment from retinal surface vitreous cortex attached to macula within a radius of 3mm Associated with distortion of foveal surface, intraretinal structures, **classification:** by extend of attachment area-**focal** <1500µm, **Broad** >1500µm: **Isolated** absence of concurrent retinal condition, **Concurrent:** associated with retinal anomaly
FTMH	Full thickness macular lesion, interrupting all layers from ILM to RPE **Classification:** By Size Small≤250µm Medium≥250 µm and ≤400µm Large ≥400µm By presence or absence of VMT **By cause :** Primary (initiated by VMT) Secondary (trauma or associated disease)
LMH	Definition: irregular foveal contour Defect in the inner fovea Intraretinal splitting Intact photoreceptor layer
Macular pseudohole	**Definition:** invaginated or heaped foveal edges Concomitant ERM with central opening No loss of retinal tissue Steep macular contour to central fovea with near normal central foveal thickness.



Optical coherence tomography (OCT) scans illustrating (A-C) vitreom acular adhesion (VMA) and (D-F) vitreomacular traction (VMT)

according to the International Vitreomacular Traction Study Classific ation System.



Management of Vitreomacular adhesion and traction:

VMA: Usually Focal VMA resolves spontaneously. Watchful waiting and monitoring by Amsler Grid evaluation is advised in these patients. Regular OCT monitoring is recommended in patients with metamorphopsia. Pharmacological vitreolysis should be considered when VMA has progressed to VMT. These agents break down the peptide bonds in laminin and fibronectin molecules which keeps the adhesion between ILM and vitreous. Collagenase,chondroitina se,hyaluronidase,plasmin, plasminogen activator are few agents used for vitreolysis. Plasmin is manufactured from patients own blood and it is very unstable. Ocriplasmin, a recombinant truncated form of human plasmin with molecular weight 27.8kDa. it is a DNA molecule which is more stable than plasmin and has emerged as new vitreolytic agent. It is recombinant protease with activity against fibronectin and laminin. MIVI TRUST studies indicate that in patients with isolated VMT without ERM, had resolution of VMT by 28 days after injection of Ocriplasmin (29.8%) versus placebo (7.7%).Ocriplasmin was approved for treatment of symptomatic VMA and VMT, including macular hole with diameter <400µ. In patients with isolated VMT, without ERM had resolution by 28 days after single injection of 0.125mg/0.1ml ocriplasmin(29.8%) as compared to placebo injection (7.7%)(9). Natural history of VMT associated with AMD, DME and retinal vein occlusion is poorly understood. If there is no release of VMT, patients can undergo PPV. 30-50% of stage I MH regress spontaneously where as only 10% stage II and III holes do so. 125 µg Ocriplasmin injection resulted in spontaneous resolution of FTMH in 40.6% (MIVI TRUST- Microplasmin for Intra Vitreous Injection-Traction Release without Surgical Treatment) of patients, within 28 days versus placebo (10.6%). Closure rates were higher in small FTMH (58.3%) as compared to medium (36.8%) and large MH(0%). Only 8.7% of eyes with FTMH with ERM had resolution of VMT after ocriplasmin injection. Ocular adverse effects of Ocriplasmin injection are vitreous floaters, photopsia, blurred vision, conjunctival harmorrhage, potential for lens subluxation, retinal breaks and dyschromatopsia (as yellow vision).ERG changes in the form of reduced 'a' and 'b' wave amplitude is reported in patients experiencing dyschromatopsia. Patients can experience transient loss of vision, which is attributed to the disruption in the ellipsoid layer (previously known as photoreceptor IS/OS junction). Wide spread retinal dysfunction can develop in patients due to its effect on laminin which is present in other layers of retina, including Bruch membrane, interphotoreceptor matrix, External limiting membrane, Outer plexiform layer, inner plexiform layer and ILM. Though the effect on

photoreceptor outer segment is transient, the action on rods may get more prolonged.

Surgical treatment: Pars Plana Vitrectomy(PPV) with ILM and or ERM peeling is the standard treatment advocated for VMT. The success of relieving VMT ranges from 80-90%. The surgery helps to improve metamorphopsia, and can also help in improving vision. But this can be associated with various complications like retinal breaks, retinal detachment, endophthalmitis, and development of cataract.

VMT and FTMH with focal VMA are called symptomatic VMT: in small macular holes (size <400µm) with VMT, pharmacological vitreolysis are indicated .

Full thickness macular Hole: If size of the hole is <400µm, pharmacological vitreolysis can be tried. Chances of FTMH closure (40.6%) at day 28 vs placebo (10.6%).

Impending macular hole: It is described in cases where there is full thickness macular hole in one eye and VMT is observed in the fellow eye.

Lamellar macular hole: round or oval reddish lesion with partial thickness foveal defect. OCT features include irregular foveal contour, intra retinal splitting, Broad VMA and tractional Macular Thickening: when VMA is > 1500µm, it can cause schisis of retinal layers.

ERM and macular pucker: The exact cause of ERM is not completely known. After PVD vitreous remnants are attached to the surface of retina (vitreoschisis), which stimulate proliferation of hyalocytes, glial cells and histiocytess on its surface cause development of ERM.

Centripetal tractional forces on retinal surface cause macular pucker.

Pseudohole: ERM with central opening can give rise to Pseudo hole formation which appear as round or oval shaped defect. Surgical membrane peeling is advised in all these condition.

Conclusion:

Clinical outcome of various vitreomacular interface anomalies depends on ability of treating physician's ability to form timely diagnosis and treatment accordingly. Untreated cases can lead to permanent damage to retina. With the availability of newer modalities of treatment patients prognosis in these cases have improved.

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