INTRODUCTION

Vitreomacular traction is the hallmark of a heterogeneous group of traction maculopathies, such as macular holes, macular pucker, tractional cystoid macular oedema, vitreomacular traction syndrome, and myopic traction maculopathy ***(Vanderbeek and Johnson, 2012).***

Myopic traction maculopathy, also known as myopic foveoschisis, is a schisis-like thickening of the retina in eyes with high myopia with posterior staphyloma. The pathologic features may also include lamellar or full-thickness macular holes and shallow foveal detachment ***(Panozzo and Mercanti, 2004).***

The mechanism of myopic vitreomacular traction is characterized by the transmission of static and dynamic antero-posterior or tangential traction forces to the macula that arises from age-related vitreous changes and pathologic epiretinal cell proliferation at the vitreoretinal interface. Imaging studies of the vitreoretinal interface and experimental investigations indicate that posterior vitreous detachment with full-thickness or partial-thickness adherences to the macula represents the pathologic prerequisite for vitreomacular traction disorders ***(Sebag, 2008).***

Patients with myopic foveoschisis may complain of worsening visual acuity with metamorphopsia or they may be relatively asymptomatic with stable visual acuity and no visual complaints ***(Panozzo and Mercanti, 2004).***

Due to the difficulties of visually assessing the transparent vitreous, biomicroscopy has been shown to underestimate the true extent to which abnormal vitreomacular adhesion exists ***(Gallemore et al, 2000).***

With the use of optical coherence tomography (OCT) imaging, the understanding of vitreoretinal traction disease pathophysiology has expanded even more. Subtle degrees of vitreoretinal traction previously not visible could be recognized as causative or contributory to the formation of macular edema ***(Akiba et al, 2000).***

The current approach in treating vitreoretinal interface is to perform a pars plana vitrectomy surgery if the severity of vision loss justifies the surgical risk ***(Aniz and Marc, 2014a).***

The internal limiting membrane (ILM) is the anatomic site of pathology that mediates vitreomacular traction forces to the retina. It has been well accepted that ILM peeling can improve the hole closure rate and postoperative best corrected visual acuity ***(Gao et al, 2013).***

Because the ILM is a barely visible membrane, identification of the ILM is a challenging step in surgery, and removal is difficult even for experienced retina surgeons. Therefore, staining the ILM with vital dyes is essential for increased visibility of the ILM and decreasing surgical trauma to the retina during ILM removal ***(Lai et al, 2009).***

Aim of the Work

To assess the role of vitrectomy with internal limiting membrane (ILM) peeling in changing visual outcomes and optical coherence tomography (OCT) findings of the macula in patients with myopic vitreomacular traction syndrome.

Diseases of the Vitreomacular interface

Vitreomacular adhesion (VMA):

The International Vitreomacular Traction Study (IVTS) defined VMA as perifoveal vitreous separation without causing any structural or functional abnormality. The posterior vitreous face lies in opposition to the fovea, often with separation of the vitreous face from the surrounding macula. It is an Optical coherence tomography (OCT) finding that is almost always the result of normal vitreous aging, which may lead to pathologic conditions. Therefore, VMA is a kind of incomplete posterior vitreous detachment (figure 1) ***(Koizumi et al, 2008).***

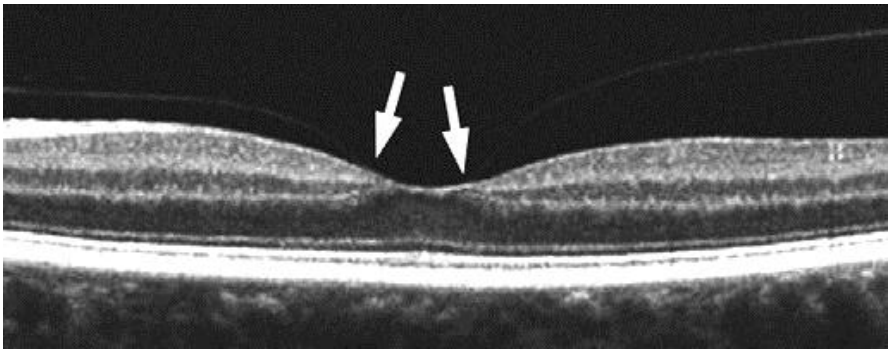

Figure (1): OCT scan illustrating VMA (arrows) with normal foveal contour ***(Duker et al, 2013)***

Vitreomacular traction (VMT):

The International Vitreomacular Traction Study (IVTS) defined VMT as the concurrence of detectable retinal anatomic changes on OCT with perifoveolar posterior vitreous detachment (PVD). All of the following anatomic criteria must appear on at least 1 OCT scan to classify an eye as having VMT:

1) Evidence of perifoveal vitreous cortex detachment from the retinal surface.

2) Macular attachment of the vitreous cortex within a 3-mm radius of the fovea.

3) Distortion of the foveal surface, intraretinal structural changes, elevation of the fovea above the retinal pigment epithelium (RPE), or a combination of them, but no full thickness interruption of all retinal layers (figure 2) ***(Johnson, 2012).***

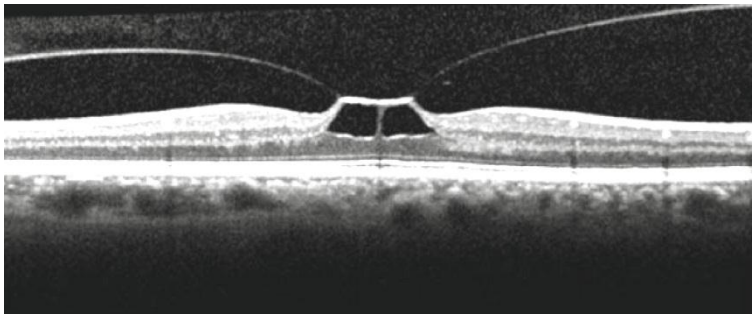

Figure (2): Optical coherence tomography (OCT) scan illustrating vitreomacular traction (VMT) with intrafoveal pseudocyst ***(Aniz and Marc, 2014c)***

Full-thickness macular hole (FTMH):

FTMH is defined as a foveal lesion with interruption of all retinal layers from the internal limiting membrane to the retinal pigment epithelium (figure 3) ***(Sonmez et al, 2008).***

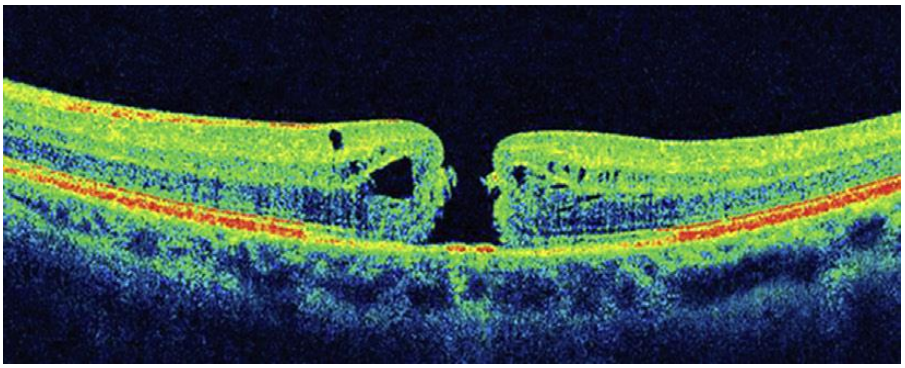

Figure (3): OCT scan illustrating Full-thickness macular hole (FTMH) ***(Duker et al, 2013)***

Lamellar macular holes (LMH):

Abortive macular holes may result in lamellar holes, in which only the inner part of the fovea is torn away from the retina. Anatomic Optical coherence tomography (OCT) -based features of LMH include the following: (1) an irregular foveal contour; (2) a defect in the inner fovea (may not have actual loss of tissue); (3) intraretinal splitting (schisis), typically between the outer plexiform and outer nuclear layers; and (4) maintenance of an intact photoreceptor layer (figure 4) ***(Witkin et al, 2006).***

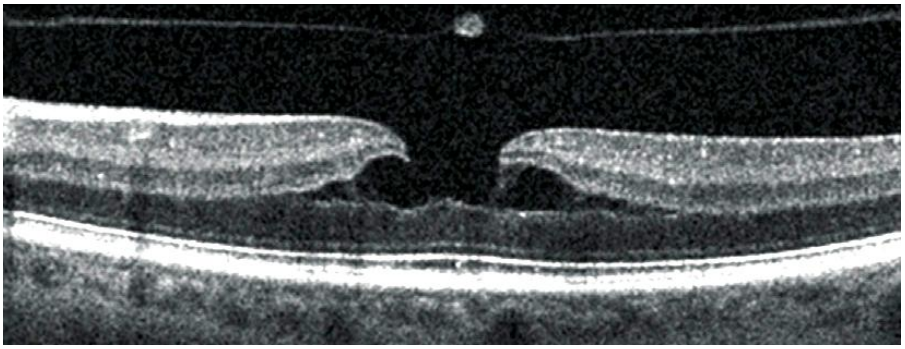

Figure (4): Lamellar macular hole ***(Duker et al, 2013)***

Myopic foveoschisis (MF):

Myopic foveoschisis is an anteroposterior distension of the retina due to the combined effects of the progressive deepening of the staphyloma and the stiffness of the retinal surface due to changes in the adherent vitreous cortex ***(Spaide and Fisher, 2005).***

Schisis of the retinal layers occurs commonly in the outer plexiform layer (outer retinoschisis). In minority of cases, it may occur within more internal retinal layers (inner retinoschisis) with detachment of the internal limiting membrane (ILM). Thus, MF is a totally different condition from congenital macular retinoschisis, which is a splitting of the retinal nerve fiber layer from the rest of sensory retina ***(Jiang et al, 2006).***

EPIDEMIOLOGY

Myopia is a disease seen at a high frequency in Asians. It is more common in women than men and results in various fundus changes from 30 years of age. Takano and Kishi first reported detection of Myopic traction maculopathy (MTM) using optical coherence tomography (OCT) in 1999. They described foveal retinal detachment and foveoschisis in patients with high myopia and posterior staphyloma ***(Takano and Kishi, 1999).***

Posterior vitreous detachment (PVD) is more common in myopic patients, occurring 10 years earlier than in emmetropia and hyperopia, resulting in myopic vitreopathy ***(Nguyen and Sebag, 2005).***

The incidence rates of myopia-specific macular complications such as myopic foveoschisis and macular holes with or without retinal detachments have not been well documented, because these complications are rare. Myopic foveoschisis was identified in 9% of patients with posterior staphyloma. In patients with a macular hole and retinal detachment, 8.9% develop the same pathology in the fellow eye within 4 years. ***(SirinVas and David, 2013)***

11–50 % of patients with myopic foveoschisis have a macular hole and/or retinal detachment within 2–3 years of follow-up without treatment. ***(Shimada et al. 2013)***

PATHOGENESIS

Myopic Traction Maculopathy (MTM):

High myopia has been noted to have tractional effects at the vitreoretinal interface and is referred to as myopic traction maculopathy (MTM) also known as myopic foveoschisis (MF) or myopic macular retinoschisis. Myopic foveoschisis is a schisis-like thickening of the retina in eyes with high myopia with posterior staphyloma (figure 5) ***(Mitry and Zambarakji, 2012).***

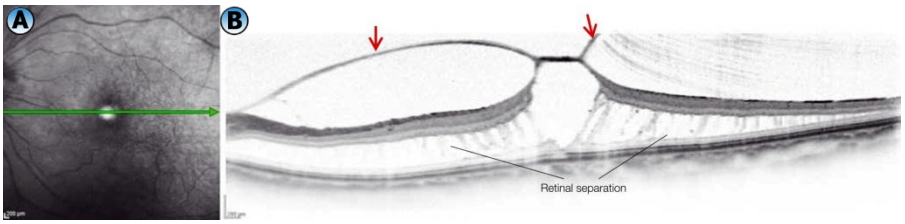

Figure (5): IR+OCT horizontal scan of the left eye of a 71-year-old female, highly myopic eye, BCVA 0.5. The posterior vitreous cortex is adhered to the surface of the fovea. The fovea is highly elevated by the centrifugal forward traction, and large foveal cystoid space and retinoschisis in the outer plexiform layer are seen ***(Nagahisa and Masanori, 2014a).***

Four major traction mechanisms were suggested in the pathogenesis of MTM: vitreomacular traction (from perifoveal Posterior vitreous detachment [PVD]), remnant cortical vitreous layer (after PVD), epiretinal membrane and intrinsic noncompliance of the internal limiting membrane (ILM) ***(Johnson, 2012).***

Lower extensibility of the retinal blood vessels and ILM, than other sensory retinal tissues, play an important role in the onset of myopic foveoschisis. The sensory retina is elongated posteriorly with posterior staphyloma formation, but the retinal blood vessels and ILM cannot follow the elongation of the sensory retina closely, and consequently the superficial layer of the retina is pulled in the longitudinal direction causing retinoschisis. Thus, myopic foveoschisis is a split between the flexible outer retina and the inflexible inner retina ***(Shimada et al, 2006).***

Myopic foveoschisis begins as retinoschisis without a retinal detachment i.e. the retinoschisis type (figure 6). A retinal detachment can start at the fovea after several months or years if there is sufficient traction i.e. the foveal detachment type (figure 7) ***(Ikuno et al, 2008).***

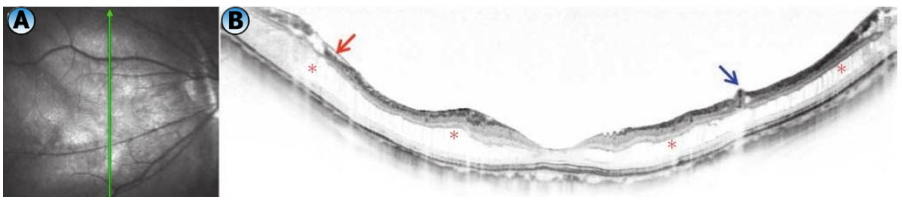

Figure (6): Myopic foveoschisis: Case without foveal detachment. A 66-year-old female, OD, BCVA 1.5, an axial length 30.31 mm. A: Color fundus photograph. B: OCT vertical scan: ILM detachment (red arrow) is observed in the left side of the OCT image. Retinoschisis is apparent over a wide area (*). Vascular microfolds (blue arrow). The choroid is thinned ***(Nagahisa and Masanori, 2014b).***

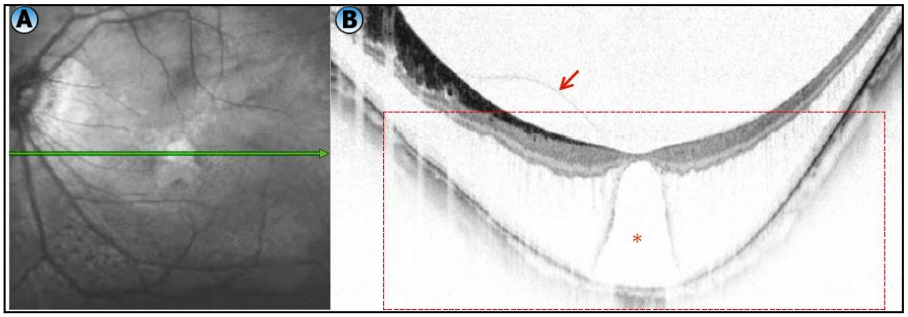

Figure (7): Myopic foveoschisis: Case with foveal detachment. A 50-year-old female, OS, BCVA 0.4, an axial length 29.45 mm A: Color fundus photograph. B: OCT horizontal scan: Extensive retinoschisis is evident in the macular area. Retinal detachment (*) is also seen in the fovea centralis. The posterior vitreous cortex is exhibited on the nasal side of the fovea centralis ***(Nagahisa and Masanori, 2014b).***

In highly myopic eyes, retinal detachment can occur without the formation of retinal breaks before the onset of macular hole retinal detachment (MHRD). This condition is seen in 10–34% of highly myopic eyes ***(Baba et al, 2003).***

The progression from myopic foveoschisis to foveal retinal detachment can be divided into 4 stages based on optical coherence tomography (OCT) findings. Stage 1 involves the localized elevation of the foveal outer retinal layers and the increased reflectivity in these layers. In Stage 2, an outer lamellar hole (a foveal detachment on OCT) develops in the fovea centralis and parafovea. Stage 3 comprises the inward expansion of the lamellar hole into the retina. In Stage 4, the inner border of the outer lamellar hole extends until it comes into contact with the innermost layer of the separated retina and retinal detachment has expanded (figure 8) ***(Shimada et al, 2008).***

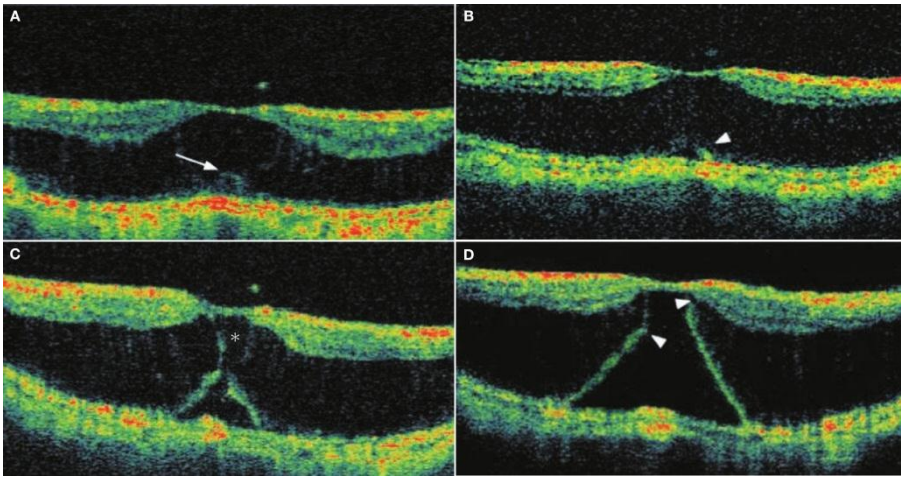

Figure (8): An example of the formation process of a macular hole in high myopia A: Stage 1, high reflectivity associated with the focal elevation of outer retinal layers can be seen. B: Stage 2, outer lamellar hole formation. C: Inward expansion of the outer lamellar hole and progression of retinoschisis. D: The outer lamellar hole progresses forward and comes in contact with the retinoschisis ***(Shimada et al. 2008).***

The occurrence of a foveal detachment (FD) and even more a breakdown in the photoreceptor layer associated with a myopic foveoschisis are at high risk of macular hole (figure 9) ***(Shimada et al, 2008).***

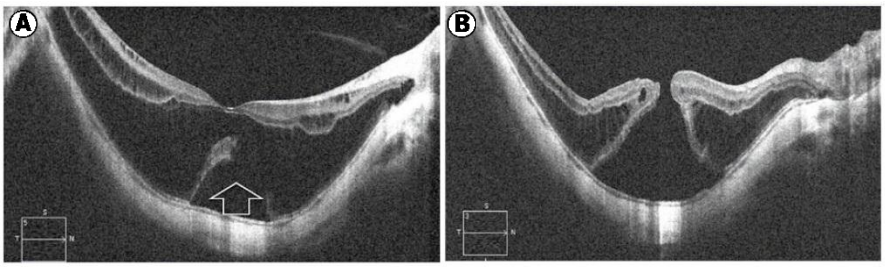

Figure (9): Evolution of a myopic foveoschisis toward a macular hole. (A) OCT showing foveoschisis with a foveal detachment (arrow). (B) Full-thickness macular hole occurred 4 months later with surrounding retinal detachment ***(Carsten et al., 2017b).***

High myopic macular holes

The pathogenesis of myopic macular holes may be different from that of non-myopic eyes because of anomalies associated with posterior staphyloma and because the vitreous cortex often remains adherent to the retinal surface. In general, the adherence of the posterior vitreous cortex to the retinal surface around the hole is wider than in idiopathic macular holes, and macular hole staging cannot be exactly applied to high myopic macular holes (figure 10) ***(Smiddy et al, 2009).***

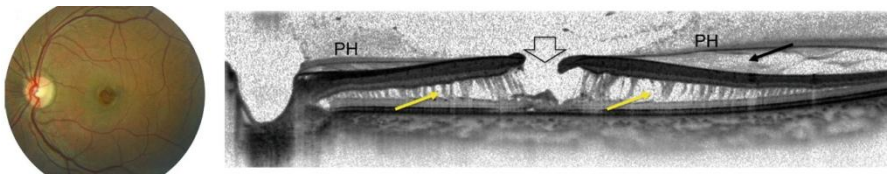

Figure (10): Small macular hole in a myopic foveoschisis. Fundus color photo showing an aspect of macular hole. The B-scan shows that the aspect of macular hole seen on color photo is mainly due to a lamellar hole (large arrow) in a foveoschisis (yellow arrows). The posterior hyaloid (PH) (posterior vitreous cortex) is delaminated in a vitreoschisis (black arrows) but still adherent to the retinal surface ***(Carsten et al, 2017b).***

Macular hole with or without retinal detachment

Interestingly, two types of macular holes are seen on optical coherence tomography (OCT) in high myopia with myopic foveoschisis, and they have distinctly different prognoses. The retinoschisis type is characterized only by retinoschisis, and no retinal detachment has started. The foveal detachment type is more advanced, and the photoreceptors are separated from the pigment epithelium (figure 11) ***(Jo et al, 2012).***

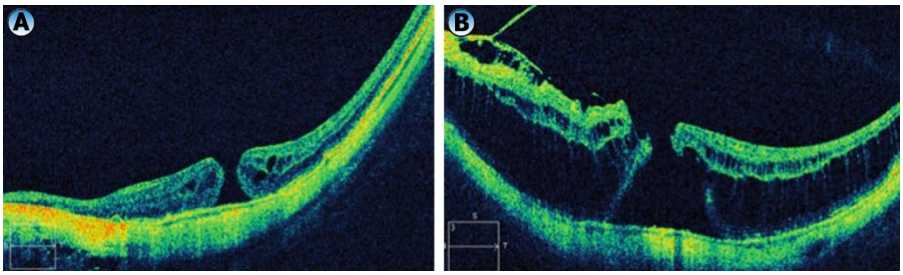

Figure (11): Two types of myopic holes are seen on the OCT images. A: The retinoschisis type. B: The foveal detachment type ***(Carsten et al, 2017c).***

In general, a macular hole rarely causes a rhegmatogenous retinal detachment. On the other hand, it is well known that myopic macular holes can be complicated by development of extensive retinal detachment. This unusual natural history for macular holes is probably determined by the combination of tangential traction of epiretinal membrane and/or antero-posterior traction of the vitreous ***(Bando et al, 2005).***