It’s the Time to Focus on the Imaging of Posterior Vitreous and Vitreoretinal Interface by Optical Coherence Tomography

Abstract
Vitreous humor plays important role in ocular ingrowth, the maintaining of ocular media transparency and tonus. It is difficult to exactly assess the relationship between the vitreous and the macula with only slit lamp bio microscopy. Spectral domain optical coherence tomography provides to evaluate the vitreomacular interface and posterior vitreous. Some vitreomacular interface diseases or the risk factors for these can be demonstrated the imaging of posterior vitreous by optical coherence tomography.

Keywords: Posterior vitreous; Imaging; Vitreoretinal interface; Vitreomacular disease; Optical coherence tomography

Abbreviations: OCT: Optical Coherence Tomography; VRI: Vitreo-Retinal Interface; VMA: Vitreo-Macular adhesion; APVD: Anomalous Posterior Vitreous Detachment; VMTS: Vitreomacular Traction Syndrome; MH: Macular Hole; MP: Macular Pucker; AMD: Age-related Macular Degeneration; DME: Diabetic Macular Edema; RVO: Retinal Vein Occlusion; DR: Diabetic Retinopathy; PVS: Posterior Vitreo-Schisis; MRS: Myopic Retino Schisis; VPTS: Vitreo-Papillary Traction Syndrome; NVD: Neovascularization Disk; NVE: Neovascularization Elsewhere; PDVR: Proliferative Diabetic Vitreo-Retinopathy; NPDR: Non-Proliferative Diabetic Retinopathy; PVD: Posterior Vitreous Detachment; ERM: Epi-Retal Membrane; CME: Cystoid Macular Edema; PPVP: Posterior Pre cortical Vitreous Pocket; TTPH: Thickened Taut Posterior Hyaloid; PPV: Pars Plana Vitrectomy; PTILM: Postvitrectomy Taut Internal Limiting Membrane

Introduction
Vitreous humor that is located between the lens and retina is a clear gel-like substance that occupies a large portion of the intraocular space (4.0 mL of ocular volume). It plays role ocular ingrowth, the maintaining of ocular media transparency and tonus, and it provides some refractive media. Vitreous is formed by water (98-99%), the meshwork of collagen fibril with hyaluronic acid. It most firmly attached to optic disc, macula, over macular blood vessels and the vitreous base[1]. It is difficult to exactly assess the relationship between the vitreous and the macula with only slit lamp bio microscopy because the vitreous is a transparent and clear tissue. Spectral domain optical coherence tomography (OCT) provides to observe and to evaluate the vitreomacular interface (VRI) and posterior vitreous. Some VRI diseases or the risk factors for these can be demonstrated by OCT of posterior vitreous.

Vitreo-Macular Adhesion (VMA) and Anomalous Posterior Vitreous Detachment (APVD)
Vitreomacular adhesion is one of the results of APVD. Although VMA's itself is not dangerous, it can cause vitreomacular traction syndrome (VMTS), and eventually severe retinal damage and visual loss. Additionally, it has been considered that the symptomatic VMA could contribute to the development of macular hole (MH) and macular pucker (MP). VMA may also be associated with neovascular age-related macular degeneration (AMD), diabetic macular edema (DME), retinal vein occlusion (RVO), and diabetic retinopathy (DR) [2-10]. According to "Classification of Vitreomacular Adhesion, Traction, and Macular Hole" by "The International Vitreomacular Traction StudyGroup", VMA is diagnosed in the presence at least one of those findings in OCT: partial vitreous detachment as indicated by elevation of cortical vitreous above the retinal surface in the perifoveal area; persistent vitreous attachment to the macula within a 3-mm radius from the center of the fovea; acute angle between posterior hyaloid and inner retinal surface; absence of changes in foveal contour or retinal morphology [2].

APVD is due to an imbalance between the degree of gel liquefaction and weakening of vitreoretinal adhesion. If the degree of vitreous liquefaction overwhelms the degree of weakening of vitreoretinal adherence, anomalous and strong vitreoretinal traction at this interface may cause VTMS, retinal or intravitreal hemorrhages in especially ischemic and diabetic retinopathies, DME, posterior vitreoschisis (PVS), myopic retinoschisis (MRS), MP, MH, periphery retinal tears/detachments, vitreopapillary traction syndrome (VPTS), neovascular AMD. Additionally, it can aggravate the neovascularization in disk (NVD) and elsewhere retina (NVE) in especially proliferative vitreoretinopathy.
Posterior Vitreo-Schisis (PVS)

Posterior vitreo-schisis is the splitting of the posterior vitreous cortex. PVS is the consequence of APVD with strong VMA, if syneresis occurs. When the posterior vitreous cortex splits, the leaving the outermost layer of PVS remains attached to the macula. The remnant anterior portion of the posterior vitreous cortex collapses forward. The split in the posterior vitreous cortex can induce some vitreomacular pathologies [11-13]. It has been reported that PVS was detected in the cases with especially DME, PDVR, retinovascular disease and Eale’s disease. PVS can occur much less frequently in the patients with neovascular AMD and nonproliferative diabetic retinopathy (NPDR). As PVS was detected in half of eyes with MH and MP, it has been considered that APVD with vitreomacular might play role in the pathogenesis of these [13-18].

Vitreo-Macular Traction Syndrome (VTMS)

Vitreomacular traction syndrome is a disorder of the VRI characterized by at least one of those OCT findings according to “Classification of Vitreomacular Adhesion, Traction, and Macular Hole” of “The International Vitreomacular Traction Study Group” [1]; an incomplete posterior vitreous detachment (PVD) (elevation of cortical vitreous above the retinal surface in the perifoveal area); persistent vitreous attachment to the macula within a 3-mm radius from the center of the fovea; acute angle between posterior hyaloid and inner retinal surface; an abnormally strong adherence of the posterior hyaloid on the macula; the anteroposterior traction exerted by the syneretic vitreous pulling at adherent sites on the macula; presence of changes in foveal contour or retinal morphology including the distortion of foveal surface; intraretinal structural changes including pseudocyst formation, elevation of fovea from the RPE; and absence of full thickness interruption of all retinal layers. Retinal thickening underlying VTMS is greatest. If VTMS involves the fovea, focal foveal distortion is greater [2,19]. VTMS can lead to formation of a full/partial-thickness MH, epiretinal membrane (ERM), and cystoid macular edema (CME). It usually occurs in cases with age-related vitreous changes and vitreous liquefaction, high myopia, neovascular AMD, DME, DR, and RVO. OCT exactly identifies and details VTMS [2,19].

Posterior Precortical Vitreous Pocket (PPVP)

The posterior precortical vitreous pocket, called also “bursa premacularis” is a liquefied lacuna anterior to the macular area. It is physiologically present in the vitreous of adults except newborns. PPVP can be confirmed by spectral-domain OCT. PPVP is observed as convex configuration posterior vitreous cortex detached from macula. The premacular thin vitreous cortex is the posterior wall of the PPVP. Its anterior wall is formed by the vitreous gel [20-26]. It has been demonstrated that PPVP plays role in some VRI disorders including idiopathic ERM, DME, PDVR, foveal cyst and MH. These pathologies may be explained by the posterior wall of the PPVP acting as an ERM on the macula or the residual premacular vitreous cortex causing a trampoline-like traction on the perifovea [20-26].

Taut Thickened Posterior Hyaloid (TTPH)

Thickened taut posterior hyaloid is firstly described by Lewis et al in the eyes with DME that had a clinically “thickened, taut, and glistening premacular posterior hyaloid” in the absence of signs of VTMS. In this case of TTPH, posterior hyaloid usually seems as a glistening reflex in ophthalmoscopy. Striae limited only to posterior hyaloid but not to retina can be observed. These striae in TTPH is not involve the retina or retinal vasculature, as distinct from eyes with ERMs. Recent reports demonstrated that pans plana vitrectomy (PPV) may be an efficacious in the treatment for DME associated with TTPH. Kaiser et al. [6] found that eyes with a clinical diagnosis of TTPH had shallow elevation of a partially detached hyaloid with VMAs. Patients with a TTPH may have the partial posterior hyaloid separation and underlying intraretinal or subretinal fluid and retinal thickening without edema as OCT finding [27-30]. Recently, Lewis et al. reported that PPV with removal of the TTPH provided a complete resolution in 80% of eyes with DME associated with TTPH [27]. In another study, it has been reported that PPV provided complete resolution in 81% of eyes with DME associated with TTPH [29]. However, the results of the surgery is depend on the duration of edema and the presence of the macular ischemia, atrophy, and postoperative ERMs [30].

Postvitrectomy Taut Internal Limiting Membrane (PTILM)

Postvitrectomy taut ILM is an OCT entity that can cause persistent DME after PPV with prior hyaloid removal. This finding can be distinguished from a TTPH and VTMS by the absence of an attached or partially attached hyaloid. Additionally, PTILM has hyper-reflective than normal reflective ILM and it has not any VMA and diffuse retinal edema [31,32]. Postvitrectomy taut ILM usually develops in first one-two months following PPV with hyaloid removal. It is nonresponsive to non-surgical therapeutic applications such as laser photocoagulation, intravitreal injections of corticosteroid and anti-VEGF agents. In focal type of PTILM, ILM exists striae while in diffuse type, there is a glistening in the posterior pole. OCT reveals the edema in our retina without manifest traction [31,32]. It has been considered that PTILM might be develop due to the tangential traction of the ILM onto the distant retina because ILM has no elasticity. Based on the histopathological examinations of PTILMs, it has been observed that PTILM has contractile cellular elements such as retinal pigment epithelium/glia cells having smooth muscle actin [31,32].

Vitreo-Papillary Traction Syndrome (VPTS)

Vitreopapillary traction syndrome is characterized by the traction of posterior hyaloid on the optic nerve head or an incomplete posterior vitreous detachment. VPTS can cause the appearance of papilledema because of tenting of the papillary rim and intrappapillary and peripapillary hemorrhages. Although VPTS may be idiopathic or isolated. It can also occur as secondary to APVD in PDVR and central RVO. In ophthalmoscopy and B scan ocular ultrasonography, optic disc traction may be overlooked. However, the traction in VPTS can be easily showed by OCT [33-40].
Table 1: Disorders of posterior vitreous or VRI and associated pathologies.

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<tr>
<th>VMA</th>
<th>APVD</th>
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<th>PPVP</th>
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<td>VMTS</td>
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<td>Idiopathic ERM</td>
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<td>Prior PPV with hyaloid removal</td>
<td>Idiopathic/Isolated</td>
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<td>Prior PPV with hyaloid removal</td>
<td>APVD in DVRP and central RVO</td>
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Conclusion

Optical coherence tomography is a noninvasive diagnostic tool for the imaging of posterior vitreous, retina, and choroid. In the usage of OCT, the interest has been concentrated on the imaging of retina, and recently of choroid. However, it's the time to focus on the imaging of posterior vitreous. Because OCT can be used for the early detection of the diseases of VRI and posterior vitreous.

References

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