PEER REVIEWED

Epiretinal Membrane Exacerbated by Vitreomacular Traction and Anomalous Posterior Vitreous Detachment

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Abstract

An anomalous posterior vitreous detachment (PVD) develops when a partial PVD exhibits persistent vitreoretinal attachment. This can contribute to a spectrum of vitreoretinal abnormalities, such as epiretinal membrane (ERM), vitreomacular traction (VMT) and macular hole, which are termed vitreomacular disorders (VMDs). These disorders often co-exist and reinforce any present vitreoretinal traction, leading to further disruption of the retinal architecture. This teaching case report explores a patient with ERM, VMT and anomalous PVD that progressed after more than 20 years of observation.

Key Words: *epiretinal membrane, vitreomacular traction, anomalous posterior vitreous detachment, vitreomacular disorders, vitreous, optical coherence tomography*

Background

Vitreomacular disorders (VMDs) represent a spectrum of vitreomacular interface abnormalities,[1] which includes epiretinal membrane (ERM), vitreomacular traction (VMT) and macular hole (MH). Each VMD encompasses a wide range of severity, from asymptomatic to vision-threatening. The introduction of optical coherence tomography (OCT) has allowed diagnosis of VMDs at their earliest and asymptomatic stages. However, inconsistencies with management exist because there are no reliable prognostic factors to predict which patients with a VMD will need surgery and no consensus on when to refer for surgery. This teaching case report explores a patient with ERM, VMT and anomalous posterior vitreous detachment (PVD) that progressed after more than 20 years of observation. It focuses on the disease process and medical management of ERM and VMT. It is appropriate for practicing clinicians at all levels, from third- and fourth-year optometry students to residents to seasoned professionals.

Case Description

An 89-year-old Caucasian male presented for his annual eye exam with gradually decreasing vision in the left eye in the past year. He denied experiencing photopsia, floaters, trauma or ocular pain or discomfort. He had ERM in both eyes, thicker in the left eye (OS), which had been monitored annually and deemed stable by various providers since a baseline visit 20 years ago **(Table 1)**.

Table 1. Click to enlarge

The patient was previously symptomatic for binocular diplopia due to an alternating intermittent exotropia. The diplopia was relieved with 5 diopters of base-in prism. He had undergone cataract surgery in both eyes (OU) 4 years prior to this visit. He had no ocular medications. His medical history was significant for hypertension, benign prostatic hyperplasia, myelodysplastic syndrome, adrenal insufficiency, chronic inflammatory demyelinating polyradiculoneuropathy-monoclonal gammopathy of uncertain significance and gastroesophageal reflux disease. None of these conditions was contributory to his ocular history. His medications were consistent for these conditions. Most notable among them were darbepoetin, hydrocortisone and intravenous immune globulin. He had no significant family ocular or medical history.

Since his routine cataract surgery, the patient's visual acuity (VA) in the right eye (OD) fluctuated between 20/20 and 20/25. Meanwhile, VA OS had decreased by approximately a line per year, from 20/25 to 20/40. By this visit, his VAs were 20/25 OD and 20/60 OS. The patient reported metamorphopsia that was more prominent OS than OD and denied reports of a scotoma. His pupils were both equal, round and reactive to light in absence of an afferent pupillary defect. His extraocular motility was unrestricted in all gazes. He had full confrontation visual fields OU. The anterior segment exam was unremarkable OU. The dilated fundus exam was significant for PVD OU and a glistening opaque membrane over the macula with retinal striations radiating toward the temporal arcades and optic nerve OS > OD.

OCT macula scan revealed a thick hyper-reflective band with surface retinal wrinkling, confirming the presence of tractional ERM OU. OCT macula scan OD was stable in central macular thickness and appearance compared to previous scans. Of note with the OCT macula scan OS, an area of focal VMT appeared along the inferotemporal arcade. The nasal portion of the vitreoretinal traction appeared to break off just superior to the area of traction **(Figure 1)**. A broad area of vitreoretinal traction temporal to the macula was pulling the temporal portion of the ERM. This portion of the ERM appeared to break off, along with the vitreoretinal traction, just inferior to the superotemporal arcade **(Figure 2)**. In addition, central macular thickness had increased, and an area of retinoschisis arose temporal to the optic nerve, along with a new drusenoid pigment epithelial detachment (PED) temporal to the fovea **(Figure 3)**. OCT angiography ruled out signs of choroidal neovascular membrane. These unexpected findings prompted a review of all previous OCT macula scans OS, which revealed that the areas of vitreoretinal traction had been present for the past 10 years and the breakage points were first observed 5 years ago. A significant increase in central macular thickness had begun 3 years prior to this visit, while the

development of retinoschisis temporal to the optic nerve appeared 1 year prior to this visit.

At this visit, the patient was diagnosed with a tractional ERM complicated by broad VMT and anomalous PVD OS. After consulting with a retina specialist about the retinal changes, observation was recommended. The patient missed his 1-month follow-up but presented 6 months later. At this visit, his VAs were stable and retinoschisis had decreased in size, but central macular thickness and the size of the PED continued to increase. At this point, vitrectomy and membrane peel was recommended. Within a month after surgery, retinoschisis had completely resolved **(Figure 4)**. At a year after surgery, his VA improved by two lines (from 20/60 to 20/40) and he noticed less metamorphopsia. The central macular thickness returned to baseline, but the PED remained stable **(Figure 5)**.

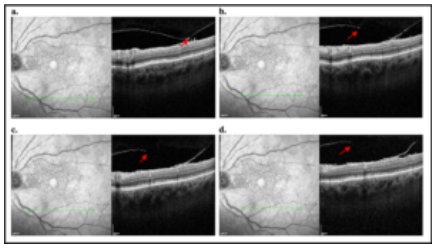

Figure 1. OCT macula scan OS in 2020 that prompted review of previous OCT scans. (a) An area of focal vitreoretinal traction (arrow) along the inferotemporal arcade. (b-d) The nasal portion of the vitreoretinal traction appears to break off (arrow) just superior to the area of traction. Click to enlarge

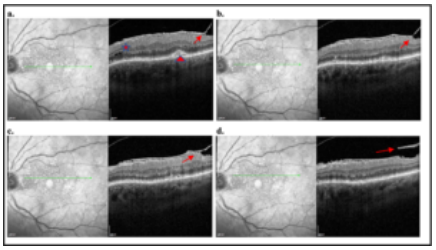

Figure 2. OCT macula scan OS in 2020 that prompted review of previous OCT scans. (a-c) A broad area of vitreoretinal traction (arrow) along the temporal portion of the epiretinal membrane. (d) This temporal portion, along with the vitreoretinal traction, appears to break off (arrow) just inferior to the superotemporal arcade. (a) Presence of retinoschisis temporal to the optic nerve (star) and a large drusenoid pigment epithelial detachment (triangle) temporal to the fovea. Click to enlarge

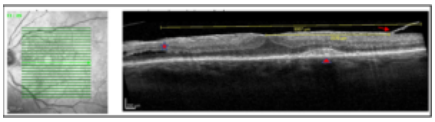

Figure 3. OCT macula scan OS in 2020 that prompted review of previous OCT scans. Blunted foveal contour with the epiretinal membrane across its surface and vitreomacular traction (arrow) at its temporal portion. This scan provides more detail of the retinoschisis (star) temporal to the optic nerve and the drusenoid pigment epithelial detachment (triangle) temporal to the fovea. The area of vitreoretinal traction is within the 3-mm radius of the fovea and measures 5,007 ?m. Click to enlarge

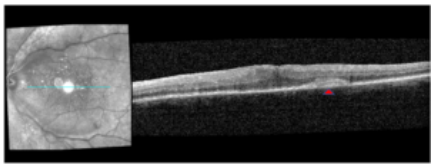

Figure 4. OCT macula scan OS 1 month after vitrectomy shows resolution of vitreoretinal traction and retinoschisis. The drusenoid pigment epithelial detachment (triangle) temporal to the fovea was still present. Click to enlarge

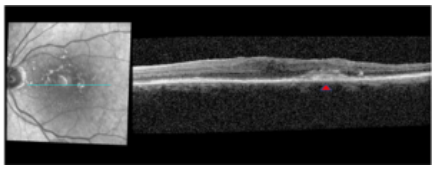

Figure 5. OCT macula scan OS 1 year after vitrectomy shows continued resolution of vitreoretinal traction and retinoschisis. The drusenoid pigment epithelial detachment (triangle) temporal to the fovea was still present. Click to enlarge

Education Guidelines

In a didactic setting, students and residents can be presented with this case and OCT images in slideshow format to develop their own primary diagnosis, differential diagnosis and treatment and management plan. Then, the discussion questions can be used to explore a deeper understanding of the vitreous, VMDs and treatment and management considerations. The learning objectives can be assessed by presenting OCT scans of ERMs and VMT at different stages and having the students and residents classify the conditions and develop a management plan based on a hypothetical patient's symptoms, pertinent clinical findings and OCT scans.

Learning objectives

1. Understand the interaction between the vitreous and retina
2. Know the natural PVD process
3. Recognize an anomalous PVD and its resultant VMDs
4. Classify ERMs and VMT syndrome
5. Increase awareness about when to refer patients with ERM/VMT for surgical options

Key concepts

1. The complications that arise from an abnormal interaction between the vitreous and retina
2. The exacerbating inter-relationships between VMDs
3. The significance of OCT in identifying the disease course and aiding management of VMDs
4. The signs and symptoms that may identify a patient with ERM and/or VMT syndrome as a candidate for surgery

Discussion questions

1. What are the components of the vitreoretinal interface?
2. What are the processes of synchysis and syneresis?
3. Which disorders are considered part of the VMD spectrum?
4. How do you classify an anomalous PVD, ERM and VMT?
5. What is the difference between vitreomacular adhesion (VMA) and VMT?
6. What proposed role does anomalous PVD play in ERM and VMT pathogenesis?
7. How does the presence of an ERM exacerbate VMT?
8. When would you observe a patient with an ERM or VMT?
9. When would you refer a patient with an ERM or VMT for surgery?

Literature Review

The vitreous and vitreoretinal interface

The vitreous body is a highly hydrated extracellular matrix within the posterior chamber of the eye.[2] With a high water content of 98-99%, its transparency allows for uninterrupted transmission and refraction of light onto the retina. The interaction between its two principal macromolecules, hyaluronic acid and collagen fibrils, produces its recognized viscoelastic property. The rope-like collagen fibrils are composed of three different collagen types: a core of collagen Type II surrounded by collagen Type IX and a hybrid of types V/XI.[3,4] The chondroitin sulfate glycosaminoglycan chains of collagen Type IX act like rungs of a ladder to bridge the collagen fibrils together and form an extended structure.[3,4] The space between the collagen fibrils is filled and maintained by a network of hydrophilic hyaluronic acid. As it attracts water, it swells and “inflates” the collagen scaffold,[3,5,6] producing the vitreous’ gelatinous consistency. Interestingly, when the collagen fibrils are separated by at least one wavelength of incident light, light-scattering is minimized, which further contributes to the vitreous’ transparency and ability to transmit light.[4,7]

An outer cortex composed of tightly packed collagen fibrils surrounds the vitreous body and is 100-300 µm thick.[3,5,7] The posterior vitreous cortex is a lamellar structure that contains a single layer of mononuclear phagocytes, called hyalocytes, approximately 50 µm from the inner limiting membrane (ILM).[7,8] Studies have shown that hyalocytes are important in stimulating cell proliferation, inducing tangential vitreoretinal contraction, and stimulating collagen gel contraction in response to platelet-derived growth factor and other cytokines.[8] The posterior vitreous cortex runs parallel to the retinal surface and superficially inserts into the ILM, creating the vitreoretinal interface.[7] Adhesion molecules

such as fibronectin, laminin and heparan sulfate keep these collagen fibers attached to the retinal surface.[7,9,10]

The natural posterior vitreous detachment process

PVD is defined as the complete separation of the posterior vitreous cortex from the retina in all areas posterior to the vitreous base.[10] A natural, innocuous PVD results when two processes work in tandem: vitreous liquefaction (synchysis) and vitreoretinal dehiscence with collapse (syneresis). Synchysis is characterized by the dissolution of the hyaluronic acid-collagen complex. Over time, there is a loss of collagen Type IX and its chondroitin sulfate chains from the collagen fibrils' surface, exposing the core of "sticky" collagen Type II.[3] Without chondroitin sulfate chains to space the fibrils apart, the now "sticky" fibrils coalesce and displace the hyaluronic acid that once filled this space. The hyaluronic acid pools water into areas adjacent to the coalesced fibrils, creating pockets of liquified vitreous called lacunae. The vitreous is approximately 12.5% liquid by age 18 and increases to 50-62% liquid by age 80-90.[3,10-12]

Concurrent with vitreous liquefaction is vitreoretinal adhesion weakening.[10] The posterior vitreous cortex has strong attachments (from strong to strongest) at the major blood vessels, macula, optic disc and vitreous base. So, the natural detachment course of the posterior vitreous cortex from the retina starts at the major blood vessels, followed by the macula, and finally the optic disc. At the completion of detachment, the posterior vitreous cortex remains attached only to the vitreous base. Though it is unknown what changes occur at the interface to weaken vitreoretinal adhesion, it is hypothesized that the collagen fibrils from the posterior cortex that insert into the ILM slowly degenerate with advanced age.

The liquified vitreous travels through fissures and holes of the posterior cortex and into the retrocortical space.[1,13] As the volume of liquified vitreous in the retrocortical space increases, it displaces the gelatinous vitreous body from its weakened attachment at the ILM, resulting in syneresis. Evidence from autopsy studies has indicated a 51% prevalence of PVD in the seventh decade, which further increases to 63% in the eighth decade.[2,10] It has been suggested that PVD evolution may occur faster in women older than 60 due to the effect of declining estrogen levels on vitreous collagen in post-menopausal women.[1]

Anomalous posterior vitreous detachment and vitreomacular disorders

If the liquified vitreous in the retrocortical space displaces the gelatinous vitreous body before vitreoretinal adhesion has sufficiently weakened, a partial PVD with focal and persistent vitreoretinal attachment(s) emerges as a condition termed anomalous PVD.[4] The sequelae of anomalous PVD depend on the position of the strongest vitreoretinal attachment and greatest liquefaction of the gel.[7] When this persistent traction occurs at the macula, VMDs transpire, which include vitreoschisis, ERM, VMT, MH, lamellar hole and myopic foveoschisis.[1] These disorders are characterized by a distortion of macular architecture, such as intraretinal pseudocyst formation, intraretinal schisis, elevation of fovea from retinal pigment epithelium (RPE) and foveal detachment.[1] They are asymptomatic until the traction is strong enough to distort the outer segments, elevate the fovea from the photoreceptors and/or disturb the spatial arrangement of cones,[7,14] manifesting into symptoms of reduced central vision, metamorphopsia, micropsia and/or macropsia.[1]

Anomalous PVD can be classified as either full-thickness or partial-thickness.[10,15] A full-thickness anomalous PVD occurs when the entire posterior vitreous cortex remains attached to the retina.[10,15] A partial-thickness anomalous PVD, termed vitreoschisis, occurs when the posterior vitreous cortex lamellae split; the outermost layer of the split remains adherent to the retina while the innermost layer collapses forward with the vitreous body.[7-10,15] Residual vitreous tissue has been found to be left on the inner retinal surface in nearly 50% of PVDs.[14,16]

Epidemiology and risk factors

Since the introduction of OCT in 1993, the ability to visualize and evaluate the vitreoretinal interface has deepened the understanding of its associated disorders. VMDs are underdiagnosed due to their typically asymptomatic nature and subtle clinical appearance. In a study of participants older than 60 with maculae deemed normal by color fundus photography or biomicroscopy, 8.4% of eyes were found to have a VMD by OCT.[17] The most common abnormality found was ERM (61 out of 984 eyes).[17]

Epiretinal membrane

The prevalence of ERM varies across studies, which can be attributed to differences in methods and protocols for grading and how to define ERM.[18] The prevalence of ERM appears to vary by race, from 2.2% in a Beijing study in rural China up to 28.9% among Latinos in a multi-ethnic study conducted in the United States.[19] However, the role of race and ethnicity remains unclear because the variations are inconsistent across studies. For example, in the United States, Multi-Ethnic Study of Atherosclerosis (MESA) data suggest that the prevalence of any ERM was highest in persons of Chinese ancestry (39.0%), whereas the data from China suggest ERM prevalence rates were much lower (2.2% – 3.4%).[20]

The original Beaver Dam Eye Study used nonstereoscopic color fundus photographs and found ERM in 11.8% of the population in at least one eye and in 2.4% of the population in both eyes.[21] In the 20-year follow-up study using OCT, a higher prevalence was documented: 34.1% in at least one eye and 30.3% in both eyes.[22] The reported prevalence of early asymptomatic ERM, termed cellophane maculopathy, has varied from 1.8% and 2.2% in China to as high as 25.1% in MESA.[20] The prevalence of the more severe form of ERM, commonly termed macular pucker, has been more consistent across studies with rates ranging from 0.7% in rural China to 3.5% among Asian Indians, 3.8% in MESA, and 3.9% in Melbourne, Australia.[20]

Studies have consistently identified age and PVD as risk factors for ERM. It is estimated that the prevalence of macular ERM is 2% in patients younger than 60 years, 12% in patients age 70 or more, and 22.5% in those age 80 or more.[7,23-25] In the Beaver Dam Eye Study, the prevalence of ERM increased with age from 28.1% in those age 63-74 years to 53.2% in those age 85 years or more.[22] PVD has been found to be present in 70% of patients in the earlier stages of ERM[15,23] and in up to 90% of patient with advanced ERM.[26] Other retinal pathologies that have been identified as risk factors for ERM are uveitis, retinal breaks, retinal vein occlusion, proliferative diabetic retinopathy and ocular inflammatory disease.[20] Cataract surgery has also been identified as a risk factor.[15,21]

Our patient's retinal changes and subsequent decreased vision developed approximately a year after cataract surgery, which points to the procedure as the agent for his ERM progression. Even though he had cataract surgery in each eye 1 month apart, the ERM progressed in only the left eye, which was most likely due to the multiple areas of anteroposterior vitreoretinal traction in that eye when none existed in the right eye.

Vitreomacular traction syndrome

Few studies have specifically addressed the epidemiology of idiopathic VMT as it tends to overlap with other retinal diseases, especially MH.[5,27] The prevalence of isolated idiopathic VMT has been estimated as 22.5 cases per 100,000 of the general population, with an incidence of 0.6/100,000 persons per year.[5,27] In a multi-center prospective study involving 1,950 eyes, VMA and VMT were detected in 38.77% and 1.07% of eyes, respectively.[28] The Beaver Dam Eye Study reported a prevalence of 26% and 1.6%, respectively, for VMA and VMT.[22,28]

As it is for ERM, age is the strongest risk factor for VMT. In the Beaver Dam Eye Study, the prevalence

of VMT increased from 1% in those age 63-74 years to 5.5% in those age 85 years or more.[22]

Classification, clinical presentation and symptoms

Epiretinal membrane

Unlike other VMDs, ERM does not have an OCT-based classification that has reached widespread consensus or mainstream use.[1] In general, ERM can be classified based on etiology [primary (idiopathic) or secondary] or histology [simple or complex (tractional or contractile)].

Primary or idiopathic ERMs are associated with vitreoretinal traction. Secondary ERMs are associated with retinal pathologies such as retinal breaks, tears or detachments, intraocular inflammation, trauma, retinal vascular diseases and retinal surgery. Interestingly, these pathologies are notable risk factors for ERM development.[7,20] Idiopathic ERM represents approximately 60% of patients with ERM.[24]

Simple ERM grows directly on the ILM and is composed of a monolayer of retinal cells.[7] It appears as a thin, glistening membrane on the surface of the retina, termed cellophane maculopathy. It is noncontractile with mild to no vision symptoms.[7,27] Complex or tractional ERM is a multi-layer of cells (fibrous astrocytes, myofibroblasts, hyalocytes, macrophages, RPE and glial cells) that is separated from the ILM by a layer of vitreal collagen Type II.[7] It appears as a thick, opaque membrane that has been proposed to exert a progressive, tangential, inward (centripetal) traction that can pull the underlying retina inward toward the ERM center resulting in macular pucker.[7,29] In some cases, this centripetal contraction can lead to the biomicroscopic slit lamp appearance of a hole, historically termed pseudohole. Unlike a true MH, a pseudohole has an intact photoreceptor layer.[1] If the traction becomes excessive, macular pucker will cause retinal changes, such as irregular wrinkling, nerve fiber layer dragging, ectopic fovea, winding corkscrew vessels surrounding the overlying ERM, or major vessel straightening and crowding.[7,29] It may even cause vitreoretinal traction and/or tractional retinal detachment.[23]

In early stages, ERM is often asymptomatic and detectable only on OCT scans.[1] Among patients with idiopathic ERM, two-thirds exhibit VA of 20/30 or better, while 85% display VA of 20/70 or better.[26] ERM becomes symptomatic when the traction involves the macula or perimacular regions.[7,23,24]

Symptoms of ERM include reduced VA, metamorphopsia, micropsia, macropsia, aniseikonia and/or dragged-fovea diplopia.[7,23,24,30] In cases where ERM is more severe in one eye, the difference in metamorphopsia, micropsia or macropsia between the eyes can lead to binocular image rivalry.[1] A patient who has reduced binocular visual quality may close one eye, even in the absence of diplopia or strabismus.[30] With our patient's history of horizontal diplopia, it was important to perform a full binocular workup to determine proper treatment. The binocular workup for our patient revealed an alternating intermittent exotropia. Therefore, prism correction was the appropriate treatment to relieve his diplopia. If the binocular workup had revealed no strabismus, his symptom of diplopia would have been most likely due to binocular rivalry. If this were the case, a surgical referral for vitreoretinal traction relief would have been most appropriate.

Vitreomacular traction

In 2013, the International Vitreomacular Traction Study (IVTS) proposed OCT-based definitions for VMA, VMT, full-thickness MH, lamellar hole and macular pseudohole, along with an OCT-based classification system for VMA, VMT and MH.[14] Advantages include clinical applicability for the assessment of vitreous state and reproducibility for comparative analysis in clinical studies.[1,5] Most notably, the classification system provides evidenced-based quantification of the pathology that correlates to treatment outcomes for MH.[1,5]

VMA is defined as a perifoveal vitreous detachment with macular attachment within a 3-mm radius of the fovea and without associated retinal deformation.[1,14,31] VMA is considered a normal stage in early PVD development. Patients with VMA are usually asymptomatic and detected incidentally.[1] In contrast, VMT is defined as perifoveal vitreous detachment with macular attachment within a 3-mm radius of the fovea that does exhibit retinal deformation.[1,14,31]

Based on the IVTS classification system, VMT is subclassified as focal (≤ 1,500 µm) or broad (> 1,500 µm). This distinction has clinical relevance because the diameter of VMA is inversely related to macular morbidity and foveal deformation.[32] The narrower the area and the higher the angle of adhesion, the greater amount of tractional force exerted and the greater the foveal deformation.[7,33] Focal VMT is associated with distorted foveal surface, foveal pseudocysts and foveal elevation, and it may lead to MH development.[1,14] Foveal pseudocysts are associated with diminished VA and vision distortion, such as metamorphopsia.[14] After the release of traction, pseudocysts generally resolve over time with little remaining vision deficit.[14] A broader area of VMA may distribute the tractional force beyond the border of the foveal region, leading to generalized thickening of the macula, intraretinal schisis and associated ERM.[1,14,32]

Anteroposterior traction from VMT normally creates a V-shape, with its “point” at the macula and traction on both sides. However, in this case, there was a single area of anteroposterior traction approximately 2,370 µm temporal to the fovea creating a J-shape (Figure 3). This temporal band continued superiorly before breaking off just before the superotemporal arcade with the posterior cortex as a continuous, yet separate layer above the retina. There was another focal area of traction along the inferotemporal arcade, where the nasal portion broke off just superior to this area, leaving a sole area of temporal traction. Because this area of persistent vitreoretinal traction occured beyond a 3-mm radius of the fovea, this was considered areas of anomalous PVD instead of VMT.

Discussion

The inter-relationship of vitreomacular disorders

While each VMD has its own pathogenesis and disease course, they are often inter-related with anomalous PVD at their incipience.

Vitreoschisis and epiretinal membrane

The effect that vitreoschisis (partial-thickness anomalous PVD) has on the retina depends on the location of the vitreous cortical split in relation to the hyalocyte layer. If the split occurs posterior to the level of hyalocytes, the hyalocytes, as part of the anterior split vitreous cortex, will detach from the retina, leaving behind a relatively thin hypocellular layer of vitreous cortex attached to the macula.[7,8] If this membrane is also attached at the optic disc, it may cause an outward (centrifugal) tangential contraction, inducing a MH or schitic changes as seen in lamellar holes.[1,7,8] Studies conducted at the VMR Institute using OCT combined with scanning laser ophthalmoscopy (SLO) have identified vitreoschisis in 53% of patients with MH and in 43% of patients with macular pucker.[35]

While the exact mechanism of ERM development is still poorly understood, two observations have emerged. First, PVD development is one of its most significant risk factors.[15,23] A PVD at any stage has been found in 80-90% of eyes with idiopathic ERM.[7] Second, ERM is characterized as a growth of fibrocelluar tissue on the ILM.[36] The type of PVD that has been proposed to induce fibrocellular proliferation is a vitreoschisis with a split anterior to the level of hyalocytes, which leaves the hyalocytes as part the vitreous cortex layer that remains attached to macula.[7,8,10,16] Gandorfer et al. have shown histologically that a thin layer of cortical vitreous is often sandwiched between the fibrocellular ERM and the ILM, confirming the role of residual vitreous material in ERM formation.[37,42] As mononuclear

phagocytes of the reticulo-endothelial cell system, hyalocytes can stimulate the migration of monocytes from the circulation and glial cells from the retina.[8,16] The proliferation of these cells onto the surface of the retina creates a relatively thick, hypercellular and contractile scaffold that allows uptake of other cells into the membrane.[1,7] The progression of ERM from cellophane maculopathy to macular pucker is considered a fibrotic process that is sustained by collagen deposition and transdifferentiation of retinal Müller cells, RPE cells and hyalocytes into myofibroblasts.[15,38] In short, hyalocytes lying on the macular surface proliferate, recruit and stimulate glial cells to proliferate upon an intact ILM. This forms an ERM, a scaffold that allows the uptake of other cells into the membrane and induces contraction.[7,39]

Full-thickness anomalous posterior vitreous detachment and vitreomacular traction

While vitreoschisis (partial-thickness anomalous PVD) can lead to ERM, a full-thickness anomalous PVD can exert enough persistent anteroposterior traction to produce VMT.[35] The deformation may be evident by changes in foveal contour, distortion of the macular architecture, intraretinal pseudocyst formation, intraretinal schisis or even elevation of the fovea from the RPE, without full-thickness defect of all the retinal layers.[1,14] When VMT creates outer retinal changes, patients are often symptomatic for reduced vision or metamorphopsia.[1,40] Progressive and excessive traction at the fovea can result in complete interruption of all neural layers, leading to a MH.[1]

Epiretinal membrane and vitreomacular traction

Since the proposed pathogenesis of both ERM and VMT involves anomalous PVD, it is of little surprise that they often co-exist. In a study of 60 eyes with ERM, a partial PVD with VMA occurred in 57% of the eyes.[41] However, this study was conducted in 1999, before the IVTS Group proposed OCT-based definitions that differentiated VMA (normal PVD stage) from VMT (abnormal consequence). In studies that utilized IVTS Group classification and definitions to study the natural history of VMT, ERM was found in 20-37.5% of eyes with VMT.[34,37]

Several studies have demonstrated that in eyes with VMT, ERM proliferates onto both the retinal surface and onto the back surface of the detached posterior hyaloid membrane.[42-44] This reinforces the VMA, preventing its separation and prolonging the tractional stress on the macula.[37] The combined forces of tangential contracture of the ERM and anteroposterior traction of the vitreoretinal attachment magnify any tractional stress on the underlying foveal structure.[14] This sustained vitreoretinal traction limits treatment options for patients with combined ERM and VMT.

Treatment and management

The goal for VMD management is to optimize vision and quality of life by preventing vision loss and minimizing metamorphopsia and/or diplopia.[20] As a spectrum, VMDs are either observed or undergo pars plana vitrectomy. A patient with VMT has the additional options of pharmacologic or pneumatic vitreolysis.

As previously noted, the presence of ERM reinforces the adhesion between VMT and the retina, contributing to the low success rate of pharmacological vitreolysis in these patients.[20,45,46] In the MIVI-TRUST trial, among the subjects receiving ocriplasmin, 8.7% and 37.4% had resolution of “symptomatic VMA” with and without ERM, respectively.[29] Among patients in the placebo group, 1.5% and 14.3% had resolution of “symptomatic VMA” with and without ERM, respectively.[29] Given the intensifying adhesion between the ERM, VMT and anomalous PVD, our patient would have been considered a poor candidate for vitreolysis. For this reason, this teaching case report focuses on whether a patient with ERM and/or VMT should be observed or referred for vitrectomy.

Observation

The current standard of care for early stage, asymptomatic ERM and VMT is observation. The risk to this conservative approach includes MH formation and further vision loss.[46] While spontaneous separation of ERM is uncommon,[7] most ERMs tend to remain stable without need for surgical intervention. Using fundus photography, the Blue Mountains Eye Study found that of patients with ERM, 39% were stable, 26% regressed and 29% progressed over a 5-year period. In a retrospective case series exploring the natural history of idiopathic ERM in 145 eyes over a 10-year period, 14.5% underwent surgical intervention with a mean time to surgery of 6.5 years.[47]

While VMT holds a higher risk for MH formation than ERM, observation is considered a viable option for VMT. Before the commercial use of OCT, the spontaneous resolution of VMT was considered uncommon. Using biomicroscopy to study the natural history of VMT over a median follow-up of 5 years, Hichiki et al. found that the spontaneous resolution of VMT occurred in 11% of eyes at a median duration of 15 months. However, in more recent studies using OCT, the incidence of spontaneous resolution has been 20-43% at a median duration of at least 9 months.[31,34,46] Dimopolous et al. studied the natural history of 46 eyes with VMT less than 1,500 µm and without ERM to match the baseline characteristics of an ideal candidate for ocriplasmin. They found that 43.5% of these eyes developed spontaneous resolution of the VMT with a mean duration of 375 days.[46] In a retrospective cohort study of 183 eyes with VMT over an average of 17 months, 60% were stable and 20% resolved with a mean time to resolution of 15 months.[34] In the same study, 12% developed MH and 8% elected to proceed with surgery for worsening symptoms, on average, by 19 and 13 months, respectively.[34]

At this time, no factors have been found to reliably predict which patients will experience spontaneous resolution of VMT and which patients will develop a MH.[1] A few studies have suggested that the following factors may be predictive of spontaneous VMT release:[48] adhesion diameter less than 400 µm,[31] a wide angle (approaching 90°) between the vitreous surface,[31] isolated inner retinal layer distortion,[49] treatment of concurrent retinal diseases with intravitreal injections[49] and VMA surface area less than 101,002 µm as calculated by OCT.[50]

General recommendations during the observation period

OCT has been key in identifying the disease course and guiding management of VMD. For cases that require finer detail, such as to differentiate ERM from either a vitreoschisis or a shallow PVD, B-scan ultrasonography and OCT/SLO imaging can be used.[13,14,16] While advancement in imaging technology has improved the quality of diagnosis, utility for identifying which patients will regress, remain stable or progress is still lacking. A standard of care for asymptomatic patients with ERM and VMT is observation for 3 months before initiating any treatment to avoid unnecessary surgery.[5,27,31] Shorter follow-up intervals with detailed OCT macula scans are recommended for patients with focal VMT or history of MH in the fellow eye.[1] In between follow-up visits, patients should be educated on the signs and symptoms of progression and advised to perform periodic at-home monocular Amsler grid testing.

Vitrectomy

Vitrectomy with membrane peel is the mainstay treatment for patients with symptomatic ERM and VMT.[1] Vitrectomy involves removing the vitreous body and mechanically separating the vitreous from the optic disc by inducing a PVD and releasing the traction of the vitreous from its attachment at the fovea.[1] This facilitates a restoration of the central and outer retinal architecture, resulting in improved neural transmission.[27] Additional steps include removal of any concurrent ERM and/or peeling the ILM.[1] Many studies have suggested that ILM peeling is significant for preventing ERM recurrence.[7,39,51] Common complications of vitrectomy are accelerated cataract progression, ERM development and retinal detachment.[45,46] Rare adverse events include infection, hemorrhage and MH.[45]

Indications for vitrectomy

Determining when to refer our patient for vitrectomy was the most ambiguous aspect of this case for two reasons. One, there are no preoperative characteristics that are reliably predictive for patients who would benefit from surgery. Two, it is unclear at what point the damage from either ERM or VMT onto the macula is irreversible. The lack of evidence-based guidelines creates uncertainty about when a referral is considered too early. Do we refer as soon as possible to minimize vision loss, or will surgery be deemed unnecessary? With no consensus on when or which patients with ERM or VMT would benefit from a vitrectomy, most surgical referrals are dictated by progressing symptoms.

Vitrectomy is often indicated for patients who experience a decrease in VA, metamorphopsia, double vision or difficulty using their eyes together.[1,20,24] A retrospective study reported that 21% of patients required surgery at 4 years if baseline VA was ≥ 20/40.[47] However, there is variance on the level of vision impairment required for intervention, and some patients may be very symptomatic with metamorphopsia despite good Snellen VA.[1,52] In such cases, the goal of surgery is to reduce metamorphopsia and improve binocularity, thereby improving quality of life.[52]

Poor prognostic factors for ERM are inner nuclear layer cysts and an associate lamellar hole at baseline.[1] Metamorphopsia is another poor prognostic factor as it is likely the result of rearranged photoreceptors.[53] Luu et al. found that ERM with greater central macular thickness and disruption of outer retinal layers were more likely to undergo surgery.[47] Kakehashi et al. identified ERMs with partial PVD without shrinkage and with VMA as having the worst visual prognosis compared to other types of ERMs, possibly due to the chronic weak vitreous traction exerted on the macula. The presence of ERM and broad-based VMA are associated with persistent VMT. As these are less likely to spontaneously resolve, vitrectomy would be the remaining option to relieve traction.[5,27] Ultimately, our patient's decreasing vision, increased macular thickness and outer retinal changes were indicators for vitrectomy.

Possible prognostic factors for favorable surgical outcome

While there are no reliable prognostic factors for a favorable surgical outcome, most surgeons define success in terms of postoperative VA. Prognostic factors associated with higher postoperative VA are higher preoperative VA, younger age, lower central foveal thickness, longer photoreceptor outer segment length [measured from the ellipsoid zone (EZ) to the RPE], intact EZ and duration of symptoms ? 6 months.[1,7,24,26,54]

Studied postoperative findings

Hartman et al. reported a restoration of the normal anatomy, on average, 4 months after vitrectomy.[38] It often takes 4-6 weeks for a patient's vision to return to preoperative level, and subsequent improvement continues over the following 3-6 months.[26] It has been reported that 60-90% of patients have VA improvement of two or more lines by 6-12 months after surgery.[26] Most metamorphopsia and, on average, scores on the National Eye Institute Visual Function Questionnaire – 25 improve postoperatively.[20] Thus, even in the absence of VA gain, patients report improved quality of life with relief from some or all metamorphopsia.[20]

For ERM specifically, 70-80% demonstrate improvement in VA of more than two lines following vitrectomy, which continues to improve up to 3 years thereafter.[1,55] Idiopathic ERM recurs in approximately 10% of cases, and re-operation is required in approximately 3% of cases.[7] In a recent study, recurrent ERM that required repeat vitrectomy occurred in 5.5% of cases, and all recurrences were at least 5 years after the initial surgery.[55]

Clinical Pearls

- OCT is more accurate than either fundus photography or biomicroscopy for detecting VMD

- VMT is defined by persistent vitreous anteroposterior traction within a 3-mm radius from the fovea
- Anomalous PVD is when persistent vitreous anteroposterior traction occurs outside a 3-mm radius from the fovea
- Persistent anteroposterior and/or tangential traction becomes symptomatic when it affects the macula and perimacular areas
- The effect that vitreoschisis has on the retina depends on the location of the vitreous cortical split in relation to the hyalocyte layer
- ERM reinforces the adhesion between VMT and macula, preventing vitreomacular separation and prolonging tractional stress on the macula
- A binocular workup is important to determine whether binocular diplopia is due to strabismus or binocular rivalry from metamorphopsia, micropsia or macropsia
- As a risk factor for ERM, one should consider a patient's vitreoretinal status before cataract surgery
- The current standard of care for early stage, asymptomatic ERM and VMT is observation for 3 months. Shorter follow-up intervals with detailed OCT macula scans are recommended for patients with focal VMT or history of MH in the fellow eye
- While there is no consensus on when to refer a patient for vitrectomy, surgery is often indicated for patients who experience a decrease in VA, metamorphopsia, double vision or difficulty using their eyes together
- On average, restoration of normal retinal anatomy is observed at 4 months after vitrectomy, and 60-90% of patients show VA improvement of two or more lines by 6-12 months after surgery

Conclusion

The majority of ERMs remain relatively stable. However, when an ERM co-exists with another VMD, any existing vitreoretinal traction is reinforced and the retinal architecture is further disrupted. Our patient's previous providers assumed his ERM would remain stable given its 20-year-plus history, his age and lack of associated symptoms. Under this impression, even when his VA began to decline and central macular thickness increased on OCT after cataract surgery, the changes were considered minimal and his management remained the same. Even though he gained two lines of VA (from 20/60 to 20/40) after vitrectomy, his acuity did not return to his baseline of 20/25. Earlier identification of the vitreoretinal traction, especially when vision began to decrease, would have prompted closer monitoring. Understanding the dynamic role of vitreoretinal traction with ERM could have led to earlier intervention and a better vision prognosis for our patient.

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