Taiwan Journal of Ophthalmology 4 (2014) 63–68

Contents lists available at ScienceDirect

Taiwan Journal of Ophthalmology

journal homepage: www.e-tjo.com

Review article

Vitreomacular interface diseases: Diagnosis and management

Ashleigh L. Levison, Peter K. Kaiser*

Cole Eye Institute, Cleveland Clinic Foundation, Cleveland, OH, USA

ARTICLE INFO

Article history:
Received 25 November 2013
Accepted 4 December 2013
Available online 13 January 2014

Keywords:
epiretinal membrane
lamellar hole
macular hole
pseudohole
vitreomacular adhesion
vitreomacular interface disorders
vitreomacular traction

ABSTRACT

This article discusses the diagnosis and management of abnormal vitreomacular interfaces disorders including vitreomacular adhesion, vitreomacular traction, epiretinal membrane, full thickness macular holes, lamellar holes and pseudoholes. Optical coherence tomography has better enabled our ability to diagnose abnormalities of the vitreoretinal interface by providing clinical information that cannot be obtained by other ophthalmic diagnostic techniques. While vitrectomy remains the most commonly performed treatment for these disorders, the recent introduction of pharmacologic vitreolysis represents the development of non-surgical treatment options of certain diseases of the vitreoretinal interface.

1. Introduction

The vitreous fills the space between the lens and the ciliary body anteriorly and the lens and the retina posteriorly. The vitreous comprises approximately 80% of the volume of the eye. It is composed of approximately 98% water and 2% proteins and an extracellular matrix. Collagen is the major structural protein; type II collagen and type IX collagen are the most common proteins, and make up 75% and 15%, respectively, of the collagen in the vitreous.[1,2] The vitreous also contains hyaluronan, chondroitin sulfate, fibrillins, and opticin.

The strongest points of attachment of the vitreous to the retina are at the optic nerve, macula, ora serrata, and around the blood vessels.[3] The equatorial and posterior vitreoretinal interfaces consist of the posterior vitreous cortex, the internal limiting membrane (ILM), and the intervening extracellular matrix.[1] The ILM is primarily composed of type IV collagen. The posterior vitreous cortex and retinal ILM are bound at their interface by this macromolecular attachment complex, which is composed of fibronectin, laminin, and other extracellular components that form a glue-like matrix. Chondroitin sulfate is present at this interface and has an important role in the strong vitreoretinal interface.[1]

The normal aging process of the vitreous gel causes the development of posterior vitreous detachment (PVD). Liquefaction of the vitreous occurs over time, thereby creating lacunae or pockets in the vitreous.[1] Synchysis (i.e., the process of vitreous gel liquefaction) first begins at approximately the age of 4 years.[2] Vitreoretinal separation normally occurs at many sites throughout the peripheral fundus. This process occurs for years prior to a final separation of the vitreous from the macula and optic nerve occurs and leads to PVD. The early stages are typically asymptomatic.[4] Posterior vitreous detachment normally results in a complete and clean separation between the ILM of the retina and the cortical vitreous.[5]

An anomalous separation of the vitreous cortex from the ILM can lead to an abnormal vitreoretinal interface. This separation can happen when liquefaction occurs faster than the detachment of the vitreous cortex or when an abnormal adhesion of the vitreous cortex to the ILM occurs.[6] Various pathologic vitreomacular interface diseases can develop when there is an anomalous PVD.[7]

Most abnormal vitreomacular interface diseases were historically diagnosed by slit lamp biomicroscopy with or without the addition of fluorescein angiography. Thus, many subtle alterations to the vitreomacular interface were often missed clinically. The introduction of optical coherence tomography (OCT) approximately two decades ago has dramatically altered the ability to diagnose abnormalities of the vitreoretinal interface by providing clinical information that cannot be obtained by other ophthalmic diagnostic techniques. In 2004, the development of spectral domain OCT, which has increased resolution and more rapid scanning

Conflicts of interest: Peter K. Kaiser is a consultant for Alcon, Novartis, Thrombogenics, and Allegro.

* Corresponding author. Cole Eye Institute, Cleveland Clinic Foundation, 9500 Euclid Avenue i-13, Cleveland, OH 44195, USA.
E-mail address: pkkaiser@gmail.com (P.K. Kaiser).

2211-5056/$ – see front matter
http://dx.doi.org/10.1016/j.tjo.2013.12.001

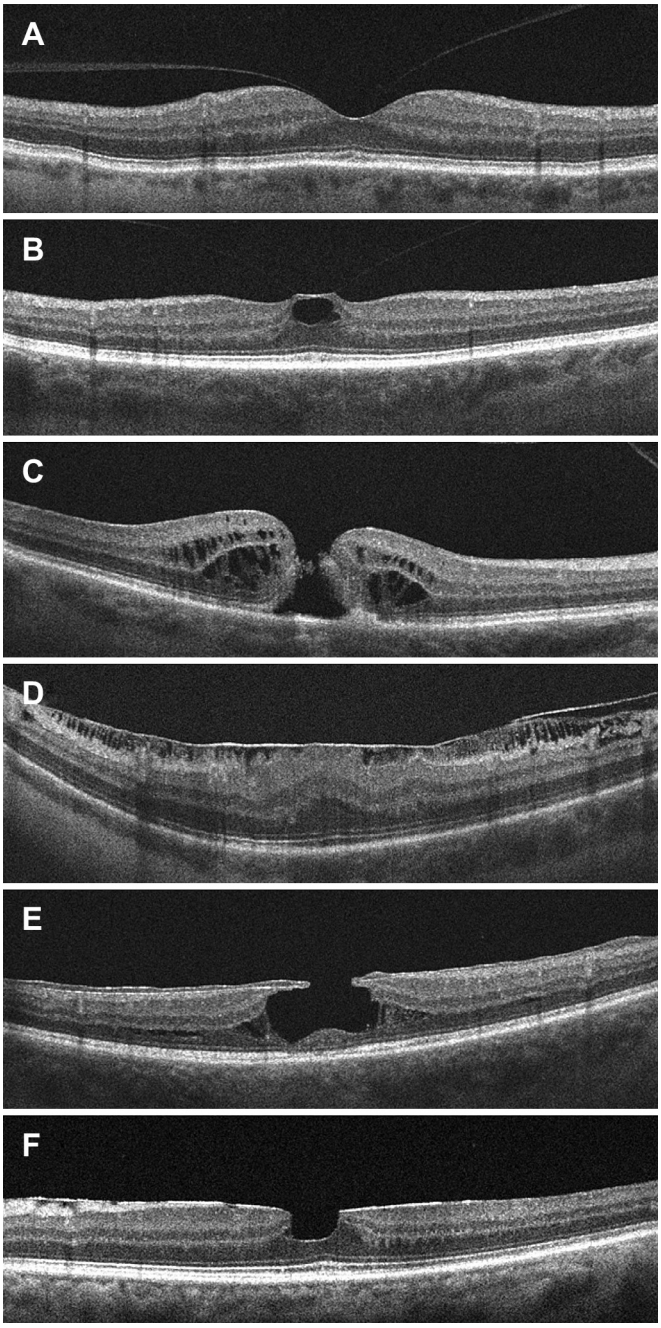

Fig. 1. Disorders of the vitreoretinal interface. (A) Vitreomacular adhesion, which is adhesion of the vitreous at the fovea without distortion of the retinal contour. (B) Vitreomacular traction, which is abnormal vitreous adhesion with excessive traction of the fovea that causes pseudocyst formation. (C) Full-thickness macular hole, which is a full-thickness retinal defect with posterior vitreous detachment. (D) Epiretinal membrane, which has cellular proliferation on the surface of the inner retina that creates traction and loss of the foveal contour. (E) Lamellar hole, which is a partial-thickness foveal defect with an irregular foveal contour and a splitting of the inner and outer retina. (F) Pseudohole, which is an epiretinal membrane with a central opening that causes a steep macular contour at the central fovea.

capabilities, has permitted physicians to visualize and monitor the vitreomacular interface with better consistency and accuracy.[8]

In this manuscript, we will explore the diagnosis and management of abnormal vitreomacular interfaces diseases such as vitreomacular adhesion (VMA), vitreomacular traction (VMT), epiretinal membrane (ERM), full-thickness macular holes (FTMH), and lamellar holes and pseudoholes (Fig. 1).

2. Vitreomacular adhesion

The diagnosis of VMA is applied to patients who have incomplete separation of the posterior vitreous with persistent attachment to the macula. This term has been broadly used to include patients with and without distortion of the retinal architecture; however, the term should only be used for patients who have an intact retinal architecture. In the past, VMA has been classified by symptomatic patients versus asymptomatic patients (i.e., based on a patient's visual complaints). With the introduction of OCT, physicians have become aware that VMA is a more common entity than was previously clinically known and may be part of the normal formation of PVD.[9]

A group of retina specialists, the International Vitreomacular Traction Study (IVTS) Group, recently defined the OCT characteristics of VMA.[5] The IVTS new definition of VMA is more restrictive than the definition previously used. On OCT, VMA is a specific stage of vitreous separation when partial detachment of the vitreous in the perifoveal area has occurred without any abnormalities to the retinal contour. The slit lamp examination is clinically normal. Eyes with VMA are subclassified by the size of the adhesion. An adhesion is either focal (i.e., less than 1500 μm) or broad (i.e., greater than 1500 μm). Focal points of dehiscence between the vitreous and retina can be present within areas of broad VMA.[5] Vitreomacular adhesion is typically asymptomatic and nonpathologic, and does not cause any apparent retinal changes.[10] It is a natural component of the development of a PVD and can therefore be considered an incomplete PVD.[9,11] Vitreomacular adhesion, although asymptomatic, has been hypothesized as playing a role in the pathogenesis of many macular conditions such as neovascular age-related macular degeneration, macular hole, and diabetic macular edema.[9]

Based on the new characterization of VMA defined by the International Vitreomacular Traction Study Group, VMA is a normal stage in the process of PVD, is not associated with symptoms, and causes no change in the retinal architecture. Treatment is not required. These patients should be observed for resolution or for the progression to vitreomacular traction or, possibly, a macular hole.[5]

3. Vitreomacular traction

In certain patients with abnormal vitreous adhesion, there can be excessive traction on the macula from the vitreous that changes the contour of the foveal surface. Findings on slit lamp biomicroscopy may be subtle, but there may be a distortion of the fovea, a blunted foveal reflex, cystic changes, or (in severe cases) subretinal fluid. Optical coherence tomography allows the direct visualization of the vitreoretinal interface; therefore, very subtle distortion of the foveal contour on OCT may be the only feature that distinguishes VMT from focal VMA when the retinal anatomy is otherwise normal.[10] In addition, there may be elevation of the retina at the fovea at the level of the retinal pigment epithelium (RPE). The combination of anatomical changes on OCT with signs of perifoveolar PVD constitutes a diagnosis of VMT.[5] In accordance with the International Vitreomacular Traction Study Group definition, VMT (like VMA) can be classified as "focal" or "broad", based on the horizontal width of the adhesion. These broad areas of attachment with traction can be associated with thickening of the macula, vascular leakage on fluorescein angiography, macular schisis, and cystoid macular edema.[5] The anatomical changes to the fovea induced by VMT can lead to reduced visual acuity,[9] metamorphopsia, and micropsia.[8]

The natural history of patients with VMT is not well established. John et al[9] performed a consecutive case series evaluating the clinical course of what they referred to as "vitreomacular