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Optical Coherence Tomography Pathologic Findings in the Vitreoretinal and Macular Interface

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Abstract

The vitreous body is bounded posteriorly by the retina and is adherent to it. Abnormalities in the adhesion between the vitreous and the macula, or vitreoretinal interface, are involved in the pathogenesis of several macular conditions. Optical coherence tomography is a noninvasive in vivo ophthalmic imaging technique, which allows for a better understanding and improved diagnosis of disease processes that involve the vitreoretinal interface. The aim of this paper is to describe optical coherence tomography findings in vitreomacular disorders, namely epiretinal membrane, idiopathic macular hole, lamellar macular hole, and vitreomacular traction syndrome, in order to assist in the diagnosis and proper treatment of these syndromes.

The vitreous body is bounded posteriorly by the retina and is adherent to it. It is formed by collagen fibrils that insert superficially into the internal limiting membrane (ILM) of the retina [1]. Attachment to the ILM is facilitated by macromolecules such as laminin, fibronectin, chondroitin and heparan sulfate proteoglycans [2]. The vitreous is firmly attached to the lens capsule, retinal vessels, optic nerve, and to the macula. Abnormalities in the adhesion between the vitreous and the macula, or vitreoretinal interface, are involved in the pathogenesis of several macular conditions.

Optical coherence tomography (OCT) is a noninvasive in vivo ophthalmic imaging technique. Technological advancements, including the introduction of high-resolution spectral-domain OCT (SD-OCT) in 2004, have allowed for a better understanding and improved diagnosis of disease processes that involve the vitreoretinal interface. Nowadays, OCT has become a valuable tool for assessment of the vitreoretinal interface.

The aim of this chapter is to describe OCT findings in vitreomacular disorders, namely epiretinal membrane (ERM), idiopathic macular hole (MH), lamellar MH, and vitreomacular traction syndrome, in order to assist in the diagnosis and proper treatment of these syndromes.

Epiretinal Membrane

An ERM is a fibrocellular membrane on the inner retinal surface that is adherent to the ILM of the retina. It is believed to arise as a result of proliferation of glial cells, retinal pigment epithelium, or hyalocytes at the vitreoretinal interface.

ERMs are classified as idiopathic or secondary to a plethora of conditions, including retinal vascular occlusive disease, diabetes mellitus, ocular inflammatory diseases, ocular trauma, ocular surgery and others.

Clinically, ERMs may have various appearances on biomicroscopy. Mild ERMs may appear as a glistening layer on the retinal surface, as an irregular light reflex – the so-called cellophane maculopathy, or as retinal striae, resulting from wrinkling of the ILM. Denser ERMs appear as a gray sheet overlying the retina. When the pathology is more advanced, contraction of the ERM may lead to retinal distortion, vascular traction, macular edema due to increased permeability of contracted vessels, and formation of a pseudo-hole. Moderate-to-severe cases of ERM contracture are also termed macular pucker.

On OCT, ERMs appear as a highly reflective layer on the inner retinal surface. They may appear adherent to the retina (fig. 1Aa) or separated from the inner retina (fig. 1Ab), partially or completely.

Since epiretinal membranes may appear completely detached from the retinal surface, they may be confused with a detached posterior hyaloid on OCT images. One distinction is the reflection caused by the two entities. The posterior hyaloid usually has a thin reflection compared to the denser reflection of an ERM. Another way to differentiate between the two is the degree of separation from the inner retina, which is usually greater for the posterior hyaloid (fig. 1B). When still not certain, one can examine an OCT slice containing the optic nerve head. The posterior hyaloid tends to adhere to it while an ERM does not.

An ERM may distort the foveal contour or lead to its flattening (fig. 1Aa). It may lead to superficial retinal folds, or a saw-tooth pattern (fig. 1Ab, C). Retinal folds should not be mistaken for choroidal folds. The latter will also include folds in the outer retinal layers. Other helpful modalities in this distinction are the infra-red image accompanying an OCT image and the B-mode ultrasound of the eye. The latter may also shed light on the etiology behind the choroidal folds.

As mentioned above, contraction of the membrane may lead to the creation of a pseudo-hole. A pseudo-hole (fig. 1C), as opposed to a MH (see p. 12), which is a retinal break or defect, appears due to thickening of the retinal layers on both sides of the foveola, may lead to the false appearance of a true partial MH. A number of features assist in recognizing pseudo holes and differentiating them from true holes. The first is the existence of a distinct ERM alongside the pseudo-hole. Second, the foveal contour tends to appear punched out, well delineated, and steepened. Another important characteristic of a pseudo-hole, which is almost pathognomonic for the condition, is verticalization of the foveal pit – its edges on both sides tend to be straight and vertical, as opposed to the rounded edges of a MH or lateral splits of a lamellar hole (see p. 14). As the pathology arises from thickening of the perifoveal retina, a moderately increased thickness of the surrounding macula will be seen, usually with a normal central foveal thickness.

Another result of contraction of an ERM is the creation of traction on the inner retinal layers, leading to the formation of macular edema. In most cases, edema in ERM is diffuse and not cystic (fig. 1Da). In the rare occasions of a cystic edema caused by an ERM, it tends to appear central and symmetric (fig. 1Db).

A final subject of significance in the interpretation of ERM images on OCT is the photoreceptor-retinal pigment epithelial (PR-RPE) layers. When looking at the outer retinal layer in an OCT image, one should note the integrity of the PR layer. PR disruption was found to be a predictor of poor visual outcome in eyes with idiopathic

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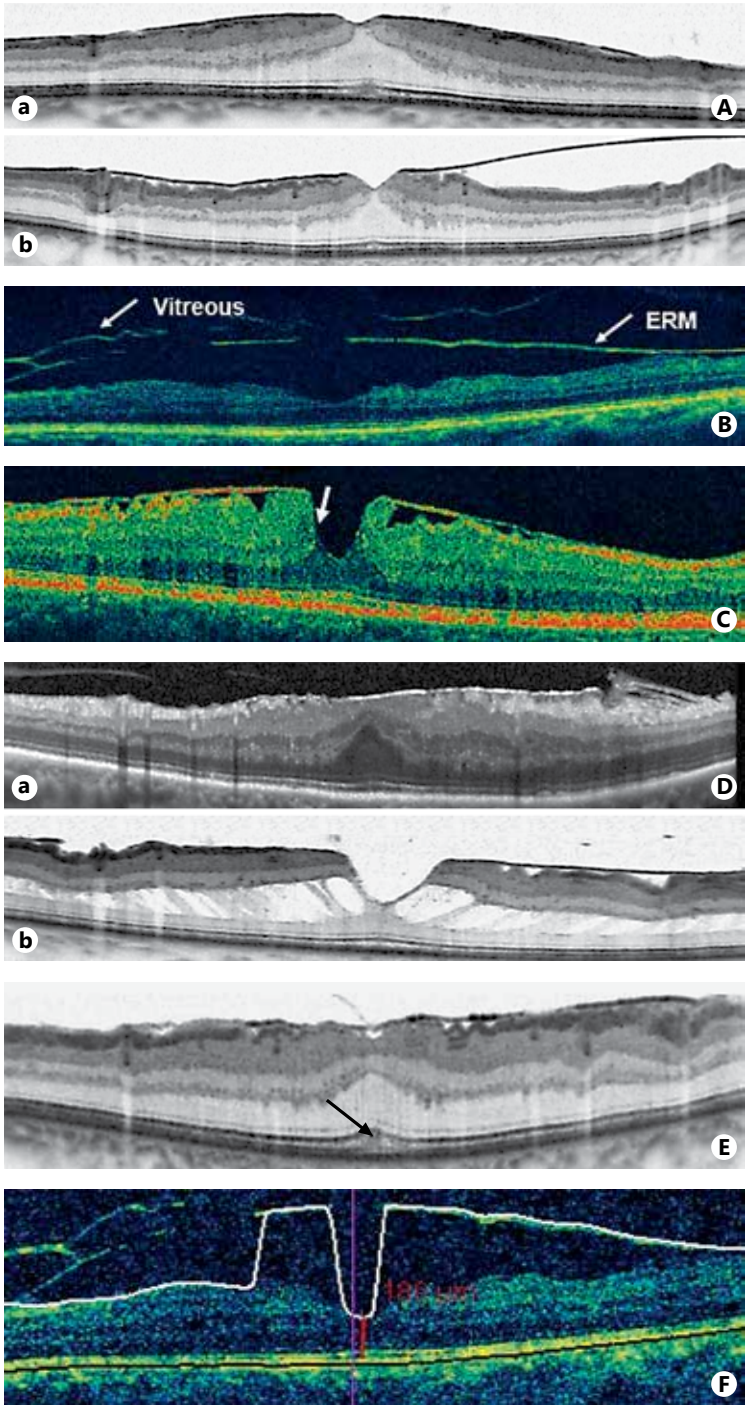

Fig. 1. A Epiretinal membrane as seen on spectral-domain OCT (SD-OCT). ERMs may appear as a highly reflective layer adherent to the retinal inner surface (**a**), or partially separated from the ILM (**b**). The membrane is creating a saw-tooth pattern in this image, seen to the left of the fovea. **B** Posterior hyaloid face versus ERM on SD-OCT. Notice the thinner reflection of the vitreous in comparison to the ERM and the degree of separation from the inner retina, which is greater for the posterior hyaloid. **C** Pseudo-hole created by an epiretinal membrane. Notice the steepening of the foveal contour, the verticalization of the foveal pit (arrow), and the increased thickness of the surrounding macula. **D a** Diffuse edema caused by an ERM on SD-OCT. **b** The rarer form of cystic edema along with an ERM, characterized by a symmetrical pattern. **E** SD-OCT image showing an ERM. Notice the complete integrity of the PR layer. Also note the subfoveal sediment between the PR and RPE layers (arrow). **F** Segmentation inner line breakdown results in inaccurate quantitative analysis.

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ERMs. Moreover, early ERM removal may prevent further progression of PR damage in those patients [3]. Another feature in these layers is the existence of reflective sediment between the PR and RPE layers which may be found in eyes with an ERM. The clinical significance of such sediments has not been proven (fig. 1E).

Finally, when interpreting a thickness map of patients with an ERM, one should not rely solely on the measurements made by the OCT software, as they can lead to erroneous treatment decisions. OCT segmentation lines are automatically drawn on the ILM as the inner line. However, segmentation inner line breakdown frequently occurs secondary to ERMs and vitreomacular traction (VMT), resulting in irregular topographic mapping and inaccurate quantitative analysis. The segmentation made by the OCT software may confuse an ERM for an ILM, leading to the false detection of macular edema (fig. 1F). SD-OCT devices allow for segmentation line correction, so the inner lines can be manually manipulated and moved to the correct location.

Macular Hole

A MH results from a vertical split in the foveal neurosensory retina. MHs are more common in females, and tend to occur in the sixth to eighth decades of life. They may be idiopathic, or may be secondary to a variety of conditions, including trauma (physical, electrical, laser), cystoid macular edema, retinal vascular diseases, macular pucker, rhegmatogenous retinal detachment, and hypertensive retinopathy. This chapter focuses on idiopathic MHs.

Investigations using OCT and ultrasonography suggest that idiopathic MHs are caused by vitreoretinal abnormalities and vitreoretinal traction [4].

Patients with MHs may complain of metamorphopsia and diminished central visual acuity.

On biomicroscopy a MH appears as a small yellow spot in the fovea. It may be differentiated from a pseudohole (see p. 12) or a lamellar hole (see p. 14) by the Watzke-Allen test, in which a thin beam of light is shined over the area of the spot. A positive test, indicating a true MH, requires that the patient perceive a 'break' in the slit beam.

In 1988, long before the introduction of OCT, Gass defined 4 stages in the development of MHs, based solely on clinical observation. He believed that idiopathic MHs development was attributed to tangential traction rather than anteroposterior forces [5]. The introduction of OCT provided better understanding of the disease pathogenesis and allowed clinicians to follow the sequence of events which lead to the development of full thickness MHs. It has led to the understanding that oblique traction, rather than tangential traction, results in MH. OCT imaging re-defined Gass' stages and is composed of 3 MH stages rater than 4.

Stage I (or impending MH) – This stage is divided into two stages. Stage Ia MH: (fig. 2Aa) foveal 'pseudocyst', or horizontal splitting (schisis), associated with a vitreous detachment from the perifoveal retina.

If the tractional forces persist, the pseudocyst might extend through the entire foveal thickness resulting in stage Ib MH (fig. 2Ab) where there is a break in the outer foveal layer of the pseudocyst.

A stage I MH may resolve spontaneously following separation of perifoveal vitreous adhesion in as many as 50% of cases. In other cases it may become a lamellar hole (see p. 14) or a full thickness MH (stage II).

When a tractional break develops in the 'roof' of the stage Ib pseudocyst a stage II full-thickness MH forms.

Stage II (fig. 2Ac) is defined as a full-thickness MH (FTMH) with posterior hyaloid (operculum) which remains attached to either side of the MH.

Stage III (fig. 2B) is defined as a FTMH along with foveal posterior hyaloid detachment. The posterior hyaloid may be seen with an operculum suspended over the hole.

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