Optical coherence tomography findings of incomplete posterior vitreoschisis with vitreomacular traction syndrome and impending macular hole: A case report

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INTRODUCTION

First reported by Green and Byrne in patients with diabetic retinopathy and vitreous hemorrhage (1), posterior vitreoschisis is a clinical entity characterized by splitting of the posterior vitreous cortex. Vitreomacular traction syndrome is a distinct clinical entity in which partial posterior vitreous detachment is present in combination with persistent macular adherence causing traction-induced visual impairment, metamorphopsia, photopsia, and micropsia (2).

Recently, optical coherence tomography (OCT) allowed visualization of incomplete posterior vitreoschisis leading to vitreomacular traction (3).

We report a case of vitreomacular traction syndrome with impending macular hole due to incomplete posterior vitreoschisis followed by OCT.

Case report

A 57-year-old woman reported blurred vision in her left eye (LE). Best-corrected visual acuity (BCVA) was 20/20 in the right eye (RE) and 20/30 in the LE. LE fundus biomicroscopic examination revealed a partial posterior vitreous detachment with macular thickening findings suggestive of a vitreomacular traction syndrome. OCT examination (Stratus OCT, Carl Zeiss Meditec, Dublin, CA) confirmed an evident increase in macular thickness in LE (mean foveal thickness: RE = 242 µm, LE = 446 µm; minimum foveal thickness: RE = 210±8 µm, LE = 441±73 µm;
**OCT findings of incomplete posterior vitreoschisis**

A total volume: RE = 7.22 mm$^3$, LE = 10.21 mm$^3$). The horizontal OCT scan (Fig. 1A: green line) showed a left eye macular morphology characterized by a foveal cystic cavity between photoreceptors and inner layers resembling an impending macular hole (Fig. 1B). At the vitreoretinal interface OCT scans showed a linear hyperreflective signal, usually referred as posterior vitreous cortex, adherent to the fovea causing vitreomacular traction. Moving the scan superiorly to the fovea, a split in the linear hyperreflective signal was evident (Fig. 2, Fig. 1A: blue line). The outer layer appeared partially attached to the retina with some bridging points causing an irregular retinal surface. The inner layer was completely detached from the retina. The split was detectable until the vascular arcades (Fig. 3, Fig. 1A: red line).

**Fig. 1** - (A) Preoperative fundus image showing the exact points of the optical coherence tomography (OCT) scans: Fig. 1B: green line, Fig. 2: blue line, Fig. 3: red line. (B) Preoperative OCT macular scan showing V-shaped vitreomacular traction and cystic foveal change.

**Fig. 2** - Peripherally to the fovea, a split in the linear hyperreflective signal is evident.

**Fig. 3** - Posterior vitreoschisis characterized by two parallel hyperreflective layers. The inner layer is completely separated from the retina while the outer layer is persistently adherent associated with an irregular internal neuroretina surface.

**Fig. 4** - Postoperative optical coherence tomography macular scan showing disappearance of any hyperreflective signal at the vitreoretinal interface. Mean foveal thickness is 245 µm.
The OCT findings were classified as incomplete posterior vitreoschisis with vitreomacular traction syndrome and impending macular hole. The patient underwent pars plana vitrectomy for progressive decrease in visual acuity with associated light metamorphopsia. We carefully observed the epiretinal membrane during vitreous surgery. Separation of vitreoretinal attachment at the fovea and removal of the inner wall of vitreoschisis was obtained during premacular vitrectomy; the outer wall was accurately peeled by means of Eckardt forceps beyond supertemporal and inferotemporal vascular branches. During peeling no dot epiretinal hemorrhages, usually related to internal limiting membrane removal, appeared. After peeling the cellophane-like macular aspect disappeared.

One month after surgery BCVA improved to 20/20 in the LE. OCT scan demonstrated a decrease in macular thickness (LE mean foveal thickness = 245 µm; minimum foveal thickness = 215 ± 12 µm; total volume = 7.41 mm³) and the disappearance of pseudocystic foveal changes. The hyperreflective linear signal at the vitreoretinal interface was no longer detectable (Fig. 4).

DISCUSSION

Posterior vitreoschisis, also known as laminated posterior vitreous cortex (4), is a spontaneous splitting of the posterior vitreous cortex. Before the OCT era posterior vitreoschisis was correctly detected by ultrasonography only when the outer layer appeared completely detached from the retina; it was thus considered a frequent cause of poor ultrasound prediction of PVD (5). OCT has been widely identified as a useful diagnostic tool for vitreoretinal interface pathologies. The association between incomplete posterior vitreoschisis and vitreomacular traction syndrome has been recently documented by means of OCT (3). However, some authors debate about the presence of vitreoschisis (6), referring to the outer hyperreflective signal partially adherent to the retina surface as the internal limiting membrane.

In our case report OCT clearly detected the split of the hyperreflective signal and documented it beyond the temporal vascular branches. In addition, vitreomacular traction caused intraretinal alterations leading to impending macular hole. To our knowledge, these findings have never been described all together. The intraoperative findings suggested the presence of a schisis cavity as in a true posterior vitreoschisis. These characteristics, together with the evidence that the internal limiting membrane thickness is thinner than the axial resolution of the Stratus OCT (8 mm) and a spontaneous internal limiting membrane detachment has never been demonstrated, are likely to support the hypothesis that posterior vitreoschisis exists and can be associated with vitreomacular traction syndrome.

REFERENCES