Recent advancements in optical coherence tomography (OCT) have identified the key role that perifoveal posterior vitreous detachments (PVDs) play in the development of macular holes, vitreomacular traction syndrome, and some aspects of diabetic macular edema. Before OCT was introduced, the anatomy of the posterior vitreous was hard to visualize during a biomicroscopic examination because of its transparency. The vitreous anatomy was studied biomicroscopically in postmortem eyes. Sebag reported that anomalous PVD causes vitreomacular traction syndrome, results in vitreoschisis with macular pucker or macular holes, and contributes to some cases of diabetic macular edema. Worst described a liquefied area of the vitreous anterior to the macula, which was observed by injecting India ink into the cisternal canal of the vitreous in postmortem eyes. We identified a posterior precortical vitreous pocket (PPVP) at autopsy in eyes in which the vitreous gel was stained with fluorescein. A PPVP is a physiologic liquefied lacuna whose posterior wall is a thin layer of vitreous cortex situated anterior to the macular area. Although PPVPs are difficult to observe with biomicroscopy, triamcinolone acetonide-assisted vitrectomy confirmed their presence. Time-domain OCT showed the vitreous cortex slightly detached from the optic disc, a complete PVD develops. An anatomical feature of the PPVP may play a role in the development of a perifoveal PVD.

In the current study, we used SD-OCT to evaluate the role of the PPVP in the development of PVD in healthy individuals.

Methods

We performed SD-OCT with noise reduction (Cirrus OCT, version 4.5; Carl Zeiss Meditec) in the right eyes of 368 healthy individuals.
volunteers (188 males and 180 females), using 9-mm horizontal and 6-mm vertical scans through the fovea. The subjects' ages ranged from 12 to 89 years (mean [SD], 57.1 [19.4] years; age <20 years in 22 subjects; 20-29 years in 23; 30-39 years in 25; 40-49 years in 46; 50-59 years in 52; 60-69 years in 91; 70-79 years in 69; and 80-89 years in 40).

This study was prospective and included consecutive subjects examined from June 2010 through December 2012. All 368 eyes were phakic. Subjects with high myopia exceeding −8.0 diopters (D) were excluded, and no eyes had vitreoretinal disease. The mean (SD) refractive error was −1.1 (2.3) D (range, −7.5 to 4.0 D). Before performing OCT, we evaluated all subjects for the presence of a PVD, performing biomicroscopy with a superfield lens (Volk Optical Inc). We defined a complete PVD as a detached posterior vitreous cortex with a Weiss ring.

During the OCT examination, we placed the scanner head backward to display the vitreous structure in the B-scan images. After obtaining SD-OCT images, we adjusted the contrast to display the gel, liquefied pocket, and cortex. All values are expressed as mean (SD) and range.
The study was conducted according to the tenets of the Declaration of Helsinki. The institutional review board ethics committee approved the study. All subjects provided informed consent after receiving a detailed explanation of the study.

**Results**

In all 227 eyes without a complete PVD, a PPVP was seen on the SD-OCT images. The condition of the posterior wall of the PPVP was classified into 5 stages according to the biomicroscopic findings and SD-OCT images (Figure 1): stage 0, no PVD with PPVP (134 eyes; mean [SD] subject age, 38.7 [16.5] years; range, 12-76 years); stage 1, paramacular PVD with PPVP (47 eyes; mean age, 55.2 [10.3] years; range, 36-77 years); stage 2, perifoveal PVD with PPVP (27 eyes; mean age, 62.0 [8.7] years; range, 46-81 years); stage 3, vitreofoveal separation with persistent attachment to the optic disc (19 eyes; mean age, 65.8 [6.2] years; range, 55-80 years); and stage 4, complete PVD (141 eyes; mean age, 73.2 [8.3] years; range, 48-89 years).

In stage 0, the premacular vitreous cortex appeared in the peripheral macula as a thin membrane that served as the posterior wall of the PPVP. In stage 1, the paramacular PVD involved separation of the vitreous cortex from part (5 eyes [11%]) or all (42 eyes [89%]) of the paramacular retina. In stage 2, a posterior wall of the PPVP had a detached cortex with a convex curve around the fovea as perifoveal PVD. In stage 3, vitreofoveal separation with persistent attachment to the optic disc, this separation was observed with an intact posterior wall of the PPVP (stage 3a) in 12 eyes (63%) and with a defect in the poste-
rior wall of the PPVP (stage 3b) in 7 (37%). The diameter of the defective area ranged from 2.5 to 4.6 mm (mean [SD], 3.5 [0.9] mm). In stage 4, biomicroscopy showed a Weiss ring and detached vitreous cortex, and no vitreous structure was seen on the OCT images. Figure 2 and Table 1 show the percentages for each stage of PVD in each decade of life. Mean ages and refractive errors for each stage are summarized in Table 2.

Discussion

Age-related PVD has been thought to be an acute event occurring when the liquefied fluid escapes through a break in the vitreous cortex into the retrohyaloid space. Uchino et al11 reported that perifoveal PVDs insidiously develop and progress to complete PVDs. Because of the limited resolution of time-domain OCT, those investigators detected a partially detached vitreous cortex but failed to show the vitreous structure. In the current study, using noise-reduced SD-OCT, we investigated the role of the PPVP and premacular vitreous cortex in early-stage PVDs.

The SD-OCT images showed a PPVP in all 227 subjects without a complete PVD. No PVDs were seen in subjects younger than 38 years (all stage 0). As we reported elsewhere,14 the posterior wall of the PPVP is a very thin vitreous cortex in younger subjects, and it thickens with age. The thickened cortex often has a laminated structure in the perimacula. First, a partial PVD occurred in the paramacula (stage 1), which preferentially occurred in the late fourth decade of life. The separation of the vitreous cortex then extended to the perifoveal area, which resulted in a perifoveal PVD (stage 2). Notably, the detached vitreous cortex around the fovea had a convex curve, which suggested its elasticity. In contrast to the other parts of the vitreous cortex, the premacular vitreous cortex is seen as a thin membrane separated from the vitreous gel by the intervening PPVP. Tangential contraction of the premacular vitreous cortex and relatively firm vitreoretinal attachment at the fovea may lead to a perifoveal PVD. Our findings and those of Uchino et al11 confirmed that a perifoveal PVD is a physiologic event in the course of development of a PVD. Johnson et al12 reported that a perifoveal vitreous detachment is the primary pathogenic event in idiopathic macular hole formation. If vitreous traction remains at the fovea at the stage of development of a perifoveal PVD, a macular hole or vitreomacular traction syndrome may ensue.

After a perifoveal PVD (stage 2), vitreomacular separation (stage 3) may occur. The foveal PVD may develop with the posterior wall of the PPVP intact (stage 3a) or with a defect (stage 3b) in the premacular vitreous cortex. The vitreous cortex is extremely thin at the fovea. The physiologic vitreofoveal adhesion may cause a defect in the thin vitreous cortex during a vitreous detachment. In a study using scanning electron microscopy,15 we observed a vitreous cortex remnant at the fovea in 44% of postmortem eyes with PVD. In the current series, 7 (37%) of 19 eyes with stage 3 (vitreomacular separation) had a defect in the posterior wall of the PPVP. These eyes may have some vitreous cortex left on the fovea. In cases of idiopathic preretinal macular fibrosis, an oval defect often is seen in the detached posterior hyaloid membrane.16 A histologic study of surgically obtained epiretinal membranes showed that a type 2 collagen layer was the major component of the epiretinal membrane.17 Those biomicroscopic and histologic findings suggested that the remnant of the posterior wall of the PPVP may be the source of the idiopathic premacular fibrosis. Using SD-OCT, Johnson17 showed that a defect in the detached premacular hyaloid membrane corresponded to the epiretinal membrane.

The vitreous cortex has a lamellar structure with a few layers.18 Splitting of the vitreous cortex was reported in some cases of vitreomacular traction syndrome20 and macular pucker.21 Thus, the outermost layer of the vitreous cortex may remain on the fovea even in vitreomacular separation with an intact PPVP (stage 3a).

The incidence of complete PVD (stage 4) increased to 44% in the seventh decade of life and 80% in the eighth decade of life from 14% in the sixth decade of life (Figure 2). Foos and Wheeler,22 reporting findings in a biomicroscopic study of postmortem eyes, noted age-related vitreous liquefaction and an increase in the incidence of PVDs in the sixth and seventh decades of life. Yonemoto et al23 examined myopic eyes using slit-lamp biomicroscopy and reported that vitreous liquefaction and PVD occurred earlier in patients with severe high myopia. In our current study, subjects with high myopia (exceeding −8.0 D) were excluded.

In conclusion, noise-reduced SD-OCT showed a PPVP in the early stage of PVD development. The premacular vitreous cortex is the posterior wall of the PPVP, which is seen as a thin membrane separated from the vitreous gel. This anatomical feature may cause trampoline-like detachment of the premacular vitreous cortex. Combined with firm vitreofoveal attachment, a perifoveal PVD forms. Although a perifoveal PVD can cause a macular hole or vitreomacular traction, it is a physiologic event during the development of age-related PVDs.

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Conflict of Interest Disclosures: None reported.

REFERENCES


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**OPHTHALMIC IMAGES**

**Initial Presentation of Pseudoxanthoma Elasticum**

Darin R. Goldman, MD; Gregory D. Lee, MD; Chirag P. Shah, MD; Jeffrey S. Heier, MD

A 52-year-old man was referred by his primary care physician for evaluation of decreased vision in his left eye. A, Characteristic “plucked chicken” skin appearance of the neck. B, Wide-field fundus imaging shows numerous angioid streaks, highlighted on indocyanine green angiography (B inset). C, Optical coherence tomography reveals a vitelliform-like subretinal cavity that did not leak on fluorescein angiography. A trial of intravitreal anti-vascular endothelial growth factor therapy did not result in any therapeutic effect.