Review

ROLE OF OPTICAL COHERENCE TOMOGRAPHY IN MANAGEMENT OF ACUTE POSTERIOR VITREOUS DETACHMENT AND ITS COMPLICATIONS

RUI WANG, MD,* CHRISTINE LOVENBERG, COA,† OLIVIA HESS, BS,‡ BOZHO TODORICH, MD, PHD†§

Purpose: Currently, no consensus exists on the role of optical coherence tomography (OCT) imaging in the setting of acute posterior vitreous detachment (PVD). The authors outline the clinical utility of OCT in the management of acute PVD and its complications.

Methods: Literature review of OCT findings in association with acute PVD and report of illustrative cases.

Results: Optical coherence tomography imaging in the setting of acute PVD can provide details of vitreoretinal interface that are difficult to appreciate on biomicroscopy alone including partial PVDs, focal vitreoretinal adhesions and traction, and subclinical macular changes. The presence of vitreous hyperreflective dots on OCT in the premacular space, especially if severe, is highly correlated with the presence of peripheral retinal breaks and development of epiretinal membrane. Advancements in OCT technology, including enhanced vitreous imaging OCT, swept-source OCT, wide-angle OCT, and widefield OCT, allow for increased resolution and expanded field of imaging of the vitreoretinal interface.

Conclusion: Optical coherence tomography imaging is an emerging standard of care in the setting of patients presenting with new flashes and floaters. The authors highlight the benefits of OCT imaging in patients with acute PVD, which includes recognition of the status of the vitreoretinal interface, assistance in identifying high-risk PVDs, and performance of risk assessment that predict future macular pathologic condition.

RETINA 43:371–378, 2023

Posterior vitreous detachment (PVD) is a slow physiologic process that involves the separation of the posterior hyaloid from the internal limiting membrane of the retina.[1,2] This process originates in the perifoveal macula and involves the liquefaction and collagen fibrillar aggregation that causes the vitreous to shrink, thus weakening vitreoretinal adhesions.[3–5] Therefore, it is integral to pathologic condition occurring at the vitreoretinal interface and plays a role in the pathogenesis of vitreoretinal disorders, such as epiretinal membrane (ERM)[6] and macular hole[7] formation, and development of vitreous hemorrhage,[8] vitreomacular traction,[7] and retinal detachments (RDs).[8]

The prevalence of PVD increases with age, with a reported incidence of 53% in patients older than 50 years and 87% among those older than 80 years.[9,10] Other risk factors for PVD formation include myopia, female gender, trauma, and intraocular inflammation. Retinal tears develop in approximately 15% of eyes with symptomatic PVD,[11] and if left untreated, they have been reported to cause RD in 33% to 55% of cases.[12,13] Given the clinical implications of

From the *Department of Ophthalmology, Pennsylvania State College of Medicine, Hershey, Pennsylvania †Lehigh Eye Specialists, P.C., Allentown, Pennsylvania ‡Stanford University School of Medicine, Stanford, California §Susquehanna Retina Center, P.C., Lemoyne, Pennsylvania

None of the authors has any financial/conflicting interests to disclose.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.retinajournal.com).

Reprint requests: Bozho Todorich, MD, PhD, Susquehanna Retina Center, P.C., 20 Erford Rd, Ste 110, Lemoyne, PA 17043; e-mail: btodorich@susquehannaretina.com

diagnosing a retinal tear associated with acute symptomatic PVD, a thorough clinical assessment is indicated, and this is one of the most common clinical presentations to a retina specialist in both academic and community practices.

Historically, clinical PVD was thought to occur when an abrupt break develops in the posterior vitreous cortex causing liquified vitreous fluid to rush into the retro-hyaloid space. Partial PVDs under this model were thought to be transient and progress rapidly within hours to days to complete PVD formation.[14] The early stages of shallow, partial PVD were difficult to detect with biomicroscopy or ultrasound, but more recently, B-scan ultrasonography with attention to perpendicularity has proven capable at detecting shallow posterior hyaloid detachments.[15,16] Additionally, the advent of high-resolution spectral-domain optical coherence tomography (SD-OCT) has provided substantial new capacity for high-resolution cross-sectional imaging of the vitreoretinal interface. Uchino et al[17] performed a series of OCT imaging on healthy patients with a mean age of 52 years old and found that early (perifoveal) stages of PVD were present in approximately 60% of eyes. Moreover, the author noted a gradual progression occurred from no PVD to early parafoveal separation, then to perifoveal PVD with residual adherence to the fovea, and finally to complete separation throughout the macular region. Detection and differentiation of these stages of PVD progression is near-impossible with conventional white-light biomicroscopy in most eyes, and thus, the evolving PVD remains subclinical for many years in earlier stages of PVD until the vitreous is fully separated from the optic nerve.[5] Nevertheless, this complex and underappreciated process of PVD evolution is thought to be a key pathogenic driver of many common vitreoretinal conditions managed in average retinal practice such as ERMs, vitreomacular traction syndrome, macular and lamellar holes, and macular edema.

Literature review was conducted on PubMed MEDLINE using keywords including posterior vitreous detachment AND optical coherence tomography, posterior vitreous detachment AND hyperreflective foci OR hyperreflective dot, posterior vitreous detachment AND epiretinal membrane.

Status of Vitreoretinal Interface

Currently, no consensus guidelines exist to address the utility of OCT imaging in the setting of acute PVD. Although the detection of Weiss ring on biomicroscopy is thought to indicate a complete PVD, it may be difficult to determine the precise completeness of vitreoretinal detachment using clinical observation alone. Wagley et al[18] conducted a cross-sectional study of 506 patients using data from the Primary Retinal Detachment Outcomes Study and found that there was a significantly higher rate of PVD identification by SD-OCT (78.4%) than clinical examination alone (51.6%) ($P \leq 0.001$). This study used a $30 \times 30°$ capture mode (roughly 8.9×7.4 mm) using the Spectralis (Heidelberg Engineering Inc; Heidelberg, Germany) machine centered on the fovea that allows the ability to examine retinal layers and the posterior vitreous surface from the optic cup to beyond the arcade, thus providing a more comprehensive assessment of vitreomacular pathologic condition.

Mojana et al[19] performed SD-OCT imaging of patients with symptomatic PVD and compared the diagnosis of partial and complete PVD with clinical examination alone. The study found that SD-OCT was equally effective at diagnosing complete PVD compared with clinical examination alone ($P = 0.683$) but was statistically more effective at diagnosing partial PVD compared with clinical examination alone ($P \leq 0.001$). Additionally, SD-OCT imaging in eyes with various degrees of posterior vitreous detachment identified macular abnormalities including vitreomacular traction (3%), ERM (6%), and dehiscence (1%), all of which were found at a higher incidence in eyes with partial PVD.

Finally, idiopathic macular holes have been postulated to be caused by tangential contraction of the premacular vitreous cortex. During the development of a PVD, the release of vitreous traction can reduce the hole diameter and resolution of subretinal fluid, resulting in spontaneous closure of macular holes. This was observed in 3 of 66 eyes (5%) of cases by Guyer et al[20] and 3 of 112 eyes (3%) of cases by Hikichi et al[21] over a follow-up period of over 4 years in patients with a Stage 3 full-thickness macular hole. These studies support the notion that SD-OCT can have utility in not only diagnosing PVDs but can also provide additional details of the vitreoretinal interface and vitreous body, including partial PVD, focal vitreoretinal adhesion and traction, and subclinical macular changes related to the PVD that are not readily apparent on conventional ophthalmoscopy.[6,22]

Vitreous Hyperreflective Dots as Biomarker of a Rhegmatogenous Event

Risk factors for retinal tears associated with symptomatic PVD have been identified on examination, including vitreous hemorrhage, vitreous pigment

("tobacco dust") in the anterior vitreous, and the presence of lattice degeneration.[23,24] More recently, the detection of vitreous hyperreflective dots (VHRDs) on SD-OCT has been found to correlate with the presence of a peripheral retinal break. These VHRDs are thought to represent red blood cells from vitreous hemorrhage or clumps of retinal pigment epithelial cells emanating from retinal breaks.[25] Sample image of patients with retinal tears and VHRDs demonstrated on SD-OCT is shown in Figure 1. In a review of 60 patients presenting with acute PVD, Todorich[26] demonstrated that VHRDs had a sensitivity of 100% and specificity of 65% in association with retinal tears. Similarly, Ansari et al[4] demonstrated a sensitivity and specificity of 65.2% and 75.4%, respectively, in association of VHRDs with retinal tears in a retrospective study of 78 patients. When eliminating all patients with a vitreous hemorrhage that can artificially cause posterior vitreous opacity, the association of VHRDs remained high with sensitivity and specificity of 63.8% and 93.7%, respectively. Oh et al[25] further found that the quantity of VHRDs was clinically relevant, and retinal tears were more frequently found in patients with severe VHRDs (VHRDs $\geq$ 15). Subsequent visits of patients with retinal tears demonstrated significant reduction in mean number of VHRDs supporting that the presence of VHRDs indicate an acute process. These studies support that the presence of VHRDs on SD-OCT especially when severe should raise clinical suspicion for a peripheral retinal break in patients presenting with symptoms of flashes and floaters. Notably, the absence of clinical vitreous hemorrhage and VHRDs indicated a 94% chance that no retinal breaks were present.[4] Thus, a "negative" or normal SD-OCT imaging in setting of an acute PVD seems to have a strong negative predictive value for a peripheral rhegmatogenous event, especially if paired with conventional ophthalmoscopy with scleral depression.

Posterior Vitreous Detachment and Association With Epiretinal Membrane Formation

Posterior vitreous detachments are strongly associated with the formation of ERM, and some degree of ERM formation has been reported in as many as 40%

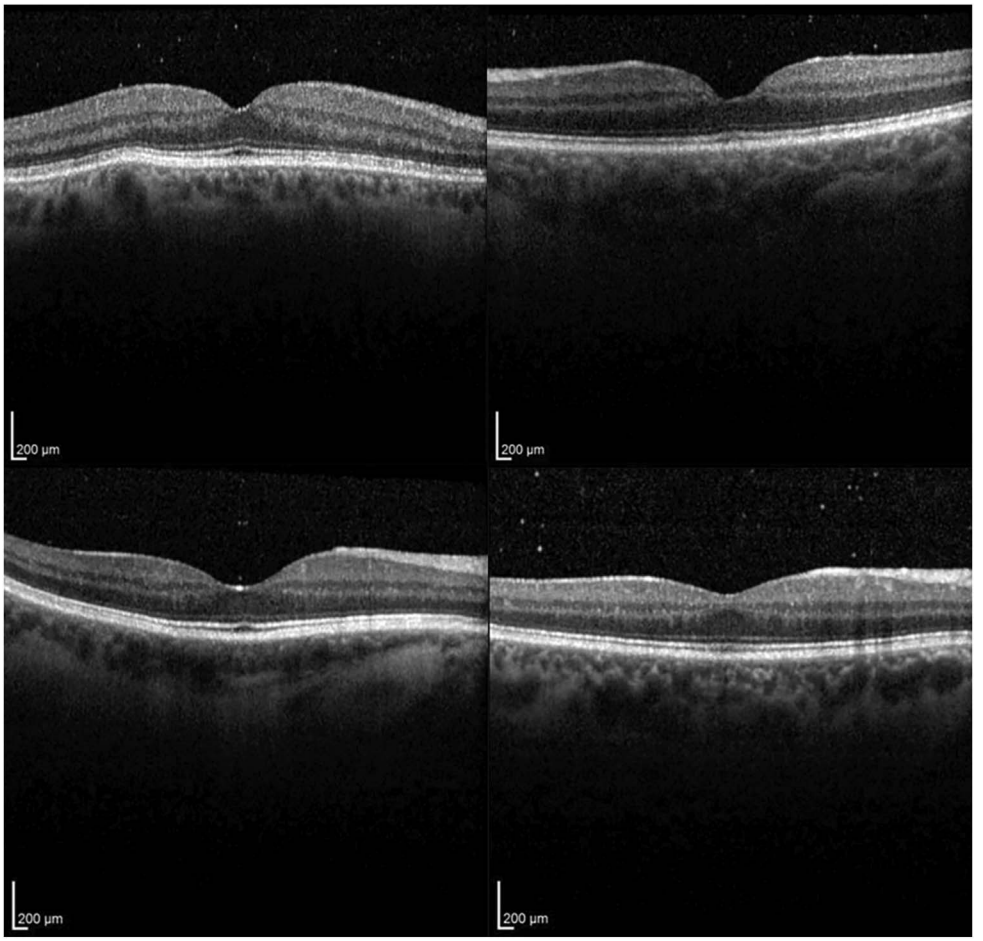

Fig. 1. Spectral-domain optical coherence tomography images demonstrating the presence of VHRDs in patients with retinal tears.

of eyes with PVD.[27] It is postulated that as the vitreous shrinks during the development of a PVD, tractional forces are created that disrupt the internal limiting membrane. This allows for migration and proliferation of the glial cells, which form an abnormal cellophane-like membrane over the macula that may thicken and contract over time.[28] In a retrospective case series, best-corrected visual acuity (BVCA) of patients with ERM and no PVD, complete PVD with collapse, or partial PVD without shrinkage were observed over a 2-year period. The absence of PVD was defined by the lack of evidence of a posterior hyaloid membrane even after ocular movement; complete PVD with collapse was defined by a highly mobile detached posterior hyaloid membrane that can be traced easily with a prepapillary glial ring observed on the posterior hyaloid membrane; and partial PVD with shrinkage was defined by the presence of a posterior hyaloid membrane that was not mobile even after ocular movement. The study found that the cases of ERM associated with a partial PVD without shrinkage had the worst visual acuity (0.17 ± 0.23) compared with the no PVD group (0.06 ± 0.14) and the complete PVD with collapse group (0.0009 ± 0.09) ($P < 0.05$ for both comparisons).[28] Furthermore, another study found that VHRDs were predictive of subsequent ERM formation with 69.9% of patients with VHRDs on presentation developing ERM over a follow-up course of at least 3 months.[26] Figure 2 demonstrates an example of a patient presenting with acute PVD and VHRDs and subsequent development of ERM 6 months later. Correct diagnosis of the type of PVD by OCT and presence of VHRDs can predict risk of subsequent ERM formation and guide patient counseling on visual prognosis.

Role of Novel Optical Coherence Tomography Technology

Advancements in OCT technology has led to novel imaging strategies and methods of imaging the vitreoretinal interface. The enhanced vitreous imaging OCT was developed to enhance the characterization of the PVD process and is optimal for evaluation and quantification of VHRDs. Enhanced vitreous imaging

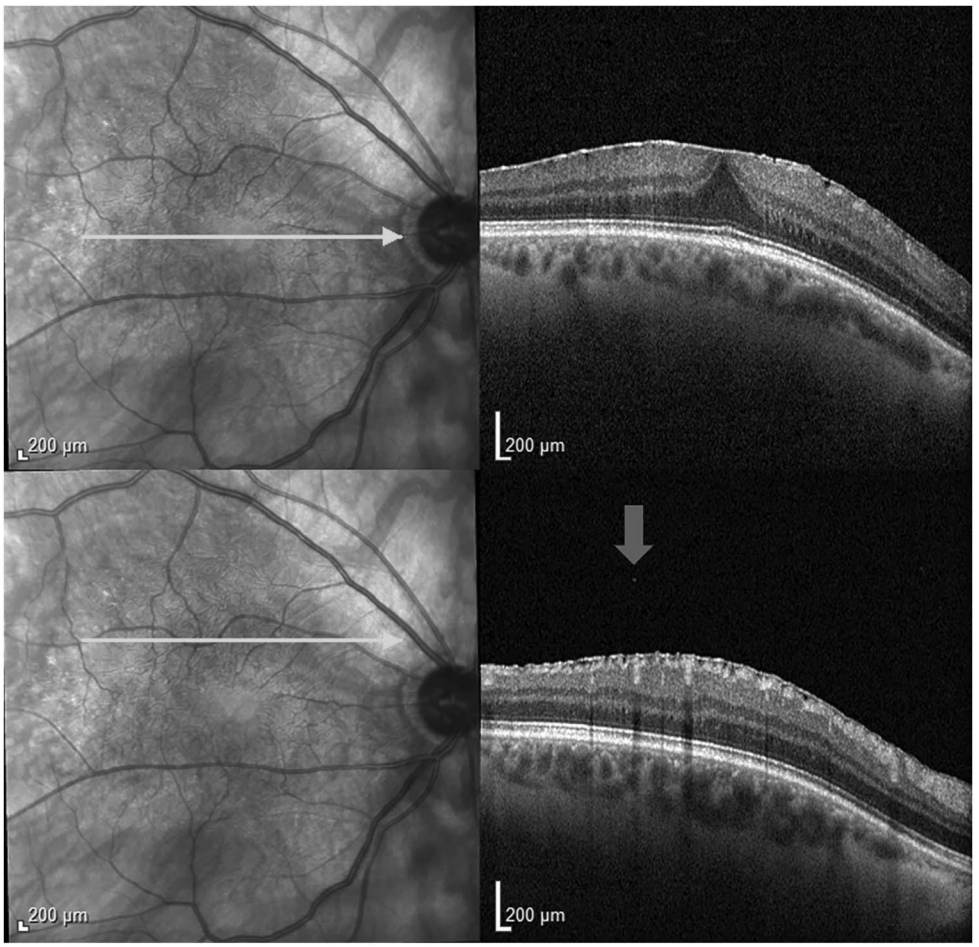

Fig. 2. Spectral-domain optical coherence tomography images demonstrating the development of ERM at the follow-up of patient. Persistence of VHRD is demonstrated by arrow.

OCT uses six radial scans with a scan width of 30° centered at the fovea and uses built-in functions, including focusing on the vitreous, positioning the retinal layers inferiorly to allow maximum imaging depth into the vitreous, and adjusting the image brightness to allow optimal imaging resolution and depth into the vitreous.[29,30] Such built-in functions to enhance imaging of the vitreous complex can be adopted for use in other OCT modalities to enhance visualization of the vitreous.

The development of swept-source OCT (SS-OCT) has resulted in increased scan speed and deeper penetration than previous modalities of OCT allowing for detailed documentation of vitreoretinal features in a 3-dimensional dataset.[31] In a retrospective study of 175 patients, Hwang et al[32] examined preoperative macular SD-OCT (6 mm OCT scan length) as compared with PVD determination during vitrectomy and found a low positive predictive value (53%) of a macular OCT scan diagnosing a complete PVD. The authors concluded that the limited depth of field on OCT was a significant limitation as the diagnosis of complete PVD relied on the absence of the premacular bursa and posterior vitreous cortex. Swept-source OCT technology, especially when the images include a wide window extending past the perifoveal region and optic nerve head, allows for more definitive diagnosis of a complete PVD. Wang et al[33] assessed the precision of diagnosing PVD by SS-OCT, biomicroscopy, and B-scan ultrasound. The study found that the agreement on PVD status by SS-OCT was similar for all three methods; however, in cases of discrepancies, a complete PVD was only visualized by SS-OCT. Notably, the SS-OCT images in the study used a 16-mm window capturing the perifoveal region and optic nerve head. The benefit of capturing the peripapillary retina is further supported in a study by Bertelmann et al,[34] which demonstrated a case of a PVD that was falsely diagnosed by OCT using a 6-mm window that was refuted when the same eye was imaged using a 12-mm horizontal window demonstrating persistent vitreous adhesion to the optic nerve head. Similarly, Moon et al[35] found that adding a peripapillary OCT scan improved the diagnostic ability of the OCT and can especially be helpful when PVD status is ambiguous on transverse OCT images.

The developments of enhanced vitreous imaging OCT and SS-OCT allow for mapping of the locus and area of vitreous detachment from the retina as well as measurement of areas of vitreoretinal adhesion. Such measurements and maps can be helpful in following vitreous detachment over time, for preoperative planning, and assessing treatment response to surgery or pharmacologic enzymatic vitreolysis.[36]

Wide-angle OCT is another emerging technology that allows for the visualization of broader vitreoretinal interface extending from the macula to the periphery. Tsukahara conducted a cross-sectional study involving montaged OCT of 144 healthy eyes and noted that PVD was observed as early as the third decade of life, and the first appearance of PVD was noted in the extramacular-peripheral vitreous where the vitreous gel is directly adherent to the posterior cortical vitreous and retinal surface rather than the perifoveal macula that previous research had suggested.[37] Additionally, >40% of eyes without observable fundus disease at PVD initiation were associated with vitreoschisis, which was demonstrated by lamellar separation of the posterior vitreous cortex noted on montaged OCT. The clinical significance of these findings in otherwise normal eyes is unknown and requires further study. Nevertheless, in pathologic settings, such as a rhegmatogenous RD, the correct identification of vitreoschisis is critical in preoperative evaluation and surgical planning. It can have profound impact on surgical approach, prognosis, visual and anatomic outcomes, and risk stratification for subsequent development of proliferative vitreoretinopathy. For example, knowing whether there is no PVD or only a partial PVD during preoperative evaluation of a patient with rhegmatogenous RD would compel a serious consideration of primary scleral buckling as the choice for mode of RD repair. Observing vitreoschisis or partial PVD on preoperative OCT imaging should compel surgeon to ensure that complete PVD is surgically induced with vitreous cutter early in the case. This is, in authors' opinion, a critical step for successful RD repair, either by direct visualization or by staining with triamcinolone acetonide or chromovitrectomy adjuncts such as such as brilliant blue or indocyanine green in cases where ILM peeling is used to ensure that all posterior cortical vitreous is removed.[38]

Finally, the adoption of angled and widefield OCT into clinical practices has allowed for enhanced visualization of retinal pathologic condition spanning greater area than the standard scan and allows for the application of OCT technology to imaging retinal periphery. Figure 3 demonstrates a highly myopic patient with blonde fundus who presented with acute PVD symptoms. The scleral depressed examination by a fellowship-trained retina specialist failed to identify any retinal breaks. The patient's macular OCT demonstrated the presence of VHRDs that prompted additional examination and imaging. Peripheral OCT scan noted a localized hyporeflective area on near-infrared reflectance and detected presence of a localized shallow RD stemming from a small horseshoe tear and associated vitreoretinal traction. These findings could

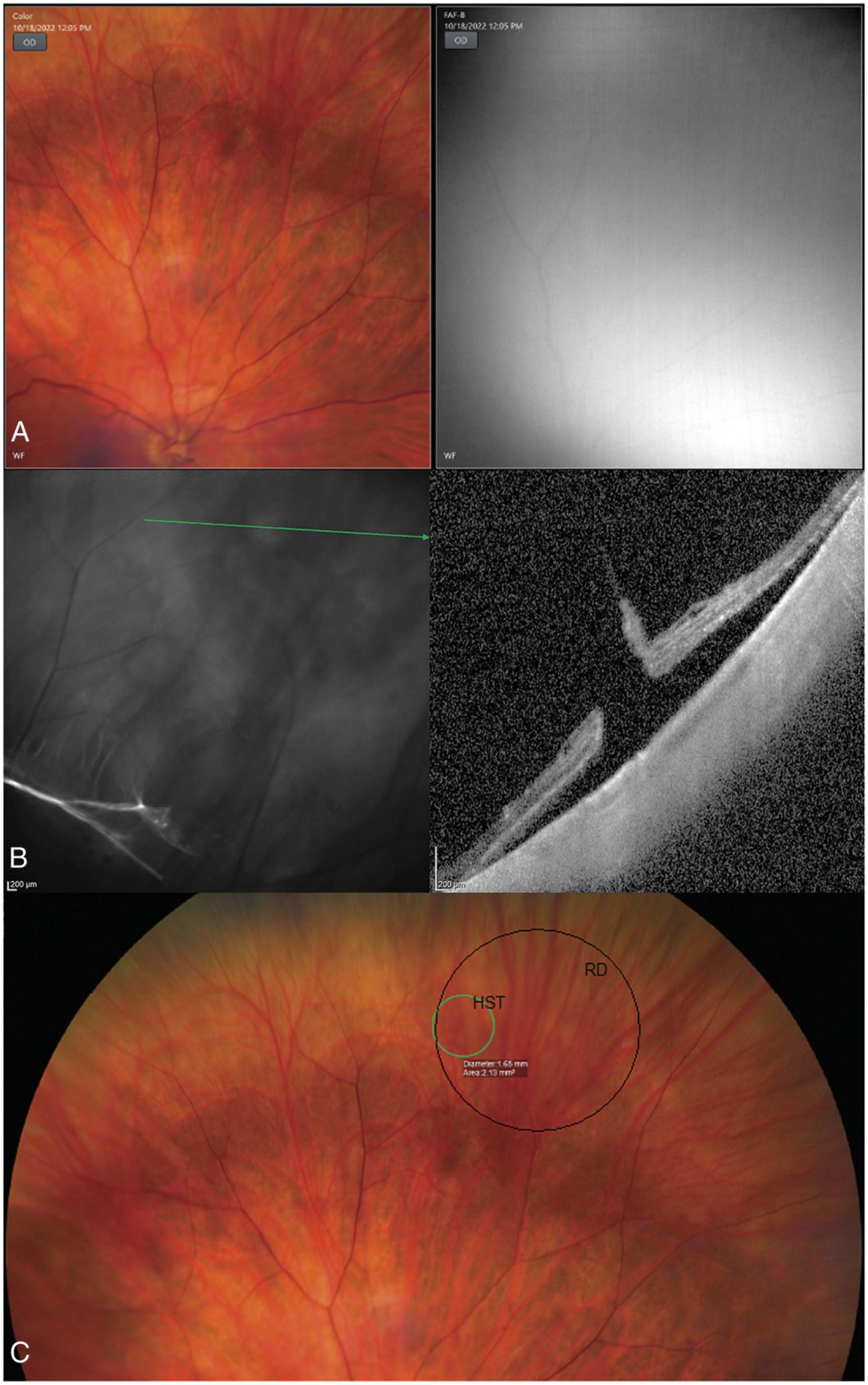

Fig. 3. A. Colored fundus photograph and fundus autofluorescence of peripheral retina at area of interest without appreciable retinal pathologic condition. **B.** Widefield OCT at area of interest demonstrates localized hyporeflective area with retinal tear and associated shallow RD and vitreoretinal traction (arrow). **C.** Colored fundus photograph of peripheral retina, with circles localizing area of horseshoe tear and RD.

neither be visualized on repeat white-light and red-free biomicroscopy or indirect ophthalmoscopy with scleral depression nor were they visible on widefield fundus photography obtained on the same visit. Patient subsequently underwent pars plana vitrectomy at which time the retinal tear and associated shallow

peripheral rhegmatogenous RD were confirmed intraoperatively and successfully repaired with endolaser and fluid–gas exchange as shown in **Supplemental Digital Content** (see **Video 1**, http://links.lww.com/IAE/B922). Additionally, when a definitive distinction cannot be made between retinoschisis and RD on clinical examination alone, widefield OCT can provide information on the location of the splitting of the retinal layers.

In conclusion, SD/SS macular OCT imaging of patients presenting with symptoms of acute PVD has underappreciated yet significant clinical utility in providing information regarding the vitreoretinal interface, identification of subclinical macular or extramacular pathologic condition, risk stratification for the presence of peripheral retinal breaks especially in the presence of severe VHRDs, and information regarding risk of subsequent ERM formation. Furthermore, a "normal" macular OCT in setting of acute PVD has a strong negative predictive value against peripheral rhegmatogenous event, especially if combined with traditional examination with scleral depression. A 30 × 30° capture mode can be easily adapted to current SD-OCT systems to allow improved visualization of the posterior vitreous surface, and SS-OCT with a wide capture window including the peripapillary retina can significantly enhance the sensitivity of diagnosing a PVD. Angled OCT and widefield OCT are additional advances that can be adopted into clinical practice to allow enhanced visualization of the retinal periphery. Further advances in OCT imaging technology will provide clinicians with even better understanding of the vitreoretinal interface and ultimately guide improved clinical management of vitreoretinal diseases. For all these reasons, we strongly believe that computerized imaging of posterior segment/OCT should be a standard of care for evaluation of patients presenting with symptoms of acute PVD.

Key words: posterior vitreous detachment, optical coherence tomography, hyperreflective dots, hyperreflective foci, retinal tear, retinal hole, epiretinal membrane, rhegmatogenous retinal detachment.

References

1. Foos RY. Ultrastructural features of posterior vitreous detachment. Albrecht Von Graefes Archiv Klin Exp Ophthalmol 1975;196:103–111.
2. Hollands H, Johnson D, Brox AC, et al. Acute-onset floaters and flashes: is this patient at risk for retinal detachment? JAMA 2009;302:2243–2249.
3. Di Michele F, Tatone A, Romano MR, Repetto R. A mechanical model of posterior vitreous detachment and generation of vitreoretinal tractions. Biomech Model Mechanobiol 2020;19:2627–2641.
4. Ansari WH, Blackorby BL, Shah GK, et al. OCT assistance in identifying retinal breaks in symptomatic posterior vitreous detachments. Ophthalmic Surg Lasers Imaging Retina 2020;51:628–632.
5. Johnson MW. Posterior vitreous detachment: evolution and role in macular disease. Retina 2012;32:S174–S178.
6. Kishi S, Demaria C, Shimizu K. Vitreous cortex remnants at the fovea after spontaneous vitreous detachment. Int Ophthalmol 1986;9:253–260.
7. Duker JS, Kaiser PK, Binder S, et al. The International Vitreomacular Traction Study Group classification of vitreomacular adhesion, traction, and macular hole. Ophthalmology 2013;120:2611–2619.
8. Novak MA, Welch RB. Complications of acute symptomatic posterior vitreous detachment. Am J Ophthalmol 1984;97:308–314.
9. Pischel DK. Detachment of the vitreous as seen with slit-lamp examination. Trans Am Ophthalmol Soc 1952;50:329–346.
10. Hikichi T, Hirokawa H, Kado M, et al. Comparison of the prevalence of posterior vitreous detachment in whites and Japanese. Ophthalmic Surg Lasers Imaging Retina 1995;26:39–43.
11. Brod RD, Lightman DA, Packer AJ, Saras HP. Correlation between vitreous pigment granules and retinal breaks in eyes with acute posterior vitreous detachment. Ophthalmology 1991;98:1366–1369.
12. Wilkinson CP. Evidence-based medicine regarding the prevention of retinal detachment. Trans Am Ophthalmol Soc 1999;97:397–404; discussion 404-406.
13. Shea M, Davis MD, Kamel I. Retinal breaks without detachment, treated and untreated. Mod Probl Ophthalmol 1974;12:97–102.
14. Foos RY, Wheeler NC. Vitreoretinal juncture: synchysis senilis and posterior vitreous detachment. Ophthalmology 1982;89:1502–1512.
15. Johnson MW. Perifoveal vitreous detachment and its macular complications. Trans Am Ophthalmol Soc 2005;103:537–567.
16. Sebag J. Imaging vitreous. Eye 2002;16:429–439.
17. Uchino E, Uemura A, Ohba N. Initial stages of posterior vitreous detachment in healthy eyes of older persons evaluated by optical coherence tomography. Arch Ophthalmol 2001;119:1475–1479.
18. Wagley S, Belin PJ, Ryan EH. Utilization of spectral domain optical coherence tomography to identify posterior vitreous detachment in patients with retinal detachment. Retina 2021;41:2296–2300.
19. Mojana F, Kozak I, Oster SF, et al. Observations by spectral-domain optical coherence tomography combined with simultaneous scanning laser ophthalmoscopy: imaging of the vitreous. Am J Ophthalmol 2010;149:641–650.
20. Guyer DR, de Bustros S, Diener-West M, et al Observations on patients with idiopathic macular holes and cysts Arch Ophthalmol 1992;110:1264–1268.Arch Ophthalmol.
21. Hikichi T, Yoshida A, Akiba J, Trempe CL. Natural outcomes of stage 1, 2, 3, and 4 idiopathic macular holes. Br J Ophthalmol 1995;79:517–520.
22. Doi N, Uemura A, Nakao K, Sakamoto T. Vitreomacular adhesion and the defect in posterior vitreous cortex visualized by triamcinolone-assisted vitrectomy. Retina 2005;25:742–745.
23. Sigelman J. Vitreous base classification of retinal tears: clinical application. Surv Ophthalmol 1980;25:59–74.
24. Boldrey EE. Relationship between floaters, light flashes, or both, and complications of posterior vitreous detachment. Am J Ophthalmol 1994;118:682–683.
25. Oh JH, Oh J, Roh HC. Vitreous hyper-reflective dots in optical coherence tomography and retinal tear in patients with acute posterior vitreous detachment. Curr Eye Res 2017;42:1179–1184.

26. Todorich B. Vitreous Hyper Reflection Foci Are Predictive Of Peripheral Retinal and Macular Pathology in the Setting of Acute PVD. Oral presentation at: American Society of Retinal Specialists; July 2019; Chicago, IL.
27. Shirakawa H, Ogino N. Idiopathic epiretinal membranes with spontaneous posterior vitreous separation (with 1 color plate). Ophthalmologica 1987;194:90–94.
28. Clarkson JG, Green WR, Massof D. A histopathologic review of 168 cases of preretinal membrane. Retina 2005;25:1–17.
29. Pang CE, Freund KB, Engelbert M. Enhanced vitreous imaging technique with spectral-domain optical coherence tomography for evaluation of posterior vitreous detachment. JAMA Ophthalmol 2014;132:1148–1150.
30. Spaide RF, Valmaggia P, Maloca PM. Imaging the vitreous with a novel boosted optical coherence tomography technique: posterior vitreous detachment. Retina 2022;42:1425–1432.
31. Toyoda F, Ota A, Tanaka Y, et al. Relationship between variations in posterior vitreous detachment and visual prognosis in idiopathic epiretinal membranes Clin Ophthalmol 2015;10:7–11.
32. Hwang ES, Kraker JA, Griffin KJ, et al. Accuracy of spectral-domain OCT of the macula for detection of complete posterior vitreous detachment. Ophthalmol Retina 2020;4:148–153.
33. Wang MD, Truong C, Mammo Z, et al. Swept source optical coherence tomography compared to ultrasound and biomicroscopy for diagnosis of posterior vitreous detachment. Clin Ophthalmol 2021;15:507–512.
34. Bertelmann T, Goos C, Sekundo W, et al. Is optical coherence tomography a useful tool to objectively detect actual posterior vitreous adhesion status? Case Rep Ophthalmological Med 2016;2016:1–5.
35. Moon SY, Park SP, Kim YK. Evaluation of posterior vitreous detachment using ultrasonography and optical coherence tomography. Acta Ophthalmol 2020;98:e29–e35.
36. Liu JJ, Witkin AJ, Adhi M, et al Enhanced vitreous imaging in healthy eyes using swept source optical coherence tomography PLoS One 2014;9:e102950.
37. Tsukahara M, Mori K, Gehlbach PL, Mori K. Posterior vitreous detachment as observed by wide-angle OCT imaging. Ophthalmology 2018;125:1372–1383.
38. Sebag J. Vitreoschisis. Graefes Arch Clin Exp Ophthalmol. 2008;246:329–332.