American Academy of Optometry: Case Report 5

Clinical Findings and Management of Posterior Vitreous Detachment

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Abstract: A posterior vitreous detachment is a degenerative process associated with aging that affects the vitreous when the posterior vitreous cortex separates from the internal limiting membrane of the retina. The composition of the vitreous gel can degenerate two collective ways, including synchysis or liquefaction, and syneresis or shrinking. Commonly, this process of separation occurs with the posterior hyaloid resulting in a Weiss ring overlying the optic nerve. Complications of a posterior vitreous detachment may include retinal breaks or detachments, retinal or vitreous hemorrhages, or vitreomacular traction. This case presentation summarizes the etiology of this ocular condition as well as treatment and management approaches.

Key Words: *Posterior Vitreous Detachment, Weiss Ring, Vitreous Degeneration, Scleral Depression, Nd:YAG Laser*

Introduction

The vitreous humor encompasses the posterior segment of the eye and fills approximately three quarters of the ocular space.[1] The vitreous is a transparent, hydrophilic, "gel-like" substance that is described as a dilute solution of collagen, and hyaluronic acid.[2,3,4] It is composed of 98% to 99.7% water.[4] As the eye matures, changes may occur regarding the structure and composition of the vitreous. The vitreous functions to provide support to the retina against the choroid, to store nutrients and metabolites for the retina and lens, to protect the retinal tissue by acting as a "shock absorber," to transmit and refract light, and to help regulate eye growth during fetal development.[3,4]

Case Report

Initial Visit (03/23/2018)

A 59-year-old Asian female presented as a new patient for examination with a complaint of a new onset of floaters and flashes of light in her right eye. She reported that she started observing a large floater or blurred spot in her vision seven days prior to her appointment and she had not noticed any improvement. Her symptoms were constant in nature, the appearance and location of the floater changed when she looked in different directions, and the floater was observed in variable locations. Additionally, she reported momentary flashes of light occasionally associated with the floaters. She noted the flashes particularly occurred in an episode two days following the floater's onset. She denied any curtain or veil over her vision. The patient's last ocular examination was in July of 2017. Her ocular history included myopia, astigmatism, and presbyopia. She denied any past ocular surgery or trauma. Family ocular history was negative. The patient's medical history was also negative. No current medications were reported. Social history was negative for tobacco, alcohol, or recreational drug use. She had no known systemic medication allergies. She was oriented to person, place, and time, and her mood was appropriate.

Spectacle corrected distance visual acuity was $20/25^{-2}$ OD and $20/20^{-1}$ OS. Spectacle corrected near visual acuity was 20/20 OD and 20/20 OS. The patient's habitual flat-top bifocal spectacle correction measured with lensometry was -2.25 -1.00 x 011 OD and -0.50 D.S. OS with a +2.75 Add OD, OS. Confrontation visual fields were full to finger counting in both eyes. Extraocular muscle testing showed full range of motion without pain or diplopia. Pupils were equal, round and reactive to light with no afferent pupillary defect noted. Manifest refraction was -2.00 -1.50 x 016 OD and -0.25 D.S. OS with a +2.50 Add, with best corrected visual acuity at distance as $20/20^{-1}$ OD and 20/20 OS and best corrected visual acuity at near through the add power as 20/20 OD and 20/20 OS. Slit lamp biomicroscopy revealed normal adnexae, lashes, puncta, and palpebral and bulbar conjunctiva in both eyes. The lids in the right eye were clean and free of debris, however, a Meibomian gland orifice capped with sebum was noted in the lower lid of the left eye. Meibography was ordered at-this-time to evaluate and monitor the condition of the Meibomian glands further. Meibography of the lower eyelids showed no

atrophy or truncation of any meibomain glands in either eye. The corneas were intact and clear through all layers in both eyes. Both irides were flat and brown in both eyes. The anterior chambers appeared deep and quiet with open angles estimated at 4+ using the Von Herrick method. One drop of Benoxinate 0.4%/Fluorescein 0.25% (Fluress) was instilled in both eyes prior to intraocular pressure measurements. Goldmann applanation tonometry measured 10 mmHg OD, 10 mmHg OS at 9:54 A.M. The patient was dilated using one drop of Mydriacyl 1%, and one drop of Phenylephrine 2.5% in both eyes at 9:59 A.M.

Upon full dilation, an evaluation of the posterior segment by slit lamp with 90D lens and by Binocular Indirect Ophthalmoscope with 20D lens was performed. Grade 1+ nuclear sclerosis was noted in the media of the lenses in both eyes. Assessment of the fundus revealed the optic nerve heads were well perfused with cup to disc (C/D) ratios of 0.25/0.25 in both eyes. The right eye had peripapillary atrophy noted in the beta and alpha zones circumferentially. A flat and intact macula was noted in both eyes. The right eye revealed a small, pinpoint hemorrhage within the temporal peripapillary zone (Figure 3). Retina and retinal vasculature was flat and intact with no pathology noted in left eye. Thorough evaluation of the vitreous demonstrated a Weiss ring overlying the optic nerve head as well as an aggregation of collagen fibrils noted in the vitreous cavity inferiorly in the right eye (Figure 1 and 2) and syneresis of the vitreous in the left eye. No vitreous hemorrhage or pigment, Schaeffer's sign, was noted in the right eye. Scleral depression was ordered at-this-time to evaluate and monitor the retina for any breaks, tears, or detachments. Scleral depression was performed with a scleral depressor and Binocular Indirect Ophthalmoscope with 20D lens without signs of any retinal breaks, tears, or detachments.

The differential diagnoses considered at-this-time of the examination included:

Vitritis
Retinal Break or Detachment
Vitreous Hemorrhage
Migraine Aura
Posterior Vitreous Detachment

- Vitritis presents with symptoms of blurry vision and a new onset of floaters. Ocular signs noted include vitreous haze, cells located in the posterior vitreous with the potential of them also being within the anterior vitreous, and sheathing around retinal vessels.
- Retinal detachment is characterized by symptoms of floaters, flashes of light, and a curtain over a part of the field of vision. Ocular signs observed are fluid within the sub-retinal space causing elevation of the retina.
- Vitreous hemorrhage presents with symptoms of sudden, painless vision loss, or sudden areas of black spots in the vision. Ocular signs noted include no view of the fundus or red reflex in severe cases, or a partially obscured view of the retina in a mild case.

- Migraine aura is characterized by symptoms of flashing scintillating lights which last for minutes, that may occur with or without headache. Ocular signs observed include normal ocular health with an improvement of symptoms over time, such as a typical aura.
- Posterior vitreous detachment (PVD) presents with symptoms of a large floater or a blurred area within vision and can be associated with flashes of light in vision. Ocular signs observed include a vitreous floater or Weiss ring and may also be associated with a retinal or vitreal hemorrhage, vitreomacular traction, and retinal breaks or detachments.

The presentation of the patient's right eye suggested a diagnosis of a complete Posterior Vitreous Detachment based on the patient's symptoms of a sudden onset of a floater or blurred spot that changed positions, as well as flashes of light and the signs noted of a Weiss ring in the right eye. Vitritis was ruled out as the patient did not demonstrate any vitreous haze or cells. A retinal detachment was eliminated as no break, tear, or detachment was noted with scleral depression. However, this differential diagnosis would remain during the two to six week observation period. A vitreous hemorrhage was excluded as the vitreous was clear of any blood and a clear view of the fundus was obtainable. Lastly, a migraine aura was ruled out as the patient did not present with an associated migraine headache and did not describe the classic aura photopsia.

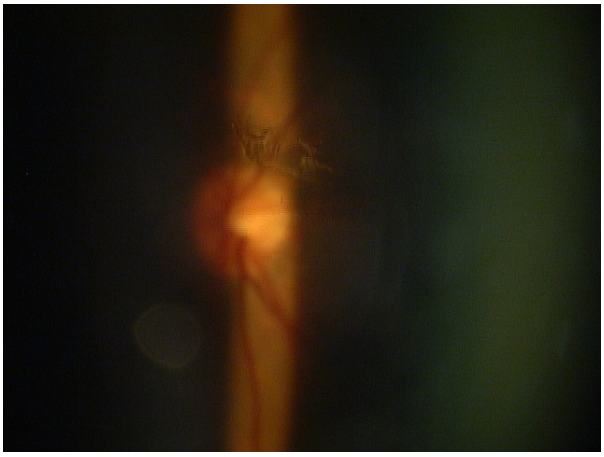

Figure 1 and 2

Anterior segment photograph of the Weiss ring noted in the right eye in 16X magnification using a 90D lens and slit lamp. Figure 2 demonstrates that the position of the Weiss ring changed location as the patient blinked or moved her eye.

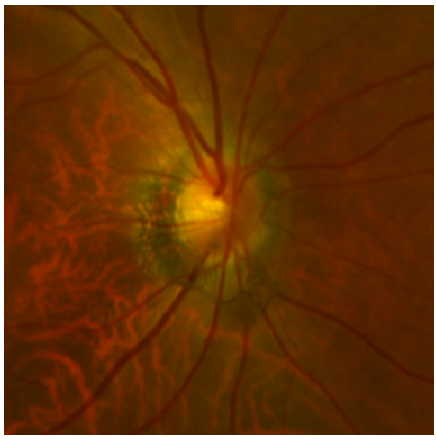

Figure 3

Posterior segment photograph of the retinal hemorrhage located temporal to the optic nerve head within the peripapillary zone.

The patient's final spectacle prescription was finalized. She was educated regarding the change in prescription compared to her habitual prescription. Also, she was educated regarding the capped Meibomian gland noted on her right eyelid. It was recommended that she begin lid hygiene with lid scrubs as well as warm compresses.

The patient was thoroughly educated and provided an educational handout regarding the exam findings of a posterior vitreous detachment, its etiology, and the associated treatment plan. The signs and symptoms of the PVD were reviewed, including that the PVD will be visible and more noticeable for the first few months, especially outside in bright light, against the blue sky, or solid, lightly colored walls. Also, the patient was educated regarding the risk of a retinal break, tear, or detachment following the PVD and was further educated on the signs and symptoms of a retinal detachment. The patient was advised to immediately report any change in symptoms and return to the clinic if noted. The after-hours and emergency phone number was provided to the patient. The patient was instructed to return to the clinic in three weeks, approximately four weeks following the onset of her symptomatic PVD to monitor further for any retinal break, tear, or detachment.

Follow Up Visit (04/20/2018)

The patient returned to the clinic four weeks later for a vitreous and retinal evaluation. She reported no new symptoms or recent flashes of light. Spectacle corrected distance visual acuity was $20/25^{-1}$ OD and $20/20^{-1}$ OS. Spectacle corrected near visual acuity was 20/20 OD and 20/20 OS. Confrontation visual fields were full to finger counting in both eyes. Extraocular muscle testing showed full range of motion without pain and diplopia. Pupils were equal, round and reactive to light with no afferent pupillary defect noted. Slit lamp biomicroscopy revealed normal adnexae, lids, lashes, puncta, and palpebral and bulbar conjunctiva in both eyes. The corneas were intact and clear through all layers in both eyes. Both irides were flat and brown in both eyes. The

anterior chambers appeared deep and quiet with open angles estimated at 4+ using the Von Herrick method. One drop of Benoxinate 0.4%/Fluorescein 0.25% (Fluress) was instilled in both eyes prior to intraocular pressure measurements. Goldmann applanation tonometry measured 9 mmHg OD, 9 mmHg OS at 9:37 A.M. The patient was dilated using one drop of Mydriacyl 1%, and one drop of Phenylephrine 2.5% in both eyes at 9:45 A.M. Upon full dilation, an evaluation of the posterior segment by slit lamp with 90D lens and by Binocular Indirect Ophthalmoscope with 20D lens was performed. Grade 1+ nuclear sclerosis was noted in the media of the lenses in both eyes. Assessment of the fundus revealed the optic nerve heads were well perfused with C/D ratios of 0.25/0.25 in both eyes. The right eye had peripapillary atrophy noted in the beta and alpha zones circumferentially. A flat and intact macula was noted in both eyes. The right eye revealed a small hemorrhage within the temporal peripapillary zone that had resolved by 50%. Retina and retinal vasculature was flat and intact with no pathology noted in left eye. Thorough evaluation of the vitreous demonstrated a Weiss ring overlying the optic nerve head as well as an aggregation of collagen fibrils noted in the vitreous cavity inferiorly in the right eye and syneresis of the vitreous in the left eye. No vitreous hemorrhage or pigment, Schaeffer's Sign, was noted in the right eye. Scleral depression was ordered at-this-time to evaluate and monitor the retina for any breaks, tears, or detachments. Scleral depression was performed with a scleral depressor and Binocular Indirect Ophthalmoscope with 20D lens without signs for breaks, tears, or detachments.

Again, the patient was thoroughly educated regarding the exam findings of a posterior vitreous detachment, its etiology, and the associated treatment plan. Also, the patient was educated regarding the risk of a retinal break, tear, or detachment following the PVD and was further educated on the signs and symptoms of a retinal detachment. The patient was advised to immediately report any change in symptoms and return to the clinic if noted. Further treatment options along with their benefits and risks were discussed with the patient at this visit, including pars plana vitrectomy and Neodymium-doped Yttrium Aluminum Garnet (Nd:YAG) laser vitreolysis. The patient reported she did not wish to proceed with either procedure at this time, but would notify the clinic if her symptoms were bothersome enough for her to consult a surgeon. The patient now was at the end of the critical period for risk of retinal tear or detachment. Therefore, she was recommended to schedule her yearly comprehensive examination in March of 2019. Little concern remained regarding the minute, resolving retinal hemorrhage as it was resolving as expected with no associated side effects and no vitreous traction remained around the optic nerve head as a complete PVD or Weiss ring was observed. The patient was advised to return to the clinic if any new symptoms began.

Discussion

The vitreous forms strong adhesions to the structures it surrounds. The firmest attachment is located at the vitreous base, located two millimeters anterior to the ora serrata.[3,4] Additional adhesions are located at the posterior lens, the optic disc, the macula, and the retinal vessels.[2,3,4] Two types of degeneration can occur within the vitreous consisting of synchysis and syneresis. Due to aging, the gel volume decreases

while the watery volume increases creating areas of liquefaction, called lacunae. This process of redistribution of collagen is called synchysis.[3,4] Next, the vitreous may also change structurally by collapsing due to collagen aggregation.[3,4] This process of the vitreous shrinking is called syneresis. As the configuration of the vitreous changes, the vitreous may be affected by traction. This traction can then result into a posterior vitreous detachment where the glial tissue is pulled away with the vitreous with the outcome of a Weiss ring, or circular area of condensation.[3,4] The pathognomonic sign for a PVD is the presence of the Weiss ring over the optic nerve head.[3] Posterior vitreous detachments may be classified as localized, partial, or complete depending on how much traction and detachment has occurred thus far.

Due to the bothersome nature of a PVD, research has been performed to evaluate the affect vitreous degenerations have on patients' quality of life. Literature found a correlation with floaters and a negative impact on life.[5,6] Further, younger patients reported they would accept a 7% chance of blindness to eliminate themselves of floaters.[6] Visual acuity, straylight glare, and contrast sensitivity have also been studied regarding the effects from vitreal floaters. Straylight, also known as the perceived glare around light, was found in patients with PVD's and present with symptoms of fuzzy or hazy vision as well as challenges driving at nighttime.[6] Contrast sensitivity function was discovered to be abnormal in patients with vitreous floaters compared to those without.[5] Lastly, patient symptoms, such as subjective visual reduction, are commonly used to help determine the probability the patient has an associated retinal break, or detachment. Subjective visual reduction has been found to parallel findings of vitreous hemorrhage, vitreous pigment, and, therefore, a potential for an underlying retinal break.[7,8] Investigators report that the risk of a complicated PVD is approximately 45% when the patient experiences subjective complaints of vision reduction.[7]

A complete and comprehensive evaluation of the retina is essential in cases with a new onset of a symptomatic PVD. First and foremost, a dilated examination must be completed. Examination with a 90D lens and slit lamp biomicroscopy is the first step that allows analysis and viewing of the vitreous, posterior pole, and mid-peripheral retina with high magnification.[9] Additionally, Goldmann 3-Mirror with biomicroscopy enables magnified views of the peripheral retina, however, it is not a dynamic view.[9] Scleral depression has been viewed as the gold standard when retinal breaks, tears, and detachments must be ruled out.[9,10] Scleral depression provides an evaluation of the presence of sub-retinal fluid while assessing the peripheral retinal with a more dynamic view.[9,10] Several barriers may exist with the use of scleral depression, including the challenging nature of the technique to perfect and utilize in the clinical setting, the time consuming nature of the clinical skill, and the poor comfort or pain the patient experiences.[9,10] As of recent, literature has utilized Optical Coherence Tomography (OCT) as a clinical tool to standardize the diagnosis of a PVD.[1,11] Montaging OCT images together have provided vast information regarding the vitreous, vitreoretinal interface, and location of where the PVD first occurs.[11]

Posterior vitreous detachments are commonly linked to the aging process and are said to be a consequence of age.[2,7] Statistics show that 65% of patients over the age of 65

years old will have developed a PVD.[2] Additionally, the prevalence of a PVD exponentially increases with increasing age. For example, patients in their fifties have a 25% of having a PVD while patients in their eighties have a 90% chance.[7] Studies, particularly the OCT studies discussed above, have determined that partial posterior vitreous detachments can begin as early as the fourth decade of life.[3] Furthermore, women have been found to have a higher prevalence of PVD at a younger age compared to men.[3] Literature correlates this to decreased hyaluronic acid synthesis due to decreased estrogen levels after menopause.[3]

Predisposing risk factors for the development of a posterior vitreous detachment include myopia, trauma, inflammation, surgery, and inherited vitreoretinal disease.[2] Investigators have noted that PVDs occur a decade earlier in patients with high myopia of -6.00 D and greater.[7] Moreover, subsequent, symptomatic retinal tears are more frequently diagnosed in myopia patients.[12] Associated findings correlated with a posterior vitreous detachment depend on the site of vitreoretinal traction and detachment. Traction on the retinal vasculature can result in a retinal hemorrhage or a vitreous hemorrhage, traction on the macula may develop into vitreomacular traction, and traction associated with the peripheral retina can lead to retinal tears.[2] Retinal tears or detachments comprise of the most vision threatening risk factor associated with a PVD. Retinal tears have been diagnosed clinically in 8-15% of presenting PVDs.[12] The risk of retinal tear increases by 62% with a concomitant vitreous hemorrhage and by 88% with associated vitreous pigment dusting, also known as Schaeffer's sign.[7] Most commonly a subsequent retinal tear will be located in the superotemporal quadrant.[12] The literature reports that the majority of retinal tears correlated with a symptomatic posterior vitreous detachment are observed two to six weeks following the initial symptoms, including new onset of flashes of light or floaters.[12] Due to this, it is essential to monitor the patient closely during this time.

Current treatment options for posterior vitreous detachment include observation, pars plana vitrectomy, and neodymium-doped yttrium aluminum garnet (Nd:YAG) laser vitreolysis. Classically, the treatment plan of choice has been observation as physicians have been reluctant to provide surgical options due to the risk to benefit ratio.[13] Stable, long-standing PVD's are perceived as an inconsequential ocular finding, therefore, performing a major ocular surgery for this condition creates a challenging scenario for surgeons.[13] A vitrectomy is a surgical procedure that removes the vitreous from the eye, thus, removing the symptomatic PVD. Micro-incision vitrectomy procedures are minimally invasive and now being used very effectively with a lower risk assessment.[13] Research shows that a small 27-gauge vitrectomy is well-tolerated by patients and has provided vast improvement to their symptoms, vision, contrast sensitivity, and overall quality of life.[6,13] Some risk is associated with this procedure, including post-operative cataracts, post-operative glaucoma, retinal tears, cystoid macular edema, macular holes, and epiretinal membranes.[13] Lastly, Nd:YAG laser vitreolysis is a procedure where the vitreous opacities are vaporized with the use of a laser. There is limited published data regarding the safety and efficacy regarding this procedure. In addition, the exact risk or side-effect profile is still being explored.[14] However, it shows promising potential as a less invasive way to improve patients' quality of life. The limited literature

reveals the potential risks of vitreolysis as being retinal holes, vitreous hemorrhages, elevated intraocular pressure, and secondary cataract formation.[14,15] As with any procedure, there are limitations to vitreolysis, such as inaccessible floaters or the inability to treat floaters due to their positioning within the vitreous.

Conclusion

In summary, when a posterior vitreous detachment is diagnosed, it is essential that a thorough dilated fundus examination with scleral depression is completed due to the risk factors associated with a new onset of PVD, including retinal or vitreous hemorrhage, vitreomacular traction, or retinal tear or detachment. It is beneficial to know statistical data regarding future risks as listed above so that patients may be educated regarding the importance of further follow up. Also, the risks and benefits of the surgical treatment procedures rather than observation only are essential to patient education due to the affect posterior vitreous detachments have on patient's quality of life, contrast sensitivity, straylight glare, and vision level.

Bibliography

1. Fincham GS, James S, Spickett C, Hollingshead M, Thrasivoulou C, Poulson AV, McNinch A, Richards A, Snead D, Limb GA, Snead MP. Posterior Vitreous Detachment and the Posterior Hyaloid Membrane. *Ophthalmology.* 2018;125(2):227-236.

2. Schechtman DL, Calderon DE. Posterior Vitreous Detachment: A Common Process with Potential for Ocular Morbidity. *Review of Optometry.* 2008;1:11-16.

3. Gurwood AS. Will this Floater Sink? *Review of Optometry.* 2014;1:90.

4. Remington LA. Aqueous and Vitreous Humors. Clinical Anatomy and Physiology of the Visual System. Third Edition. Butterworth-Heinemann, an imprint of Elsevier Inc. 2012:109-122.

5. Garcia GA, Khoshnevis M, Yee KMP, Nguyen-Cuu J, Nguyen JH, Sebag J. Degradation of Contrast Sensitivity Function Following Posterior Vitreous Detachment. *American Journal of Ophthalmology.* 2016;172:7-12

6. Milston R, Madigan MC, Sebag J. Vitreous floaters: Etiology, diagnostics, and management. *Survey of Ophthalmology.* 2016;61(2):211-227.

7. Mangan R. Urgent Flashes and Floaters. *Review of Optometry.* 2016;4:30-33.

8. Hurst J, Johnson D, Law C, Schweitzer K, Sharma S. Value of subjective visual reduction in patients with acute-onset floaters and flashes. *Canadian Journal of Ophthalmology.* 2015;50(3):265-268.

9. Arjmand P, Hurley B. Flashes and floaters: a survey of Canadian ophthalmology residents' practice patterns. *Canadian Journal of Ophthalmology.* 2017;52(5):453-457.

10. Shukla SY, Batra NN, Ittiara ST, Hariprasad SM. Reassessment of Scleral Depression in the Clinical Setting. *Ophthalmology.* 2015;122(11):2360-2361.

11. Tsukahara M, Mori K, GehlbachPL, Mori K. Posterior Vitreous Detachment as Observed by Wide-Angle OCT Imaging. *Ophthalmology.* 2018;6420(17):33234-33237.

12. Crim N, Esposito E, Monti R, Correa LJ, Serra HM, Urrets-Zavalia JA. Myopia is a risk factor for subsequent retinal tears in the course of a symptomatic posterior vitreous detachment. *BMC Ophthalmology.* 2017;17(1):226

13. Sommerville DN. Vitrectomy of vitreous floaters: analysis of the benefits and risks. *Current Opinion in Ophthalmology.* 2015;26(3):173-176.

14. O'Day R, Cugley D, Chen C, Fabinyi D. Bilateral posterior capsule injury after Nd:YAG laser vitreolysis: unintended consequence of floaters treatment. *Clinical and Experimental Ophthalmology.* 2018.

15. Koo EH, Haddock LJ, Bhardwaj N, Fortun JA. Cataracts induced by neodymium-yttrium-aluminum-garnet laser lysis of vitreous floaters. *British Journal of Ophthalmology.* 2017;101(6):709-711.