Vitreous Opacities: Diagnosis, Classification, and Management Options

BY PAULO EDUARDO STANGA, MD, AND URSULA REINSTEIN

T o understand why vitreous opacities occur, it is crucial to have knowledge of the anatomy of the vitreous. It is a gel-like substance that consists of water, extracellular matrix, and parallel nonbranching collagen fibers that are naturally arranged in an anteroposterior direction. The vitreous cortex is a peripheral shell of densely packed collagen fibrils.1-3 Hyaluronan (HA) which fills the gaps between the collagen fibrils and allows the gel to remain in its native state, is denser in the vitreous cortex. There is no direct connection between the posterior vitreous and the retina. The vitreous cortex is, however, adherent to the internal limiting membrane (ILM) and most firmly attached where the ILM is the thinnest, along the vitreous base, optic disc, macula, and retinal blood vessels.4

The physiological function of the vitreous is unclear. What we do know, however, is that it helps to regulate eye growth and shape.5-7 It creates a barrier to the cellular migration and diffusion of large macromolecules to maintain transparency, and it lowers oxygen concentration in the lens.8,9

VITREOUS OPACITIES AND FLOATERS: AN AGE-RELATED CHANGE INFLUENCED BY MYOPIA

As the eye ages, the vitreous becomes more fibrillar, type IX collagen in the vitreous is lost, and the aggregation of collagen fibrils leads to liquefaction. The packed bundles of collagen fibrils form visible fibers that become more numerous, thicker, and more irregular with increasing age. By the age of 40, the firm connection between HA and collagen fibrils loosens. By the age of 80, roughly 62% of the vitreous has liquefied, and the risk for posterior vitreous detachment (PVD) increases (Figure 1).

In the 1970s, Eisner and Worst described the presence of membranelles and cisterns, respectively, in the vitreous, and in the 1980s Sebag and Balazs identified fibers in the vitreous.10-12 About 10 years later, Kishi and Shimizu described the formation of pockets in the vitreous.13 Vitreous floaters present as cloud-like or linear shadows or spots that move with eye and head movements.14

Vitreous opacities can also result from the separation between the vitreous cortex and the retinal surface, along with the anterior displacement of the posterior vitreous cortex. They also result from the high density of the collagen fibers in the vitreous cortex, which causes light to scatter.15

A sudden onset of vitreous opacities is usually associated with acute PVD. Risk factors for PVD include aging, high myopia, and lens surgery. Furthermore, PVDs occur more often in older women compared to younger women and men due to the loss of estrogen associated with menopause.16 An acute PVD can induce a sudden onset of floaters or a cloud-like floating opacity.

Eyes with myopic vitreopathy are at increased risk for vitreous opacities as structural changes start at an earlier age compared to emmetropic eyes. PVD can occur between 10 to 13 years earlier in high myopic versus emmetropic eyes. As the HA molecules dissociate from the collagen fibers, the fibers form bundles that are seen as floaters.17

Vitreous opacities are more visible against a light or bright background. This is problematic today because of the increasing use of backlit screens.

About 44% of patients with myopic vitreopathy present with clinically significant floaters. The number of individuals with myopia is steadily increasing. By 2050, nearly 5 billion people will present with myopia, and 1 billion people will suffer from high myopia.18

DIAGNOSIS OF PVD

The observation of a Weiss Ring on biomicroscopy is confirmation of posterior pole PVD. However, it may not indicate whether the PVD is partial or complete. In addition, Weiss rings cannot always be visualized, especially as they may disintegrate or migrate into the periphery.

We (Stanga et al.) first described the use of swept-source OCT to visualize in-vivo anatomical features in the cortical vitreous such as the Bursa Premacularis and Space of Martegiani as well as the vitreoretinal interface.19 PVD can be objectively diagnosed using ultrasound, which allows visualization along the entire axis of the eye, or with swept-source OCT.20

Figure 1. The eye as an embryo (A), in middle age (B), and in old age (C) (Reprinted from Sebag J, Balazs EA. Morphology and ultrastructure of human vitreous fibers. Invest Ophthalmol Vis Sci. 1989;30:1867-1870).
PVD can start in the posterior pole or in the periphery. PVD can occur acutely, with a sudden onset of vitreous opacities and/or floaters created by the shadow cast by the cortical vitreous and/or the Weiss ring. Patients often describe a “moving cloud” in front of their eyes or “bothersome floaters.” In eyes with PVD, the spherical posterior vitreous surface becomes smaller, forming surface folds that may increase light scattering, thus contributing to the reduced quality of vision.

**VISION-DEGRADING MYODESOPSIA AND TREATMENT OF VITREOUS OPACITIES AND FLOATERS**

Vitreous opacities can significantly impact vision. As the size and number of vitreous floaters increases, so do the symptoms associated with this visual phenomenon, which include diminished contrast sensitivity and increased stray light (Figure 2).  

Many patients who experience vitreous opacities and floaters present for treatment. In our experience, the higher the education level of the patient, the more likely patients are to pursue treatment to increase quality of life. If left untreated, some patients can even experience varying levels of depression and/or anxiety.

**MEDICAL INTERVENTION**

Medical intervention is often ignored and brushed off as a nuisance. Unfortunately, until today most patients continue to be
told that no treatment is available and that they must learn to live with their symptoms. Today, this is far from correct.

There are several reasons to account for this incorrect line of thinking, including the following:

- Vitreous opacities are not always visible to the examiner, meaning that they are therefore undiagnosed and the symptoms dismissed.
- The impact of vitreous opacities is still not well understood, and we still have difficulty to correlate number, size and degree of vitreous opacities and floaters to the symptoms and need of treatment.
- Better vitreous visualization technologies and techniques must become available to enhance our ability to better diagnose and understand the impact on vision of both vitreous opacities and PVD.

It is therefore imperative that we urgently educate patients about vitreous opacities and floaters and the risk, even if low, of retinal tears associated with an acute PVD and the need for an urgent vitreoretinal examination by a retinal specialist that includes indirect ophthalmoscopy with 360° scleral indentation through fully dilated pupils. This is the only way to examine the retina up to the ora serrata.

We must continue to be objective and proactive when diagnosing a PVD, incorporate ultrasonography and swept-source OCT as part of the routine examination of symptomatic patients, and be aware that a sudden onset of vitreous opacities does not automatically mean the presence of a PVD.

**CASE EXAMPLE**

A PVD can have an impact on the development and intensity of symptoms associated with vitreous opacities. The following case example illustrates this point:

A 57-year-old artist presented with symptoms of acute floaters in the right eye. On examination, cross-sectional 23 mm single-scan widefield OCT confirmed a PVD and a Weiss ring (Figure 3A). A 3D rendering of the widefield OCT scan clearly showed the detached posterior cortical vitreous (Figure 3B). Creases were visible in the posterior cortical vitreous, as was the Weiss ring.

We also used a new imaging technique (IP in process) that we are developing in conjunction with an imaging company which in this instance showed quite clearly that the patient was suffering from vision-degrading myodesopsia. This is a condition where the vitreous floaters are drifting in front of the macula as the eye moves thus significantly impacting the patient’s visual field at a distressing level.

We used the same visualization modalities to examine the patient’s asymptomatic left eye. There were no signs of PVD, and the vitreous appeared clear (Figures 4A and 4B). The difference in perception of symptoms between the two eyes of this patient is that the right eye shows a PVD whereas the asymptomatic eye does not.

**TREATMENT OPTIONS**

**Nd:YAG Laser Vitreolysis.** This treatment option for vitreous opacities has been available for several years. It has, however, been used mainly by anterior segment surgeons rather than retinal specialists. The main reason is that, historically, Nd:YAG laser technologies were developed for posterior capsulotomy rather than for the treatment of vitreous floaters.

Most recently, however, advances in Nd:YAG laser technologies have created the potential for Nd:YAG laser vitreolysis to be safer and more efficient. One such technology is the Ultra Q Reflex with True Coaxial Illumination (Ellex; Figure 5), which provides on- and off-axis visualization beyond the posterior capsule in both the anterior and posterior vitreous. It is therefore possible with this technology to treat both posterior and anterior vitreous floaters.

In this laser technology, the beams of light and laser follow the same path, allowing us to visualize and target vitreous floaters located deeper or in the more posterior vitreous. With the Ultra Q Reflex laser, we can not only target and disrupt vitreous opacities by vaporizing or breaking them up into smaller and less symptomatic ones but also target strands of collagen fibers attached to vitreous floaters, which sometimes allow the vitreous floaters to displace away from the visual axis.

The Ultra Q Reflex laser has a very short pulse width (nanoseconds) and a small spot size (microns) to cut and vaporize vitreous...
floaters at energies between 3 and 5 mJ, as per our use. The treatment should not be used, however, if the opacity is too close to the retina, and a 3-mm safety distance should be maintained between the focal point of the treatment and the lens to avoid collateral damage. The use of a posterior offset setting for vitreous floaters in the anterior vitreous is also recommended to reduce the risk of lens damage. It is also recommended to use an anterior offset setting when targeting vitreous floaters close to the retina. Furthermore, it is essential to ensure prior to every treatment session that there are no peripheral retinal lesions that could expose the eye to retinal detachment. Indirect ophthalmoscopy with 360º scleral indentation through fully dilated pupils is the only way to achieve this. It is also important to discuss with patients that some may require more than one laser session to achieve the desired reduction in symptoms.22

**Limited Pars Plana Vitrectomy.** Another treatment option for vitreous opacities, floaters and vision-degrading myodesopsia is limited pars plana vitrectomy.23 In a nutshell, this surgical technique is different from the traditional full vitrectomy in that we do not actively induce an intraoperative PVD nor remove the anterior and peripheral vitreous.

**CONCLUSION**

In our experience, Nd:YAG laser vitreolysis for vitreous floaters using the Ultra Q Reflex is a safe and effective nonsurgical treatment option. It is ideal for the alleviation of symptoms secondary to a Weiss ring or to a few and well-defined centrally located vitreous floaters.

We must continue to work together to improve the visualization and diagnosis of vitreous opacities and floaters through better imaging.

It is also critical to educate the public and patients about vitreous floaters and acute PVD and its risks, as well as not to dismiss the symptoms and incapacitation that can be caused by vitreous opacities and floaters.

Nd:YAG laser vitreolysis may not be the best option for every patient, but we should certainly have the capacity to present this treatment option to our patients.

---


PAULO EDUARDO STANZA, MD
■ Director, The Retina Clinic London, United Kingdom
■ Retina Lead and Vitreoretinal Surgeon, London Vision Clinic, United Kingdom
■ Professor of Ophthalmology, Institute of Ophthalmology, University College of London, United Kingdom
■ p.stanza@theretinacliniclondon.com
■ Financial disclosures: Consultant and unrestricted research grant (Lumibird Medical)

Lumibird Medical: Provision of Tango Reflex™ YAG Laser (Ellex) and Absolu™ Ultrasound units (Quantel Medical).

URSULA REINSTEIN
■ Retina Research Associate, The Retina Clinic London, United Kingdom
■ Financial disclosure: None

DISCLAIMER:
This article is intended for EU readers. It is not intended for readers in the following regions: US, Canada.