Reassessing the Surgical Treatment of Floaters

Pars plana vitrectomy remains the safest and best approach.

BY J SEBAG, MD, FACS, FRCOphth, FARVO

Age-related changes in vitreous were first reported in 1917,[1] and 12 years later Duke-Elder theorized that the liquefaction process may result in aggregation of residual proteins.[2] The resulting phenomenon of floaters remains a complex if underappreciated problem to the current day.

The healthy transparent state of the vitreous body (Figure 1) results from hyaluronan keeping collagen fibrils separated to allow light transmission with minimal to no scattering. Over time, hyaluronan dissociates from the collagen, allowing the latter to crosslink and aggregate,[3-5] ultimately forming fibrous structures that course from the vitreous base to the posterior pole (Figure 2A). This process is accelerated in myopia, resulting in a destabilized vitreous body with crosslinked collagen fibers and liquefaction of the gel. One specific type of collagen, type IX, diminishes with age, further contributing to the liquefaction process,[6] with the end result an increase in the volume of liquefied spaces[7] and an increase in optically dense areas[8] in both age-related vitreous degeneration (Figure 2B) and myopic vitreopathy.

Because floaters are widespread in myopia and commonly result from the natural aging process especially after posterior vitreous detachment (PVD), they are often considered a benign problem and not a disease. Thus, patients are told that they just have to adjust. Studies are beginning to indicate otherwise, however, and vitreoretinal surgeons currently have an effective and relatively safe means to address floaters: pars plana vitrectomy (PPV).

ETIOLOGY AND CLINICAL CHARACTERISTICS

Floaters result from light scattering by structures within the vitreous body as well as from the posterior vitreous cortex following PVD (Figure 3). They can be caused by exogenous sources (ie, hemorrhage, inflammation), but the natural aging process of the eye is a significant contributor to this phenomenon. As vitreous liquefies with age (a process resulting from the aforementioned molecular rearrangement of hyaluronan and collagen that actually begins in childhood), vitreous collagen fibrils crosslink and aggregate to form linear and focal opacities that can interfere with photon transmission to the retina.

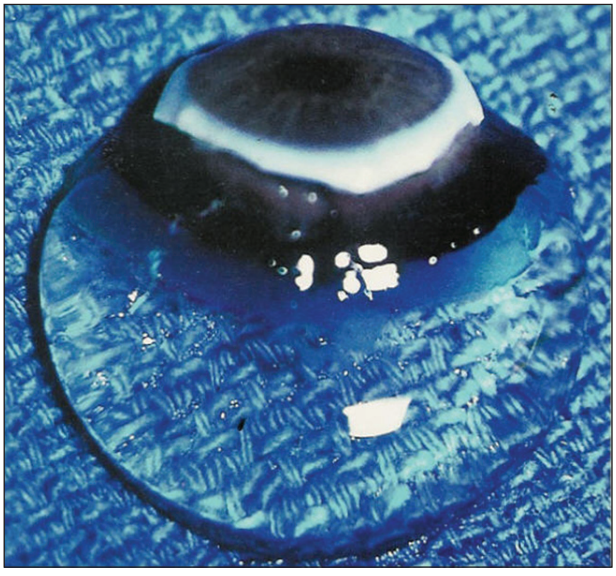


Figure 1. Vitreous body from a 9-month-old human, dissected of the sclera, choroid, and retina but still attached to the anterior segment. [Cover Photo: Sebag J. The Vitreous – Structure, Function, and Pathobiology. Springer-Verlag: New York, 1989.]

A concurrent phenomenon with vitreous liquefaction due to the aging process is weakening at the interface of the posterior vitreous cortex and the inner limiting membrane (ILM) of the retina.[9,10] When posterior vitreous detachment occurs, the posterior vitreous cortex is no longer anchored to the retina and moves about in the center of the eye with head or eye movement, creating moving shadows that are perceived by patients as floaters.

The prognosis of vitreous liquefaction is more guarded in patients with certain risk factors. All individuals with

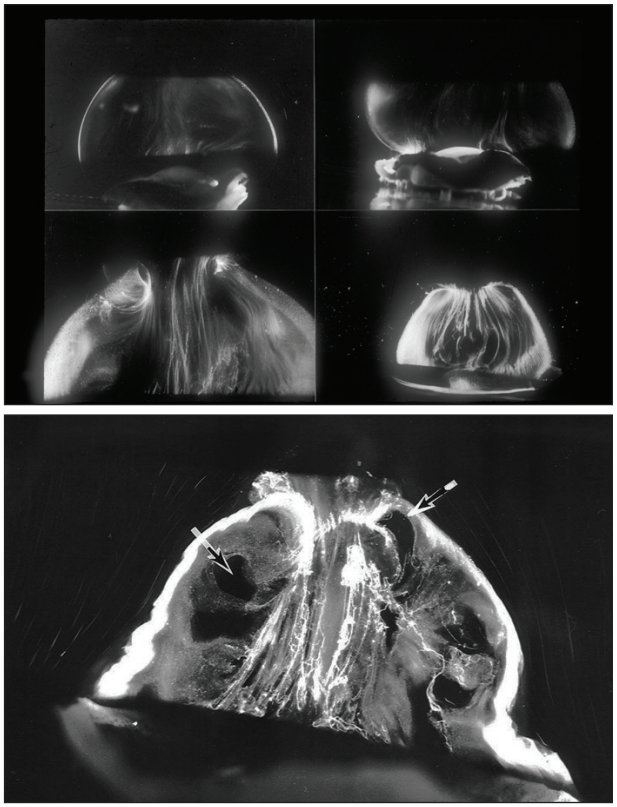
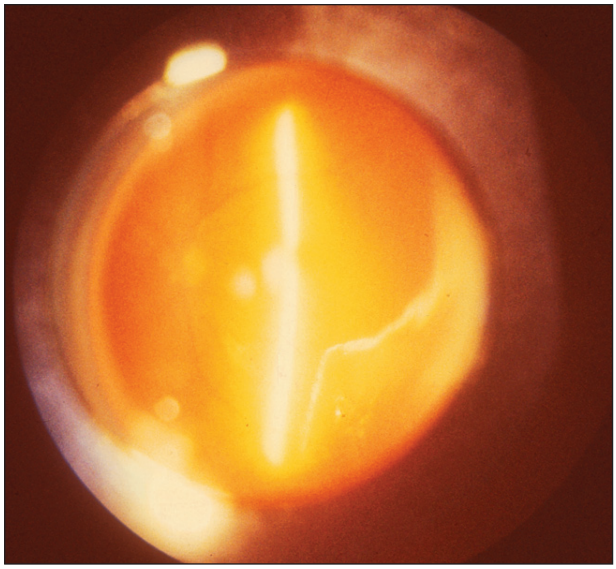


Figure 2. (A) Dark-field slit microscopy of human vitreous structure in youth and middle-age. The anterior segment is at the bottom and the posterior pole is at the top of these images. The top 2 images are from children aged 6 (top left) and 11 (top right) years. Apart from the peripheral vitreous cortex, which is known to have a high density of collagen fibrils, there is little or no light scattering in the vitreous body. The images in the lower panel are from humans 56 (lower left) and 58 (lower right) years of age. There are visible fibers with an anteroposterior orientation coursing from the vitreous base (lower right area of lower right image) to the posterior pole. (B) Dark-field slit microscopy of vitreous structure in an 88-year-old woman. Aggregation and tortuosity of vitreous fibers with adjacent pockets of liquid vitreous (arrows) called lacunae can be seen. [From Sebag J. The Vitreous – Structure, Function, and Pathobiology. Springer-Verlag: New York, 1989.]

axial myopia and postmenopausal women are predisposed to a higher incidence of PVD.[10] Indeed, myopic vitreopathy is associated with PVD some 10 to 15 years earlier in life than in emmetropes.[10] The most commonly reported etiologies associated with floaters are PVD, myopic vitreopathy, and asteroid hyalosis.[11-13] Marfan syndrome, Ehlers-Danlos syndrome, and diabetic vitreopathy are also known to be associated with the aggregation of vitreous collagen fibers that results in glare due to light scatter.[3,9,10,12]



[Courtesy of Clement Trempe, MD]

Figure 3. Preset lens biomicroscopy image of posterior vitreous detachment.

Symptoms associated with floaters may include the description of gray, linear, hair-like structures with round points that appear more prominent against bright backgrounds (a white wall or clear sky), translucent strings, or "spider web-like" images that are more prominently noticed during head or eye movement. The physical presence of inhomogeneities in the vitreous body can usually be appreciated clinically via ultrasonography.[14-16] Dynamic light scattering, a laser-based nanodetection method for measuring particles in the cornea, lens, aqueous, and vitreous, has been tested as a way to detect the earliest changes of diabetic vitreopathy,[10] myopic vitreopathy,[12] and age-related vitreous degeneration.[17,18] Use of ultrasonography in this indication is not limited by media opacity,[19] and studies are ongoing at Columbia University and the Riverside Research Institute to quantify ultrasound evaluation and provide an objective clinical index of the structural abnormalities causing floaters.

RECONSIDERING THE CLINICAL SIGNIFICANCE OF FLOATERS

Patient complaints of floaters are often dismissed or downplayed. Because floaters most often result from the body's own aging process, there may be a perception that this inevitability simply requires the patient to neurally adapt to a new normal. Yet studies indicate that floaters are a more insidious condition than is often appreciated. A survey by Waggle and associates indicated that the deleterious effect on quality of life as a result of floaters is comparable to or worse than that of age-related macular degeneration, diabetic retinopathy, or

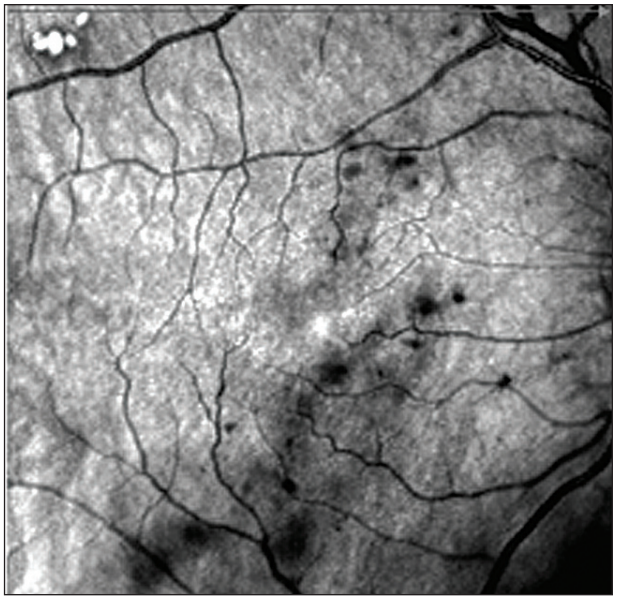


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Figure 4. Scanning laser ophthalmoscope imaging of focal vitreous floaters.

glaucoma.[20] The scores indicating disruption to quality of life due to floaters were more severe even than those for mild angina, mild stroke, colon cancer, or asymptomatic HIV infection. These survey findings add perspective to patient reports of symptomatic floaters.

No further proof of the impact of floaters is needed than to consider the lengths to which some patients will go to get treatment. Despite a lack of evidence for its efficacy, some patients are willing to accept the risks associated with Nd:YAG laser vitreolysis in hope that it offers relief. Furthermore, as this is an unproven treatment, it is not covered by Medicare or third-party payers, and patients must bear the out-of-pocket expense.

Although there is no literature objectively quantifying the disruption of visual acuity secondary to floaters, a physical object in the vitreous body will block photon transmission and generate light scatter with a variety of untoward effects. One such phenomenon is known as straylight, sometimes described as disability glare. Patients experiencing straylight may report symptoms of hazy vision, decreased color, difficulty recognizing faces, and glare. It is not hard to appreciate how these may contribute to lower quality of life and quality of vision. Moreover, light scatter causes a decrease in contrast sensitivity, which causes difficulty with judging distances, driving at night, and mobility.[21] In a study using a validated measure of contrast sensitivity function, patients with bothersome floaters were found to have a 67% reduction in function compared with age-matched controls.[22]

The degradation of contrast sensitivity explains why patients with floaters are unhappy and complain bitterly to a heretofore unmoved medical profession. However, with this new understanding of how vision is affected by floaters, and with the ability to quantify these effects, clinicians now have the ability to change the management of floaters for the better.

THERAPEUTIC MANAGEMENT

One proposed therapeutic approach to managing eyes with floaters is to perform vitreolysis with an Nd:YAG laser; however, the efficacy of this application is controversial.[23-25] In a single-center retrospective review of 42 eyes of 31 patients who underwent 54 procedures, Nd:YAG vitreolysis eradicated floater symptoms in less than a third of patients and led to worse symptoms in 7.7%.[21] Furthermore, Nd:YAG laser is ineffective and potentially unsafe for treatment of posteriorly located vitreous opacities.[25]

Vitrectomy provides a definitive cure to floaters, although the surgery is not without risk, albeit quite minimal. Pars plana vitrectomy has been associated with the development of cataracts, endophthalmitis, retinal tears and detachments, glaucoma, vitreoretinal hemorrhage, and macular edema.[26] Given the risk of complications,[27-29] it is reasonable to reserve this option for patients who experience significant visual disturbances resulting from floaters and who understand and are willing to accept the associated risks. It should be noted that modern 25-gauge instrumentation and techniques help reduce the risks of these surgical complications.[22,26,30]

Pars plana vitrectomy can be both effective and safe as a means of resolving floaters. A number of studies demonstrate that successful PPV improves functional measures of straylight and contrast sensitivity. Mura et al showed that measurements of straylight improved after vitrectomy, and patients experienced improvements in facial recognition and glare hindrance.[31] In a prospective series of 16 cases, measurement of contrast sensitivity function normalized 1 week following minimally invasive vitrectomy and remained stable through 3 to 9 months of follow-up.[7] In a continuation of this study after publication, the results have been duplicated in an additional 30 patients (J.S. unpublished data). In addition to these clinical results, several studies indicate a significant improvement in patient quality of life following PPV to address floaters.[22,32-37]

Delaney et al reported complete resolution of floater-related symptoms in 93.3% of patients after PPV in a series of 42 eyes of 31 patients.[8] Complications of note in this series included postoperative cataract development that was later resolved with successful phaco-

emulsification surgery, and postoperative retinal detachment occurring 7 weeks after a combined vitrectomy and phacoemulsification. Another study reported an overall low rate of retinal detachment following PPV that increased slightly during long-term follow up.[38] Nevertheless, these and other studies indicate a small but not insignificant rate of complications associated with PPV. It is the physician's responsibility to educate patients about these and all potential complications associated with surgery.

That said, the author's own experience in over 100 consecutive cases has encountered no cases of endophthalmitis or retinal detachment. One vitreous hemorrhage cleared spontaneously after 2 weeks. Owing to use of a minimally invasive approach, cataracts formed in 9 of 68 (13%) phakic eyes, which compares very favorably to the reported incidence of 50% to 76%. The mean age was 61 years, and no patients under the age of 53 developed cataracts. It took on average 14 months after PPV before cataract surgery was needed (abstract accepted for presentation at Association for Research in Vision and Ophthalmology Annual Meeting; May 5, 2014; Orlando, FL).

CONCLUSION

The impact of floaters on patients' quality of vision and quality of life can be significant. As such, it is incumbent upon ophthalmic surgeons to offer safe and effective management of this condition. Current evidence supports the use of minimally invasive PPV. Future surgical improvements will make PPV for floaters an even safer and more effective therapeutic approach, while the development of optical methods to counteract or neutralize the visual effects of floaters will provide other options. The application of pharmacologic vitreolysis is intriguing, although not without the potential to worsen floaters in some eyes.[39-50] ■

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