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Major review

Vitreous floaters: Etiology, diagnostics, and management

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ABSTRACT

Vitreous is a hydrated extracellular matrix comprised primarily of water, collagens, and hyaluronan organized into a homogeneously transparent gel. Gel liquefaction results from molecular alterations with dissociation of collagen from hyaluronan and aggregation of collagen fibrils forming fibers that cause light scattering and hence symptomatic floaters, especially in myopia. With aging, gel liquefaction and weakened vitreoretinal adhesion result in posterior vitreous detachment, the most common cause of primary symptomatic floaters arising from the dense collagen matrix of the posterior vitreous cortex. Recent studies indicate that symptomatic floaters are not only more prevalent, but also have a negative impact on the quality of life that is greater than previously appreciated. We review the literature concerning management of symptomatic vitreous floaters, currently either with observation, vitrectomy, or Nd:YAG laser. Published evidence is consistent with a low-risk profile and excellent success rate for floater vitrectomy, particularly with sutureless small gauge instruments and a limited core vitrectomy without PVD induction. Nd:YAG laser treatment of floaters, reported less commonly, claims resolution of floaters ranging between 0% and 100%; however, both peer-reviewed literature and assertions on web-based nonpeer-reviewed laser vitreolysis sites remain to be substantiated, and at present only vitrectomy has proven value. Prospective studies using objective, quantitative outcome measures are required to assess the relative efficacy and safety of these two procedures as well as new therapies such as pharmacologic vitreolysis.

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1. Introduction

The vitreous body is an extracellular matrix that is highly hydrated and relatively acellular except in the periphery where hyalocytes reside in the vitreous cortex. Vitreous consists of 98% water and macromolecules, the most important being collagens and hyaluronan, organized in an exquisitely clear gel (Fig. 1).[7,64] Floaters arise from molecular changes within the vitreous body and at the vitreoretinal interface that occur throughout life and ultimately attain sufficient prominence to alter vitreous structure. Structural changes within the vitreous body can result from inflammation, vitreoretinal dystrophies, myopic[27] and diabetic vitreopathy,[28] but most commonly stem from aging.[93] In addition to altering the internal structure of the vitreous body, aging also weakens vitreoretinal adhesion.

The strength of attachment of the vitreous body to the retina depends on the topographic location. The strongest zone of vitreous attachment is at the vitreous base where there is a relatively higher concentration of collagen and lower concentration of hyaluronan, resulting in a more condensed and solid vitreous consistency.[90] The densely packed basal collagen fibrils align perpendicular to the retina and insert through discontinuities in the inner limiting membrane (ILM) to anchor on Müllerian glia and astroglia, forming an unbreakable adhesion.[26,102] In other regions of the vitreoretinal interface, collagen fibrils are orientated parallel to the ILM. Although the mechanism of attachment here is not completely understood, it is thought to result from interface macromolecules including laminin, fibronectin, and chondroitin as well as heparin sulphate proteoglycans, forming a glue-like adhesion.[32,38,43,65] The presence of different types of collagen may also contribute to this attachment.[63] Vitreoretinal adhesion has been found to be stronger over areas where the ILM is thinner, including the margin of the optic disk (where the ILM of Elschnig may be supported by few astrocytes)[35] over retinal blood vessels, and in a 500 to 1500 μm disk-shaped zone surrounding the fovea.[25,26] The strength of vitreoretinal adhesion may also relate to the thickness of the posterior vitreous cortex, in particular over retinal blood vessels and at the macula where there is a thinner vitreous cortex related to the rarefaction of the collagen fibrils.[32,70]

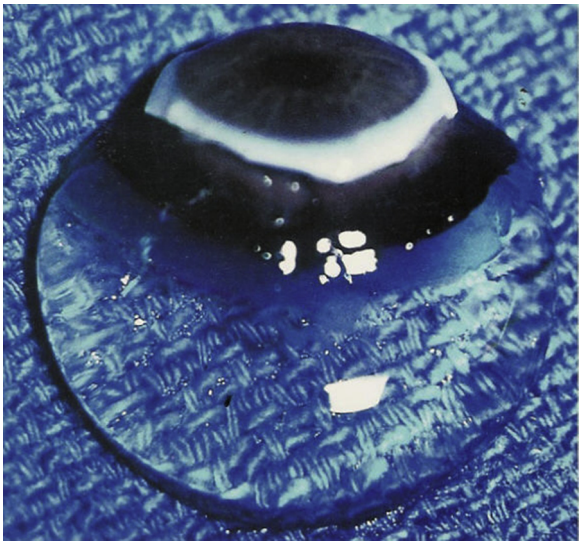

Fig. 1 – Human vitreous body. The vitreous body is attached to the anterior segment with the sclera, choroid, and retina dissected away. This specimen is from a 9-month-old child and thus the vitreous body is a solid gel and maintains its shape in spite of being situated on a surgical towel exposed to room air. (From Sebag J: *The Vitreous—Structure, Function, and Pathobiology*. Springer, New York, 1989; cover photo.)

At birth, the human vitreous body is a colloidal gel; however, with aging, liquefaction occurs within the vitreous body that can subsequently coalesce into pockets, called lacunae or cisterns. Recent swept source optical coherence tomography (OCT) imaging of posterior vitreous structure in young individuals has confirmed the presence of the bursa premacularis originally described by Worst that is unrelated to aging, although cisterns are believed to result from aging. Vitreous gel liquefaction likely results from dissociation of hyaluronan from its association with collagen, allowing cross-linking and aggregation of collagen fibrils (Fig. 2) into macroscopic fibers that scatter incident light (Fig. 3).[70,75,93] Lacunae, on the other hand, are regions devoid of collagen fibrils, owing either to aggregation and displacement of collagen to the periphery of the lacuna, or possibly enzymatic destruction of collagen, transforming the gel vitreous to a liquid consistency,[49] facilitating collapse. Lacunae (Fig. 4) increase vitreous heterogeneity, scatter light (especially at gel-liquid interfaces) and can disturb vision, if severe. Vitreous gel liquefaction increases with age, being first evident at the age of 4 years.[24,45,93]

Posterior vitreous detachment (PVD) occurs as a separation of the posterior vitreous cortex from the ILM of the retina, that begins posteriorly and progresses up to the posterior border of the vitreous base. Johnson[39] describes perifoveal PVD as a slow insidious process until vitreopapillary separation. PVD is a common age-related process caused by a combination of vitreous liquefaction and vitreoretinal dehiscence allowing liquid vitreous to enter through a cortical defect into the retrocortical (preretinal) space,[24] triggering a so-called rhegmatogenous PVD. PVD is more common with increasing age and in postmortem studies is reported at an incidence of 63% by the eighth decade.[24,93,106] There is also purported to be a higher incidence of PVD in older women, which may relate to differences in biochemical composition of vitreous from hormonal changes at menopause.[12,24,93] Risk factors for earlier PVD include myopia and collagen disorders such as Marfan and Stickler syndrome.[83] PVD at younger ages not only induces the phenomenon of floaters, but also, due to firm vitreoretinal adhesion to an irregular posterior vitreous base, results in retinal tears and rhegmatogenous retinal detachment.[77,83]

2. Etiology of vitreous floaters

Floaters, previously called myodesopsia (Greek) and muscae volitantes (Latin), are visual phenomena caused by vitreous