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Review

Ageing of the vitreous: From acute onset floaters and flashes to retinal detachment

Xhevat Lumi[a], Marko Hawlina[a], Damjan Glavač[b], Andrea Facskó[c], Morten C. Moe[d], Kai Kaarniranta[e], Goran Petrovski[c,d,*]

[a] Eye Hospital, University Medical Centre, Ljubljana, Slovenia
[b] Institute of Pathology, Faculty of Medicine, University of Ljubljana, Slovenia
[c] Department of Ophthalmology, Faculty of Medicine, University of Szeged, Hungary
[d] Center for Eye Research, Department of Ophthalmology, Oslo University Hospital and University of Oslo, and Norwegian Center for Stem Cell Research, Norway
[e] Department of Ophthalmology, Institute of Clinical Medicine and Kuopio University Hospital, University of Eastern Finland, Finland

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ABSTRACT

Floaters and flashes are most commonly symptoms of age-related degenerative changes in the vitreous body and posterior vitreous detachment. The etiology and pathogenesis of floaters' formation is still not well understood. Patients with acute-onset floaters, flashes and defects in their visual field, represent a medical emergency with the need for same day referral to an ophthalmologist. Indirect ophthalmoscopy with scleral indentation is needed in order to find possible retinal break(s), on-time treatment and prevention of retinal detachment. The molecular and genetic pathogenesis, as well as the epidemiology of the ageing changes of the vitreous is summarized here, with view on the several treatment modalities in relation to their success rate and side-effects.

Contents

* Corresponding author at: Department of Ophthalmology, University of Szeged, Korányi fasor 10-11, 6720 Szeged, Hungary. Tel.: +36 62 544 573; fax: +36 62 545 487.
E-mail addresses: xhlumi@hotmail.com (X. Lumi), marko.hawlina@kclj.si (M. Hawlina), damjan.glavac@mf.uni-lj.si (D. Glavač), facsko.andrea@med.u-szeged.hu (A. Facskó), m.c.moe@medisin.uio.no (M.C. Moe), Kai.Kaarniranta@kuh.fi (K. Kaarniranta), petrovski.goran@med.u-szeged.hu (G. Petrovski).

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1. Introduction

Ageing of the vitreous is a complex biochemical and structural process. Floaters are opacities in the vitreous body which cast shadows onto the retina. Patients see them as small moving spots or specs in the visual field. They may appear as lines, circles, dots, cobwebs, clouds, flies or of any other shape (Fig. 1).

Floaters move as the eye moves, but do not follow eye movements precisely. When attempted to look directly at them, the floaters seem to move away, while blinking does not get rid of them. They are mostly seen when looking at something bright like white paper, plain white wall or blue sky. The perception of floaters is known as *myodaeopsia* (*muscae volitantes* in Latin) (Cline et al., 1997).

Floaters usually begin to appear as few small spots, becoming much dense upon time. In most cases, vitreous opacities occur as a result of degenerative changes in the vitreous body. Vitreous liquefaction (*synchisis senilis*) provokes condensation of the vitreous collagen fibers and posterior vitreous detachment (PVD) (Wagle et al., 2011). More dramatic condition is an acute onset of floaters, the most common cause of which is PVD, having a prevalence of 24% among adults aged 50–59 years and 87% in those over 80 years old (Hikichi et al., 1995).

Flashes of light or lightening streaks appear independently or sometimes together with floaters. They are usually noticed in dim light, at night or in a dark room. They can be induced by eye movements. The most common cause of flashes is also PVD (Hikichi et al., 1995).

PVD is involved as inciting event in most cases of rhegmatogenous retinal detachment (RRD) (Banker and Freeman, 2001). By definition, RRD is described as separation of the neurosensory retina from the underlying retinal pigment epithelium by an accumulation of fluid (Feltgen and Walter, 2014).

This review focuses on the degenerative changes – biochemical and pathological, occurring in the vitreous throughout ageing and the symptoms associated with floaters and flashes of light. In addition, PVD and RRD are discussed here in the context of their role or appearance in acute-onset floaters and flashes. Finally, treatment modalities for managing floaters, PVD and RRD are presented.

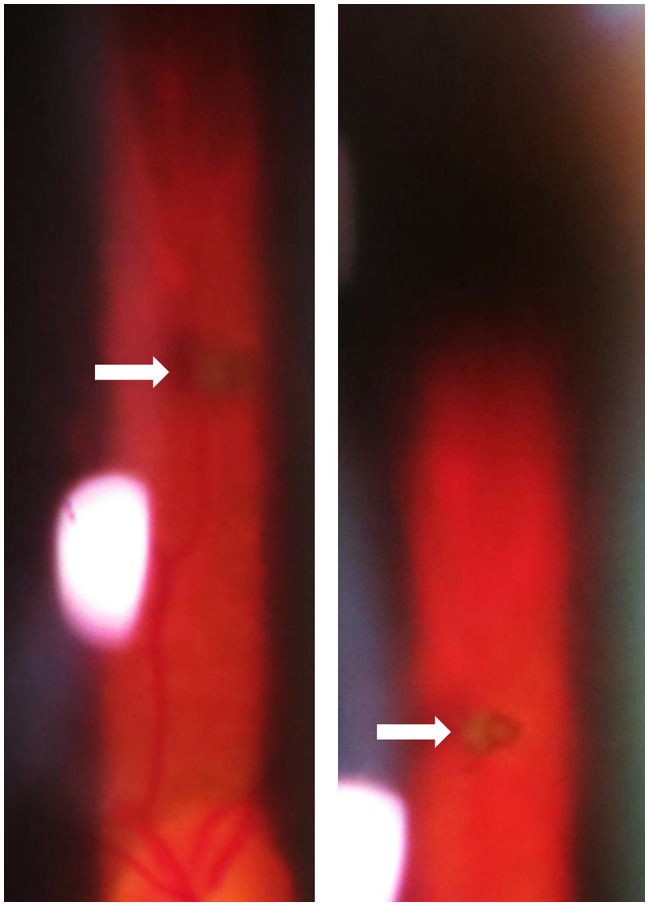

Fig. 1. Slit lamp imaging of floaters in the vitreous body (arrow points at the floater).

2. Pathogenesis of floaters

Degenerative changes in the vitreous body start at an early age. Vitreous liquefaction which destabilizes collagen fibrils has been detected at age 4 years and 12.5% of the vitreous is liquefied at age of 18 (Balazs and Denlinger, 1982). The most common etiologic causes of floaters are age-related and myopia-induced liquefaction of the vitreous gel (Sebag et al., 2014). This liquefaction induces collagen aggregation into visible fibrils and, at a later stage, leads to collapse of the vitreous body (Sebag, 1989).

Anatomically and biochemically, the human vitreous body is a complex structure (de Nie et al., 2013). At least 98% of its content is water and only 0.1% is made up from macromolecules (Bishop, 2000). The most important macromolecules are collagens II and IX, glycosaminoglycans (GAGs) like hyaluronic acid (hyaluronan, HA), proteoglycans (PGs), and also non-collagenous glycoproteins (Ponsioen et al., 2010) (Fig. 2). Water binds to HA. The ageing process leads to two structural changes: depolymerization of HA, which causes release of water and loss of collagen IX. Absence of collagen IX provokes aggregation of collagen II fibrils (syneresis) which leads to formation of fluid filled lacunae (synchisis) (de Nie et al., 2013) (Fig. 2). Collagen filaments aggregation and condensation results in formation of larger fibrils, which float in lacunas of liquefied vitreous giving the patients the perception of floaters. The speed at which these vitreous changes happen depend on age, environmental factors, exposure to sunlight, oxidative effects and HA-collagen interaction (Roth et al., 2005). In patients older than 70 years of age, at least 50% of the vitreous is liquefied (Foos and Wheeler, 1982). Interference of the floaters with the visual axis produces patient discomfort. The number of floaters may increase with age, which has impact on the quality-of-life as well.

There have been many other causes of floaters described before, such as vitreous hemorrhage in proliferative retinopathies, including diabetic retinopathy, sickle cell, venous occlusion, Eales disease (Alan et al., 2009) as well as, myopia (Balazs and Denlinger, 1982),

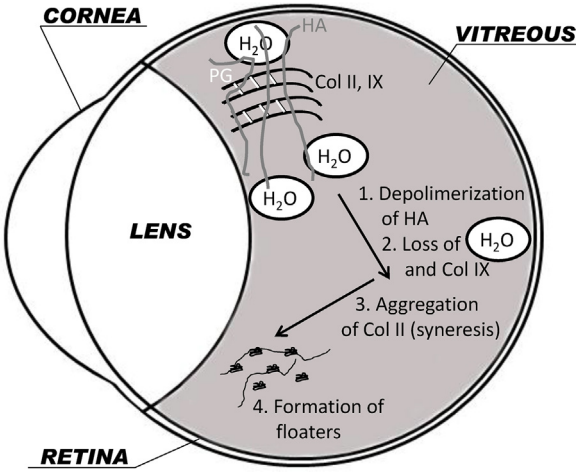

Fig. 2. Pathogenesis of floaters (Col II, IX: Collagen II, IX; HA: hyaluronic acid, PG: prostaglandins).