Vitreous floaters – etiology, diagnostics and treatment

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ABSTRACT

Vitreous floaters are one of the most common complaint of ophthalmological patients. The aetiology of floaters includes pathological process (vitreous haemorrhages, injuries, diabetics or uveitis) and also natural age-related changes in vitreous body. Diagnosis and treatment depends on the severity of the symptoms. Process of posterior vitreous detachment requires particularly careful monitoring, because of potential complications such as retinal tears or retinal detachment. Supplementation with anti-glycation and antioxidant substances allows to reduce complains of patients with vitreous floaters.

Key words: vitreous floaters, vitreous body, PVD, posterior vitreous detachment
INTRODUCTION
Vitreous floaters affect many people of different ages. They cause discomfort at near and far distances. Patients report problems reading, using the computer, telephone or driving.

Floaters, described by patients as flying flies, clouds, zigzags, threads, specks or dots, are best seen when looking at a bright background, called “miodesopsia” (fig. 1).

The etiology of vitreous floaters
The risk of vitreous opacities is associated with some pathological conditions such as vitreous hemorrhage in diabetes or after ocular trauma, uveitis, increasing age, or posterior vitreous detachment (PVD). Vitreous hemorrhage is also associated with non-proliferative diabetic retinopathy, but spontaneous absorption of exudative lesions is visible within a few weeks. A careful inspection of the retina is necessary for the detection any tears or vitreoretinal traction that may lead to retinal detachment. In case of dense hemorrhages, an ultrasound examination is performed for accurate eyeball imaging and possible surgical intervention (fig. 2). Vitrectomy is indicated for long-standing, dense vitreous hemorrhages. Vitreous hemorrhages may also develop because of blunt trauma to the eyeball. In such a case, it is important to find the cause of the lesion and intervene appropriately. Patients often report vitreous opacities caused by amyloidosis or due to inflammatory response following capsulotomy or intravitreal injections (fig. 3). With ageing, the vitreous body undergoes structural changes. Vitreous degeneration results from a progressive reorganization of the hyaluronic acid and collagen molecular networks. The vitreous undergoes the phenomenon of synchysis and syneresis. Initially, vitreous liquefaction occurs because hyaluronic acid separates from the collagen fibers. Then, collagen fibers aggregate together, forming bundles within the space filled with liquefied vitreous [2]. Once a rupture occurs in the cortical part of the vitreous body, vitreous fluid leaks through the tear and the vitreous body separates from the retina. Patients commonly report PVD because of its frustrating symptoms. PVD affects about 60% of people over the age waves, and provides physical support to the retina. The vitreous body allows oxygen to diffuse from the retinal vessels into the anterior segment of the eyeball. The central part of the vitreous body is occupied by the core made up of interconnected bundles of collagen fibrils separated by hydrated hyaluronic acid molecules.

The central vitreous is surrounded by the vitreous cortex. The vitreous is attached to the posterior surface of the lens with the Wieger’s ligament (which disappears around the age of 30 years); the space between is called the Berger’s space. From the back, the vitreous body is firmly attached to the retina at the vessels, at the periphery of the optic nerve disc, in the periorbital area, and at the pars plana of the ciliary body about 2 mm anterior to the ora serrata and and 3–4 mm posterior to it. Here, the collagen fibers are densely packed together and arranged in parallel to each other. Owing to laminin and fibronectin, the vitreous is firmly attached to the anterior surface of the retina – the limiting membrane.

THE COMPOSITION OF THE VITREOUS BODY
The vitreous body is mainly composed of water (99%), type II and IX collagen, hyaluronic acid and ascorbate. It accounts for about 80% of the volume of the human eye and assists vision, supports the eyeball, absorbs the shock
Oxidative stress is caused by an imbalance between oxidation processes and the activity of the antioxidant system. Reactive oxygen species are neutralized by enzymatic and non-enzymatic antioxidants such as tocopherol, ascorbic acid, glutathione, carotene, superoxide dismutase, catalase and glutathione peroxidase. Increased levels of ROS disrupt vitreous collagen fibers in the process of hyaluronic acid depolymerization leading to collagen aggregation. The process of oxidative stress is exacerbated by hyperglycemia, which causes non-enzymatic glycation and glycoxidation. Deterioration of collagen in the vitreous can lead to vitreous degeneration, pathological changes in the vitreoretinal junction, and microcirculatory abnormalities in diabetic retinopathy [6]. Recent studies have suggested that antioxidants such as ascorbic acid and collagen fiber glycation inhibitors (l-lysine, procyanidin) may reduce visual complaints associated with vitreous opacities [7–9]. L-lysine and inositol have been found to have good anti-glycating effects and may have beneficial effect in vitreoretinal diseases. Inositol was found to be useful in inhibiting collagen glycoxidation. It reduces the formation of collagen cross-linking, which can help maintain its structure. Inositol is synthesized in the body and can be found in nuts, beans, and yeast. L-lysine is an essential amino acid found in cheese, eggs, milk, fish, cocoa, meat and soy. The body does not have the ability to synthesize it, unlike endogenous amino acids. The antiglycation effect of l-lysine is stronger than of inositol. Recent studies indicate the important role of l-lysine in preventing collagen glycation and in the treatment of eye diseases, particularly of vitreous body [10].

Some antioxidants (e.g., vitamins) must also be supplied from external sources. Vitamin C is one the most potent antioxidants, found mainly in fruits and vegetables. It is responsible for neutralizing free radicals, and its deficiency leads to impaired collagen synthesis, decreased immunity, fatigue or anemia. Selenium is an essential component of many enzymes, including a powerful antioxidant – glutathione peroxidase. It can be found in seafood, fish, chickpeas or milk. Selenium deficiency is associated with nervous system disorders, thyroid diseases, immune system disorders, and an increased risk of developing civilization diseases or cancer. Zinc, a cofactor of about 200 enzymes, is extremely important for the proper functioning of the body. Zinc is also a component of another important antioxidant – superoxide dismutase. Zinc can be found in cheese, porridge, pumpkin or sunflower seeds. Zinc deficiency may lead to many health problems, including skin lesions, impaired wound healing, and decreased immunity. Furthermore, zinc helps maintain normal vision. The effects of antioxidant deficiency prove their importance for the proper functioning of the entire body; therefore,
it is worth ensuring an adequate supply of antioxidants in the diet, both in food and supplementation. There are commercially available ready-made preparations containing necessary ingredients: antioxidants and protein glycation-inhibiting flavonoids and l-lysine. The positive role of a properly composed diet in the prevention of many diseases, including eye diseases, has been recently increasingly recognized. The 2018 study included 463 patients (334 women and 129 men) aged 14–61 years reporting the presence of visual disturbances related to vitreous floaters/opacities. The study group received a 3-month supplementation – 1 capsule daily containing l-lysine (125 mg), hesperidin from bitter orange extract, proanthocyanidin from grape extract (23.75 mg), vitamin C (40 mg) and zinc (5 mg). According to the subjective assessment in the questionnaire, nearly 91% of the respondents showed a reduction in their reported complaints – a slight improvement in 25.9%, a moderate improvement in 27.6%, a significant improvement in 28.4%, and a very significant improvement in 8.7% of people. Favorable results were observed in both women and men, especially in the group below 50 years of age [11].

Visual comfort improvement has been reported after the use of potassium iodine eye drops in the treatment of troublesome vitreous floaters [12]. Potassium iodine is also recommended in vitreous pathologies such as post-hemorrhagic floaters – it supposedly accelerates their absorption [13]. Potassium iodine binds free radicals, speeds up metabolism and has anti-inflammatory properties, so it has long been used in the treatment of many eye diseases. Iodine is essential for the proper synthesis of fT3 and fT4, and its deficiency can lead to hypothyroidism. The recommended intake of iodine depends on age, gender or lifestyle, and its increased supply is required during pregnancy and lactation. Foods high in iodine include fish, especially sea fish, shellfish, eggs, and dairy products. Including commercially available potassium iodine eye drops in the treatment of post-hemorrhagic vitreous lesions should be considered. PVD is a long-term process and can last up to several months.

We distinguish partial and complete PVD (fig. 4–6). Its course can be chronic or acute. Symptoms that require urgent ophthalmology follow-up include flashes (photopsia), opacities, or blurred vision. Posterior vitreous detachment may also be accompanied by vitreous hemorrhage due to possible retinal tears. Periodic ophthalmic follow-up is recommended to monitor the progression of lesions and urgently intervene. If a retinal tear is present, it needs to be treated with a laser. This is an indication for urgent photocoagulation, as retinal tear can lead to retinal detachment. Such complications occur in about ⅕ of patients presenting with symptomatic PVD.

**DIAGNOSTICS**

Vitreous opacities can be visualized on ultrasound biomicroscopy. Patients often describe the Weiss ring as a blurred ring-shaped opacity. On B-scan ultrasound, PVD is described as a thin, hyperreflective line at the border of the vitreous body. It is also visible on OCT imaging.

**TREATMENT**

Since there are no consensus guidelines on the treatment of vitreous floaters, this process is not easy. Laser vitreolysis or vitrectomy are recommended if symptoms are particularly bothersome [14]. Nd:YAG laser vitreolysis uses laser energy to fragment vitreous opacity and shift them off the optical axis [15]. Sometimes, for better results, the treatment should be repeated. Vitreolysis is recommended to treat single, centrally located opacities [16], with a distance more than 3 mm from the retina. Contraindications to vitreolysis include young age, glaucoma, small floaters located peripherally or too close to the retina, many small opacities,
Completely detached vitreous body visualized on OCT.

Vitrectomy is indicated if vitreous opacities prevent the diagnosis of serious retinal pathologies. Vitrectomy should also be considered in patients who persistently complain that opacities adversely affect their quality of life. Complications of vitrectomy include cataract formation, increased risk of developing primary open angle glaucoma, retinal tear or detachment, vitreous hemorrhage, and endophthalmitis.

The results of the procedure are very good, with about 90% of patients reporting improvement, reduction or resolution of their complaints. The decision about the treatment should be undertaken together with the patient after considering all possible complications and benefits.

CONCLUSIONS

Vitreous floaters negatively affect patients’ health-related quality of life [20]. Therefore, they require attention and intervention not only due to their severity, but also because of the level of patient frustration [21, 22]. In our ophthalmic practice, we may encounter patients complaining on vitreous floaters lowering patient’s visual comfort. In such cases, we should be understanding, offer proper supplementation with proven efficacy, and, in the case of complications, recommend appropriate treatment.

Figures: from the authors’ own materials.

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