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Indian Journal of Clinical and Experimental Ophthalmology

Journal homepage: www.ijceo.org

Review Article

Vitreous floaters: review and prophylactic strategies

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Abstract

The extensive use of screen-containing devices has significantly increased screen-induced ocular degeneration. Studies show that up to 90% of people who spend over three hours daily on digital screens experience symptoms like headaches, blurred vision, and eye discomfort. Even minor refractive errors can severely impact the quality of life and lead to more serious conditions.

The significance of studying floaters lies not only in their diagnostic relevance but also development of prophylactic strategies Vitreous floaters, affecting about 30% of individuals over 50, are a common sign of declining ocular health. If untreated, they can lead to retinal detachment, which affects roughly 1 in 10,000 people annually and can cause permanent vision loss. Although not immediately threatening, floaters cause significant discomfort, disrupting visual fields and daily activities, and negatively impacting mental health. Up to 50% of those with persistent floaters report decreased quality of life.

This review evaluates the pathophysiology of vitreous floaters and explores both conventional and alternative therapeutic approaches. Modern interventions like vitrectomy and laser vitreolysis show a success rate of 85-90% but are invasive. Ancient medicinal systems like Ayurveda and Homeopathy offers different perspectives targeting oxidative stress and rejuvenating ocular heath, hence integrating ancient medicinal system with modern treatment opens new paths towards the prophylaxis of the condition.

Keywords: Floaters, Vitreous, Visual field, Posterior eye region, Ocular degeneration, Treatment.

Received: 14-01-2025; **Accepted:** 01-10-2025; **Available Online**: 16-12-2025

1. Introduction

In the digital age, prolonged screen exposure has led to a rise in degenerative eye disorders across all age groups. Among these, vitreous floaters are common and often associated with myopia, vitreous degeneration, inflammation, or trauma. They represent clumped collagen fibers suspended in the vitreous humor.[1] Though usually benign, floaters may signal pathologies such as posterior vitreous detachment (PVD) or retinal detachment, requiring prompt attention. While painless, they cause visual discomfort, reduced concentration, and mental stress.

This review explores the frequency, pathophysiology, and management of vitreous floaters. Current treatments target underlying causes rather than the floaters themselves. Main approaches include vitrectomy—partial or total removal of the vitreous gel through a 25-gauge needle—and Nd:YAG laser vitreolysis, which vaporizes vitreous opacities using laser energy. Antioxidant and vitamin supplementation (retinol, tocopherol acetate, lutein, zeaxanthin) can mitigate oxidative stress but not eliminate existing floaters.[2]

Because surgical options are invasive, they are reserved for severe cases. Mild floaters are managed conservatively with counselling and nutritional advice. Hence, new minimally invasive treatments—including drug and enzyme delivery systems capable of crossing ocular barriers—and integrative therapies combining modern pharmacology with Ayurveda are being explored to improve patient comfort and outcomes.

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https://doi.org/10.18231/j.ijceo.9752.1761366757

2. Formation of Vitreous Floaters

The occurrence of vitreous floaters is prominent in younger age, older age and those with refractive disorders however the mechanism of formation differs. In the case of younger patients, the floaters are formed as a result of collagen which is the protein found in vitreous forms into a cluster and assembles in hiatus. In comparison with elderly patients these floaters are less in number and are undeviating in nature.

In elderly patients, the floater etiology is usually associated with vitreous superseding during regular eye motion and dispersing light forming a shadow on the retina that is recognized as odd irregular twisted dark grey lines in the visual field. Patients with floaters usually complain that the visualization of floaters is more prominent in light-colored backgrounds and very much visible when seen towards the sky or when seen towards an illuminated light-colored background.

For patients with refractive disorders like myopia the floater symptomology is associated with a degenerated vitreous. Degenerative vitreous refers to the loss of structural integrity of the vitreous that is a normal vitreous can be compared with jelly-like consistency but degenerated vitreous is similar to that of a less viscous jelly. Vitreous degeneration is prominent in myopics of -6 and above. Marfans, Ehlers-Danlos, Sticklers syndrome, and diabetic retinopathy may also induce pathologic variation in the vitreous integrity.

Recently formed PVD disperses lights and imparts diversified shadows on the retina. This is due to the formation of the Weiss Ring. The Weiss ring is identified as neuroglial tissue of protuberance origin that has been bonded to the posterior vitreous cortex. A distend fundus examination is required to analyze acute floater symptomology to rule out the possibility of retinal hemorrhage, and retinal breaks.[4]

3. Mechanism of Floater Formation

The mechanism of floater formation is characterized by the vitreous liquification as a result of macromolecular metamorphosis. The vitreous constitution is such that collagen fibrils are arranged in such a way that they are neither too close nor distant from each other. The fibrils are organized such that there is enough space for the passage of light and also the integrity of the jelly's nature is maintained. The space between the collagen fibrils is maintained by the hyaluron.

The vitreous jelly structure is upheld by the collagen fibers consisting of type 2,5/11,9. Hyaluron functions by possessing the water molecules and keeps the collagen fibrils away from each other, this sometimes results in the forming of a 'hiatus '. The collagen fibrils tend to cluster when the amount of hyaluron decreases. Studies have determined that type 2 collagen is protected by the type 9 collagen from being vulnerable to the surface of collagen fibrils. This is due to the chondroitin sulfate boundary groups. The quantity of collagen type 9 proteoglycan reduces with age which causes type 2 collagen to be revealed and results in lateral amalgamation thus promoting the liquefaction process. The vitreous matter is subjected to two morphological changes with age that result in light dispersion which in turn increases the capacity of liquid room and aggregation due to agglutination of vitreous collagen (**Figure 1**). The emergence of vitreous liquid is seen during the 1st five years of life. The liquefaction process reaches up to 20% by puberty which is further increased by 50% by the age of 65-75 yrs.[6]

Because surgical options are invasive, they are reserved for severe cases Mild floaters are managed conservatively with counselling and nutritional advice. Hence, new minimally invasive treatments—including drug and enzyme delivery systems capable of crossing ocular barriers—and integrative therapies combining modern pharmacology with Ayurveda are being explored to improve patient comfort and outcomes.[3]

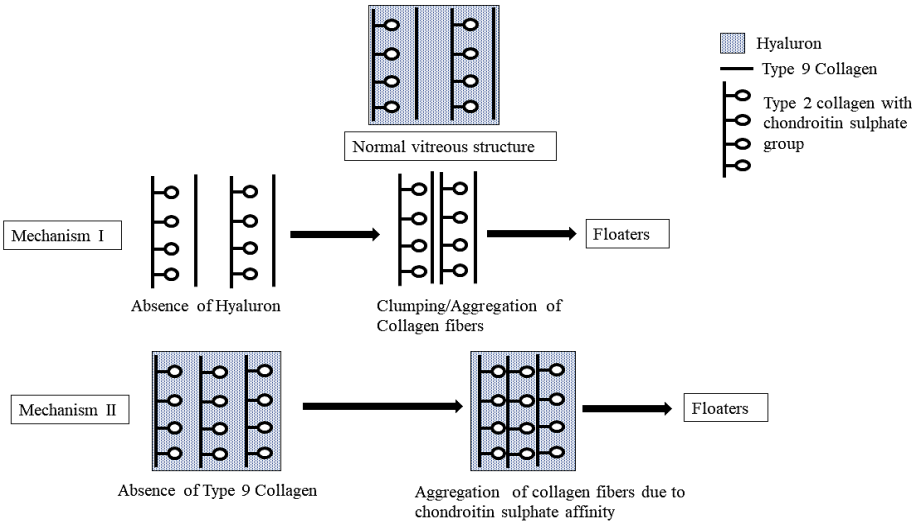


Figure 1: Mechanism of floater formation

The **Figure 1** shows two separate mechanisms of floater formation

4. Classification of Vitreous Floaters

4.1. Based on emergenc[6]

4.1.1. Primary vitreous floaters

Vitreous floaters that are visceral to the vitreous body are termed as primary floaters. Stuffed bundles form noticeable fibers that 1st materialize in the central vitreous having an untwisted alignment. The probability of their appearance is more prominent in people with axial myopia, these types of floaters tend to get plenty, rigid and asymmetrical with age. As the age progresses the vitreous matter liquefies and forms a hiatus, the walls of which obstruct photon conveyance to the retina subscribing to the sensation of floaters. Primary vitreous floaters cause disruption and dispersion of light and appear as stationary intense dark lines dots within the visual area.

4.1.2. Secondary vitreous floaters

Secondary floaters are obscurity in the vitreous matter whose genesis is estranged from the vitreous matter consisting of macromolecules, amyloids, or cells. The most common cause of secondary floaters is preretinal or vitreous bleeding which results in unexpected arrival of floaters and foggy vision.

4.2. Based on appearance

4.2.1. Black or grey dots

Vitreous floaters may appear as tiny black or grey dots in the visual field. These may be formed as black or grey dots themselves or may be due to fragmentation of a larger floater such as squiggy lines types or thread type. The intensity of black or grey dots will depend upon the magnitude of the clumping of vitreous proteins.

4.2.2. Squiggy lines

These types of floaters appear as irregularly coiled and twisted lines. This may be formed by a single degenerated collagen fiber or a combination of two collagen fibers, such floaters are longer than the previous type.

4.2.3. Ring shaped

Such type of floaters appears as a ring in the visual field. The rings may appear single or multiple, multiple rings might even get entangled which gives the appearance similar to the Venn diagram.

4.2.4. Thread-like strands

This type of floater looks like strands of threads freely floating in the visual field. The appearance of a single thread type floater is usually rare, however appearance of multiple thread like floaters is more prominent.

4.2.5. Cobweb types

The appearance of cobweb-type floaters is a complex cobweb-type-looking structure. Such types of floaters appear more than any other type causing the most obstruction in vision. These types of floaters exist as single cobweb structures, its fragments, or in combination with any other type of floater.

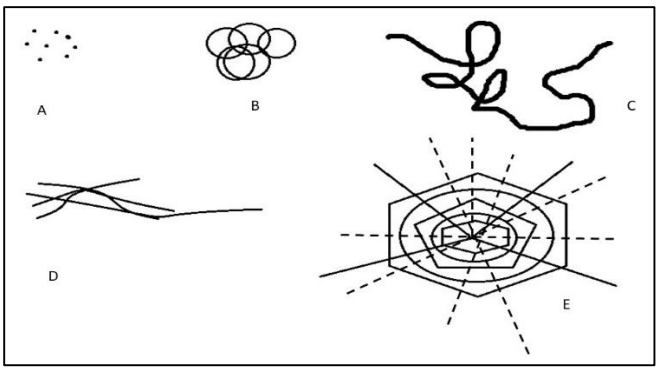


Figure 2: Visual appearance of different types of vitreous floaters (A: Dots type floater, B: Ring type, C: Squiggy lines, D: Tread type, E: Cobweb type)

4.3. Causes of vitreous floaters

4.3.1. Retinal detachment

Partitioning of the neurosensory retina (NSR) from basal retinal pigment epithelium (RPE) results in retinal detachment. The emergence of these 2 layers originates from the neuroectoderm that appears through the optic vesicle during embryonic propagation. The optic cup is formed as the optic vesicle intussuscepts and the 2 layers come in juxtaposition. The inner layer metamorphoses into the NSR and outer in RPE. Non-real morphological intersection exists between the cells of two layers. Therefore, the forces of bonding of NSR to RPE are delicate and once deluged, retinal detachment results that reinstitute the prospective space between the two layers.[7]

4.3.2. Types of retinal detachment

1. Rhegmatogenous retinal detachment (RRD) is identified by a retinal interruption. The separation of the retina from the basal choroid occurs as a result of the accumulation of fluid from the vitreous, which leaks and transits through the retinal rupture into the prospective space under the retina. This condition requires surgical intervention.
2. Tractional Retinal Detachment (TRD) is identified by scarring that hauls the retina from its linkage. This may require surgical assistance depending on the magnitude of RD. The most common causes of TRD are diabetes, EalesOs disease, sickle cell, retinopathy, and trauma.
3. Exudative and serous retinal detachment are prominent as a result of an oddity in water transit

across the retinal pigment epithelia or in its blood flow.[8]

4.3.3. Myopia

Shortsightedness is the common most refractive disorder experienced by people wherein there is the inability to see distant objects clearly. This is caused as a result of image formation in front of the retina; to obtain a normal vision the image should be formed on the retina itself. The image formation is dependent on the corneal thickness or the lens flexure or the eye is too protracted. Clement's myopia is 0D to -1.5D, Modest myopia is -1.5D to -6D, and exorbitant myopia is -6 and above. -8 D and more are results in pathological myopia although retinal illness, glaucoma, and cataracts the analogous menaces to the vision that can emerge in patients with clement and exorbitant myopia.[9]

4.3.4. Posterior vitreous detachment

PVD refers to the detachment of the posterior vitreous cortex from the retina's internal limiting membrane. Diagnosis is made using dilated slit-lamp biomicroscopy and optical coherence tomography (OCT). It is an age-related vitreous change that can initiate pathological processes at the vitreoretinal interface.

PVD typically begins near the macula when liquefied vitreous moves into the subhyaloid space, causing traction and separation from the retina. Complications include retinal tears, vitreous hemorrhage, and optic disc bleeding, often due to strong vitreoretinal traction during sudden eye movements.

Prevention includes regular eye checkups, lifestyle modification, and antioxidant supplementation. Treatment focuses on complications—vitrectomy for traction or non-resolving hemorrhage, and laser photocoagulation or cryotherapy to seal retinal tears. Early diagnosis is crucial to prevent permanent vision loss.[10]

4.3.4.1. Classification of posterior vitreous detachment

The classification is done on the basis of absence or presence of PVD.[11]

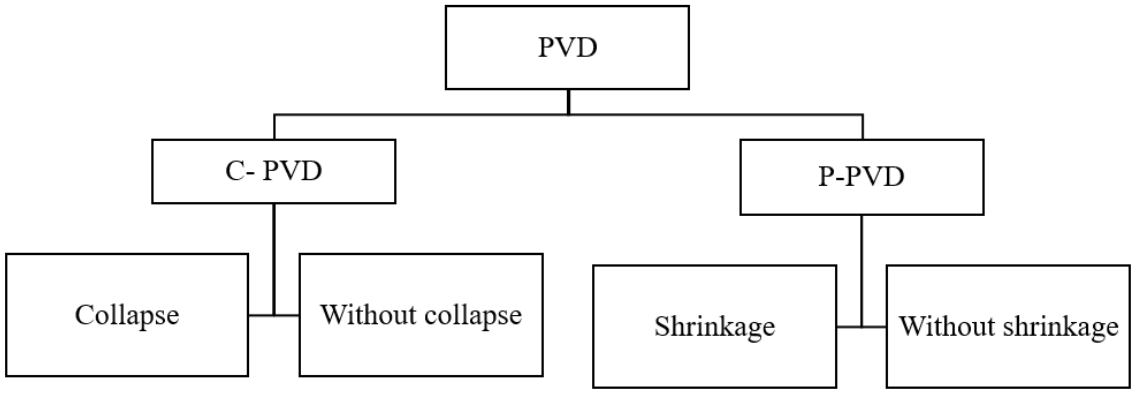
Normal eye with no PVD displays asteroid hyalosis which propagates diagnosing the nonappearance of a PVD.

C-PVD with collapse is identified by a stationary massively emancipated posterior hyaloid layer that is outlined easily and a prepapillary gilal halo is noticed on the posterior hyaloid membrane.

C-PVD without collapse is identified by a shallow emancipated hyaloid membrane is outlined anterior to the retina. The eyes of young patients with paramount retinal vein barricade and uveitis also recurrent have this type of PVD

P-PVD with shrinkage is identified by retinal vascular arcade along with neovascular proliferative tissue. Post ocular motion the posterior hyaloid layer along with the neovascular proliferative tissue was not stationary. Robust vitreous traction results due to the vitreous variation to the retina along the retinal vascular colonnade.

P-PVD without shrinkage is identified by a posterior hyaloid layer that is outlined superiorly (overturned). However, the inferior vitreous did not dissociate from the inferior retina post-ocular motion.[12]



Note: C-PVD refers to complete posterior vitreous detachment and P-PVD refers to partial vitreous detachment

Figure 3: Flowchart displaying classification of PVD

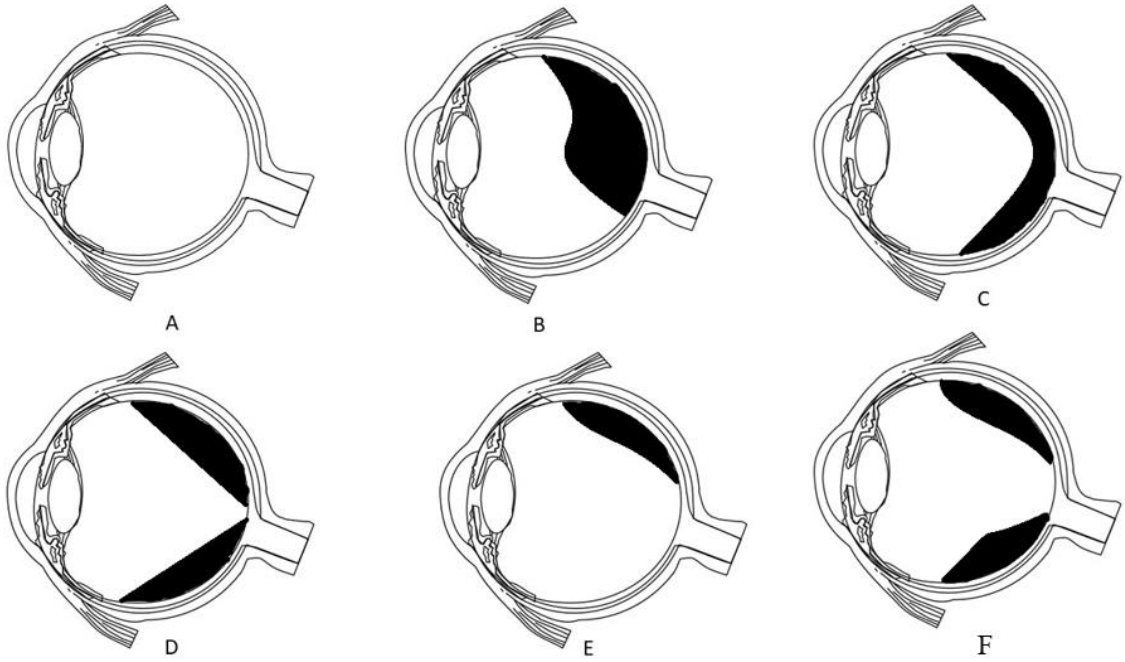


Figure 4: Different types of PVD (A: No PVD, B: C PVD with collapse, C: CPVD without collapse, D: P PVD with shrinkage, E: P PVD without shrinkage, F: P PVD without shrinkage

4.3.5 Uveitis

Uveitis is inflammation of the uveal tract—iris, ciliary body, and choroid—often extending to the retina, vitreous, and optic nerve. It is classified as anterior, intermediate, posterior, or panuveitis, and can be acute or chronic.[13]

In developing countries, about half of all cases are infectious, while in developed regions they are mainly autoimmune or idiopathic. Infectious uveitis results from microbial invasion or immune-mediated reactions. Diagnosis is supported by aqueous humor PCR and Goldmann–Witmer coefficient tests for microbial DNA detection. Toxoplasma gondii causes focal chorioretinitis, and herpes viruses (HSV, VZV) lead to recurrent anterior uveitis or necrotizing retinitis.[14,15]

Noninfectious uveitis is commonly linked to HLA-B27–associated spondyloarthropathies, Vogt–Koyanagi–Harada disease, sympathetic ophthalmia, and Behçet's disease. The anterior form is most frequent, particularly in males with ankylosing spondylitis. Sarcoidosis should be suspected in granulomatous uveitis. Early classification and targeted therapy are vital to avoid complications and irreversible visual loss.[16]

5. Existing Treatment Techniques

The existing treatment techniques available today are mainly invasive approaches and are recommended only in critical situations; a flow diagram representing all the treatment strategies is shown in **Figure 4**. Floaters as such aren't harmful hence invasive approaches aren't employed in every situation instead the patients are prescribed vitamins that help battle oxidative damage. Consumption of vitamins for a regular course is beneficial in maintaining ocular health but it doesn't eliminate the floaters hence usage of vitamins as treatment for floaters isn't specific but only supportive.

The classification of existing treatment techniques can be done as follows

1. Floater-specific treatment
2. Disease-specific treatment
3. Supportive treatment
4. Alternative medicine treatment

5.1. Floater-specific treatment

It specifically targets vitreous floaters and aims in eliminate of the vitreous floaters by surgical and laser methods. The methods include the following

5.1.1. Vitrectomy

The extraction of the vitreous jelly is done by vitrectomy, the surgical procedure performed by the surgeons. The terminology of vitrectomy consists of 'vitrec' referring to vitreous and 'tomy' referring to incision and evacuation in medical diction.

Vitrectomy can be performed to extract the entire vitreous jelly referred to as complete vitrectomy or specifically the floaters are extracted referred to as floater-only vitrectomy. Post extraction of vitreous jelly the void is compensated by replacing the vitreous with either saline solution or silicone oil.

5.1.2 Pars plana vitrectomy

Vitrectomy involves surgical removal of the vitreous humor under local or general anesthesia. After inserting an eyelid

speculum, the surgeon examines the fundus using a microscope or ophthalmoscope. Trocar and cannulas are introduced 3–4 mm from the corneal limbus at the 2, 10, and inferotemporal clock positions. An infusion cannula placed inferotemporally maintains intraocular pressure and visualization.

The procedure begins with core vitreous extraction. If the posterior hyaloid remains attached to the macula or optic disc, a PVD is induced by suction. Steroids may aid visualization, and a bimanual technique using a light probe can be applied when clarity is poor. In phakic patients, lens protection is critical during peripheral vitreous removal. After extraction, the retina is inspected for tears or detachment near sclerotomy points to prevent postoperative complications. Removed vitreous may be analyzed pathologically when required.[17]

5.1.3. Small incision vitrectomy

The 25-gauge vitrectomy also termed as small incision vitrectomy is a nominal invasive surgical strategy adopted for the removal of the vitreous. Eugene de Juan came up with this technique in 2002. The basic procedure remains the same as the previous method however the size of the instruments used in the procedure is greatly reduced. The tools have a lumen diameter of 0.5mm. The introduction of the trocars is done trans-concjuctiviatlly and transversally. The advantage of using these latest instruments is that it eliminates the need to suture the instruments to the sclera to keep them stationary in one place. In this procedure, the conjunctiva is not exposed as a result of which there is decreased anterior region injury during the procedure.[18]

5.1.4. YAG laser vitreolysis

Advancements in medical technology have led to direct evaporation and anatomized vitreous opacities by the implementation of the laser. This process is termed as 'laser vitreolysis'.[19] In this method, UltraQReflex laser is utilized where an apex energy of 7mJ is used. The synchronization is done by observing the plasma development with the evolution of gas bubbles. This is done by application of 3mJ energy at first and titrating to a suitable level. To conduct the laser vitreolysis process post intraocular pressure determination the eye on which the procedure has to be performed is dilated with phenylepinephrine (2.5%), and tropicamide (1%). Before the application of YAG laser proparacaine is administered and an ocular karickhoff 12mm vitreous lens containing goniosol is applied. The complexity and number of floaters determine the number of laser shots to be shot at the patient and hence is completely varying. In most of the cases, the laser shots are terminated only after the vaporization of Weiss ring and all other prominent visual opacities. Each is operated per session to avoid divulging controls.[20]

Complications

1. Focal cataracts and extended elevation of the intraocular pressure were most commonly observed. In some cases, the focal cataracts were confined to the visual axis and some had developed posterior capsule rupture.
2. Extended elevation of intraocular pressure
3. Retinal detachment
4. Laser-induced transient posterior pole retinal bleeding
5. Elevated number of visual floaters[21]

5.2. Disease-specific treatment

It involves the treatment of the underlying cause of floaters. Floaters may be caused due to trauma, or diabetic retinopathy. Most complicated eye diseases usually result in retinal tear or retinal detachment hence specifically treating retinal tear or retinal detachment reduces the further occurrence of eye floaters but does not help in the elimination of existing floaters.

5.3. Treatment of retinal tear

5.3.1. Photocoagulation

Photocoagulation ("photo" = light; "coagulation" = thickening) uses laser energy to fuse the retinal pigment epithelium with the retina, sealing tears and reattaching tissue. It is performed during or after scleral buckling surgery using a laser coupled to an ophthalmoscope for precise targeting.

Laser absorption by pigment epithelium generates localized heat, welding the retina to the underlying layer. Difficulties arise in retinal degeneration, miosis, cataract, or vitreous haemorrhage. Trans-scleral diode laser photocoagulation transmits energy through the sclera with scleral depression to enhance contact. The desired burn is light gray-white; excess power may rupture Bruch's membrane. Despite being labour-intensive, it offers precision and is effective even in previously buckled eyes. Photocoagulation provides high accuracy, minimal collateral injury, and rapid adhesion (within 24 hours), making it superior to cryopexy or diathermy.[22]

5.3.2. Cryopexy

Retinal complications can be improved by the retinal cryopexy. The terminology evolves from the Greek diction wherein 'Cryo' means icy cold. The procedure employs freezing cold temperatures to treat the injured retina. Post anaesthesia a pen-like probe is mildly pushed against the eye. The tip of the cryoprobe is chilled by a foot-operated cryo – instrument. The freezing effect is transmitted through the ocular walls to the retina. Usually, cryopexy is safe however risks are prominent.

Post-procedure, analgesia, redness, inflammation, bruising, and rupturing are the most frequent adverse

consequences. In some cases, cryopexy is unable to reverse retinal detachment and additional surgical intervention is required. Critical and sparse complications include septicaemia, haemorrhage, scarring, loss of vision, and the loss of an eye. When cryopexy is suggested, the benefits of prophylaxis exceed the risks.[23]

5.3.3. Treatment of retinal detachment

Retinopexy prevents or treats retinal detachment and is performed under local anesthesia by a retinal specialist. Pupils are dilated with phenylephrine (2.5%) and tropicamide (1%) for optimal visualization. Minor irritation is managed with counseling and anti-allergic measures.

After anesthesia, the patient is positioned for best access. Retinal breaks are located with magnified imaging; small ones are identified by adjusting light and head position. Once localized, cryotherapy (cryopexy) is applied to freeze and adhere the retina to underlying tissues. Controlled probe placement and temperature prevent tissue injury. A pneumatic gas bubble is injected into the vitreous cavity to tamponade the break, and patient head position is adjusted so the bubble presses against it for sealing. Postoperatively, patients are monitored for pain or vision distortion. Follow-ups confirm reattachment. In rare cases, a second gas injection may be required. Properly executed, retinopexy is minimally invasive, precise, and effective for restoring retinal integrity and preventing vision loss.[24]

5.3.4. Treatment of uveitis

5.3.4.1. Topical and systemic corticosteroids

The treatment strategy for the management of chronic anterior uveitis includes long term administration of topical corticosteroids; also, routine use of mydriatics ensures that the pupil is kept dilated and restrict the development of synechia which may result in cataract development and increased intraocular pressure.

In cases of patients with chronic uveitis, corticosteroids are the pillars of treatment strategy. Corticosteroids are administered for the prophylaxis of molecular oedema and in cases of visual activity less than 6/12.12.13. Patient should be administered with relevant doses to decide whether the macular oedema is reversible. Thus, maximum dose (1.0 – 1.5 mg/kg body weight/ day of prednisolone must be administered for 2-3 weeks. If this strategy is irresponsive then a secondary agent like cyclosporin may be administered for next 4-6 weeks. If corticosteroids are irresponsive the dose is adjusted by 5 mg/ week until the lowest dose keeps the vision is determined. A secondary agent is not required in cases if the dose is more than or equal to 15 mg/day but if relapse episodes are continuous there or second drug may be indicated.[25]

5.3.4.2. TNF- α inhibitors

The next set of indications to be indicated in non- infectious uveitis is the use of biologic responses adjusters. These medications are considered by including or shifting when other immune suppressive agent is ineffective in acquiring a state of quiescence, in cases where very continuous flares (<3flares /year) or if they are not well condoned. Tumour necrosis factor alpha inhibitors are most commonly used for treating uveitis. TNF- α is a vital cytokine responsible for ocular inflammation and tissue necrosis.

Campath 1H (alemtuzumab) is a human derived monoclonal antibody which identifies the pan -lymphocyte antigen CD52. It is indicated in refractory manifestation of orbital inflammation including revocation in 80%. However, it is limited use in healthy patients with uveitis as it can have chronic side effects like haemolytic anaemia and thrombocyte paena.

Rituximab is a quixotic monoclonal anti-CD20 antibody CD20 is a β cell distinct surface antigen expressed on all β cells. The indication of Rituximab in rheumatoid arthritis gave promising results although the role of β cells was not evident. In Rheumatoid arthritis β cells are considered to have a role in production of auto antibodies T cells activation and producing cytokines. This one evidence in literature of its triumphant use in case of treatment resistant uveitis and it has also been triumphantly used in refractory ophthalmic wegness granulomatosis.[26]

5.3.4.3. NSAID

The inhibition of cyclo-oxygenase is done by NSAIDS. Through this mechanism lessens inflammatory arbitrator are producing outright to reticence of arachidonic acid breakdown and thus there is reduced devastation of blood aqueous barrier and limits the development of aqueous humour proteins and prostaglandins. There are fewer clinical proofs which support the indication of topical NSAID in uveitis, although in vivo studies have shown promising results with respect to aqueous penetration and efficacy similar to prednisolones acetate 1% in prophylaxis of uveitis.[27]

5.4. Supportive treatment

Supportive treatment includes non-surgical methods for the treatment of vitreous floaters. It includes the prescribing of antioxidants and certain plant and fruit derivatives. The main objective of supporting treatment is maintaining eye health and preventing degeneration of vitreous tissue.

‘Oxidative strain’ is a chronic situation wherein ROS vandalizes antioxidant armour activity. The situation arises when antioxidant protection is insufficient and or ROS production is extreme. The dainty balance between ROS production and ROS rummage accompanied by antioxidants results in oxidative stress. Progressive cell malfunctions

followed by unsuppressed reactions of ROS with cellular proteins, lipids, and other cellular components lead to the situation.

Oxidation strain can be correlated with aging, an array of age-related chronic diseases inclusive of various ocular pathology such as cataracts, glaucoma, age-associated molecular degeneration, and diabetic retinopexy.[28]

5.4.1. Role and mechanism of action of antioxidants

The posterior eye—including the vitreous, retina, choroid, and optic nerve—is metabolically active. The retina converts light to neural signals and consumes more oxygen per gram than any other tissue, generating abundant reactive oxygen species (ROS). These are countered by antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), glutathione (GSH), vitamin C, lutein, and zeaxanthin.

Ocular melanin, found in the iris, choroid, and retinal pigment epithelium, acts as a free radical scavenger, reducing photo-induced oxidative stress and neutralizing lipofuscin, a major ROS source. The vitreous body, comprising 80% of eye volume, contains SOD, GPX, CAT, transferrin, GSH, uric acid, vitamins A and C, zinc, and selenium. Zinc protects sulfhydryl groups and induces antioxidant enzyme production, while selenium supports antioxidant selenoproteins.[29]

Vitamin C neutralizes hydrogen peroxide and hydroxyl radicals, converting to dehydroascorbate, which is recycled by GSH or NADPH. Glutathione detoxifies ROS via catalase and glutathione peroxidase, protecting retinal and lens proteins.

Carotenoids such as lutein and zeaxanthin quench singlet oxygen, scavenge radicals, and prevent lipid peroxidation. Concentrated in the retina and lens epithelium, they stabilize membranes and limit oxygen diffusion. Their perpendicular alignment within membranes allows them to neutralize ROS efficiently, maintaining membrane integrity and preventing oxidative retinal degeneration.[30]

5.4.2. Eye patch[31]

Cellular rejuvenation is associated with oxygen, an essential source of energy. If the oxygen demand is not met tan this results in hypoxia leading to cellular inactivity. Screen-induced radiation is a prominent contributor to ocular cellular hypoxia. Oxygen can be utilized for treating the root cause. Degraded tissue repair can be achieved by oxygen delivery to the degraded tissue which can overturn the resultant dysfunctionality/ disease.

The functional dressing is from TO2MCo (Miaoli County, Taiwan model BXX01. The dressing comprises sodium peroxide, sodium hydroxide, aluminium powder, and oxalic acid. By imbibing the water from the applied environment, it generates hydrogen. The patch not only functions to safeguard the wound but also amalgamates oxygen and hydrogen to provide a perfect environment for healing the affected area, block the free radical attack and decrease the susceptibility to septicaemia.

5.5. Alternative treatment

5.5.1. Ayurveda

Ayurveda is an ancient form of medicine practiced in the Indian subcontinent. The Ayurvedic form of medicine is completely natural using herbs-derived medication in the form of *rassa Bhasma* etc. The methodology of Ayurveda pivots on maintaining the equilibrium of the body, mind, and soul to encourage health and well-being. Ayurveda accentuates the use of natural treatments, including medicinal plants, dietary changes, yoga, and meditation. The Ayurvedic system is founded on the abstract of three fundamental energies or *Dosha- Vata, Pitta, and Kapha* each of which rules specific bodily functions. In Ayurveda, the disease is cured by maintaining the equilibrium among these *doshas* and modifying lifestyle choices to the individual unique constitution (*prakriti),* and prevent disease through hostilic living.

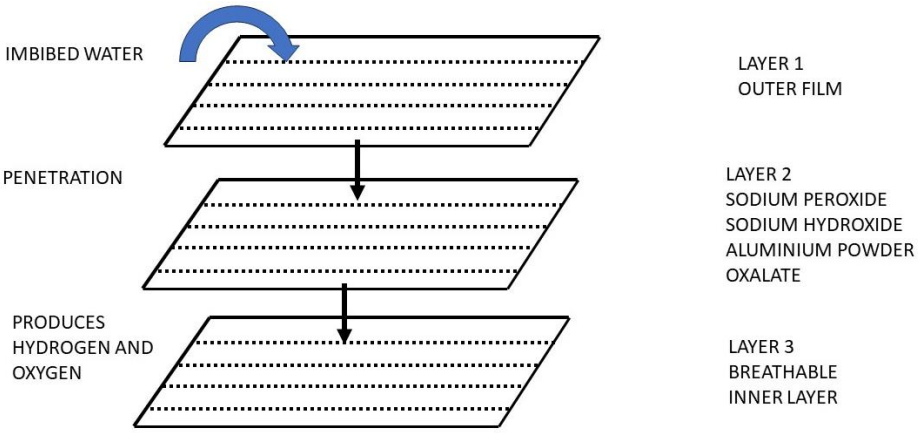


Figure 5: Structure of functional dressing

5.5.2. Netra tarpana

Netra Basti or Akshitarpana is an ancient procedure that is Brihmana in nature and is generally followed by Kriya in Netra Chikista. Tarpana is beneficial for healthy as well as the ill person. The ocular diseases categorized under Dristigarta Rogas are treated by 'Tarpana', a singhdha- kriya. The procedure is usually employed in computer vision syndrome, macular degeneration, myasthenia gravis, and flaccid eyelids.[32]

5.5.3. Procedure of Netra Tarpana[33]

5.5.3.1. Practice Karma

Akshitarpana is employed post-essential purification procedures (cleansing). The procedure is practiced on as propitious day either in the early or late afternoon later to the consumption and digestion of food by the patient. The patient is let to get adjusted to a luminous, dust-free, spacious room. A leek-proof barricade is formed surrounding the eye by a Pali (wall) and is then capped with Masha paste. According to Vagbhata a height of 2 Angula can be achieved by the Pali.

5.5.3.2. The Passta Karma

The Ghrita is maintained for a definite period through a pore in the dough barrier in proximity to the external canthus. Soon after the eye is abluted with lukewarm water. Kapha should be eliminated with Shirovirechana (Nasya) and Dhoompana. When administering Kapha anhiliating medication it has been activated by the persuasiveness of Ghrita. The patient must safeguard himself from intense light, wind, mirrors, and kinetic lights.

Snehadharanakala is known as the interval of detainment. Depending on the magnitude of Dosha Prakopa and the diseases Adhishtana, Snehadharanakala is employed.

5.5.3.3. Sessions of procedure or Tarpana Avadhi

According to the Acharya Sushruta, the treatment should be implemented for 1, 3, or 5 days till Samkaya Tarpita Lakshana are identified without considering the state of the eye. As stated by Acharya Jeffata Tarpana should be implemented for one day for Vataja diseases, 3 days for Pittaja matters, and 5 days for Khapaya matters. Tarpana can be implemented every day, alternate days or with a sequence of 2 or 3-day implementation followed by 2 or 3-day gap based on dosha and magnitude (Teevratha) of the vyadhi and health (swasthya).

5.5.3.4. Triphala Ghrita[34]

Shrarangdhara the first ancient Ayurvedic text defines the category of Snehakalpa which is utilised in the Kriya Kalpa procedure known as Tarpana, a provincial ocular revitalizing and cherishing therapy. Snehakalpana (medicated Ghrita) distinctive oily dosage form employed for local as well as circulatory administration. The contents of Triphala ghrit is given in **Table 1**.

Table 1: Contents of Triphala Ghrita

S. No.	Content	Latin name/English name
1	*GoGhrita*	Cow's Ghee
2	Aja-Kshira	Goat's Milk
3	*Bibhitaki*	Terminalia bellirica
4	*Haritaki*	Terminalia chebula
5	*Amalaki*	Embilica officinalis
6	Pippali	Piper longum Linn.
7	Draksha	(Vitis vinifera Linn.),
8	*Vasa*	Adhathoda Vasica
9	Daruharidra	Berberisaristata
10	Punarnava	Boerhavia diffusa
11	Shweta Chandana	Santalum album
12	Maricha	Piper nigrum
13	Sunthi	Zingiber officinale Rosc.
14	Haridra	Curcuma longa
15	Meda	PolygonatumVerticillatum
16	Neel Kamal	Nelumbo nucifera
17	Bala	Sida cordifolia
18	Kakoli	Roscoea procera
19	Kshir kakoli	Lilium Polphyllum
20	Bringraj	Eclipta alba Hassk
21	Sharkara	Sugar

5.5.4. Homeopathy

Homeopathy, founded by Samuel Hahnemann in the 18th century, is based on the principle of "like cures like", where a substance causing symptoms in health can cure similar symptoms when highly diluted. Remedies are often diluted beyond molecular presence, believed to stimulate the body's healing response. Despite limited evidence beyond placebo, homeopathy remains widely practiced.[35]

For eye floaters, remedies are selected individually after evaluation. Common options include:

1. Phosphorus – For black floaters, flashes, or colored spots linked with migraine or declining vision.
2. *Natrum Mur* – For zigzag lines or misty, blurred vision.
3. *Argentum Nitricum* – For brown floaters, photophobia, or gray spots.
4. *Physostigm*a – From *Physostigma venenosum*, for myopia-related floaters.
5. Cyclamen – From cyclamen europaeum, for fluctuating yellow-green or sparkling floaters.
6. Belladonna – From "Deadly Nightshade," for inflammatory floaters with burning or tearing.

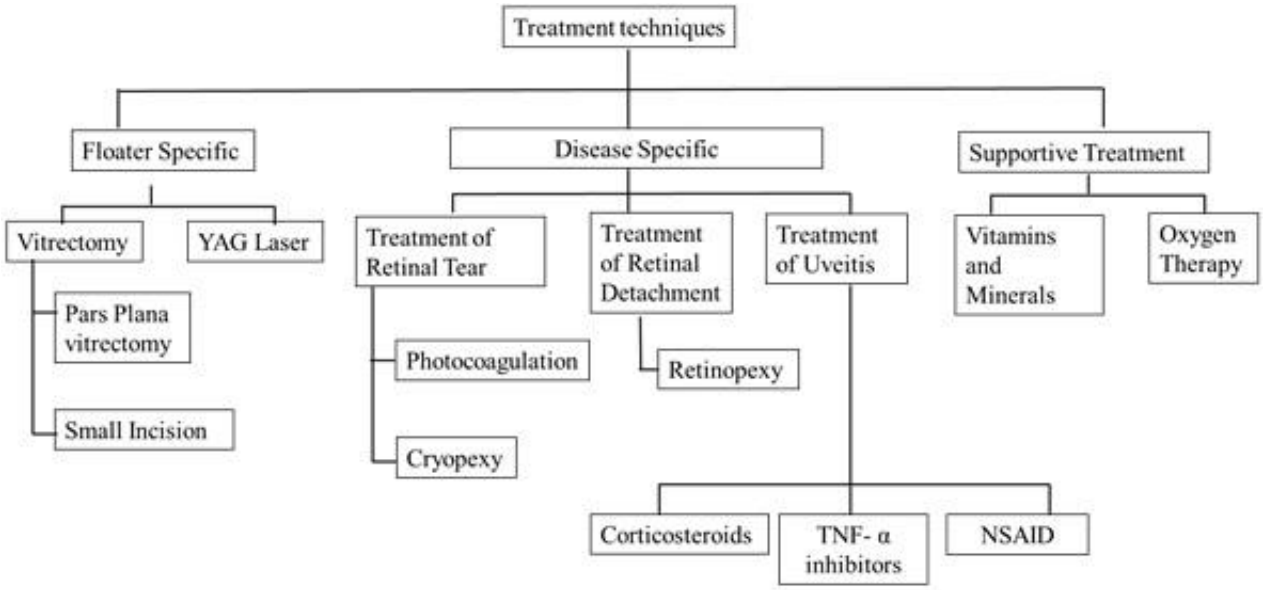


Figure 6: Shows the classification of all the treatment techniques available for treating floaters (Source: Authors Compilation)

5.6. Psychological perspective of eye floater[36]

The psychological fitness of the human mind is greatly influenced by the physical fitness of the human body which is free from diseases and is functionally able to carry out regular activities. Being diagnosed with an untreatable or complicated medical condition puts a human mind in psychological distress which affects his behaviour and cognitive abilities. Vitreous floaters although is not a painful condition the constant appearance of floaters in the visual field causes irritation and frustration. It is seen that patients diagnosed with symptomatic vitreous floaters are more prone to anxiety, and depression. The psychological perspective may be different for every individual. To understand the psychological perspective, we can divide the patients with floaters into the following groups

5.7. Patients who understand the science of floaters

Patients who understand about the floaters may generally be more prone to psychological distress as these types of patients know that there are no proper medications to treat floaters hence, they can get more frustrated as they are aware that they will have to experience the floaters lifelong. Nowadays people tend to access and evaluate their medical conditions through the internet. Coming across unwanted or irrelevant information can tend to create more fear and can lead to depression. Some people might accept that since there is no specific treatment, they have to bear the symptoms of floaters lifelong and will ultimately adapt to the new visual field changes.

5.8. Patients who do not understand the science of floaters

Patients who do not understand the science of floaters will tend to be more comfortable with floater symptoms as they will be informed by their doctors that there is no floater-specific treatment to be used for mild symptoms and hence will accept the fact and move on although the feeling of irritation might remain due to the constant movement of floaters. Patients who do not understand the science of floaters might be influenced by the patients who know about floaters. The diagnosis of the cause of floaters might not be accurate and there might be a wrongful transfer of information to the patient who does not understand the science of floaters hence making him susceptible to psychological distress like anxiety or depression.

6. Limitations

The recent studies provide only short term results hence, long term and focused research is needed to understand vitreous floaters progression and optimize treatment. While surgical and non-surgical methods show promise, comparative trials remain limited. Additionally the impact of prolonged screen exposure requires deeper experimental evaluation. Traditional systems like Ayurveda and Homeopathy may offer supportive benefits but robust clinical validation is essential for effective integration with modern medicine.

7. Conclusion

The review reveals that vitreous floaters can affect all age groups, with those having existing refractive pathologies being more prone. Floaters can also result from injuries and other pathological conditions. While there are no treatments specifically targeting floaters, symptom management typically involves supportive measures like vitamins, and in severe cases, surgical interventions. This highlights their high prevalence and impact as upto 50% of those with persistent floaters report a significant reduction in quality of life due to visual and psychological stress

Floaters are often caused by free radical damage, particularly prevalent among younger individuals due to prolonged screen exposure. Our review assessed the effectiveness of current treatments based on treatment

specificity and patient compliance. Although surgical options, such as vitrectomy, exist, they are considered only in extreme cases due to their invasive nature. This reflects the limited and invasive nature of treatments that are available but are also to be used only in severe cases.

In conclusion, there are no specific treatments available for floaters, though surgical methods can be used in severe scenarios, hence the need for prophylactic and integrative approaches becomes critical combining preventing measures such as reduced screen time and antioxidant supplements with ancient medicine systems like Ayurveda which offer different methodologies for managing floaters and maintaining ocular health.

8. Source of Funding

All authors acknowledges that no funding was provided for this study.

9. Conflicts of Interest

The authors declare no conflicts of interest.

10. Author Contributions

I would like to sincerely thank all my co-authors for their invaluable contributions toward the completion of this review article. Special thanks to Dr. SN Surme (MBBS, DGO), Dr. Prakash Kuncolienkar [M.S. (Oph), DOMS (CPS)] and Dr. Vishakha Moghe (BAMS) for their valuable guidance and support.

References

1. Webb BF, Webb JR, Schroeder MC, North CS. Prevalence of vitreous floaters in a community sample of smartphone users. *Int J Ophthalmol*. 2013;6(3):402–5. https://doi.org/10.3980/j.issn.2222-3959.2013.03.27
2. Al-Asadi SZ. Vitreous floaters and photopsia as predictors of vitreoretinal pathology. *Basrah J Surg*. 22013;19(1):50–5. https://doi.org/10.33762/bsurg.2013.73618
3. Sendrowski DP, Bronstein MA. Current treatment for vitreous floaters. *Optometry*. 2010;81(3):157–61. https://doi.org/10.1016/j.optm.2009.09.018
4. Milston R, Madigan MC, Sebag J. Vitreous floaters: etiology, diagnostics, and management. *Surv Ophthalmol*. 2016;61(2):211–27. https://doi.org/10.1016/j.survophthal.2015.11.008
5. Ghazi NG, Green WR. Pathology and pathogenesis of retinal detachment. *Eye (Lond)*. 2002;16(4):411–21. https://doi.org/10.1038/sj.eye.6700197
6. Kakehashi A, Takezawa M, Akiba J. Classification of posterior vitreous detachment. *Clin Ophthalmol*. 2014;8:1–10. https://doi.org/10.2147/OPTH.S54021
7. Jalali S. Retinal detachment. *Community Eye Health*. 2003;16(46):25–6.
8. Gabbey AE. Retinal detachment. Healthline. 2023. Available from: https://www.healthline.com/health/retinal-detachment#types-and-causes
9. Fredrick DR. Myopia. *BMJ*. 2002;324(7347):1195–9. https://doi.org/10.1136/bmj.324.7347.1195
10. Hobbs H. Nearsightedness (Myopia). Healthline. 2022. Available from: https://www.healthline.com/health/nearsightedness
11. Johnson MW. Posterior vitreous detachment: evolution and complications of its early stages. *Am J Ophthalmol*. 2010;149(3):371–82.e1. https://doi.org/10.1016/j.ajo.2009.11.022
12. Kakehashi A, Takezawa M, Akiba J. Classification of posterior vitreous detachment. *Clin Ophthalmol*. 2014;8:1–10. https://doi.org/10.2147/OPTH.S54021
13. van Laar JAM, Rothova A, Missotten T, Kuijpers RWAM, van Hagen PM, van Velthoven MEJ. Diagnosis and treatment of uveitis; not restricted to the ophthalmologist. *J Clin Transl Res*. 2015;1(2):94–9.
14. Gamalero L, Simonini G, Ferrara G, Polizzi S, Giani T, Cimaz R. Evidence-based treatment for uveitis. *Isr Med Assoc J*. 2019;21(7):475–9.
15. Sève P, Cacoub P, Bodaghi B, Trad S, Sellam J, Bellocq D, et al. Uveitis: Diagnostic work-up. A literature review and recommendations from an expert committee. *Autoimmun Rev*. 2017;16(12):1254–64. https://doi.org/10.1016/j.autrev.2017.10.010
16. De Smet MD, Taylor SRJ, Bodaghi B, Miserocchi E, Murray PI, Pleyer U, et al. Understanding uveitis: the impact of research on visual outcomes. *Prog Retin Eye Res*. 2011;30(6):452–70. https://doi.org/10.1016/j.preteyeres.2011.06.005
17. Omari A, Mahmoud TH. Vitrectomy. StatPearls - NCBI Bookshelf. 2023. https://www.ncbi.nlm.nih.gov/books/NBK551668/
18. Spandau U, Heimann H. Practical Handbook for Small-Gauge Vitrectomy. Springer eBooks. 2018. https://doi.org/10.1007/978-3-319-89677-9
19. Shah CP, Heier JS. YAG Laser Vitreolysis vs Sham YAG Vitreolysis for Symptomatic Vitreous Floaters. *JAMA Ophthalmol*. 2017;135(9):918. https://doi.org/10.1001/jamaophthalmol.2017.2388
20. Hahn P, Schneider EW, Tabandeh H, Wong RW, Emerson GG. Reported complications following laser vitreolysis. *JAMA Ophthalmol*. 2017;135(9):973. https://doi.org/10.1001/jamaophthalmol.2017.2477
21. Williams GA Jr, Aaberg TM Jr. Laser coagulation. *Retina*. Fourth Edition. https://www.sciencedirect.com/topics/medicine-and-dentistry/laser-coagulation
22. Pautler S. Retinal Cryopexy. Scott E. Pautler, M.D. Tampa. 2022. Available from: https://www.scottpautlermd.com/retinal-cryopexy/
23. Pneumatic Retinopexy Procedure. process.st. 2025. Available from: https://www.process.st/templates/pneumatic-retinopexy-procedure/
24. Chan CK, Lin SG, Nuthi ASD, Salib DM. Pneumatic retinopexy for the repair of retinal detachments: a comprehensive review (1986–2007). *Surv Ophthalmol*. 2008;53(5):443–78. https://doi.org/10.1016/j.survophthal.2008.06.008
25. McCluskey PJ. Regular review: management of chronic uveitis. *BMJ*. 2000;320(7234):555–8. https://doi.org/10.1136/bmj.320.7234.555
26. Lin P, Suhler EB, Rosenbaum JT. The future of uveitis treatment. *Ophthalmology*. 2013;121(1):365–76. https://doi.org/10.1016/j.ophtha.2013.08.029
27. Lyon F, Gale RP, Lightman S. Recent developments in the treatment of uveitis: an update. *Expert Opin Investig Drugs*. 2009;18(5):609–16. https://doi.org/10.1517/14728220902852570
28. Rodella U, Honisch C, Gatto C, Ruzza P, Tóthová JD. Antioxidant nutraceutical strategies in the prevention of oxidative stress related eye diseases. *Nutrients*. 2023;15(10):2283. https://doi.org/10.3390/nu15102283
29. Ma L, Lin XM. Effects of lutein and zeaxanthin on aspects of eye health. *J Sci Food Agric*. 2010;90(1):2–12. https://doi.org/10.1002/jsfa.3785
30. Fan WS, Huang SY, Nguyen HT, Ho WT, Chao WH, Lin FC, et al. Design of a functional eye dressing for treatment of the vitreous floater. *J Pers Med*. 2022;12(10):1659. https://doi.org/10.3390/jpm12101659
31. Sah MP, Parajuli S, Acharya S, Tribhuvan University, Bhusal N. A review on Netra Tarpana – An Ayurveda method of eye care. *World J Pharm Res*. 2021:1779–87. https://doi.org/10.20959/wjpr20211-19784

32. Kumar DM. Netra Tarpana: an Ocular Therapy in Ayurveda for Maintenance of Eyes. *Int J Res Appl Sci Eng Technol*. 2022;10(7):1800–3.
33. Manani YL. Pharmaceutical Evaluation of Triphala Ghrita. pharmaayurved.in. 2025. Available from: http://www.pharmaayurved.in/
34. DrHomeo.com. Eye Floaters - A homeopathic treatment for black spots before eyes. 2025. Available from: https://www.drhomeo.com/homeopathic-treatment/eye-floaters-homeopathic-remedies/
35. Vinita G. Homeopathic remedies for treating eye floaters! Lybrate. 2025. Available from: https://www.lybrate.com/topic/6-homeopathic-remedies-for-treating-eye-floaters/f636a0d8bf8a072fce75471d6f8332a1
36. Kim YK, Moon SY, Yim KM, Seong SJ, Hwang JY, Park SP. Psychological distress in patients with symptomatic vitreous floaters. *J Ophthalmol*. 2017;2017:3191576. https://doi.org/10.1155/2017/3191576

Cite this article: Revankar HS, Bobde S, Bhilegaonkar S. Vitreous floaters: review and prophylactic strategies. *Indian J Clin Exp Ophthalmol*. 2025;11(4):601–612.