VITREOUS HEMORRHAGE

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1. Core Definition and Classification

Vitreous hemorrhage (VH) is a significant and often sudden ophthalmological condition characterized by the extravasation of blood into the vitreous humor, the clear, gelatinous substance that fills the large space between the lens and the retina of the eye. This bleeding arises most commonly from the rupture of fragile, newly formed blood vessels (neovascularization) originating from the retina or optic disc. Because the vitreous is normally transparent, the presence of blood creates opacities that dramatically interfere with the transmission of light to the retina, resulting in acute, painless vision loss. The severity of the visual impairment is directly proportional to the density and volume of the hemorrhage present within the vitreous cavity.

The immediate effect of VH is the physical blockage of the visual axis. When the hemorrhage is minimal, a patient may experience only scattered floaters or mild haziness. However, in cases of dense hemorrhage, vision can be reduced to mere light perception, constituting a medical emergency requiring prompt assessment. Clinically, vitreous hemorrhages are often classified based on their location, which dictates their potential rate of absorption and associated risks. A preretinal or subhyaloid hemorrhage lies between the retina and the posterior face of the vitreous gel; these are typically well-demarcated, boat-shaped, and often clear spontaneously relatively quickly as the blood settles inferiorly.

Conversely, an intragel hemorrhage, where blood diffuses throughout the fibrous structure of the vitreous body, tends to absorb much more slowly due to the restricted motility of the blood cells within the dense gel matrix. Furthermore, VH can be categorized by chronicity--acute, recent, or old--which is important for predicting treatment response. The most frequent and medically significant cause of VH, especially in working-age adults in developed countries, is complications arising from **diabetic retinopathy**, specifically the proliferative stage where neovascularization is rampant. Understanding this underlying etiology is paramount, as recurrent hemorrhage is likely unless the root cause is addressed.

2. Pathophysiology and Mechanism of Injury

The fundamental mechanism underlying vitreous hemorrhage involves the development and subsequent rupture of abnormal, fragile blood vessels. In conditions such as **proliferative diabetic retinopathy** (PDR), chronic ischemia (lack of oxygen) in the retina triggers the release of potent angiogenic growth factors, most notably Vascular Endothelial Growth Factor (VEGF). This leads to neovascularization--the formation of new, weak blood vessels that grow along the posterior surface of the retina and extend into the vitreous gel. Unlike healthy capillaries, these new vessels lack the necessary tight junctions and pericyte support, making them extremely susceptible to leakage and rupture.

The mechanical forces exerted on these brittle vessels are typically the immediate trigger for bleeding. The most common mechanical trigger is the process of **posterior vitreous detachment (PVD)**. As the eye ages, the vitreous gel naturally liquefies and shrinks, separating from the retina. If the vitreous gel is abnormally adherent to the sites of neovascularization, the separation process can exert traction, causing a shearing force on the fragile vessels. This traction tears the vessels, allowing blood to stream into the vitreous cavity. The presence of significant fibrovascular proliferation--scar tissue intertwined with new vessels--greatly exacerbates the potential for tractional forces during PVD.

Another significant pathophysiological factor is the rapid increase in intraocular pressure or systemic blood pressure, such as occurs during strenuous activity, coughing, or the Valsalva maneuver. While these actions alone rarely cause VH in a healthy eye, they can be sufficient to rupture already compromised neovascular membranes in an eye afflicted with PDR or other vascular diseases. Furthermore, traumatic injury to the globe, whether blunt or penetrating, can directly tear retinal or choroidal vessels, leading to massive and immediate vitreous bleeding. The presence of blood subsequently initiates a complex inflammatory and oxidative cascade within the vitreous, potentially leading to long-term complications such as **ghost cell glaucoma** and hemosiderosis.

3. Primary Etiologies

While vitreous hemorrhage is a symptom rather than a primary disease, identifying its underlying cause is essential for effective management and prevention of recurrence. By far the most prevalent etiology in the adult population is **diabetic retinopathy**. Approximately 30% to 50% of all cases of spontaneous vitreous hemorrhage are attributable to the advanced stage of this condition, where extensive neovascularization has occurred. Patients with poorly controlled Type 1 or Type 2 diabetes are at the highest risk, particularly those who have failed to undergo timely panretinal photocoagulation (PRP) treatment. The extent of the underlying proliferative disease is the single strongest predictor of future hemorrhage risk.

Beyond diabetes, several other vascular and anatomical conditions contribute substantially to the incidence of VH. Retinal vascular occlusions, including **Central Retinal Vein Occlusion (CRVO)** and Branch Retinal Vein Occlusion (BRVO), are important secondary causes. These conditions lead to large areas of retinal ischemia, which, similar to diabetic retinopathy, stimulate massive VEGF production and subsequent neovascularization, often in the iris (rubeosis iridis) and retina. Other causes related to vascular pathology include retinal arterial macroaneurysms, which can rupture and bleed profusely, and sickle cell retinopathy, which causes peripheral vascular occlusion and subsequent neovascularization.

Trauma and anatomical anomalies account for the remainder of cases. Blunt trauma, such as a punch or severe fall, can cause a contrecoup injury leading to retinal tears and associated hemorrhage. Penetrating injuries directly violate the globe, inevitably causing bleeding. Furthermore, tears in the retina--often associated with **PVD**--can tear adjacent healthy vessels, leading to hemorrhage that is sometimes referred to as 'sentinel bleeding.' Rare, non-retinal causes include Terson Syndrome, characterized by intraocular bleeding secondary to sudden increases in intracranial pressure (e.g., following subarachnoid hemorrhage), and various rare inflammatory conditions.

4. Clinical Presentation and Symptoms

The defining characteristic of vitreous hemorrhage is its typically sudden and painless onset. Patients do not usually report acute ocular pain unless the hemorrhage is associated with a secondary complication, such as acute retinal detachment or neovascular glaucoma. Instead, the symptoms are purely visual, reflecting the mechanical obstruction of light transmission by the blood cells suspended in the vitreous. The presentation is highly variable, ranging from minor visual annoyance to complete blindness in the affected eye, depending on the volume and location of the bleed.

In mild to moderate cases, patients frequently report seeing "floaters," which are often described as dense, mobile spots, clouds, or "cobwebs" that drift across the visual field with eye movement. A classic description, particularly in larger bleeds, is the sensation of a "shower of soot" falling through the vision. As the hemorrhage becomes denser, the visual symptoms escalate to profound blurring, haziness, or a generalized red tint (erythropsia). If the hemorrhage is massive, the patient will experience a complete or near-complete cessation of useful vision, often perceiving only the ability to distinguish light from dark, termed 'light perception only' (LP).

During an ophthalmic examination, the physical signs are equally distinctive. The most important sign is the reduction or complete absence of the normal red reflex when examining the eye with an ophthalmoscope, as the blood blocks the view of the underlying fundus. Slit-lamp examination may reveal red blood cells (RBCs) in the anterior vitreous face, indicating a fresh, often liquid, hemorrhage. When PVD is the cause, the examiner may detect a boat-shaped hemorrhage lying anterior to the retina, corresponding to the space between the retina and the detached posterior hyaloid face. This presentation often suggests a self-limiting bleed, whereas diffuse opacity suggests a more chronic or widespread hemorrhage within the vitreous gel.

5. Diagnosis and Assessment

The initial diagnosis of vitreous hemorrhage is typically made based on the patient's history of acute, painless vision loss combined with the inability to visualize the retina clearly during ophthalmoscopy. However, the critical diagnostic task is not merely confirming the presence of blood, but determining its source and ruling out concomitant sight-threatening pathology, particularly **retinal detachment (RD)**. Since the blood opacity precludes direct visualization of the retina, specialized imaging techniques are essential.

The gold standard for assessing the eye when the fundus is obscured by dense VH is **B-Scan Ultrasonography**. This non-invasive test uses sound waves to generate a cross-sectional image of the internal ocular structures. A B-scan can accurately confirm the presence of blood (often appearing as mobile echoes within the vitreous cavity) and, crucially, determine whether the hemorrhage is complicated by a concurrent retinal detachment. The mobility of the blood echoes can also provide clues regarding the chronicity and potential for spontaneous clearing. B-scan is indispensable for surgical planning, as it provides the surgeon with a map of the retinal anatomy prior to intervention.

Once the hemorrhage begins to clear--or if the hemorrhage is initially mild--other diagnostic tools become useful for identifying the underlying cause. **Fundus photography** and **Fluorescein Angiography (FA)** are used to map the extent of underlying vascular disease, such as areas of ischemia, microaneurysms, and active neovascularization that require targeted laser treatment. Optical Coherence Tomography (OCT) is also employed once the media clears sufficiently to assess the integrity of the macula, determine the presence of tractional forces, and identify macular edema, which significantly affects visual prognosis. Systemic workup, including blood pressure monitoring and evaluation of HbA1c levels, is mandatory, especially if diabetes is suspected or confirmed as the primary cause.

6. Management and Treatment Modalities

The management strategy for vitreous hemorrhage is highly individualized, dictated by the underlying etiology, the density of the blood, and the status of the macula. Initial management for most spontaneous hemorrhages not associated with traumatic injury or immediate retinal detachment is typically conservative and involves observation. The goal of this initial phase is to allow the blood to settle and clear spontaneously, which often occurs within 2 to 6 weeks.

Conservative treatment typically involves instructing the patient to maintain a semi-recumbent position (head elevated) and restrict strenuous physical activity. Gravity helps the blood settle inferiorly, improving central vision and potentially allowing for a view of the superior retina where the bleed source often lies. The primary surgical intervention for non-clearing or complicated VH is **Pars Plana Vitrectomy (PPV)**. PPV involves surgically removing the opacified vitreous gel and blood and replacing it with a balanced salt solution, gas, or silicone oil. PPV is indicated immediately if there is evidence of a concurrent retinal tear or tractional retinal detachment, or if the hemorrhage remains dense and stationary for more than three months, significantly delaying visual rehabilitation.

Following spontaneous clearing or surgical removal of the vitreous blood, the treatment shifts focus to addressing the underlying pathology to prevent recurrence. For cases caused by PDR, this involves extensive retinal treatment. Panretinal Photocoagulation (PRP) uses laser energy to ablate the ischemic peripheral retina, thereby reducing the metabolic demand and decreasing the production of VEGF, leading to the regression of neovascularization. Alternatively, or often in conjunction with PRP, intravitreal injections of anti-VEGF agents (e.g., bevacizumab, ranibizumab) are used to rapidly suppress active neovascular growth and stabilize fragile vessels. These adjunctive treatments are crucial for ensuring long-term success and mitigating the high risk of repeat hemorrhaging associated with proliferative retinopathy.

7. Prognosis and Long-Term Outcomes

The prognosis for visual recovery following vitreous hemorrhage is highly variable and directly correlates with the etiology and the timely intervention regarding underlying retinopathy. When VH results from minor causes, such as a small PVD-related tear without significant underlying vascular disease, the prognosis is generally excellent, with most patients achieving good visual acuity once the blood clears spontaneously. However, the prognosis is guarded when the hemorrhage is secondary to advanced **proliferative diabetic retinopathy (PDR)**, especially if significant maculopathy or tractional retinal detachment has already occurred.

Long-term complications, particularly in cases of chronic or recurrent VH, can significantly limit final visual acuity. One notable complication is **ghost cell glaucoma**, which occurs when old, rigid red blood cells (ghost cells) clog the trabecular meshwork of the eye, leading to elevated intraocular pressure and potential optic nerve damage. Another risk is **ocular hemosiderosis**, resulting from the breakdown of hemoglobin, where iron deposits can cause toxic damage to the retina and permanently impair photoreceptor function, particularly if the blood remains in the vitreous cavity for six months or longer.

Ultimately, preventing recurrence is the most critical factor in securing a favorable long-term outcome. Patients who receive comprehensive treatment for their underlying proliferative disease--whether through PRP, anti-VEGF therapy, or successful PPV combined with endolaser treatment--have a much lower rate of future VH episodes. Conversely, patients with systemic conditions like poorly controlled diabetes or severe hypertension who fail to manage their systemic health often face repeat hemorrhages, requiring multiple interventions and leading to poorer ultimate visual outcomes due to cumulative retinal damage.

Further Reading

Vitreous Hemorrhage - Wikipedia
American Academy of Ophthalmology (AAO) on Vitreous Hemorrhage
National Center for Biotechnology Information (NCBI) - Vitreous Hemorrhage Etiology and Management