DETACHED RETINA

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Detached Retina (Retinal Detachment)

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1. Core Definition

A **detached retina**, medically termed **retinal detachment**, is a severe ophthalmic condition characterized by the physical separation of the neurosensory layer of the retina from the underlying retinal pigment epithelium (RPE). This crucial separation occurs in the potential space between these two layers, known as the subretinal space. The retina, the light-sensitive tissue lining the back wall of the eye, functions much like the film in a camera, translating light into neural signals that the brain interprets as vision. When the neurosensory layer detaches, it is cut off from the vital nutritional and metabolic support provided by the RPE and the underlying choroidal blood supply. This ischemia and deprivation rapidly lead to the dysfunction and eventual death of photoreceptor cells (rods and cones), resulting in partial or complete vision loss in the affected eye, making retinal detachment a true medical emergency requiring immediate surgical intervention to preserve sight. The urgency of treatment is directly related to whether the detachment involves the macula, the small central area of the retina responsible for sharp, detailed central vision.

The term "separation" refers specifically to the cleavage plane that forms between the photoreceptor layer, which is part of the neurosensory retina, and the RPE. Historically, this relationship was viewed as a strong adhesion, but physiologically, the connection relies on osmotic forces and the active transport mechanisms of the RPE to maintain a dry subretinal space. When fluid--either vitreous fluid or exudative fluid--accumulates in this space, these adhesive forces are overwhelmed, leading to the physical lifting of the retina. This mechanical disruption prevents the photoreceptors from functioning effectively, as they rely on the RPE for waste product removal and essential nutrient delivery, including Vitamin A derivatives necessary for the visual cycle. Therefore, the longer the duration of the detachment, particularly involving the central retina, the poorer the final visual acuity prognosis, even after successful reattachment surgery.

2. Anatomy and Pathophysiology

Understanding the pathology of retinal detachment requires a brief review of retinal anatomy. The retina is a complex, multilayered structure composed of ten distinct layers. The outer layers, including the photoreceptors, rely heavily on the choroid--a vascular layer beneath the RPE--for oxygen and nutrient supply. The **retinal pigment epithelium** (RPE) serves as a critical biological barrier and transport system, sitting between the highly vascular choroid and the avascular photoreceptor outer segments. The RPE also manages the phagocytosis of spent photoreceptor outer segments and contributes to the establishment of the blood-retinal barrier.

In pathological conditions, the fundamental mechanism involves the accumulation of fluid in the

subretinal space, separating the neural retina from the RPE. This fluid accumulation can arise from several distinct processes, which form the basis for classifying the different types of retinal detachment. Once separated, the photoreceptors quickly enter a state of metabolic distress. They are no longer able to recycle photopigments efficiently or receive adequate oxygenation, leading to acute cellular damage. Furthermore, the fluid itself contains inflammatory mediators which contribute to further damage and the potential for **proliferative vitreoretinopathy** (PVR), a severe complication involving the growth of contractile membranes over the retinal surface, which can lead to permanent, rigid scarring and recurrent detachment.

3. Classification and Types

Retinal detachments are fundamentally categorized into three primary types based on the underlying etiology and mechanism of fluid accumulation. Accurate classification is critical as it dictates the appropriate surgical management strategy:

Rhegmatogenous Retinal Detachment (RRD): This is the most common form, typically accounting for 90% or more of cases. The term "rhegma" means tear or break. RRD occurs when a full-thickness retinal break (a tear or hole) allows liquefied vitreous gel from the vitreous cavity to pass through the break and enter the subretinal space, thereby physically peeling the neurosensory retina away from the RPE. RRD is often preceded by **posterior vitreous detachment** (PVD), a common age-related event where the vitreous gel shrinks and separates from the retina. If the vitreous traction on the retina is strong at the point of separation, it can induce a tear, initiating the detachment process. High myopia, ocular trauma, and previous cataract surgery are significant risk factors for RRD.

Tractional Retinal Detachment (TRD): TRD occurs due to the physical pulling forces exerted by fibrovascular or fibrous membranes that form on the inner surface of the retina and contract. These membranes are typically associated with ischemic retinopathies where new, abnormal blood vessels grow in response to oxygen deprivation (neovascularization). The most prevalent cause of TRD is advanced **proliferative diabetic retinopathy**, where uncontrolled diabetes leads to the formation of these contractile membranes. Other causes include penetrating trauma or sickle cell retinopathy. In TRD, separation is due to mechanical pulling, often without an initial retinal break, although combined tractional-rhegmatogenous detachments are also common.

Exudative (or Serous) Retinal Detachment (ERD): ERD occurs when fluid leaks from damaged blood vessels in the choroid or retina and accumulates under the retina without the presence of a retinal break or tractional forces. The RPE barrier function fails, or the choroidal vessels become excessively permeable. Common systemic or ocular conditions that cause ERD include inflammatory disorders (e.g., Vogt-Koyanagi-Harada syndrome, scleritis), tumors (e.g., choroidal melanoma or metastatic disease), or severe hypertension. Treatment for ERD is often non-

surgical, focusing instead on addressing the underlying disease causing the choroidal leakage or RPE dysfunction.

4. Etiology and Risk Factors

While the immediate cause of a rhegmatogenous detachment is a retinal tear, several predisposing factors significantly increase an individual's risk of developing the condition. Age is a primary factor; as the vitreous naturally liquefies and shrinks with age (PVD), the risk of retinal tears increases. The vast majority of spontaneous RRDs occur in individuals over 50 years old. However, certain anatomical and medical conditions accelerate this risk.

High **myopia** (nearsightedness) is one of the strongest independent risk factors. Highly myopic eyes are structurally longer, which causes the retina to be stretched thinner and often leads to peripheral retinal degeneration (lattice degeneration). This thinned, compromised retina is more susceptible to tears during PVD. Additionally, ocular trauma, such as blunt force injury to the eye or head, can cause immediate tears or induce later detachments. Previous eye surgery is also a significant concern; while modern surgical techniques minimize risk, procedures like cataract surgery or YAG laser capsulotomy can precipitate PVD and subsequent retinal detachment, particularly in the months following the operation.

Systemic diseases also play a crucial role, particularly in tractional and exudative types. **Diabetes mellitus**, especially when poorly controlled over many years, leads to proliferative diabetic retinopathy (PDR), which is the leading cause of tractional retinal detachment globally. PDR involves the proliferation of fragile, abnormal vessels that anchor to the vitreous, and subsequent contraction pulls the retina off the RPE. Other conditions, such as Stickler syndrome or Marfan syndrome, which are inherited connective tissue disorders, are associated with abnormal vitreous and retinal structure, often leading to retinal detachment in younger individuals.

5. Key Symptoms and Clinical Presentation

The onset of **retinal detachment** is typically acute, and the symptoms serve as crucial warning signs that warrant immediate ophthalmic examination. The classic triad of symptoms includes photopsia, increased floaters, and a visual field defect, often described as a shadow or curtain. Recognition of these symptoms by the patient is paramount for timely intervention.

Photopsia refers to the perception of sudden, bright flashes of light. These flashes occur because the mechanical traction or pulling on the neurosensory retina stimulates the photoreceptors, causing them to fire electrical signals even in the absence of external light stimuli. Patients often report these flashes as being transient or localized to the peripheral vision. A sudden and dramatic increase in **floaters**--small specks, threads, or cobwebs that drift across the field of vision--is another hallmark. Floaters are caused by cellular debris released into the vitreous when the retina

tears, often including blood cells if a retinal vessel is torn (vitreous hemorrhage), or condensed collagen fibers associated with PVD. The sudden appearance of a large number of fine, dust-like floaters is sometimes referred to as a "shower of tobacco dust," indicative of RPE cells released into the vitreous, a severe sign of rhegmatogenous detachment.

As the detachment progresses, the patient experiences a noticeable reduction or loss of vision corresponding to the area of the detached retina. This is frequently described as a **curtain or shadow** moving across the field of vision, starting peripherally and progressing centrally. If the macula--the central area responsible for detailed vision--becomes detached, the patient will experience a profound and sudden drop in central visual acuity. Macula-off detachments carry a significantly worse prognosis for recovery of high-quality central vision, even after successful surgery, highlighting why identifying the preceding symptoms (flashes and floaters) before macula involvement is critical for optimal visual outcome.

6. Diagnosis and Examination

Diagnosis of **retinal detachment** requires a comprehensive ophthalmic evaluation by an ophthalmologist, focusing on visualizing the peripheral retina to locate the exact site of the tear and determine the extent and type of detachment. The gold standard diagnostic tool is indirect ophthalmoscopy, often performed after pupillary dilation. This technique allows the clinician to obtain a wide, stereoscopic view of the entire retina, which is essential for identifying subtle tears or holes, especially those located far in the periphery.

The examination often utilizes a **slit lamp examination** coupled with specialized contact lenses (e.g., three-mirror contact lens) to provide high magnification views of the vitreoretinal interface and peripheral retina, helping to confirm the presence of fluid, traction, or predisposing lattice degeneration. In cases where the view of the retina is obscured by media opacity, such as dense cataract or significant vitreous hemorrhage, B-scan ultrasonography is employed. This non-invasive imaging technique uses sound waves to generate cross-sectional images of the eye's internal structures, allowing for visualization of the detached retina and measurement of its extent, thereby confirming the diagnosis and guiding surgical planning.

7. Treatment Modalities

Treatment for **retinal detachment** is overwhelmingly surgical and must be performed rapidly to prevent permanent vision loss. The choice of procedure depends heavily on the type of detachment, the location and number of retinal breaks, and the presence of proliferative vitreoretinopathy (PVR). The primary goal of all surgical techniques is to reattach the neurosensory retina to the RPE and seal the retinal break(s).

One common approach is the **Scleral Buckle (SB)** procedure. This involves placing a silicone

element (a sponge or band) onto the sclera (the white outer wall of the eye) outside the eye. This element indents the eyewall inward, effectively pushing the RPE and choroid closer to the detached retina, thereby closing the retinal break and relieving vitreous traction. Cryopexy (freezing) or laser photocoagulation is then applied around the break to create a permanent scar that welds the retina to the underlying tissue. The subretinal fluid is often drained concurrently.

A second major technique, often preferred for complex detachments, vitreous hemorrhage, or PVR, is **Pars Plana Vitrectomy (PPV)**. In this procedure, the vitreous gel is surgically removed through small incisions in the pars plana (the area just behind the iris). Removing the vitreous relieves traction on the retina. The surgeon then internally drains the subretinal fluid, flattens the retina, and applies endolaser photocoagulation around the breaks. To hold the retina in place while the laser scars form, the vitreous cavity is filled with a temporary tamponade agent, such as a gas bubble (sulfur hexafluoride or perfluoropropane) or silicone oil. The gas bubble is naturally absorbed by the body, while silicone oil requires a second surgical procedure for removal.

A simpler, often outpatient procedure suitable for specific, uncomplicated RRDs is **Pneumatic Retinopexy (PR)**. This involves injecting a small gas bubble directly into the vitreous cavity, followed by positioning the patient's head so that the bubble floats up and presses against the retinal break. Cryopexy or laser is then applied to seal the break. PR relies heavily on patient cooperation to maintain specific head postures for several days post-procedure to ensure the bubble properly seals the break.

8. Further Reading

Retinal Detachment: Symptoms and Causes (Mayo Clinic)
Retinal Detachment (Wikipedia)
Understanding Retinal Detachment (American Academy of Ophthalmology)