

Retinal Migraine

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Retinal Migraine

Primary Disciplinary Field(s): Neurology, Ophthalmology

1. Core Definition

A Retinal Migraine (RM), also known historically as ophthalmic migraine or monocular visual disturbance preceding a headache, is a rare and distinct form of migraine characterized by repeated bouts of transient, monocular visual disturbance, which may include partial or complete vision loss. Unlike the common visual aura associated with a typical migraine with aura, which usually affects both visual fields (though often perceived as one-sided), the visual symptoms of RM are strictly confined to one eye. The International Headache Society (IHS) classifies Retinal Migraine under the category of complicated migraines, specifically defined by the presence of monocular visual symptoms fully reversible within one hour, confirmed by examination, and often associated with a subsequent headache phase. This condition is crucial to differentiate from other forms of temporary vision loss, such as transient ischemic attacks (TIA) or amaurosis fugax, which signal vascular pathology that requires immediate medical intervention. The rarity of the condition necessitates a thorough diagnostic workup to exclude underlying serious ophthalmic or vascular disorders.

The visual symptoms experienced during a Retinal Migraine episode are fundamentally similar to those described in classical migraine aura but are strictly unilateral. Patients frequently report phenomena such as **scintillations** (shimmering or flashing lights), **scotoma** (temporary blind spots), or, in severe cases, complete **monocular blindness**. While the source content noted that these disturbances generally occur without actually disrupting functional vision--a key characteristic of typical aura--Retinal Migraine is defined precisely by the disruption of functional vision in one eye, though this disruption is by definition temporary. The episode of visual loss typically resolves spontaneously within minutes, but rarely may last up to sixty minutes. The transient nature of the symptoms, combined with the presence of classic migraine triggers and the subsequent or concurrent headache, helps to establish the diagnosis of RM.

The diagnosis of Retinal Migraine is primarily clinical, adhering to the stringent criteria set forth by the IHS. A critical component of this definition is the reversible nature of the visual deficit; any permanent vision loss stemming from a migraine episode would typically be classified as a retinal infarction (stroke) secondary to migraine rather than a Retinal Migraine itself. Furthermore, although the visual symptoms and triggers often share commonalities with **ocular (acephalic) migraines**, the defining feature of RM remains the strict unilaterality of the visual event, confirming its origin within the vascular supply or neuronal activity of the affected retina or optic nerve pathway specific to that eye.

2. Etymology and Historical Development

The concept of a migraine affecting the eye specifically has been recognized for centuries, though precise delineation from other types of transient vision loss is relatively modern. Early physicians often grouped all forms of transient monocular visual obscuration, or amaurosis fugax, together. It was only through increasing specialization in neurology and ophthalmology during the 20th century that distinctions began to be drawn based on associated symptoms and outcomes. The term "retinal migraine" emphasizes the presumed localization of the underlying pathology--specifically, within the **retinal circulation** or nerve layer--as opposed to the cerebral cortex, which is the site of typical migraine aura.

Initial attempts to classify these monocular events were hampered by the difficulty in visually observing the retinal circulation during an acute attack. Researchers hypothesized that these symptoms were caused by a transient vasospasm of the central retinal artery or its branches, leading to temporary ischemia of the retinal tissue. This hypothesis underpinned much of the early nomenclature. The introduction of standardized classification systems, such as those developed by the IHS, provided the necessary structure to distinguish RM definitively from other neurological and vascular events. The third edition of the International Classification of Headache Disorders (ICHD-3) formalized the diagnostic criteria, insisting on specific reversibility and recurrence to secure the diagnosis.

The historical difficulty in diagnosis stems from the necessity of ruling out more common and dangerous causes of monocular vision loss. Prior to modern imaging and diagnostic techniques, many cases of TIA or carotid artery disease presenting with monocular symptoms might have been misattributed to benign migraine events. The evolution of the diagnosis reflects a growing understanding that while RM is benign in terms of systemic vascular risk compared to TIA, it still represents a localized vascular instability that warrants careful clinical evaluation to prevent potential complications, including permanent visual field defects or retinal infarction, though these outcomes remain exceptionally rare.

3. Key Characteristics

Retinal Migraine possesses several unique characteristics that differentiate it from the far more common migraine with visual aura. The fundamental distinction is the **monocular presentation**; the visual disturbance is strictly perceived in only one eye, and if that eye is closed, the disturbance vanishes. This contrasts sharply with typical migraine aura, which is defined by visual phenomena that cross the midline and affect both eyes' visual fields simultaneously, indicating a cortical (occipital lobe) origin.

Monocular Visual Symptoms: The visual disturbance is confined to a single eye. Symptoms range from positive phenomena (e.g., shimmering lights, fortification spectra) to negative

phenomena (e.g., scotoma or complete blindness, known as amaurosis). The intensity can vary dramatically between episodes.

Reversibility and Duration: Symptoms are transient, typically lasting between five and sixty minutes. The complete and full reversibility of the visual deficit is a mandatory criterion for diagnosis. Prolonged vision loss suggests a complication, such as retinal infarction.

Association with Headache: The visual episode usually occurs immediately before or during the headache phase of a typical migraine attack. The headache itself often possesses classic migraine features: unilateral, pulsating, moderate to severe intensity, and exacerbated by physical activity. However, some patients may experience the monocular visual symptoms without an associated headache, though this is less common.

Triggers: RM shares common triggers with other forms of migraine, including stress, hormonal fluctuations (especially in women), sleep deprivation, certain foods (e.g., aged cheese, nitrates), and intense exercise. Exposure to bright light or high altitudes may also precipitate an attack.

Furthermore, the recurrent nature of the episodes is characteristic. A diagnosis of Retinal Migraine requires at least two attacks fulfilling the strict diagnostic criteria. The recurrence rate is highly variable among individuals, ranging from infrequent attacks separated by years to monthly episodes. Given the potential for irreversible visual damage, even mild, recurrent symptoms necessitate ongoing monitoring by both a neurologist and an ophthalmologist.

4. Pathophysiology and Causes

The underlying pathophysiology of Retinal Migraine remains a subject of ongoing debate, though the leading theory centers on **vasospasm** within the retinal arterial system. This hypothesis suggests that similar to the cerebral vasoconstriction and subsequent vasodilation seen in the brain during a classic migraine, the retinal arteries undergo a temporary, severe constriction. This constriction reduces blood flow (ischemia) to the retina, leading to the transient loss of photoreceptor function and subsequent visual symptoms. Once the spasm releases, blood flow is restored, and vision returns entirely.

An alternative, though less widely accepted, theory proposes that Retinal Migraine is an ophthalmic manifestation of **Cortical Spreading Depression (CSD)**, the underlying neurological event thought to generate typical migraine aura. CSD involves a slow wave of neuronal and glial depolarization that spreads across the cortex. If this wave were somehow able to propagate into the retina--which is, embryologically, an extension of the central nervous system--it might cause the transient functional disturbance specific to one eye. However, the direct evidence supporting retinal CSD is limited, and the vascular theory remains more robust due to observational evidence of retinal artery narrowing during acute attacks in some patients, though such observations are

rare.

There are also contributing factors related to vascular vulnerability. Patients with RM are sometimes found to have underlying conditions that predispose them to vascular instability, such as Raynaud's phenomenon, or evidence of platelet aggregation abnormalities. While RM is generally considered benign, the fact that it involves temporary ischemia means that individuals with pre-existing vascular risk factors (e.g., hypertension, hyperlipidemia, smoking) are theoretically at higher risk for complicated episodes leading to permanent damage. This highlights the importance of managing systemic vascular health in patients diagnosed with Retinal Migraine.

5. Clinical Presentation and Diagnosis

The clinical presentation of Retinal Migraine is dramatic, often causing significant anxiety for the patient due to the sudden, temporary loss of vision. The patient typically reports that the vision in one eye becomes affected--often described as looking through muddy water, seeing a patchy blind spot, or experiencing complete darkness (**amaurosis**). These symptoms are highly characteristic and must be distinguished from the binocular visual symptoms of typical migraine aura, where the patient may perceive a zigzag pattern or shimmering lights that respect the vertical midline.

The diagnostic process requires a thorough exclusion of serious vascular pathology. Because transient monocular vision loss (TML) is a hallmark symptom of several potentially life-threatening conditions, clinicians must rule out these differential diagnoses urgently. Key conditions to exclude include: **carotid artery dissection**, TIA due to emboli from the carotid arteries or heart (leading to amaurosis fugax), optic neuritis, and in older patients, giant cell arteritis (GCA). This usually involves extensive testing, including neuroimaging (MRI/MRA), cardiac evaluation, and Doppler ultrasound of the carotid arteries to assess for stenosis or plaques.

The formal IHS ICHD-3 criteria for Retinal Migraine mandate the following:

At least two attacks fulfilling criteria B and C.

Migraine with aura criteria fulfilled for the aura, which is characterized by fully reversible monocular positive and/or negative visual phenomena (e.g., scintillations, scotoma, or blindness).

Visual symptoms confirmed to be monocular during an attack by the patient's description (i.e., symptoms persist when the unaffected eye is covered) or by clinical examination.

Headache fulfilling criteria for Migraine with or without aura begins during the visual symptoms or within 60 minutes thereafter.

Not better accounted for by another ICHD-3 diagnosis, particularly TIA or other vascular causes.

Only after exhaustive testing confirms the absence of underlying cardiovascular or embolic sources for the TML can a definitive diagnosis of Retinal Migraine be safely assigned.

6. Treatment and Management

Management of Retinal Migraine is generally divided into acute abortive treatment and long-term prophylactic management, particularly because the risk, however small, of permanent vision loss is present. The primary goal of acute treatment is to address the associated headache and shorten the episode, while prophylactic treatment aims to reduce the frequency and severity of attacks.

For the acute episode, standard migraine treatments are often used for the headache phase. However, there is controversy regarding the use of vasoconstrictive medications, such as **triptans** (e.g., sumatriptan) and ergot alkaloids, during the acute visual phase of RM. Since the underlying cause is hypothesized to be vasospasm, administering a potent vasoconstrictor during an RM attack theoretically carries a risk of worsening retinal ischemia or prolonging the attack, potentially increasing the risk of retinal infarction. Therefore, many clinicians advise caution or contraindicate the use of these drugs until the visual symptoms have fully resolved. Instead, simple analgesics or Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) may be recommended for headache relief.

Preventive therapy is typically initiated if attacks are frequent (more than two per month), disabling, or if there is concern about progression to permanent vision loss. Prophylactic agents used for RM overlap significantly with those used for other migraine types:

Beta-blockers: Such as propranolol, which help stabilize vascular reactivity.

Calcium Channel Blockers: Drugs like verapamil, which are thought to be particularly effective in vascular-mediated migraines and may directly counter retinal vasospasm.

Anticonvulsants: Topiramate and valproate may also be used, depending on co-morbidities.

Lifestyle modification, including trigger identification and avoidance, stress management, and maintaining regular sleep and dietary schedules, is also crucial for reducing the frequency of Retinal Migraine episodes.

7. Significance and Impact

Retinal Migraine, while rare, carries significant clinical weight primarily due to its potential as a mimic of more serious conditions and the psychological burden it places on patients. The sudden onset of blindness, even temporary, is intensely frightening and often leads patients to seek emergency medical care, contributing to the strain on healthcare systems through extensive diagnostic workups designed to rule out stroke or TIA.

Furthermore, RM serves as an important conceptual link between neurological disorders (migraine) and vascular disorders (retinal ischemia). Its study provides insights into the complex interplay between the central nervous system's control of vascular tone and the peripheral circulation. For

the individual patient, the impact of RM goes beyond acute visual loss; the constant fear of recurrence and the small but real risk of permanent vision impairment severely impact quality of life. Counseling and reassurance, emphasizing the general reversibility of the condition once serious causes are excluded, are vital components of the long-term management strategy.

8. Debates and Criticisms

A significant debate surrounding Retinal Migraine centers on whether it is a distinct, retinal-based entity or simply a severe form of typical visual aura that the patient misinterprets or localizes to one eye. Critics argue that true, transient vasospasm severe enough to cause ischemia in the retina without causing permanent damage is physiologically questionable, given the end-artery nature of the retinal circulation. They suggest that some cases diagnosed as RM may actually be atypical forms of cortical aura that only temporarily affect the visual pathway supplying one eye's visual field, or they represent benign cases of TML not truly related to the migraine mechanism.

Another critical debate involves the safety of vasoconstrictive treatments. As discussed, the theoretical risk of inducing permanent retinal infarction when treating an acute RM attack with triptans is a major clinical concern, leading to varied practice patterns among neurologists and ophthalmologists. This pharmacological controversy underscores the need for clear, evidence-based guidelines regarding acute abortive therapy specific to RM, differentiating it from standard migraine treatment protocols. Until clearer pathogenesis is established, physicians must weigh the benefit of treating the associated debilitating headache against the potential, albeit low, risk of precipitating permanent vision loss.

Further Reading

[International Classification of Headache Disorders \(ICHD-3\)](#)

[American Academy of Ophthalmology: Retinal Migraine](#)

[National Center for Biotechnology Information \(NCBI\): Pathophysiology and Clinical Management of Retinal Migraine](#)