

Most Cases Labeled as “Retinal Migraine” Are Not Migraine

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Background: Monocular visual loss has often been labeled “retinal migraine.” Yet there is reason to believe that many such cases do not meet the criteria set out by the International Headache Society (IHS), which defines “retinal migraine” as attacks of fully reversible monocular visual disturbance associated with migraine headache and a normal neuro-ophthalmic examination between attacks.

Methods: We performed a literature search of articles mentioning “retinal migraine,” “anterior visual pathway migraine,” “monocular migraine,” “ocular migraine,” “retinal vasospasm,” “transient monocular visual loss,” and “retinal spreading depression” using Medline and older textbooks. We applied the IHS criteria for retinal migraine to all cases so labeled. To be included as definite retinal migraine, patients were required to have had at least two episodes of transient monocular visual loss associated with, or followed by, a headache with migrainous features.

Results: Only 16 patients with transient monocular visual loss had clinical manifestations consistent with retinal migraine. Only 5 of these patients met the IHS criteria for definite retinal migraine. No patient with permanent visual loss met the IHS criteria for retinal migraine.

Conclusions: Definite retinal migraine, as defined by the IHS criteria, is an exceedingly rare cause of transient monocular visual loss. There are no convincing reports of permanent monocular visual loss associated with migraine. Most cases of transient monocular visual loss diagnosed as retinal migraine would more properly be diagnosed as “presumed retinal vasospasm.”

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After the development of the ophthalmoscope permitted visualization of the retina in vivo, Galezowski (1) observed retinal changes suggesting infarction in patients presenting with visual symptoms and presumed migraine headaches. Working with Charcot in Paris, Galezowski had heard Féré (2,3), one of Charcot’s residents, report that cerebral infarction may be related to migraine. Galezowski (4) hypothesized that a permanent “retinal or optic nerve affection,” similar to changes described in the brain by Féré, could be secondary to migraine. In 1892, Galezowski (4) used the term “ophthalmic megrim” to describe permanent monocular visual loss associated with migraine headache. Subsequently, Fisher (5,6) in 1952 and 1971 and Walsh and Hoyt (7) in 1969 suggested that the eye itself can be affected by migraine.

In 1970, Carroll (8) introduced the term “retinal migraine” to describe 15 patients with transient and persistent monocular visual loss. None of the patients, however, had associated headaches. Authors have since used the term “retinal migraine” to describe a multitude of monocular visual symptoms, including events without associated headache and those resulting in persistent visual loss. Some authors have used other terms, including “anterior visual pathway migraine,” “ocular migraine,” “ophthalmic migraine,” and “monocular migraine” (9–17).

In the original (1988) classification of headache, the International Headache Society (IHS) included “retinal migraine” as a subtype of migraine (18) and provided strict diagnostic criteria. These criteria stated that retinal migraine could be diagnosed only in the presence of fully reversible monocular visual disturbances associated with

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typical migraine headache and with a normal neuro-ophthalmic examination between attacks. As with all forms of migraine, other causes had to be excluded. The revised 2004 IHS classification (19) also included retinal migraine. As in the 1988 classification, retinal migraine was considered sufficiently atypical as an aura that it was not classified under “migraine with aura” but listed as a separate entity (Table 1). The diagnostic criteria were unchanged (Table 2), again requiring “at least two attacks” of “fully reversible” monocular visual symptoms “associated with migraine headache” (Table 3).

Spreading depression (SD), as described by Leão (20) in 1944, is an excitation wave followed by depression of neuronal activity propagating through gray matter with a velocity of approximately 3 mm/min that investigators have observed in almost all gray matter regions of the central nervous system (21). Functional imaging and magnetoencephalographic studies strongly suggest that cortical SD constitutes the biological basis for the occipital aura that precedes headache in migraineurs (22,23). SD is often cited as the cause of retinal migraine.

TABLE 1. Classification of migraine according to the International Headache Society criteria

- 1.1 Migraine without aura
- 1.2 Migraine with aura
 - 1.2.1 Typical aura with migraine headache
 - 1.2.2 Typical aura with non-migraine headache
 - 1.2.3 Typical aura without headache
 - 1.2.4 Familial hemiplegic migraine
 - 1.2.5 Sporadic hemiplegic migraine
 - 1.2.6 Basilar-type migraine
- 1.3 Childhood periodic syndromes that are commonly precursors of migraine
 - 1.3.1 Cyclical vomiting
 - 1.3.2 Abdominal migraine
 - 1.3.3 Benign paroxysmal vertigo of childhood
- 1.4 **Retinal migraine**
- 1.5 Complications of migraine
 - 1.5.1 Chronic migraine
 - 1.5.2 Status migrainosus
 - 1.5.3 Persistent aura without infarction
 - 1.5.4 Migrainous infarction
 - 1.5.5 Migraine-triggered seizure
- 1.6 Probable migraine
 - 1.6.1 Probable migraine without aura
 - 1.6.2 Probable migraine with aura
 - 1.6.3 Probable chronic migraine

Adapted from The International Classification of Headache Disorders, 2nd edition (19).

We have reviewed the reported cases attributed to retinal migraine to determine whether they meet the strict criteria set out by the IHS.

METHODS

Using Medline and textbooks, we searched for articles mentioning “retinal migraine,” “anterior visual pathway migraine,” “ophthalmic migraine,” “ocular migraine,” “retinal vasospasm,” “transient monocular visual loss,” “visual loss and migraine,” “visual field defects in migraine,” and “retinal spreading depression.” Applying the most recent IHS criteria (Table 2) (19) for retinal migraine to all reported patients, we separated them into three categories: definite retinal migraine, probable retinal migraine, and possible retinal migraine (Table 4). Articles were not included in Table 4 if they lacked sufficient information for adequate classification. We also excluded patients with underlying diseases known to produce migraine-like symptoms, such as cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), antiphospholipid antibody syndrome, or systemic lupus erythematosus (24–27).

To be included as definite retinal migraine, the patients must have had at least two episodes of transient monocular visual loss associated with or followed by a migraine headache.

RESULTS

We discovered 60 articles describing 142 patients with transient or persistent visual symptoms attributed to

TABLE 2. International Headache Society criteria for retinal migraine

- Description: Repeated attacks of monocular visual disturbance, including scintillations, scotomata, or blindness, associated with migraine headache
- Diagnostic criteria:
- A. At least two attacks fulfilling criteria B and C
 - B. Fully reversible monocular positive and/or negative visual phenomena (scintillations, scotomata, or blindness) confirmed by examination during an attack or (after proper instruction) by the patient’s drawing of a monocular field defect during an attack
 - C. Headache, fulfilling criteria B–D for 1.1 *Migraine without aura** begins during the visual symptoms or follows them within 60 minutes
 - D. Normal ophthalmologic examination between attacks
 - E. Not attributed to another disorder†

Adapted from The International Classification of Headache Disorders, 2nd edition (19).

*See Table 3.

†Other causes of transient monocular visual loss must be excluded.

TABLE 3. International Headache Society criteria for migraine without aura

Description: Recurrent headache disorder manifesting in attacks lasting 4–72 hours. Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity, and association with nausea and/or photophobia.

Diagnostic criteria:

- A. At least 5 attacks fulfilling criteria B–D*
- B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated)
- C. Headache has at least two of the following characteristics:
 1. Unilateral location
 2. Pulsating quality
 3. Moderate or severe intensity
 4. Aggravation by or causing avoidance of routine physical activity (for example, walking or climbing stairs)
- D. During headache at least one of the following:
 1. Nausea and/or vomiting
 2. Photophobia and phonophobia
- E. Not attributable to another disorder

Adapted from The International Classification of Headache Disorders, 2nd edition (19). Migraine without aura is 1.1 in this classification.

*The criteria for headache associated with retinal migraine require “at least two attacks fulfilling criteria B and C” in Table 2.

retinal migraine (4–13,24–57). Among these 142 patients, 39 (from 25 articles) had persistent visual loss (4,6–8, 11,25,27–45). Among these 39, there was central retinal artery occlusion in 11 (4,6,7,11,25,33,36,42), cilioretinal artery occlusion in 4 (38,43), branch retinal artery occlusion in 9 (31,35,37,39,43), focal retinal ischemia in 1 (32), central retinal vein occlusion in 2 (27,40), ischemic optic neuropathy in 6 (34,41,44), optic atrophy in 5 (4,28), and no explanation in 1 (6,45). Also among these 39 patients with persistent visual loss, 10 (4,6,7,27,31,32,33,34,45) initially presented with recurrent transient monocular visual loss associated with headaches consistent with presumed retinal migraine; these 10 patients developed permanent visual loss from 6 weeks to 20 years after the onset of transient visual loss.

Of the 103 patients with transient visual loss attributed to retinal migraine, only 16 had clinical manifestations that were actually consistent with retinal migraine (Table 4) (45–53).

Among the many articles attributing transient monocular visual loss to retinal migraine (or equivalent terms for this condition), we found 12 patients with well-documented segmental retinal vasospasm of arteries or veins

evident on ophthalmoscopy during an attack of transient monocular visual loss (7,13,56–63). Only 1 of these 12 patients had headache during or immediately after the visual loss (57), but the pain did not conform to IHS-defined migraine. Two of the patients had a history of migraine without aura (56,63), and one had previous episodes of cluster headache (13), but no headaches temporally associated with the monocular visual loss. The duration of visual loss varied from seconds to 4 hours, but most episodes lasted a few minutes. All but one patient (59) had only negative visual phenomena.

DISCUSSION

Our review indicates that retinal migraine, as currently defined in the IHS classification, is exceedingly rare. We acknowledge that a literature review can only approximate accuracy, because many reports had incomplete descriptions or were published before the 1988 IHS Headache Classification. In addition, not all patients with retinal migraine have been reported. The authors of this article have examined several patients with monocular visual loss who meet the criteria for a diagnosis of retinal migraine.

The typical visual aura of migraine occurs in a hemifield rather than a single eye, given that the aura originates from the occipital lobe (primary visual cortex) (64). Many patients report this visual experience as monocular (19,65,66), as they may attend only to the visual phenomena seen in the temporal field, perhaps because the temporal field is larger than the nasal field. Headache typically follows the aura, although there can be a migrainous aura without headache (18,19). Gradually expanding binocular scintillations, scotomas, and zig-zag lines (“scintillating scotoma” or “fortification scotoma”), with a duration usually between 5 and 20 minutes and not more than 60 minutes, are diagnostic of a migrainous visual aura (64,65,67), particularly if the aura is followed by headache. In 1971, Richards (67) attributed the zig-zag visual phenomena in the aura to the columnar organization of the visual cortex.

By contrast, monocular visual phenomena typically originate in the retina, choroid, or optic nerve (68). The positive visual phenomena are usually simpler than those that originate in the occipital lobe. They consist of phosphenes, flashing lights, flickering, or a “rain shower” (68–74). Because these visual manifestations may be caused by any process that impairs ocular or optic nerve blood flow, IHS criteria require that “other causes” be excluded before establishing migraine as the diagnosis (18,19,75).

The attribution of monocular visual loss to migraine is based on the concept of retinal SD, first described by Gouras (76) in 1958 in the frog retina. In 1966, Martins-Ferreira and de Castro (77) recorded similar changes in the

TABLE 4. Previously published patients with transient monocular visual loss suggesting retinal migraine

	Authors (year)	Age/ Gender	Number of episodes	Eye involved	Type of visual symptoms	Duration of visual symptoms	Headaches	Side of headache
1	Grosberg et al. (2006)	42/W	Multiple	OS	Positive and negative	7–8 minutes	Follow visual symptoms	NA
2	Grosberg et al. (2006)	35/M	Multiple	OS	Negative	5 minutes	Follow visual symptoms	Ipsilateral
3	Gan et al. (2005)	40/M	Multiple	Either	Negative	5–10 minutes	Follow visual symptoms	NA
4	Guidetti et al. (2005)	9/M	Multiple	OD	Negative	2–3 minutes	During or following visual symptoms. Some without headache	Bilateral
5	Lewinshtein et al. (2004)	12/W	Multiple	OD	Positive and negative	<30 minutes	Follow visual symptoms	Ipsilateral
6	Lewinshtein et al. (2004)	13/M	Multiple	OD	Positive and negative	3 minutes	No details	NA
7	Hachinski et al. (1973)	17/M	Multiple	Either	Positive and negative	15 minutes	No details	Ipsilateral
8	Corbett (1983)	26/M	Multiple	Either	Negative	Seconds to minutes	Some with binocular visual aura. Some without headache	NA
9	Joffe (1971)	18/M	Multiple	OD	Negative	NA	Some with binocular visual aura	Ipsilateral
10	Joffe (1971)	18/M	Multiple	Either	Negative	NA	No details	Ipsilateral
11	Joffe (1971)	34/M	Multiple	OS	Negative	NA	Some with binocular visual aura	Bilateral
12	Kupersmith et al. (1979)	53/W	2	OS	Negative	60 minutes	Some with binocular visual aura	Ipsilateral
13	Kupersmith et al. (1979)	21/M	2	OD	Negative	5 minutes	No details	NA
14	Kupersmith et al. (1987)	26/W	Multiple	OD	Negative	60 minutes	No details	Ipsilateral
15	Kupersmith et al. (1987)	16/W	Multiple	OD	Negative	NA	No details	Ipsilateral
16	Kupersmith et al. (1987)	28/W	Multiple	OS	Negative	20 minutes	Some with binocular visual aura	Bilateral

NA, Not available. Only patients with at least two episodes of transient monocular visual loss and headaches suggestive of migraine are included. None of these patients had permanent visual loss. Patients 1–5 had retinal migraine fulfilling IHS-ICHD2 criteria. We designated these patients as “definite retinal migraine.” Patients 6–7 had headaches that could not be diagnosed definitely as migraine, as defined by IHS-ICHD2. We designated these patients as “probable retinal migraine.” Patients 8–16 are reported in insufficient detail and so are designated “possible retinal migraine.” Most had migraine with binocular visual aura, as well as monocular episodes. Patient 6 had a nerve fiber layer infarct during an episode of transient monocular visual loss.

optical signal during SD in the chick retina. This model of SD has since been extensively investigated in vitro, mostly on the chick retina (78). The technique involves removal of the eye from the animal, followed by separating the retina from the vitreous and placing the eyecup in buffered solution. Mechanical stimulation is applied to the periphery of the retina and a wave, similar to that described by Leão (20) propagates in a circular path through the whole tissue. This wave is accompanied by a reversible voltage shift that induces changes in the intrinsic optical properties of the tissue (20,23). Variations in light scattering allow the

observer to visualize the circling wave as an enlarging dark circle invading the retinal tissue (Fig. 1) (77,78).

Retinal SD has only been demonstrated in vitro in avascular chick and frog retina. No in vivo models have demonstrated retinal SD, nor has it been demonstrated in any animal with retinal vasculature. The isolated retina in animal experiments is not representative of the human eye in vivo, which is vascularized and supported by the highly vascular choroid. SD would seem unlikely in the choroid, a blood-filled sponge, or optic nerve, where dysfunction generally produces visual loss and rarely simple

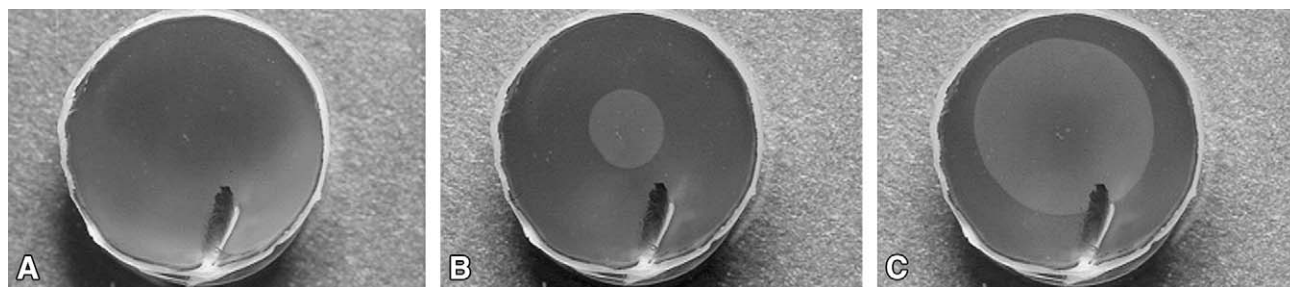


FIG. 1. Photographic appearance of light-scattering changes produced by retinal spreading depression in a chick eyecup immersed in bathing solution at different time intervals after mechanical stimulation at the center of the delineated circle. **A.** Before stimulation. **B.** 40 seconds after stimulation. **C.** 100 seconds after stimulation. (Adapted from: Martins-Ferreira and Nedergaard (78) with permission from Elsevier.)

phosphenes. Although some of the monocular visual symptoms reported as retinal migraine progress and propagate at the same speed as cortical SD, retinal SD has never been demonstrated in mammals. Scientists studying retinal SD have never suggested any clinical correlation with migraine in humans.

Retinal vasospasm can produce transient monocular visual loss. However, in the vast majority of well-documented cases of retinal vasospasm, the clinical presentation is not suggestive of migraine. Only 1 of the 12 well-documented patients with ophthalmoscopically seen vasospasm uncovered in our search had headache during or immediately after the visual loss (57), and the pain did not conform to IHS-defined migraine. All but one of these patients (59) had only negative visual phenomena.

Based on our review, we find no basis for considering migraine to be the cause of permanent monocular visual loss unless the patient had previous transient monocular visual episodes consistent with IHS-defined retinal migraine. Two recent reviews suggesting that permanent monocular visual loss is present in up to 50% of patients with retinal migraine included patients who did not have IHS-defined retinal migraine before the visual loss (45,79). The various retinal lesions reported in presumed retinal migraine include central retinal artery occlusion, branch retinal artery occlusion, localized retinal ischemia, central retinal vein occlusion, and optic atrophy. These conditions cannot reasonably be explained by a single unifying mechanism such as SD. What authors have referred to as “retinal migraine” probably represents a large heterogeneous group of underlying disorders involving the retina, choroid, or optic nerve.

Based on our review, retinal migraine is unlikely to be the cause of transient or persistent monocular visual loss. Most reported cases attributed to this condition have not met strict IHS criteria. Moreover, there are no studies in humans to suggest that the retina is subject to SD, the process believed to underlie the binocular visual aura emanating from the visual cortex in migraine. We suggest that most patients reported to have had retinal migraine as

the cause of transient monocular visual loss would be better labeled as having had “presumed retinal vasospasm (80).”

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