Case 1: A 55-year-old male presented with a two- to three-day history of flashing white lights in the periphery of his vision. The flashes were very quick, lasting only a few seconds, and were associated with a “fly” in his vision. He is nearsighted and was wearing glasses at the time of the visit. He denied a history of head or ocular trauma. His medical history includes mild hypertension and he was on a mild diuretic.

Case 2: A 30-year-old female experienced an episode of flashing lights that lasted 20 minutes, starting at the periphery of the vision in each eye and moving towards the centre. The lights were white, zigzagged and she noticed that when she focused on the image, it appeared to move, “like pixels — you know when they are trying to hide someone’s face on television.” The vision had resolved itself upon arrival to the office and the patient was experiencing a slight headache. She is otherwise healthy and using an oral contraceptive.

Case 3: A 66-year-old female presented with a history of split-second flashes of white light and transient episodes of decreased visual acuity. She could not describe the flashes well, only that they were like twinkles rather than sharp flashes, and white. The greying out of her vision was far more disturbing than the lights. She is hypertensive and taking three medications.

Case 4: A 24-year-old female presented with a history of multiple-coloured evanescent balls of light that were constantly in motion in her central field. Her normal vision is severely impaired. The episodes lasted approximately 30 minutes and were followed by a headache. She was not taking medication and is otherwise healthy.

What’s your diagnosis?
WHAT’S YOUR DIAGNOSIS?

These four cases represent the kind of cases that appear in my office. The five essentials to diagnosis are: history, history, history, physical examination and diagnostic tests, in that order.

The first case is the very typical flashing lights related to vitreous degeneration. The appearance of the light being white, arc-like, in the periphery of the vision, and lasting only a split-second is the norm. The patient will report that they think they see “a reflection off of their glasses” or a lightning bolt. The flash will often appear when they go into a dark room, just before they turn the overhead light on. The flashes may be exacerbated by rapid eye movement, and may even occur when eyes are closed. The appearance of a typical floater afterwards is the giveaway.

Vitreous degeneration occurs in myopes at an earlier age, but will likely occur in most people at some time. The vitreous is formed in the sixth week of gestation and is necessary for the formation of the primitive retina. It pushes the two layers of the primitive optic cup together, and allows for the formation of the anterior portion of the fetal eye. The vitreous fills the posterior chamber during adult life, contains a few cells, but has limited function. Indeed, after surgical removal of the vitreous (known as vitrectomy) the eye still functions very well. Unfortunately, the vitreous does not stay in its intact form throughout life, and tends to disintegrate. Bits of condensed vitreous become apparent as floaters in the gel. The vitreous body, however, is contained within a sac that is loosely adherent to the macula, retinal vessels, and optic disc. It is tightly attached to the vitreous base, behind the lens. As the vitreous degenerates, it collapses and traction can occur between it and the underlying retina. The retina, being a specific sensory organ that only perceives light, “cries out” when mechanically pulled —“Light, light, light” — hence the flashes. As long as the vitreous traction does not result in a retinal hole, there is no treatment. If, however, there is a hole in the underlying
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retina and fluid to percolate into the hole, a retinal detachment will result (Figure 1). The hope is to identify the hole, and seal it with laser surgery or cryopexy prior to detachment. A patient with symptoms suggestive of vitreous degeneration must immediately have a careful peripheral retinal examination.

The second case is recognized as migraine aura, which can occur in three forms. First, the typical or classic migraine aura starts at the temporal periphery of each eye and progresses to the centre. The lines may be irregular but moving; some people report it to be “like heat on a road” or obscuration of fine details (pixels) centrally. The episodes last 20 to 30 minutes and are typically followed by headache, nausea, vomiting or photophobia. The visions are caused by the release of catecholamines which produce vasospasms and the result in anoxia to the vision centres (usually occipital). The subsequent vasodilatation and release of kinins cause the headache, nausea, and photophobia. There are frequently migraine triggers involved that may include diet, barometric pressure changes, hormonal influences, or even stress. I ask my patients to keep a diary of these episodes, and include everything eaten in the 24-hour period preceding the attack, include weather details, and include any stresses they may have been under at that time.

The second, less common form is the acephalic migraine. This is a migraine without a headache. Again, the typical aura lasts 20 to 30 minutes, but no headache follows. The aura is predominantly uncoloured and in the periphery of the vision. Not all vasospasms are followed by dilation and there may be no headache present.

The third form of migraine affecting the eyes is the ocular migraine. The symptoms are uniocular and may vary in pattern. More typical of the migraine phenomenon affecting the central retinal artery is negative scotoma, but the patient may still experience a sense of “brightness,” as the central retina is anoxic. The causes may be similar to a classic migraine.

The third case, presented of an elderly person with twinkles in the vision, is highly suggestive of vascular occlusive disease. While most cases of amaurosis fugax present with a uniocular absence of vision, sometimes Hollenhorst plaques

Figure 2: Amaurosis Fugax
Adapted from: Handbook of Ocular Disease Management. www.revoptom.com/handbook.htm
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can be seen with just sparkles in the vision (Figure 2). In the elderly, transient obscurations of vision must be taken very seriously. The presence of diabetes, hypertension or generalized atherosclerosis may alert the physician to this possibility. In the presence of jaw claudication or symptoms of polymyalgia rheumatica, a Westergren sedimentation rate and a C-reactive protein should be taken to rule out giant cell arteritis. I routinely order these tests, as well as Doppler studies of the carotid arteries, if the patient is over 65 years old. Ten to 15% of patients with amaurosis fugax will eventually develop a central retinal artery occlusion. The associated annual stroke rate is 2%. Most patients are managed with acetylsalicylic acid, and, of course, the controversy over endarterectomy rages on.

The fourth case, where the patient experienced coloured balls of light in the centre of the vision, is suggestive of occipital epilepsy. Occipital seizures are very hard to distinguish from migraines. The presence of colour, as well as the spherical shape of the lights, is suggestive. Occipital seizures are frequently followed by postictal headaches, often indistinguishable from migraines. Most patients with occipital seizures are misdiagnosed as having basilar migraine, or acephalic migraine, and treatment is delayed for years. Treatment with carbamazepine is effective in preventing seizures.

Other causes of photopsia include retinitis, the phosphenes of quick eye movement, arteriovenous malformations, and certain drugs. Clomiphene citrate is reported to cause flickering lights and shimmering and this may persist even after stopping the medication. Flashing lights have also been reported in connection with thyroid ophthalmopathy. These are probably phosphenes brought on by globe compression secondary to the taut inferior recti muscles.

My approach to a patient with symptoms of flashing lights is to first get an adequate history (Table 1). The following points are key:

1. **Location of the flashes**: temporally would be migraine or retinal; centrally would more likely be occipital lesion or embolic phenomenon;
2. **One eye or both**: one eye suggests retinal, whereas both eyes suggest the lesion is cerebral, *i.e.*, migraine or occipital;

### Table 1

**Adequate history-taking should include:**

- What is the location of flashes?
- Do flashes occur in one eye or both?
- How long do the flashes last?
- What is the colour of the flashes?
- Does the patient have associated medical conditions?
- Does the patient have associated eye conditions?
- Is the patient taking any medications?
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3. Duration of flashes: twinkling with greyed out spots suggests emboli; lightning quick in the periphery suggests vitreous syneresis; 20 to 30 minutes of a migraine and variable suggest occipital seizure;

4. Colour of flashes: the presence of colour suggests occipital lesion; white flashes are more commonly seen with vitreous traction on retina. The zigzagged lines of migraine usually lack colour;

5. Associated medical conditions: formed seizures would most likely suggest occipital seizures; a history of migraine headaches in the past suggests migraine; hypertension, diabetes or polymyalgia rheumatica suggests vascular causes; hyperthyroidism would lead one to expect the phosphenes of Grave’s Disease;

6. Associated eye conditions: myopia, or eye trauma suggests a vitreous traction source; and

7. Use of medications: Clomiphene: digitalis can cause a yellowing of the vision, but there should be no flashing. Formed visual hallucinations are the side effect of some medications, including topical beta blockers.

A careful dilated peripheral retinal examination is mandatory in all cases of flashing lights. Diagnostic tests should be ordered based on the severity of the neurologic symptoms, their frequency and duration. Doing a careful screening could save your patient time, effort and worry. It could also save their vision. Dx

Suggested Reading