

The woman who saw the light

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Since her first psychotic break 7 years ago, Ms. G sees dancing lights and splotches continuously. What could be causing her visual hallucinations?

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HISTORY Visual disturbances

Ms. G, age 30, has schizoaffective disorder and presents with worsening auditory hallucinations, paranoid delusions, and thought broadcasting and insertion, which is suspected to be related to a change from olanzapine to aripiprazole. She complains of visual disturbances—a hallucination of flashing lights—that she describes as occurring “all the time, like salt-and-pepper dancing, and sometimes splotches.” Her visual symptoms are worse when she closes her eyes and in dimly lit environments, but occur in daylight as well. Her vision is otherwise unimpaired.

The visual disturbances started with Ms. G’s first psychotic break at age 23 and have persisted continuously. She reports that at first, the spots were more intense, like an incessant strobe light. These symptoms are aggravated by sleep deprivation and anger and improve only with topiramate, 100 mg/d, which was prescribed a year earlier after a neurologic consultation but discontinued because of cognitive side effects.

Ms. G also complains of dull, aching, continuous headaches in the center of her forehead, which do not wake her, are not worse in the morning, and do not vary with the intensity of her visual symptoms. The headaches are incorporated into her system of delusions; she believes they “drain the electricity from her head” and make her feel better. She denies a

history of migraines. Ms. G has a history of experiencing well-formed visual hallucinations, such as the devil, but has not had them for several years.

She had 5 to 10 episodes over the last 2 years that she describes as seizures—“an earthquake, a huge whooshing noise,” accompanied by heart palpitations that occur with stress, in public, in bright lights, or whenever she feels vulnerable. These episodes are not associated with loss of consciousness, incontinence, amnesia, or post-ictal confusion and she can stop them with relaxation techniques such as deep breathing and reminding herself to calm down. The last episode was 2 months ago and seemed unrelated to her visual phenomena. She also complains of occasional paresthesias in her extremities but denies dizziness, presyncope, or blurry or double vision.

Ms. G is obese and has no history of head injury or birth defects. Her family history is negative for epilepsy; however, her mother has bipolar disorder and father has schizophrenia. Her medications are phentermine, 37.5 mg/d for short-term management of

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obesity, oxcarbazepine, 600 mg/d for supposed partial seizures, sertraline, 100 mg/d for depression, aripiprazole, 10 mg/d, and nizatidine, 150 mg/d, for gastroesophageal reflux disease. She does not use alcohol, cigarettes, or illicit drugs. Ms. G obtained a master's degree in health communications and works as a research assistant at a local medical school.

Which condition does Ms. G's presentation suggest?

- migraine
- occipital lobe seizure
- arteriovenous malformation
- retinal detachment
- all of the above

The authors' observations

Visual hallucinations are not distortions of reality but original false perceptions that co-occur with real perceptions.¹ They vary from simple, elemental hallucinations involving flashes of light or geometric figures to more complex elaborations such as seeing crawling insects or a flock of angels.² Hallucinations can originate anywhere in the visual system from the lens to the visual cortex and can be vascular, toxic, electrophysiologic, structural, or neurochemical manifestations of neurologic, psychiatric, or ophthalmologic disorders. Depending on the hallucination's etiology, treatment varies from emergent ophthalmologic examination and retinal surgery to antipsychotic therapy to simple observation. Photopsia is a perception of flashing lights, and its etiology varies; causes can be categorized as emergent or nonemergent.

Emergent causes of photopsia typically have a sudden onset, often can be diagnosed from associated symptoms or medical history, and must be recognized quickly to prevent permanent vision loss. Posterior vitreous detachment and retinal tearing can manifest as flashes of light,

floaters, or blurred vision in the peripheral visual field that are worse in dim illumination and localizable by the patient. Retinal detachment can cause photopsia, floaters, or a "curtain" or "shadow" moving over the visual field, which may be accompanied by central or peripheral vision loss.³ Severe myopia is a risk factor for retinal detachment.

New-onset photopsia can suggest cytomegaloviral retinitis in an immunocompromised patient, and ocular-vascular crisis in a patient with sickle cell disease. New-onset or recurrent photopsia with bilateral blurred vision, ataxia, vertigo, dysarthria, or drop attacks may be caused by vertebrobasilar artery insufficiency (VBAI). Photopsia has been reported as the presenting symptom in carotid artery dissection.⁴ Photopsia correlated with eye movement implicates the retina or optic nerve. All of these etiologies except for VBAI should be referred to an ophthalmologist for indirect ophthalmoscopy and evaluation.

Nonemergent causes of photopsia include:

- drug toxicity
- migraine headache
- thyroid ophthalmopathy
- arteriovenous malformations
- seizures
- psychiatric disorders.

Digoxin, cocaine, paclitaxel, and clomiphenes may have ocular side effects, including photopsia. The first symptoms of digitalis toxicity often are visual and include photopsia, yellow or green discoloration of the visual field, halos, and the appearance of frost over objects.⁵ Signs of cocaine intoxication can include visual hallucinations such as photopsia, shadows, moving objects, and insects crawling and co-occurring euphoria, hypervigilance, impaired judgment, and autonomic changes such as tachycardia, pupillary dilation, hypertension, and nausea.⁶ High-dose paclitaxel infusion has been reported to cause photopsia.⁷ Clomiphenes can cause

Clinical Point

Treatment of hallucinations varies from emergent ophthalmologic exam and retinal surgery to antipsychotic therapy to simple observation

Table 1

Diagnoses suggested by photopsia phenomena

Feature	Suggested diagnosis
Appearance	
Twinkling lights with grey spots	Emboli
Zigzag lines	Occipital epilepsy
White flashes	Vitreous traction on the retina
Flickering lights and shimmering	Clomiphene use
Temporally located flashes of light, floaters, or blurred vision that is worse in dim illumination	Posterior vitreous detachment and retinal tearing
Location	
Temporal	Migraine or retinal pathology
Central	Occipital lesion or embolus
Monocular	Retinal pathology
Binocular	Cerebral cause
Duration	
Lightning quick	Vitreous detachment
Lasts up to 30 minutes	Migraine or occipital epilepsy

Clinical Point

Seizure activity in the occipital cortex and adjacent areas can produce static lights and stars

flickering lights and shimmering that persists after discontinuing the medication.⁸

Migraine with aura, which includes photopsia 39% of the time,⁹ often involves an expanding central visual disturbance or scotoma, and usually is confined to 1 visual field but may involve both. The aura typically lasts 10 to 20 minutes and often is followed by headache. Patients often report a history of episodic stereotyped auras and headache. Ocular examination is normal.

Ophthalmic migraine, also known as ocular or retinal migraine, is thought to be caused by transient vasospasm of the choroidal or retinal arteries and can be precipitated by postural changes, exercise, and oral contraceptives. It manifests as a gradual visual disturbance in a mosaic scotomata pattern that enlarges, producing total unilateral visual loss lasting from minutes to an hour, and—misleadingly—can be associated with minimal or no headache. Ocular examination is normal, and a personal or family history of migraine confirms the diagnosis.

Seizure activity in the occipital cortex and adjacent association areas can produce static light and stars. In a prospective study

of 18 patients with occipital epilepsy, visual seizures lasted from a few seconds to 3 minutes—although rarely 20 to 150 minutes—and occurred in multiple clusters a day or week.¹⁰ All but 2 patients had secondary generalized tonic-clonic convulsions. Occipital seizures often are misdiagnosed as the visual aura of migraine, particularly when elementary visual hallucinations are followed by post-ictal headache and vomiting.¹¹ Lights with a color or a spherical shape suggest occipital epilepsy. Consider ordering an electroencephalogram (EEG) if the clinical diagnosis is unclear.

Which approach would you chose to evaluate Ms. G's photopsia?

- EEG
- thyroid function tests
- urine toxicology
- ophthalmology consultation
- all of the above

EXAMINATION No obvious cause

During a mental status exam, Ms. G is pleasant, cooperative, alert, and oriented to person,

Table 2

Photopsia differential diagnosis: Look for co-occurring symptoms

Co-occurring symptoms	Suggests
Seizure activity associated with flashing lights	Occipital epilepsy
Yellow or green discoloration to the visual field, halos, and appearance of frost over objects	Digitalis toxicity
Shadows, moving objects, and insects crawling with co-occurring euphoria, hypervigilance, impaired judgment, and autonomic changes such as tachycardia, pupillary dilation, hypertension, and nausea	Cocaine intoxication
Floaters, or a 'curtain' or 'shadow' moving over the visual field, which may be accompanied by central or peripheral vision loss	Retinal detachment
Bilateral blurred vision, ataxia, vertigo, dysarthria, or drop attacks	Vertebrobasilar artery insufficiency

place, and time. Her speech is fluent, her affect is full, and she describes her mood as a "sensitive, overwhelmed state." Her thoughts occasionally are tangential, and she perseverates on guilty, embarrassed, and paranoid thoughts. She has auditory hallucinations and experiences photopsia during the interview, but denies other visual hallucinations. She shows no deficits in attention, concentration, memory, language, calculations, visuospatial abilities, or general fund of knowledge.

Her physical exam shows pupils that are equal, round, and reactive to light with normal extraocular movements, intact visual fields, 20/20 visual acuity in both eyes with glasses, and sharp optic disc margins with no retinal abnormalities apparent on fundoscopic exam. The rest of her physical and neurologic exam is normal. Her complete blood count, electrolytes, kidney, liver function tests, urinalysis, and serum toxicology screens are normal. She has no history of immunodeficiency.

An MRI performed a year earlier showed mild diffuse congestion of a left maxillary sinus but no other intracranial abnormalities, and a waking EEG with sphenoidal electrodes was normal.

The authors' observations

Evaluation of patients with photopsia includes taking a history and performing an eye examination including visual acu-

ity testing, visual field testing, and fundoscopic examination. The appearance, location, and duration of photopsia may help narrow the differential diagnosis and identify emergent cases (*Table 1 and Table 2*). Seizure activity associated with flashing lights suggests occipital epilepsy. A history of hypertension, diabetes, or polymyalgia rheumatica points to vascular causes, and hyperthyroidism suggests that flashing lights may be phosphenes of Graves' disease. Myopia or eye trauma could indicate vitreous traction. Prescription medications and illicit drug use may point to a toxic etiology.¹²

OUTCOME Diagnosis of exclusion

Ms. G's visual disturbance resembles vitreous detachment or classic migraine; however, the onset is not sudden, her fundoscopic exam is normal, and she has no discrete episodes with concomitant headache. Ms. G's improvement with topiramate is inconsistent with an ophthalmic origin because such photopsia should not improve with medication. It is consistent with recurrent seizures or status epilepticus of the occipital cortex; however, she did not experience multiple discrete episodes a day or generalized seizures experienced by most occipital seizure patients, and her EEG and clinical history are not consistent with seizures. Because

Clinical Point

The appearance, location, and duration of photopsia may help narrow the differential diagnosis and identify emergent causes

Clinical Point

A perceptual disturbance related to schizoaffective disorder is the most likely etiology of Ms. G's photopsia

Related Resources

- Ophthalmology Web. www.ophthalmologyweb.com.
- Aleman A, Larøi F. Hallucinations: the science of idiosyncratic perception. Washington, DC: American Psychological Association; 2008.

Drug Brand Names

Aripiprazole • Abilify	Oxcarbazepine • Trileptal
Clomiphene • Clomid, Serophene	Paclitaxel • Onxol, Taxol
Digoxin • Lanoxin	Phentermine • Adipex-P, Inoamin
Nizatidine • Axid	Sertraline • Zoloft
Olanzapine • Zyprexa	Topiramate • Topamax
	Ziprasidone • Geodon

Disclosure

The authors report no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products.

we cannot rule out occipital lobe epilepsy, we refer Ms. G for repeat EEG and routine ophthalmologic exam, both of which are normal.

Perceptual disturbances are common in schizoaffective disorder and schizophrenia,¹³⁻¹⁵ which we consider the most likely etiology of Ms. G's photopsia, given her normal neurologic and ophthalmologic examinations. We switch her from aripiprazole to ziprasidone, 40 mg/d, discontinue phentermine, and taper and discontinue oxcarbazepine.

Bottom Line

Because psychiatrists often are consulted to evaluate patients experiencing visual hallucinations, it is important to rule out neurologic and ophthalmologic causes before determining that photopsia has a psychiatric origin. A thorough history with attention to onset, duration, frequency, and associated symptoms can help differentiate between emergent and nonemergent causes of photopsia, and prompt appropriate referrals for electroencephalogram or ophthalmologic examination.

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