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Transient Visual Obscurations: Clues to Localization

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Transient visual obscurations – ie, blurred vision or scotomas lasting <24 hours – are most commonly caused by lesions in the retinocortical component of the visual pathway. Identification of the specific cause of the visual loss can be difficult. Examination begins with the determination if the visual loss was monocular or binocular. Monocular visual loss is usually caused by ocular ischemia, and binocular transient visual loss is most commonly caused by migraines. The objectives of this issue of *Ophthalmology Rounds* are to ensure that readers are able to formulate a series of questions to pose to the patient presenting with transient visual obscurations and to come up with the differential diagnosis and an appropriate management plan.

CASE STUDY

A 74-year-old woman presents with a recent history of 2 episodes of a grey shadow "coming down on the right eye." Each episode lasted approximately 1 minute until the shadow lifted. How would you proceed in working up this patient?

Etiology

Transient visual obscurations are reported as blurred vision or scotomas lasting less than 24 hours. They are caused by lesions anywhere along the retino-cortical component of the visual pathway. Patients who experience transient visual obscurations present frequently to ophthalmologists in general practice and should be approached in a methodical, stepwise manner to initiate appropriate investigation. The goal of the clinician is to localize the symptoms to a specific region in the visual pathway and then to come up with a differential diagnosis and management plan for possible etiologies (Figure 1).

The first step in this process is to determine whether the event involved one or both eyes. Monocular transient visual loss suggests a prechiasmal lesion, whereas binocular transient visual loss suggests a chiasmal, postchiasmal, or bilateral prechiasmal lesion. Determining whether visual loss was monocular or binocular, however, can be challenging as patients often have difficulty distinguishing between transient monocular vision loss and binocular hemianopic defects, where the eye with the temporal defect is usually blamed. It is important to specifically question the patient whether one eye was covered during the episode as well as if there were difficulties with reading during the episode, which would suggest a binocular etiology.

Pinpointing an exact etiology of transient visual loss can be difficult and the knowledge of the most common etiologies is useful in deciding which patient to investigate further (Table 1). The prevalence of transient visual loss was around 1% among the participants in the Framingham study who were specifically questioned about symptoms of transient ischemic attacks (TIAs),[1] and the etiology was determined to be due to stroke or TIA in 24%, ocular disease in 17%, transient monocular blindness in 10%, migraine in 14%, and in 22% the cause was not determined. Lavallée et al[2] found that more than one-third of patients admitted for a suspected TIA also experienced transient visual symptoms.

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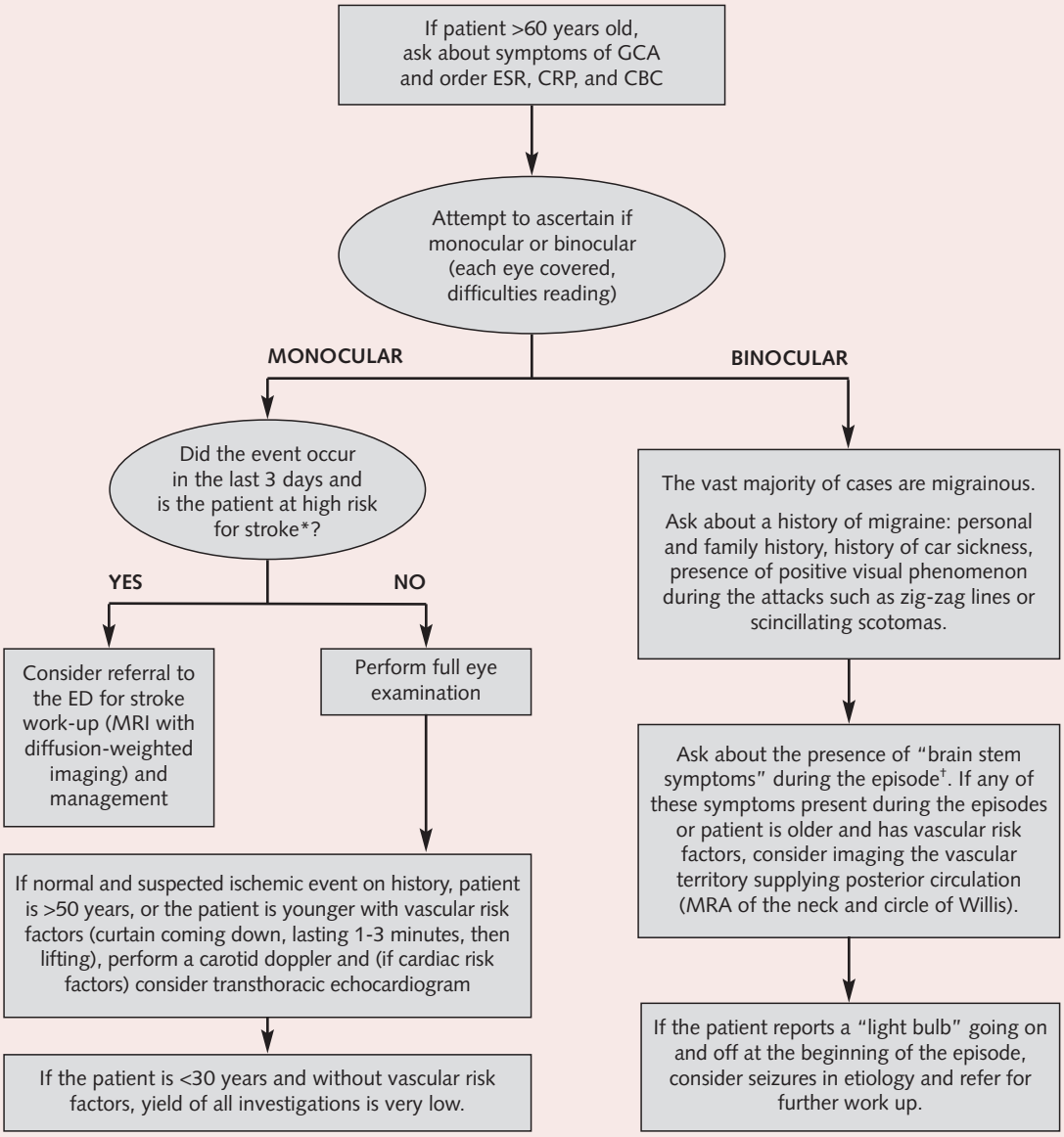
Figure 1: Steps in the investigation of a transient visual obscuration



* Age >60 years, history of hypertension, presence of other neurological symptoms during the episode

† Diplopia, dysphagia, dread, dizziness, disequilibrium, dysarthria, dysphagia, drop attacks, vertigo, weakness, tinnitus, hearing loss

GCA = giant cell arteritis; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; CBC = complete blood count; ED = emergency department; MRI = magnetic resonance imaging; MRA = magnetic resonance angiography

Transient Monocular Visual Loss

While transient visual loss in one eye may be the result of a variety of neurological and ophthalmic conditions, the principal cause is ocular ischemia.[3-5] There are generally 5 ways in which an eye can become briefly ischemic:[4]

- Embolism originating in a stenotic cervical carotid artery or the heart
- Carotid or ophthalmic artery stenosis involving a transient drop in blood pressure
- Imminent blockage of the retinal or optic nerve circulation
- Optic nerve edema that impedes retinal circulation
- Vasospasm of retinal arterioles

The eye, like the cerebral hemisphere, is the target of emboli from the heart, aortic arch, cervical carotid artery, and the systemic veins via paradoxical emboli.[6] The ophthalmic artery may also be a source of emboli.[7] Stenosis or occlusion of the large arteries of the anterior circulation of the brain may produce ocular ischemia and the drop in systemic blood pressure may sufficiently decrease perfusion to the eye to cause transient monocular (or binocular) visual loss.

Hypercoagulable states have also been associated with transient monocular and binocular visual loss by

Table 1: Most common monocular and binocular causes of transient visual loss

Monocular

Vascular
- Internal carotid artery stenosis, occlusion, or dissection
- Common carotid artery stenosis, occlusion, or dissection
- Ophthalmic artery stenosis, occlusion, or dissection
- Aortic arch atheroma
- Cardioembolic source (arrhythmia and structural abnormality)
- Vasculitis (giant cell arteritis)
- Arterial vasospasm
- Hypercoagulable state
- Systemic hypoperfusion

Neurologic
- Retinal migraine
- Elicited repetitive daily blindness

Ophthalmic
- Papilledema and optic disc edema
- Optic disc drusen
- Optic neuritis
- Orbital masses and foreign bodies
- Age-related macular degeneration
- Intermittent angle-closure glaucoma
- Uveitis-glaucoma-hyphema syndrome
- Corneal basement membrane dystrophy
- Tear film dysfunction and dry eye

Binocular

Vascular
- Transient ischemic attacks
- Bilateral carotid artery stenosis or occlusion
- Systemic hypoperfusion

Neurologic
- Migraine aura
- Occipital seizures
- Posterior reversible encephalopathy syndrome
- Angiography and contrast media exposure
- Head trauma

Ophthalmic
- Papilledema and optic disc edema
- Optic disc drusen
- Age-related macular degeneration

precipitating thrombosis in the circulation supplying visual pathways.[8]

Any condition that produces optic disc swelling (unilateral or bilateral) may give rise to obscurations of vision (monocular or binocular) that last a few seconds. The most common cause is elevated intracranial pressure, which leads to axoplasmic stasis at the optic nerve head and impaired signal transmission.[9] Assumption of upright posture may aggravate the visual symptoms by causing a critical drop in ocular perfusion.

Spasm of the retinal vessels has become an accepted explanation for episodes of transient monocular visual loss in patients under 40 years of age,[10,11] as well as in older patients in whom conventional studies are negative.[12,13] These patients often also have a history of migraine.[14] Ophthalmoscopic examination is usually negative[15] unless examined during an attack when transient narrowing of retinal arterioles or veins may be visualized.[12,16]

Less conventional causes of transient monocular visual loss include intermittent angle closure glaucoma, orbital or retro-orbital arteriovenous fistula and impending optic nerve or retinal vascular occlusion.[6,17] Anatomic anterior chamber abnormalities may lead to episodic plugging of the aqueous drainage channels by the iris. Rapidly rising intraocular pressure causes optic nerve ischemia and the patients often experience acute periocular and brow pain that may be incorrectly attributed to migraine.[17,18] Intermittent monocular vision loss may rarely be caused by an orbital or cavernous sinus arteriovenous fistula that diverts arterial blood intended for the eye into orbital or cavernous sinus veins.[19]

Hypoperfusion of the ciliary arteries supplying the optic nerve and/or retina, and hypoperfusion of the central retinal artery and its branches, will often produce transient ischemia exacerbated by upright posture.[20] The patient may also report seeing sparkling lights. In most cases of impending arterial occlusions, ophthalmoscopy is normal. However, if there is an impending ciliary artery occlusion, especially associated with giant cell arteritis, the optic disc may be slightly swollen, reflecting ischemic axoplasmic stasis and there could be cotton wool spots present in the posterior pole indicating global ischemia of the retina.

Chronically reduced perfusion to the eye may be evidenced by signs of ocular ischemic syndrome, including cotton wool spots and distended retinal veins with peri-venous hemorrhages concentrated in the retinal periphery. There is often decreased intraocular pressure initially due to the reduction in the perfusion of the ciliary body and a mild anterior chamber reaction. If the eye continues to suffer from ischemia, neovascularization of the iris and anterior chamber angles will ensue.

Evaluation

The evaluation of patients with spontaneous transient monocular visual loss consists of the following:
- History
- Ophthalmic examination
- Laboratory investigations
- Echocardiography of the carotid arteries and heart

History

The patient's description of the visual characteristics of an attack seldom allows the physician to determine its cause.[21,22] The duration of symptoms can be a useful localizing feature: transient visual obscurations from

papilledema usually last seconds, thromboembolic events (TIAs) usually last 1–15 minutes and very rarely up to an hour, and migrainous auras usually last for 10–20 minutes.

Visual loss that descends on a portion of the visual field (usually like a curtain or less commonly as an ascending shade) is highly suggestive of retinal ischemia. Positive visual phenomena such as zigzag scintillations (fortification) are characteristic of visual cortex migraine. Conversely, seizures involving the visual pathways may also present with positive visual phenomena (often like a light bulb suddenly starting to blink) but are typically maximal at the onset and then fade off.

Transient visual obscurations due to embolic events are usually painless. The presence of pain should alert the clinician to an alternative etiology, such as migraine, angle closure glaucoma, or giant cell arteritis. Association with transient contralateral hemiplegia is very suggestive of severe carotid occlusive disease. Dysarthria and diplopia are often present in the context of vertebrobasilar insufficiency producing brain stem as well as occipital lobe ischemia. Headache, scalp tenderness, and jaw claudication suggest impending ciliary occlusion in association with giant cell arteritis. Eye or brow pain can accompany intermittent angle closure glaucoma or ciliary ischemia in association with giant cell arteritis. Neck pain is often present in cervical carotid dissection. Presyncopy indicates underlying hypotension or hyperviscosity syndromes. Simultaneous contralateral hemisensory or motor findings are seen in the setting of acute cervical carotid or carotid siphon occlusion.

Ophthalmic examination

This is a critical step in identifying ocular signs of systemic embolism (retinal intra-arteriolar plaques, branch artery occlusions, cotton wool spots, Roth spots) and in identifying signs of nonembolic causes (optic disc edema, impending retinal vascular occlusion, chronic ocular ischemia, arteriovenous fistula, angle closure).

Laboratory investigations

Common hypercoagulable states can be ruled out with conventional screening laboratory studies (Table 2). Patients who lack conventional arteriosclerotic risk factors, including patients who are younger than 40 years, should be evaluated with the more extensive hypercoagulability work up.

Echocardiography of the carotid arteries and heart

Evaluation of carotid arteries for the presence of stenosis should be performed in patients whose history suggests thrombogenic or embolic etiology of transient visual loss. Yield of the carotid doppler is very low in young patients without underlying ischemic risk factors. The yield is higher in older patients, but the lesions seen on echocardiography may have no role in the pathogenesis of ocular TIA. In patients with higher suspicion of embolic events, transesophageal rather than transthoracic echocardiography should be performed. If transthoracic echocardiography is negative, a "bubble study" looking for the presence or patent foramen ovale should be considered.

Table 2: Standard screening tests for common hypercoagulable states

Basic
- Complete blood count
- Platelet count
- Erythrocyte sedimentation rate
- Protein electrophoresis
- Prothrombin and partial thromboplastin times
- Connective tissue panel
- Urinalysis

More extensive[a]
- Protein S and C deficiency
- Prothrombin
- Antithrombin III
- Factor V Leiden
- Lupus anticoagulant
- Anticardiolipin antibodies
- Homocysteine

[a] For patients without the typical arteriosclerotic risk factors

Management

When considering the management of patients with transient monocular visual loss and high-grade cervical stenosis, one must look at the evidence. It has been shown that endarterectomy significantly reduces the future risk of stroke in symptomatic patients if stenosis is >70%.[23-26] However, the estimated 2-year risk of ipsilateral stroke is only 16.6% for patients with ocular TIAs versus 43.5% for patients with hemispheric TIAs.[27] Furthermore, patients with ocular TIAs did not suffer a single major stroke with deficit persisting beyond 90 days. This nullifies the benefit of endarterectomy with a surgical risk of stroke and/or death of ≥4%.[28-30] Therefore, antiplatelet agents are favoured over endarterectomy in treating ocular TIA alone.[31]

A cardiac embolic source is unlikely to be the etiology of transient visual loss in otherwise asymptomatic patients without the history of a cardiac disorder; therefore, echocardiography should not be ordered routinely but be reserved for patients with a history of structural heart abnormalities or patients in whom these abnormalities are suspected. Transesophageal echocardiography has a much higher yield than transthoracic echocardiography in patients in whom there is a

high pretest probability of structural heart defects.[32]

Transient Binocular Visual Loss

By far, the most common reason for binocular visual loss is migraine. Migrainous binocular visual loss is usually accompanied by flickering zigzags that migrate across the visual hemifield of both eyes over 20–30 minutes. They are not always on the same side in different attacks and visual symptoms are usually, but not always, followed by headaches. The onset of symptoms is typically during the first 3 decades of life. Up to 37% of migraines can occur without positive phenomena such as scintillations.[33,34] A pure visual deficit is especially common in children, present in up to 77% of cases,[35] and it usually disappears within 20–30 minutes. The associated headache that typically follows migrainous visual loss lessens in severity with advancing age and may disappear altogether (acephalgic migraine).[33,36] Numbness or dysphasia may follow the deficit by several minutes. By contrast, all neurological manifestations in vertebrobasilar TIA occur simultaneously.[33]

The less common etiologies of binocular visual loss are occipital lobe ischemia usually due to embolism originating in the vertebrobasilar artery or in the heart, or severe vertebrobasilar artery stenosis. If occipital lobe stenosis is the underlying etiology of binocular visual loss, there is often accompanying brainstem ischemia with other neurological symptoms of brainstem dysfunction (eg, diplopia, hemiplegia).

Visual impairment is reported in 40%–50% of patients with vertebrobasilar TIAs,[37,38] and headache is present in 10% of vertebrobasilar TIAs.[37] Other symptoms of brainstem ischemia include the 6 D's – disequilibrium, diplopia, dysarthria, dysphagia, drop attacks, and dread – as well as vertigo, amnesia, nausea, numbness, weakness, tinnitus and hearing loss.[37] This can be caused by emboli or episodic hypotension superimposed on proximal vertebral artery stenosis.

Seizures originating in the occipital lobes and evoked by an underlying structural abnormality (eg, arteriovenous malformation, tumour) is another rare cause of binocular visual disturbance. Patients with occipital lobe epilepsy usually report stationary sparkles lasting up to several minutes; the sparkles can rarely be coloured, and there is often an underlying homonymous hemianopic defect, necessitating performance of formal visual fields in all patients presenting with a history of transient visual loss.

To conclude, in the absence of papilledema, transient binocular visual loss usually reflects the dysfunction of the visual cortex caused by migraine, vertebrobasilar transient ischemia, or seizure. The visual characteristics and accompanying manifestations help to determine the cause.[36-47]

CASE STUDY (cont.)

This patient's visual loss is monocular in nature, and the symptoms point to ocular ischemia as the most likely etiology responsible for transient visual loss. The ophthalmologist should pose detailed questions about her ischemic risk factors as well as the presence of any symptoms of giant cell arteritis. S/he should order a standard blood work up, including complete blood count, erythrocyte sedimentation rate, and C-reactive protein, even in the absence of other systemic symptoms of giant cell arteritis. S/he should also request a carotid ultrasound because the patient is older than 40 years.

If all of the investigations noted in the case study are normal and there is a high suspicion of structural heart defect, transesophageal echocardiography should be obtained. If the entire work up is normal, the patient should be counseled about the vascular risk factor control and if there are no contraindications (ie, gastric ulcer) and in the presence of other vascular risk factors, 81 mg of acetylsalicylic acid daily should be suggested.

Summary

Transient visual obscurations refer to a sudden, temporary monocular or binocular loss of vision that is often caused by a temporary disruption of blood supply to the eye or visual pathways. It can also result from a wide variety of other causes, including neurological (eg, migraine) and ophthalmic conditions (eg, optic disc edema). As the possible causes are so varied and physical examination is often unremarkable, it is crucial to obtain an accurate account of the episode. The first and most important step is to determine if the visual loss was monocular or binocular, keeping in mind that transient homonymous visual field loss will often be mistaken for monocular visual loss. In monocular episodes, the etiology is usually abnormalities in the eye or optic nerve and in binocular episodes the abnormality is in the chiasmal or post chiasmal visual pathways or in both eyes or both optic nerves. As some causes of transient visual obscurations, such as TIAs and giant cell arteritis, are true medical emergencies, the clinician should have a low threshold for urgent evaluation of patients who may have these conditions.

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