Primer of Work Up for Transient Visual Loss: Important to understand the mechanism!

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Mechanisms of Transient Visual Loss - *Monocular*

1. Decrease in ocular perfusion pressure
    a. Fixed arterial stenosis with drop in blood pressure
    b. Vascular occlusive disease (giant cell arteritis) with drop in blood pressure
    c. Severe transient hypotension (orthostatic or heart dysrhythmia)
    d. Vasospasm of central retinal artery
    e. Venous stasis retinopathy with cilioretinal artery hypoperfusion and drops in BP
    f. Acute rise in intraocular pressure (angle closure, pigmentary dispersion, immediately following intravitreal injection)
2. Embolic (cholesterol, platelet emboli, calcific), rarely fat emboli or talc particles
3. Compressive – gaze evoked TVL from optic nerve meningioma, orbital air,
4. Uthoff's phenomenon (e.g. after demyelination)
5. Hyphema – uveitis-glaucoma-hyphema (UGH) syndrome

Mechanisms of Transient Visual Loss – *Binocular;* orthostatic or heart dysrhythmia (negative, darkening of vision) vs. occipital activation from migraine or seizure (positive visual phenomena)

1. Decrease in ocular perfusion pressure – transient hypotension
2. Decrease in visual cortex occipital pole perfusion pressure – transient hypotension
3. Migraine – usually white out of vision or positive visual phenomena, often homonymous but the patient thinks it is one eye (e.g. right eye transient vision loss may be right homonymous phenomena)
4. Occipital seizure – usually positive visual phenomena but may be negative if post-ictal

History: It follows the understanding of the mechanism of transient visual loss

Bilateral vs Unilateral

Bilateral – retina vs. occipital cortex; is center of visual field going out first (occipital drop in perfusion pressure) or is peripheral visual field going out first (retinal drop in perfusion pressure)? The retina macula arterioles have the highest perfusion pressure, so drops in blood pressure affect the peripheral retina first. The occipital pole in visual cortex often lies in a watershed zone of perfusion between the distal branches of the posterior cerebral artery and the middle cerebral artery, so that drops in blood pressure often affect the occipital pole first (corresponding the central visual field).

Unilateral – which part of the visual field went out – peripheral, first, then central (implies drop in ocular perfusion pressure), altitudinal (implies embolic), area of vision spared (central due to cilioretinal artery) or central field only – may be involvement of cilioretinal artery.

Duration (seconds, minutes, hours, day)

Transient visual obscurations (seconds) – often occurs in the context of a swollen optic nerve due to severe papilledema with superimposed postural drops in blood pressure

Amaurosis (minutes) – usually due to emboli
Hours: Uveitis Glaucoma Hyphema Syndrome (UGH syndrome) from haptic of intraocular lens rubbing against back of iris after cataract surgery, occipital lobe epilepsy (post-ictal temporary blindness after the seizure)

Characteristics of the episode

Darkening of vision (negative phenomena)? – usually ischemic
White out of vision (positive phenomena)? – usually migraine
"Positive" complex visual phenomena – sparkles, colors, geometric shapes (migraine like aura)

What was the person doing at the time?

Valsalva from coughing or laughing or straining – may be paradoxical embolism through patent foramen ovale and Valsalva may accentuate right to left shunt of venous thrombi (e.g. from extremity). Sometimes an intense coughing episode may dislodge an intimal plaque or cause a dysrhythmia.

change in posture – orthostatic drop in blood pressure

sitting at rest – suspect either embolism or drop in blood pressure due to dysrhythmia

rubbing their eye – may cause transient spike of intraocular pressure with resulting decrease in ocular perfusion pressure and decrease in blood flow

transient visual loss precipitated by eye movement in extreme gaze – suspicious for compressive optic neuropathy in the orbit (e.g. optic nerve sheath meningioma)

transient visual loss with increase in body temperature (environmental, exercise, hot shower) – suspect demyelination with Uthoff's phenomena from delayed nerve conduction caused by increases in core body temperature

looking at a bright light and then seeing an afterimage – may be due to photoreceptor dysfunction. Dark spot in the center of vision may also be noticed on awakening in patients with macular disease due to increase in metabolic demand of photoreceptors in darkness; the membrane dark current is maintained by Na+/K+ pump which is ATP energy dependent.

post-prandial, after eating ("steal phenomena" in the setting of flow significant stenosis)

occurring with exposure to bright daylight – increase in metabolic demand that cannot be met by increases in blood flow due to flow significant arterial stenosis

palpitations, irregular heartbeat – transient drops in blood pressure with reduced ocular perfusion pressure, decrease in blood flow; if there is a superimposed arterial stenosis, then the transient visual loss may be unilateral

darkness – angle closure from mydriasis and pupil block with raised IOP

What is the pattern of the vision loss?

Peripheral field goes out first – consider retinal hypoperfusion episode due to transient hypotension (peripheral retinal arterioles have a lower perfusion pressure compared to macular arterioles)

Central field goes out first – consider occipital pole of visual cortex (lying within a watershed zone) responding to transient hypoperfusion in the setting of a drop in blood pressure.
Regional visual field change such as altitudinal loss – common with embolic cause

Were there any other symptoms at the time?

Light headedness – think about transient hypotension
Unilateral weakness or numbness (TIA) – consider embolic cause
Global amnesia or cognitive dysfunction or motor activation - consider seizure activity

<u>Exam</u>

Anterior segment – signs of causes of acute rises in IOP (pigmentary dispersion, narrow angles) or signs of hyphema or microhyphema, iris transillumination defects, IOL haptic rubbing on posterior iris surface.

Posterior segment – signs of emboli (platelet, calcific, cholesterol), venous stasis retinopathy with cilioretinal artery. Digital pressure on globe during exam to determine if small increases in IOP can collapse the central retinal artery due to low ocular perfusion pressure (e.g. carotid stenosis); sometimes this will reproduce the transient darkening of vision. Disc edema associated with raised intracranial pressure may give rise to transient visual obscurations with drops in blood pressure brought on by postural changes or Valsalva maneuver.

Neck bruits

Pulse – any irregularities due to dysrhythmia?

<u>Laboratory testing</u>

Vascular imaging – Duplex ultrasound of carotid and vertebral arteries (is there a significant stenosis or the presence of intimal plaques? But this only samples the cervical portion of the carotid – need MRA or CTA or angiogram to evaluate more distal vascular pathology.

Cardiac echography – is there an embolic source (valves) or patent foramen ovale (bubble study)?

Holter monitor to discover transient dysrhythmia +/- 24 hour ambulatory blood pressure monitoring to uncover transient episodes of hypotension.

Fluorescein angiography – is there a delay in choroidal filling (giant cell arteritis)? Is there a prolonged arteriole-venular filling time indicative of low ocular perfusion pressure?

CT and/or MRI to evaluate for any compressive lesion adjacent to the optic nerve. After trauma with orbital fractures, assess if there is intra-orbital air.

Blood testing for coagulopathies (antiphospholipid antibodies, clotting disorders)

Causes that you will need to think about/differentiate:

Unilateral or Bilateral Transient Visual Obscurations (TVO's); black outs lasting seconds:

1. Optic nerve edema due to papilledema (may be asymmetric and only having them on one eye)
2. Optic nerve drusen
3. Bleaching of the retina from having just looked at a bright light

Unilateral Amaurosis

1. Transient decrease in blood supply to the retina or optic nerve due to an embolism (could be cholesterol, calcific, or platelet) – think about the source (heart PFO, heart valve, aorta, carotid, distal to carotid
2. Transient decrease in blood supply to the retina due to hypoperfusion from a drop in blood pressure (may have a superimposed narrowing of the carotid artery or posterior ciliary artery on one side, e.g. giant cell arteritis) or increase in venous pressure from a transiently hypo-perfused cilioretinal artery in the context of a Central Retinal Vein Occlusion with a sudden drop in blood pressure, or episode of heart dysrhythmia with transiently reduced cardiac output and lowering of blood pressure
3. Metabolic demand increases in the setting of ocular ischemic syndrome (amaurosis with exposure to bright light or post-prandial, after a meal)
4. Transient decrease in blood flow to the eye due to increase in intraocular pressure from angle closure glaucoma, intravitreal gas with chronically increased IOP and superimposed transient drops in blood pressure
5. Transient decrease in blood flow to the retina or optic nerve due to orbital blood or air or mucocele.
6. Transient decrease in blood flow to the retina due to vasospasm of central retinal artery (especially in the setting of migraine)
7. Gaze evoked amaurosis due to orbital tumor such as optic nerve sheath meningioma

Bilateral Amaurosis – drops in blood pressure

1. Retinal – should be peripheral field blacking out first and closing into central field.
2. Occipital visual cortex (need to understand concept of watershed zone in the occipital pole depending on that person's dual blood supply from the posterior cerebral artery and middle cerebral artery – usually center of field goes out first

Long Duration Transient Unilateral Visual Loss – hours to days

1. Blood in anterior chamber (due to IOL haptic rubbing on iris) – usually described as a white out of constant haziness of vision like looking through a fog
2. Blood in vitreous – can still be from IOL haptic if the posterior lens capsule is not intact or can be from a source of vitreous hemorrhage (retinal tear, neovascularization)
3. Occipital seizure with post-seizure loss of neuronal firing due to membrane potential hyperpolarization after the seizure