TRANSIENT VISUAL LOSS

Tina R. Porzukowiak OD, FAAO
Midwestern University, Arizona College of Optometry
Glendale, AZ

COURSE DESCRIPTION
This course focuses on the history, associated symptoms, ophthalmic examination, differential diagnosis (DDx), and work-up for the patient with monocular and binocular transient visual loss (TVL). Ocular and systemic etiologies are reviewed.

LEARNING OBJECTIVES
1. Develop a systematic approach to the patient with transient visual loss
2. Understand the importance of a thorough history in the DDx
3. Review the components of the ocular examination
4. Formulate a DDx for monocular and binocular TVL
5. Initiate a work-up
6. Understand the treatment options

CME QUESTIONS
1. A 9 y.o. Native American male presents with recurrent episodes of swirls and bubbles in both eyes for < 2 minutes. What is the MOST likely etiology?
   a. Migraine aura without headache
   b. Occipital lobe seizure
   c. Ocular migraine
   d. Transient ischemic attack (TIA)

2. A 35 y.o. white female presents with recurrent h/o binocular scintillating scotoma for 20 minutes that moves and marches in the vision; a headache follows with associated nausea & phonophobia. What is the MOST likely etiology?
   a. Acephalgic migraine
   b. Migraine headache with aura
   c. Occipital lobe TIA
   d. Retinal migraine
3. An 80 y.o. male presents with monocular TVL OD for 5 minutes; there is no associated HA. What is the MOST likely etiology?

a. Acephalgic migraine  
b. Occipital lobe ischemia  
c. Retinal migraine  
d. TIA

4. A 40 y.o. black female presents with intermittent, painless episodes of monocular TVL OD for <10 seconds when looking to the right. What is the MOST likely etiology?

a. Carotid artery dissection  
b. Ocular migraine  
c. Orbital mass  
d. TIA

5. An elderly female presents with an acute central retinal artery occlusion OD. What is the MOST concerning concomitant pathology?

a. Carotid artery dissection  
b. Cerebral aneurysm  
c. Stroke  
d. Tumor

KEYWORDS

1. Amaurosis fugax  
2. Ischemia  
3. Migraine  
4. Seizures  
5. Transient visual loss

INTRODUCTION

Transient visual loss is the sudden loss of visual function (partial or complete) in one or both eyes that lasts less than 24 hours. It most often stems from transient vascular compromise to the eye or afferent visual pathway in the brain. A systematic approach to these cases is necessary for accurate diagnosis & management. This course will highlight the importance of a thorough history to guide the differential diagnosis, work-up, treatment, and management.

HISTORY

The history is the most important element of the neuro-ophthalmic examination. Table 1 summarizes the questions to ask of a patient presenting with TVL.
**HISTORY QUESTIONS**

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Table 1 TVL history

**Monocular versus binocular**

*Monocular* visual loss localizes to a prechiasmal or anterior circulation problem whereas *binocular* visual loss implies a chiasmal, retrochiasmal, or posterior circulation issue. It is important to stress that the patient’s visual perception of monocular versus binocular may be faulty in homonymous visual loss. If the patient describes a temporal hemifield loss in one eye, consider a nasal hemifield defect in the fellow eye. Figure 1 illustrates the anterior circulation and Figure 2 highlights the posterior circulation.

**Figures 1 & 2**

*Figure 1* illustrates the anterior circulation where blood is supplied to the anterior part of both cerebral hemispheres of the brain and the eyes. The blood is carried from the heart through the internal & external carotid arteries to supply the brain; the internal carotid artery (ICA) branches into the ophthalmic artery & further to the central retinal artery to supply the eye. The external carotid artery is important for collateral circulation for the orbit & brain when there is severe ICA stenosis.

*Figure 2* illustrates the posterior circulation where blood is supplied to the posterior part of the cerebral hemispheres (including the occipital lobe) as well as the brainstem and cerebellum. This is traditionally referred to as the vertebrobasilar system: 1) basilar artery, 2) left vertebral artery, and 3) right vertebral artery. The posterior cerebral arteries are also included in this system.
**Age**

If the patient is less than age 50, the most likely etiology is migraine or vasospasm. The exception to this rule is pregnant females with eclampsia where the concern is high for hypercoaguable states; a work-up is warranted. Patients greater than 50 are most likely to be suffering from cerebrovascular disease or giant cell arteritis (GCA).

**Onset**

The precise date of onset gives a sense of the timing and longevity of the symptoms.

**Duration**

*Transient visual obscurations (TVO)* are often exacerbated by postural change and common in patients with optic disc drusen, congenital disc anomalies, optic neuropathy, or papilledema; the timing of TVL is typically less than ten seconds. TVL lasting seconds to minutes is common for occipital lobe seizures. Bilateral occipital lobe ischemia presents with several minutes of TVL. Two to 30 minutes but less than 60 minutes of TVL is characteristic of thromboembolic disease or TIA; the average is 15 minutes. Migraines last 20-30 minutes on average and typically not longer than 60 minutes.

**Pattern of loss & recovery**

The pattern of TVL and recovery is not specific, but some generalizations may be helpful in determining the underlying etiology. A curtain coming down over one eye and lifting is the most common description of carotid artery disease; this has also been described in central retinal artery vasospasm. Carotid artery disease has also been described as altitudinal onset and disappearance, closing in of vision or tunnel vision in one eye, or sudden loss of vision in one eye.

TVL attack precipitated by heat, exercise, or physical activity is known as *Uhthoff symptom* which is commonly associated with optic neuritis.

Occipital lobe ischemia has been described as abrupt, bilateral, complete loss of vision or homonymous hemianopsia. It has also been described as bilateral, simultaneous white outs of vision. Gradual peripheral constriction or closing in of vision has been noted in systemic hypotension and occipital lobe ischemia.

Bilateral, transient, geometric pattern or hexagonal chicken wire pattern that precedes or occurs during TVL has been described in occipital lobe dysfunction like migraine, ischemia, or seizure.

Transient binocular visual loss noted as a small scotoma in homonymous portions of the visual field surrounded by jagged, luminous, shimmering edges is called a *scintillating scotoma*. The scotoma enlarges over 20-30 minutes but less than 60 minutes and then gradually disappears. The visual loss may enlarge to a complete homonymous hemianopsia. A hemicranial, throbbing headache follows known as a *migraine with aura*. 
A migraine aura without headache, formerly known as acephalgic migraine, presents as binocular TVL as described above; no headache follows. This is typically seen in patients with a strong personal or family history of migraine.

Occipital lobe seizure is described as bilateral, recurrent, and brief (seconds to 2 minute) episodes of visual loss with positive phenomena such as colored or swirling photopsia, bubbles, or a white out of vision like a flashbulb going off. Visual phenomena may be diffuse or isolated to a hemifield. Rarely, purely negative spells of visual blackening occur.

Gaze-evoked

Gaze-evoked TVL is indicative of an orbital mass like cavernous hemangioma or optic nerve sheath meningioma. Compression of the orbital vasculature and reduction of blood supply is often monocular in nature. This disturbance has also been seen with foreign bodies and thyroid eye disease.

Preceded by light exposure / photostress

Carotid artery disease can be an underlying cause of TVL preceded by light exposure. The clinician is cautioned to differentiate this phenomenon from the normal, physiologic photostress recovery in contrast to that seen in maculopathies.

Associated symptoms

It is important to inquire about additional symptoms that occurred before, during, or after an episode of TVL to further narrow the differential diagnosis. Common associated symptoms are summarized in Table 2.

Positive visual phenomena, known as photopsia, originate from the retina or the brain. Retinal photopsia are typically unilateral, white, lightening-like flashes that last for less than one second; they are commonly noted with retinal breaks or epiretinal membranes. Neurologic photopsia are generally bilateral, white or colored, positive visual disturbances lasting greater than a few seconds to several minutes but usually less than 1 hour. Clinical examples include migraine and occipital lobe seizure.

TVL exacerbated by postural change is seen in elevated intracranial pressure (ICP), GCA, or systemic hypotension. Patients with elevated ICP typically experience TVO presenting as less than 10 seconds of transient visual blurring or dimming. Other symptoms include intracranial noises or pulse synchronous tinnitus, headache, diplopia, nausea and emesis.

In addition to TVL, patients with GCA may experience a temporal headache, jaw claudication, weight loss, fever, malaise, decrease appetite, scalp tenderness, diplopia, myalgia, and arthralgia.
ASSOCIATED SYMPTOMS

- Arthralgia
- Decreased appetite
- Diplopia
- Dizziness
- Dysarthria
- Emesis
- Exacerbation with postural change
- Exacerbation with Valsalva
- Fever
- Focal weakness
- Headache
- Intracranial noises
- Jaw claudication
- Loss of consciousness
- Malaise
- Nausea
- Neck pain
- Phonophobia
- Photophobia
- Photopsia
- Presyncope
- Raynaud phenomenon
- Scalp tenderness
- Skin or joint changes
- Tinnitus
- Weight loss

Table 2 Common symptoms occurring before, during, or after TVL.

The scintillating scotoma of migraine as seen in Figure 3 may also be associated with headache, photophobia, phonophobia, nausea, or emesis.

Figure 3 Scintillating scotoma of migraine.
Occipital lobe TIA or stroke may present with a frontal brow headache, loss of consciousness, dizziness, diplopia, focal weakness, dysarthria, or confusion. Global perfusion problems or ischemia of the brainstem or cortex can present as loss of consciousness, dizziness, diplopia, focal weakness, dysarthria, or confusion.

TVL associated with Raynaud phenomenon, arthralgia, skin or joint changes is most concerning for a connective tissue disease. Figure 4 depicts the variable presentations of Raynaud phenomenon affecting the fingertips.

![Figure 4 Raynaud phenomenon commonly associated with connective tissue disease.](image)

TVL associated with neck pain is concerning for carotid artery dissection.

Presyncope presenting as lightheadedness, muscle weakness, and feeling faint is associated with systemic hypotension and hyperviscosity syndromes.

**OPHTHALMIC EXAMINATION**

The patient’s medical history guides the examiner to the TVL etiology. Pay close attention to a history of hypertension, diabetes mellitus, dyslipidemia, tobacco use, cardiovascular disease including ischemic heart disease, valve disease, atrial fibrillation, cerebrovascular disease, and TIA, polymyalgia rheumatica, connective tissue disease (particularly systemic lupus erythematosus (SLE)), rash or previous miscarriage (as in antiphospholipid antibody disease) and known ophthalmic disease. A personal or family history of migraine, hematologic abnormality or hypercoaguagule state, stroke, brain tumor, and/or aneurysm may also be contributory.

The ocular examination should focus on acquisition of the best-corrected visual acuity, identification of an afferent pupillary defect and perimetry. Blood pressure, heart rate, and carotid auscultation for bruit are indicated. NOTE: The absence of a bruit may indicate complete occlusion of the carotid artery or normal carotid blood flow; the presence of a bruit often indicates turbulent blood flow. The fundus examination is critical for the assessment of optic nerve anomalies, the presence or absence of retinal
emboli, and the presence or absence of vascular occlusive disease. A photostress test may indicate macular ischemia.

**DIFFERENTIAL DIAGNOSIS MONOCULAR TVL**

Monocular TVL can be divided into orbital, ocular, systemic and non-organic etiologies; table 3 summarizes the DDx.

<table>
<thead>
<tr>
<th><strong>Orbital</strong></th>
<th><strong>Non-organic / medically unexplained visual loss</strong></th>
<th><strong>Systemic / Vascular</strong></th>
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<tr>
<td>Foreign body</td>
<td>Aortic arch atheroma</td>
<td>Aortic arch atheroma</td>
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<td>Mass</td>
<td>Cardioembolic source</td>
<td>Cardioembolic source</td>
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<td>Orbital inflammatory disease</td>
<td>Carotid artery dissection, occlusion, stenosis</td>
<td>Carotid artery dissection, occlusion, stenosis</td>
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<td>Thyroid eye disease</td>
<td>Cocaine &amp; other illicit drugs</td>
<td>Cocaine &amp; other illicit drugs</td>
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<td>Ocular</td>
<td>Connective tissue disease</td>
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<td>Acute iritis</td>
<td>Elicited repetitive daily blindness</td>
<td>Elicited repetitive daily blindness</td>
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<td>Acute vitritis</td>
<td>Eosinophilic granulomatosis with polyangiitis</td>
<td>Eosinophilic granulomatosis with polyangiitis</td>
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<td>Age-related macular degeneration</td>
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<td>Anterior basement membrane dystrophy</td>
<td>Granulomatosis with polyangiitis</td>
<td>Granulomatosis with polyangiitis</td>
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<td>Blepharospasm</td>
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<td>Central retinal artery vasospasm</td>
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<td>Dry eye syndrome</td>
<td>Intravascular lymphoma</td>
<td>Intravascular lymphoma</td>
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<td>Hyphema</td>
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<td>Ophthalmic artery dissection, occlusion, stenosis</td>
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<td>Intermittent angle closure glaucoma</td>
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<td>Systemic hypoperfusion</td>
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<td>Macular ischemia</td>
<td>Takayasu arteritis</td>
<td>Takayasu arteritis</td>
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<td>Photostress retinal pigment epithelial cells</td>
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<td>TIA (anterior circulation)</td>
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<td>Disc at risk</td>
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<td>Optic nerve head edema / papilledema</td>
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<td>Retinal ischemia</td>
<td>Uveitis-glaucoma-hyphema syndrome</td>
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<td>Venous stasis retinopathy</td>
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*Table 3* DDx monocular TVL.

**Orbital**

Orbital disease may produce a gaze-evoked monocular TVL especially in downgaze with reading. This is commonly seen with cavernous hemangioma, optic nerve sheath menigioma, foreign body, thyroid eye disease, orbital inflammatory disease, and other orbital masses. Gaze-evoked TVL may be elicited by
having the patient moves the eyes and hold each position of gaze for 5 seconds noting any change in visual function or pupillary activity.

**Ocular**

Ocular pathology can present with monocular TVL of varying duration. Patients who cannot keep their eyes open as in blepharospasm may complain of TVL. Dry eye syndrome can also present with varying visual symptoms that typically improve with blinking or artificial tear supplementation. Isolated TVL associated with eye pain has been seen with recurrent corneal erosion particularly with concomitant anterior basement membrane dystrophy. Fluctuations in refractive error and tear film disruption of keratoconic hydrops is associated with complaints of TVL.

TVL lasting 15-20 minutes (occasionally up to 7 hrs) associated with erythropsia and color desaturation have been described in hyphema cases. Uveitis-glaucoma-hyphema (UGH) syndrome typically presents with gradual onset of TVL with slow recovery (hours to days) characterized by a diffuse “misting” of vision with or without eye pain.

Periodic occurrences of dull, aching, periorbital or ocular pain accompanied with TVO, colored halos, and/or conjunctival hyperemia should prompt gonioscopic evaluation for intermittent angle closure glaucoma.

Acute iritis and vitritis has been known to mimic amaurosis fugax.

Monocular TVL characterized by a sudden curtain of darkness descending over one eye resulting in 2-30 minutes of impairment is common with retinal emboli. At resolution, the curtain may ascend or dissolve like a clearing fog. The three most common types of emboli are cholesterol, platelet-fibrin, and calcium. Other less common varieties include cardiac tumors (myxoma), fat (long bone fractures, pancreatitis), sepsis, air, talc (crack cocaine abuse), silicone, and depot drugs (corticosteroids).

![Hollenhorst cholesterol embolus](image)

**Figure 5** Hollenhorst cholesterol embolus lodged at the bifurcation of a retinal artery. Additional work-up is necessary to ascertain the source of the embolus with the most common site being the ipsilateral carotid artery and less likely the aortic arch.
The first presenting sign of retinal ischemia may be TVL. Monocular TVL lasting seconds to minutes with recovery to normal vision may be indicative of an impending central retinal vein occlusion (CRVO). Progressive restriction of peripheral vision lasting seconds to 2 minutes precipitated by postural change from sitting to standing may be indicative of retinal hypoperfusion secondary to cardiac arrhythmias or severe stenosis of the great vessels.

*Ocular ischemic syndrome (OIS)* is characterized by mid-peripheral dot and blot hemorrhages and may cause monocular TVL particularly with exposure to bright light. Recurrent orbital or facial pain that improves when the patient lies down is highly suggestive of carotid occlusive disease. Venous stasis retinopathy, as seen in OIS, is associated with a vascular occlusion anywhere from the heart to the eye. Patients may also describe progressive visual loss lasting 10-20 minutes. In patients over the age of 50 with new-onset iritis, OIS should be considered. Patients typically describe a dark or black shade that spreads across the visual field with a more gradual onset than that of embolic, monocular TVL; the phenomenon typically lasts for seconds to minutes. It can be precipitated after meals, postural change, or sexual activity.

Photostress recovery is abnormal in macular ischemia. This phenomenon is commonly seen in age-related macular degeneration (ARMD). The retinal pigment epithelium (RPE) and photoreceptor interaction is anatomically deranged causing abnormal processing of light as it bleaches the rhodopsin in the photoreceptors. It is important to recognize that all humans can experience physiologic photostress of RPE cells; the photostress recovery test will be normal.

Retinal detachment may present with complaints of monocular TVL characterized by a shade descending over the vision or parts of the peripheral vision. Postural change may worsen or restore the vision temporarily depending on the location of the detachment. Post-scleral buckle repair has also been reported to elicit TVL.
Retinal migraine, also known as ocular migraine, presents with at least 2 attacks of recurrent, monocular aura seen as a fortification spectrum (zigzag figure) that appears near central fixation & gradually spreads or marches left or right to assume a crescent shape with angulated scintillating edges leaving variable areas of absolute or relative scotoma. Attacks develop over 5-20 minutes, but are usually less than 60 minutes. The aura is followed by a headache. Attacks may also present as complete or partial monocular blindness. This group of patients typically has a strong personal or family of migraine and are generally <50 years old. It is a diagnosis of exclusion. A thorough work-up is warranted. Treatment includes aspirin, ibuprofen, verapamil, or diltiazem. Migraine prophylactic agents may be tried. Ergot and triptan usage should be avoided to prevent further vasoconstriction and risk of permanent visual loss. Patients should be counseled to avoid potential migraine triggers, tobacco cessation, discontinuation of oral contraceptives, healthy diet, and stress elimination.

Central retinal artery vasospasm or retinal vasospasm patients have no history of migraine yet experience recurrent, monocular TVL due to retinal vasculature attenuation or vasospasm. The first episode often affects young, healthy patients less than the age of 40. The TVL usually lasts between 5-60 minutes. It may be induced by exercise or sexual intercourse. This is a diagnosis of exclusion. A thorough work-up is warranted. These patients will have no ophthalmologic, serologic, constitutional, or major atherosclerotic risk factors. Their chance for future stroke is low. Treatment for recurrent attacks is often calcium-channel blockers such as verapamil or nifedipine.

Optic nerve head anomalies or disorders such as drusen, coloboma, morning glory syndrome, or peripapillary staphyloma may be associated with monocular TVL lasting 10-30 seconds. Optic nerve ischemia as in arteritic anterior ischemic optic neuropathy (AION) secondary to GCA can also present with monocular TVL. Optic nerve edema, optic neuritis, or papilledema patients may experience TVO described as “gray-outs” or “black-outs” of vision for <10 seconds often precipitated by postural change. The episodes can involve one or both eyes; the vision often clears completely. “Disc at risk” is used to describe an anatomical variant characterized by an extremely small or no optic nerve cup which places the patient at risk for ischemic events; figure 6 depicts an example.

Figure 6 A small, crowded optic nerve cup indicative of a “disc at risk” for ischemic events.
Non-organic / medically unexplained visual loss

The examiner is reminded to include medically unexplained vision loss in the DDx for monocular TVL; this is a diagnosis of exclusion.

Systemic / Vascular

After obvious ocular and orbital causes have been ruled out, cardiovascular & other systemic etiologies must be considered. Ischemia is the most common cause of monocular TVL which typically presents with negative visual phenomena and less likely positive visual phenomena. Amaurosis fugax (AF) is a subtype of monocular TVL attributed to ischemia or vascular insufficiency; it is characterized by sudden, painless, temporary visual loss lasting 2-30 minutes followed by complete recovery.

All patients over 50 years old with TVL should be evaluated for the vasculitis GCA. Episodes may last minutes to hours. TVL was the presenting symptom in 10-15% of monocular cases and 27-35% of binocular cases.

ICA dissection should be considered in patients with painful ipsilateral, monocular TVL particularly if associated with Horner syndrome and contralateral neurologic signs.

Carotid artery disease resulting in stenosis or occlusion may present with hypotension as changes in posture or neck position temporarily reduce blood flow through the artery. Monocular TVL is typically progressive lasting for 5-10 minutes and may be precipitated by standing up or looking at bright lights. Postprandial monocular TVL has been described in severe carotid narrowing, presumably a vascular steal phenomenon. TVL has also been reported after sexual activity.

Ophthalmic artery stenosis, occlusion, or dissection can also present with monocular TVL.

Anterior circulation TIA is defined as a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction. Monocular TVL frequently accompanied by other focal neurologic symptoms such as hemiparesis, paresthesia, dysarthria, confusion, or aphasia which are usually more alarming to the patient than the vision loss. Positive visual phenomena have been reported, but negative symptoms are more common. TIA results from: 1) primary arterial stenosis or occlusion, 2) secondary occlusion due to embolism from a distant source, or less commonly, 3) arterial dissection resulting in primary stenosis or distant embolism.

Aortic arch atheroma and cardiac sources like arrhythmia or structural abnormality may present with monocular TVL.

Hyperviscosity and hypercoaguable states are blood disorders which tend to be more common in younger to middle aged individuals with a history of bruising or bleeding, family history of stroke at a young age, arterial or venous thrombosis, deep venous thrombosis (DVT), thrombophlebitis, pulmonary embolism (PE), miscarriage, preeclampsia, or placental infarction leading to stillbirth or preterm delivery. Antiphospholipid antibody syndrome may present with monocular TVL; rash is common. Polycythemia vera,
associated with a JAK2 mutation, multiple myeloma, protein S deficiency, protein C deficiency, factor V leiden deficiency, and connective tissue disease (SLE) should be included in the DDx.

Syphilis has been reported to cause monocular TVL.

Arteriovenous malformation (AVM) can present with monocular TVL causing ophthalmic artery steal which compromises the ocular blood flow leading to ocular ischemia. The TVL may alternate from one eye to the other.

Granulomatosis with polyangiitis (GPA), formerly known as Wegener granulomatosis, Takayasu arteritis, and eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg-Strauss Syndrome, has been reported to cause monocular TVL.

EGPA is an allergic granulomatosis angiitis that may affect many organs but most commonly the lungs, skin, kidneys and heart. Many patients also have asthma. They may have had a pre-existing, newly diagnosed, or recently worsening case of asthma when they are diagnosed with EGPA. The disease can involve the central and peripheral nervous systems, gastrointestinal (GI) system, musculoskeletal system, and the optic nerves. Asthma is the most common sign of this disorder; others include hay fever, sinusitis, rash, GI bleed, severe pain & numbness in the hands or feet, lymphadenopathy, fatigue, night sweats, cough, weakness, arthralgia, joint edema, diarrhea, nausea, emesis, dyspnea, congestive heart failure, hemoptysis, arrhythmia, and hematuria. It is rare, and there is no known cure. Steroids & immunosuppressants are often prescribed. Diagnosis typically occurs between the ages of 38-52 in patients with a history of asthma, chronic sinusitis, or allergies. Patients may have an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP); a positive temporal artery biopsy (TAB) assists in confirmation of diagnosis. Cases of Singulair use & monocular TVL have led to EGPA diagnosis.

Cocaine & other illicit drugs have been reported to cause monocular TVL.

Intravascular lymphoma (IVL) presenting as monocular TVL for approximately 5 hours followed by non-arteritic anterior ischemic optic neuropathy (NAION) is uncommon. IVL is a rare form of lymphoma in which there is a tropism for the lumina of small vessels, including those of the central nervous system (CNS), ultimately leading to multiple areas of non-territorial infarct, similar to CNS vasculitis. The patient’s initial acute visual loss is a true ischemic optic neuropathy, but is unique in that the ischemia results from IVL invading the posterior ciliary arteries.

Systemic hypoperfusion from decreased cardiac output or systemic hypotension rarely causes monocular TVL; it is usually binocular TVL.

Elicited Repetitive Daily Blindness is a rare, inherited autosomal dominant disorder characterized by brief episodes of fully reversible monocular TVL elicited by exposure to bright light, eyelid pressure, and postural change. It is associated with familial hemiplegic migraine and childhood epilepsy; no treatment is warranted.

**MONOCULAR TVL WORK-UP**
An emergent ophthalmic examination is warranted for all TVL patients. Those with gaze-evoked TVL should obtain orbital neuroimaging. Treat an ocular etiology accordingly. Additional work-up may be necessary to elicit a systemic etiology. The following is a general guideline for the care of these patients. If the dilated fundus findings include normal fundi, retinal embolus, retinal artery occlusion, cotton wool spots, venous stasis retinopathy or disc edema, proceed with a work-up. Table 6 summarizes testing considerations for the classic vascular etiology work-up.

### Table 6: Summary of Potential Testing for Suspected Vascular Etiology for Monocular TVL

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<th>Work-Up Category</th>
<th>Testing Considerations</th>
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<td>Consultation</td>
<td>Cardiology, Neurology</td>
</tr>
<tr>
<td>Imaging / Testing</td>
<td>Carotid duplex ultrasound, CT vs. MRI head / neck, CTA vs. MRA head / neck, EKG / TEE, Holter monitor, Transcranial Doppler</td>
</tr>
<tr>
<td>Serology</td>
<td>BUN, Cardiac enzymes (CK, TnI, TnT), CBC w/ diff, CMP, Creatinine, CRP, Fasting glucose (included in CMP), Fasting lipid panel, PT / PTT, Westergren ESR</td>
</tr>
</tbody>
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CT = computed tomography, MRI = magnetic resonance imaging, MRA = magnetic resonance angiography, EKG = electrocardiogram, TEE = transesophageal echocardiogram, BUN = blood urea nitrogen, CK = creatinine kinase, TnI = troponin I, troponin T, CBC w/ diff = complete blood cell count w/ platelet differential, CRP = C-reactive protein, PT = prothrombin time, PTT = partial prothromboplastin time, ESR = erythrocyte sedimentation rate

Look for vascular risk factors such as advanced age, hypertension, hypotension & syncope (possibly iatrogenic from overly vigorous treatment of hypertension or from other medications), ischemic heart disease, diabetes, dyslipidemia, tobacco use, and sleep apnea.

If signs and symptoms of giant cell arteritis are present or the patient is >50 y.o., order a CBC w/ differential, Westergren ESR, and CRP.

A neurologic consultation may be necessary if the patient presents with contralateral symptoms or signs of neurologic dysfunction.
A cardiovascular consultation is warranted to thoroughly evaluate the carotid arteries and the heart. A carotid work-up may include carotid duplex ultrasonography and/or transcranial Doppler (TCD) and brain MRA or CTA. Look for stenosis or occlusion secondary to atheroma, dissection, fibromuscular dysplasia, radiation arteriopathy, and arteritis. An EKG and/or TEE is warranted. Look for arrhythmia, congenital heart defects like patent foramen ovale (PFO), and cardiac sources of emboli in the ventricles, atria, interatrial septum, cardiac valves and aorta. In some cases, Holter monitoring may be needed to assess for paroxysmal atrial fibrillation.

In patients suspected of TIA, stroke, or retinal artery occlusion, a STAT referral to the emergency department is warranted to initiate a stroke protocol. Serology typically includes CBC w/ diff, ESR, CRP, PT / PTT, CMP, BUN, creatinine, fasting glucose, fasting lipid panel, and cardiac enzymes. TIA is a major risk factor for ischemic stroke with the greatest risk immediately following the event. Crescendo TIA (recent, recurrent TIA) requires urgent evaluation. Cranial nerve, somatic motor strength, somatic sensory, speech and language, and cerebellar system testing should be performed. The most common findings for TIA include diplopia, hemianopia, monocular blindness, dysconjugate gaze, facial drooping, lateral tongue movements, dysphagia, and vestibular dysfunction. Finger-to-nose and heel-to-shin movement cerebellar testing may reveal past pointing or ataxia. Motor testing may reveal spasticity, clonus, rigidity or unilateral weakness in the face, tongue, and upper or lower extremities.

In the presence of normal carotid and cardiac studies, other causes must be ruled out. Look for autoimmune diseases such as SLE and Sjögren syndrome; serology for antinuclear antibody (ANA) should be ordered. Look for hyperviscosity and hypercoaguable states. A hyperviscosity work-up might include serum viscosity, CBC w/ peripheral blood smear, total protein (TP), albumin, CMP, serum protein electrophoresis (SPEP) and urine protein electrophoresis (UPEP). Consider a coagulopathy work-up including chest x-ray (CXR), brain CT / MRI, panculture, and urinalysis (UA). A hypercoaguable work-up might include CBC w/ differential, Westergren ESR, ANA, anticardiolipin IgG and/or IgM, anti-ß2 glycoprotein I (B2GPI), SPEP, PT or international normalized ratio (INR), PTT, protein S, protein C, anti-thrombin III, factor V Leiden, Von Willebrand Factor, platelet aggregation test, prothrombin gene mutation, factor VIII, homocysteine, and JAK2 mutation. Look for syphilis; order FTA-ABS and RPR. Look for GPA or EGPA; order p-ANCA & c-ANCA studies.

For younger patients, if there is clinical suspicion for CNS infection, drug intoxication, or clotting disorder, additional work-up is warranted and may include RPR, lumbar puncture with opening pressure and CSF analysis, urine drug screen, and hypercoaguable serology. In the majority of young patients (< 50 years) with monocular TVL who are otherwise healthy, the diagnostic yield of extensive testing is very low. The extent to which these patients should be subjected to diagnostic investigation is debated and should be decided individually.

**VASCULAR MONOCULAR TVL TREATMENT**

Treatment of vascular monocular TVL is aimed at prevention of irreversible vision loss (as in central retinal artery occlusion), stroke, and myocardial infarction (MI). Table 7 highlights the management.
Table 7 Monocular TVL management considerations for vascular etiologies

DIFFERENTIAL DIAGNOSIS BINOCULAR TVL

Binocular TVL can be subdivided into the major categories of migraine, occipital lobe lesion, ischemia, seizure, and other etiologies.

Migraine

The most common cause of binocular TVL is *migraine with aura*. Symptoms typically present as a small scotoma in homonymous portions of the visual field surrounded by jagged, luminous, shimmering edges; this is known as a scintillating scotoma. The scotoma enlarges over 20-30 minutes (usually less than 60 minutes) and then gradually disappears. The visual loss may enlarge to a complete homonymous hemianopsia. The migraine aura is often noted to occur on different sides of the visual field at various times. A hemicranial, throbbing headache follows that may be associated with photophobia, phonophobia, nausea, or emesis.
Migraine aura without headache, formerly known as acephalgic migraine, is common in patients with a strong personal or family history of migraine. A binocular visual aura occurs which may be accompanied with visual loss without the headache. This phenomenon is often confused with TIA in the elderly. A diagnostic dilemma arises when an elderly patient without a history of migraine has a visual aura without headache. If they describe a typical visual aura and have an unremarkable examination including formal visual fields, neuroimaging may be performed but extensive investigations are not routinely indicated. Atypical features should prompt an evaluation for vertebrobasilar ischemia.

Complicated migraine is a rare migraine variant where the visual field loss may remain permanent.

Basilar type migraine may have an aura producing transient cortical blindness or homonymous visual field loss without positive visual phenomenon. It is often associated with vertigo, diplopia, ataxia, loss of consciousness, and subsequent headache. An evaluation for vertebrobasilar ischemia is indicated especially in older patients with vascular risk factors.

Occipital lobe lesion

Lesions associated with binocular TVL may include occipital lobe tumor, AVM, or venous sinus thrombosis. Patients may experience episodic headaches & TVL. Scintillating scotomas that mimic migraine may be noted. If attacks always occur on the same side or the headache precedes the aura, a structural lesion must be ruled out. Visual field defects are often noted particularly homonymous hemianopsia.

Occipital lobe ischemia

Binocular TVL causing occipital lobe ischemia include embolic, vasculitic, or hypoperfusion etiologies. Transient, sudden onset of bilateral complete vision loss only lasting a few minutes may be caused by bilateral occipital lobe TIA. Transient homonymous hemianopsia may be caused by unilateral occipital lobe TIA. Positive visual phenomena have been reported. TIA is most common in the elderly. A brow headache is possible at the time of the visual symptoms.

Binocular TVL lasting minutes to hours may rarely occur with GCA due to vertebrobasilar insufficiency (VBI) or impending bilateral AION. VBI leading to brainstem ischemia may be accompanied with dysarthria, dysphagia, vertigo, diplopia, or ataxia. VBI leading to cerebral ischemia may be accompanied with hemiparesis, hemisensory loss, or aphasia.

Infrequent causes of posterior circulation TIA include subclavian steal syndrome or “bow-hunter” syndrome. Bow hunter syndrome results from vertebral artery compression with neck rotation.

Systemic hypotension can result in binocular TVL. Symptoms include lightheadedness, syncope, chest pain, or palpitations. Common associations include vasovagal attack, hypovolemia, cardiac arrhythmia, valvular heart disease, aortic stenosis, and orthostatic hypotension.

Occipital lobe seizure
Bilateral, recurrent, simple, positive visual phenomena such as colored lights, swirling photopsia, bubbles, or visual white outs “like a flashbulb going off” represent binocular TVL associated with occipital lobe seizure. Rarely do purely negative spells of blackening out of vision occur. Visual phenomena may be diffuse or isolated to the contralateral hemifield. Episodes are brief (lasting seconds to 2 minutes). Postictal blindness known as status epilepticus amaurotics may occur and persist for hours, days (rare), or weeks (rare). Occipital lobe seizures are common in children and often benign. They may also occur in patients with posterior reversible encephalopathy syndrome (PRES), metabolic encephalopathy (hypercalcemia), AVM, neoplasm (neuroepithelial tumors, astrocytoma), malformations of cortical development (cortical dysplasia), prior head trauma, metabolic disease (mitochondrial disorders), and localized infection (bacterial abscess). They may also be idiopathic. In adults, an occipital lobe seizure is often associated with tumor, AVM or trauma.

Other etiologies

PRES presents as transient cortical blindness usually associated with abrupt increase in blood pressure as with malignant hypertension and pre-eclampsia. It may also occur in patients with renal failure or those of immunosuppressive treatments such as tacrolimus and cyclosporine. Symptoms include headache, altered mental status, and seizures. Neuroimaging shows bilateral cortical and subcortical edema in the occipital and parietal-occipital regions. Treatment is directed toward the underlying cause. Vision typically recovers in one to two weeks.

Transient cortical blindness is possible after a minor, blunt (usually occipital) head trauma. This finding is most common in children or adolescents. Vision loss develops within minutes after the event. Neuroimaging is usually normal and should be obtained to evaluate for intracranial hemorrhage. Vision is regained within minutes to hours with no specific intervention.

Bilateral carotid artery stenosis rarely presents as binocular TVL on exposure to bright light or photostress. TVL is more commonly seen as monocular.

Abnormal photostress recovery resulting in binocular TVL is possible in age-related macular degeneration and other bilateral maculopathies.

Porphyria is a rare, hereditary disorder where heme, an important part of hemoglobin, is synthesized improperly. Symptoms include abdominal pain or cramping, light sensitivity causing rashes, blistering, and scarring of the skin (photodermatitis), and problems with the nervous system like seizures, mental disturbances and nerve damage. Binocular TVL has been reported.

Preeclampsia with binocular TVL lasting 24 hours must be evaluated for hyperviscosity & hypercoaguable states.

Transurethral resection of the prostate (TURP) procedure has been associated with binocular TVL on rare occasions. Temporary vision loss with normal or nonreactive pupils may occur with irritability, confusion, bradycardia, nausea, hypertension, dyspnea, and seizures during or after treatment. This is most likely
due to excessive absorption of non-electrolyte irrigating fluid though the prostate venous sinuses into the general circulation. Glycine toxicity affecting the optic nerves or cortex is the suspected mechanism.

Drug toxicity is a rare etiology of TVL. Cyclosporine or tacrolimus can result in cerebral blindness lasting hours or days; PRES has been reported. Patients on interferon-α for myeloma or interleukin-2 therapy for malignancy or HIV disease may also develop binocular TVL.

Rarely, transient cortical blindness as a complication of cerebral and cardiovascular angiography procedures has been reported. Symptoms include headache, memory disturbance, and altered mental status during or shortly after contrast dye exposure. As long as infarction is excluded, a full recovery of vision can be expected in hours to days without intervention.

Other etiologies include connective tissue disease, hypertensive encephalopathy associated w/ renal disease, papilledema, optic disc drusen, and GCA.

**BINOCULAR TVL WORK-UP**

Upon completion of the urgent ophthalmic examination, additional work-up may be necessary to elicit an etiology. The following is a general guideline for the care of these patients.

Look for bilateral disc edema and evaluate for papilledema accordingly. Patients with bilateral TVO suspected of elevated intracranial pressure warrant MRI brain w/ & w/o contrast. If the MRI is normal, a lumbar puncture (LP) should be performed with opening pressure and CSF analysis.

Look for treatable causes of cerebrovascular disease & sources of emboli. If binocular TVL is present on exposure to bright light, order a carotid Duplex. For patients suspected of occipital lobe ischemia (vertebrobasilar TIA), investigate for a cardiac embolic source (echocardiogram) in addition to neuroimaging (MRI brain w/ & w/o contrast using FLAIR & DWI and MRA head and neck w/ contrast). Order a CBC w/ diff, Westergren ESR, & CRP to investigate for GCA. **Use the guidelines for vascular monocular TVL treatment for preventative stroke and MI prophylaxis as outlined in Table 7.**

Neurology consultation may be warranted for patients that do not fit the International Headache Society classification for migraine. Order an MRI brain w/ & w/o contrast using FLAIR & DWI and MRA head and neck w/ contrast. If neuroimaging is normal, consider occipital lobe seizures or atypical migraine. Electroencephalogram monitoring (EEG) may be necessary.

For patients with migraine symptoms plus signs of connective tissue disease, order Connective Tissue Diseases Profile (CONN).

In younger patients who present with binocular TVL associated with preeclampsia, look for hyperviscosity and hypercoaguable states. A work-up might include CBC w/ differential, Westergren ESR, ANA, anticardiolipin IgG and/or IgM, B2GPI, SPEP, PT/INR, PTT, protein S, protein C, and Factor V Leiden.
SUMMARY

A careful history, clinical examination, and systematic approach to the diagnosis, treatment, and management of TVL are proposed. The clinician may benefit from the general guidelines outlined here in developing the differential diagnoses. Patient necessity for additional testing should be evaluated on a case-by-case basis. Humphrey visual field testing is particularly useful in these cases and helps to direct neuroimaging studies when warranted. The reader is reminded that retinal or ocular migraine, retinal vasospasm, and acephalic migraine are diagnoses of exclusion and appropriate work-up should be completed before attributing visual loss to these entities. Elderly patients presenting with visual aura-like symptoms should be evaluated critically for TIA. The reader is also directed to the cited references for additional resources.

CME ANSWERS
1. b
2. b
3. d
4. c
5. c

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Transient Visual Loss

Tina R. Porzukowiak OD, FAAO

Please silence all mobile devices and remove items from chairs so others can sit. Unauthorized recording of this session is prohibited.
Transient Visual Loss

Tina R. Porzukowiak OD, FAAO

No financial disclosures
Learning Objectives

*Develop* a systematic approach for the pt w/ TVL

*Understand* the importance of hx in the DDx

*Review* the components of the ocular exam
Learning Objectives

*Formulate* a DDx for monocular or binocular TVL

*Initiate* a w/u

*Understand* tx options
Pre-Test
A 9 y.o. Native American male presents with recurrent episodes of swirls and bubbles in both eyes for < 2 minutes. What is the MOST likely etiology?

A. Migraine aura without headache
B. Occipital lobe seizure
C. Ocular migraine
D. Transient ischemic attack
A 35 y.o. white female presents with recurrent h/o binocular scintillating scotomomas x 20 minutes duration that moves and marches in the vision; a *headache* follows with associated nausea & phonophobia. What is the MOST likely etiology?

A. Acephalgic migraine  
B. Migraine headache w/ aura  
C. Occipital lobe TIA  
D. Retinal migraine
An 80 y.o. male presents with monocular TVL OD x 5 minutes; there is no associated HA. What is the MOST likely etiology?

A. Acephalgic migraine
B. Occipital lobe ischemia
C. Retinal migraine
D. TIA
A 40 y.o. black female presents with intermittent, painless episodes of monocular TVL OD of <10 seconds duration when looking to the right. What is MOST likely etiology?

A. Carotid artery dissection
B. Ocular migraine
C. Orbital mass
D. TIA
An elderly female presents with an acute CRAO OD. What is the MOST concerning concomitant pathology?

A. Carotid artery dissection
B. Cerebral aneurysm
C. Stroke
D. Tumor
Pattern of Loss & Recovery

**Monocular**
- Altitudinal
- Tunnel vision
- Sudden loss / black out
- Uhthoff sx

**Binocular**
- Sudden loss / black out
- White out
- Tunnel vision
- Homonymous hemianopsia
- Geometric / hexagonal “chicken wire”
- Scintillating scotoma
- Swirling photopsia, bubbles, “flash bulb going off”
Monocular or Binocular

Age

Onset

Duration

Pattern of loss & recovery

Gaze-evoked

Preceded by light exposure

Associated symptoms
Hypertension
Diabetes
Dyslipidemia
Tobacco smoking
Cardiovascular / Cerebrovascular disease
Polymyalgia rheumatica
Miscarriage / stillbirth
Connective tissue disease
Ophthalmic disease
Migraine
Hematologic abnormality
Aneurysm
Brain tumor
Stroke
The history is the most critical portion of your exam.
< 60 seconds NORMAL
> 60 seconds MACULAR DZ
DDx Monocular TVL

Orbital

Ocular

Vascular / Systemic

Nonorganic
Orbital
Ocular
Vasospasm

Retinal / Ocular Migraine

Central Retinal Artery

Vasospasm

Retinal / Ocular Migraine
Vascular or Systemic
Granulomatosis with polyangiitis
Takayasu arteritis
Intravascular Lymphoma
Eosinophilic Granulomatosis with Polyangiitis
Nonorganic
Monocular TVL Work-Up

Gaze-evoked → Ocular problem → Treat
Monocular TVL Work-Up

Normal fundus
Retinal emboli
Retinal artery occlusion
Cotton wool spots
Venous stasis retinopathy
Disc edema
Vascular Risk Factors

Age
Hypertension
Hypotension & syncope
Dyslipidemia
Diabetes
Ischemic heart disease
Tobacco use
Sleep apnea
Giant Cell Arteritis

CBC w/ diff
Westergren ESR
CRP
TAB
Neurology Consult

Contralateral signs or symptoms of neurologic dysfunction
Cardiology Consult

**Carotid & Intracranial Arteries**
- Carotid Duplex
- Transcranial Doppler (TCD)
- Magnetic Resonance Angiography (MRA)
- Computed Tomography Angiography (CTA)

**Heart**
- Electrocardiogram (EKG)
- Trans-esophageal echocardiogram (TEE)
- Holter monitor
Transient Ischemic Attack

CBC w/ diff
ESR
CRP
PT / PTT
Serum electrolytes
BUN
Creatinine
Fasting glucose
Fasting lipid panel
Cardiac enzymes (CK & troponin)
In the presence of normal carotid and cardiac studies, other causes must be ruled out.
Young Patients

- CNS Infection
- Drug Intoxication
- Hyperviscosity / Hypercoaguable state
- Lupus
- Sjögren syndrome
- Syphilis
- Granulomatosis with polyangiitis

- Lumbar puncture
- Urine drug screen
- Hyperviscosity w/u Hypercoagulopathy w/u
- ANA
- Ro-SSA / La-SSB
- RPR / FTA-ABS
- p-ANCA & c-ANCA
Hyperviscosity Work-Up

Serum viscosity
CBC w/ peripheral blood smear
Total protein (TP)
Albumin
CMP
Serum & urine electrophoresis

Consider coagulopathy w/u
CXR
Brain CT / MRI
Panculture
UA
# Hypercoaguable Work-Up

<table>
<thead>
<tr>
<th>CBC w/ diff</th>
<th>Protein S</th>
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<tbody>
<tr>
<td>ESR</td>
<td>Protein C</td>
</tr>
<tr>
<td>ANA</td>
<td>Anti-thrombin III</td>
</tr>
<tr>
<td>Anticardiolipin IgG / IgM</td>
<td>Factor V Leiden</td>
</tr>
<tr>
<td>Anti-β2 glycoprotein I</td>
<td>Prothrombin gene mutation</td>
</tr>
<tr>
<td>Serum protein electrophoresis (SPEP)</td>
<td>Factor VIII</td>
</tr>
<tr>
<td>PT or INR</td>
<td>Homocysteine</td>
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<tr>
<td>PTT</td>
<td>JAK2 mutation</td>
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<tr>
<td>Platelet aggregation test</td>
<td>Von Willebrand factor</td>
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</tbody>
</table>
In the majority of young patients < age 50 with monocular TVL who are otherwise healthy, the diagnostic yield of extensive testing is very low.
Treatment is aimed at prevention of irreversible vision loss, stroke & MI.
Vascular Monocular TVL Treatment
ASA 50-325 mg/d

ASA 25 mg + extended release Dipyridamole 200 mg bid

Clopidogrel 75 mg qd
“bioidentical” hormones
progesterone
“natural” hormones
Premarin
Provera
estrigen
DDx Binocular TVL

- Migraine
- Occipital Lobe Lesion
- Occipital Lobe Ischemia
- Occipital Lobe Seizure
- Other
Migraine
Occipital Lobe Lesion
Occipital Lobe Ischemia
Occipital Lobe Seizure
Other
Binocular TVL Work-Up

- Serology
- MRI
- Lumbar Puncture
Cardiology Consult

**Carotid Arteries**
- Carotid Duplex

**Vertebrobasilar Ischemia**
- TIA/GCA Serology
- MRI brain w/ & w/o contrast
  - FLAIR & DWI
- MRA head & neck w/ contrast

**Heart**
- Electrocardiogram (EKG)
- Trans-esophageal echocardiogram (TEE)
- Holter monitor
Neurology Consult

May be warranted for patients who do not fit the International Headache Society classification for migraine.

MRI brain w/ & w/o contrast
FLAIR & DWI

MRA head & neck w/ contrast
Binocular TVL with preeclampsia

Look for hyperviscosity & hypercoaguuable states.
Vascular binocular TVL treatment is the same as monocular TVL.
Post-Test
A 9 y.o. Native American male presents with recurrent episodes of swirls and bubbles in both eyes for < 2 minutes. What is the MOST likely etiology?

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A. Carotid artery dissection
B. Cerebral aneurysm
C. Stroke
D. Tumor
What Have We Learned?

The history is the most important component of the exam to differentiate TVL.

Ask the patient: “Did you cover each eye?”

Visual field testing can help direct neuroimaging.

Additional testing should be evaluated on a case-by-case basis.
What Have We Learned?

Elderly patients presenting with symptoms of acephalgic migraine with no prior history of migraine should be critically evaluated for TIA.

Patients with evidence of retinal artery occlusion should be evaluated emergently for stroke.
What Have We Learned?

Young patients presenting with monocular TVL are more likely to have a benign cause.

Young patients with binocular TVL & preeclampsia should undergo a work-up for hyperviscosity & hypercoaguable states.
What questions do you have?
Tina R. Porzukowiak OD, FAAO
tporzu@midwestern.edu
623.537.6000
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