Transient Monocular Visual Loss

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• PURPOSE: **To provide a practical update on the diagnosis and management of transient monocular visual loss (TMVL).**
• DESIGN: **Perspective.**
• METHODS: **Review of the literature.**
• RESULTS: **TMVL is an important clinical symptom. It has numerous causes but most often results from transient retinal ischemia. It may herald permanent visual loss or a devastating stroke, and patients with TMVL should be evaluated urgently. A practical approach to the evaluation of the patient with TMVL must be based on the patient's age and the suspected underlying etiology. In the older patient, tests should be performed to investigate giant cell arteritis, atherosclerotic large vessel disease, and cardiac abnormalities. In the younger patient, TMVL is usually benign and the evaluation should be tailored to the particular clinical setting.**
• CONCLUSIONS: **Specific prevention strategies are tailored to the most likely cause of TMVL and the patient's underlying risk factors. Prevention of a future event should begin in the ophthalmologist's office with education and aggressive treatment of artherosclerotic risk factors. (Am J Ophthalmol 2005;140:717–722. © 2005 by Elsevier Inc. All rights reserved.)**

TRANSIENT MONOCULAR VISUAL LOSS (TMVL) IS THE preferred term for abrupt and temporary visual loss in one eye.[1,2] The term "amaurosis fugax" (from the Greek, amauros, meaning "dark," and the Latin, fugax, meaning "fleeting") does not specify whether the visual loss occurred in one eye or both,[1,3,4] and some authors have suggested that "amaurosis fugax" implies that the visual loss is of ischemic origin.[3] As indicated in Table 1, there are non-ischemic causes of TMVL.[5,6] The term "transient monocular blindness," introduced by CM Fisher[3] is also not ideal because it implies that vision was entirely lost when most episodes of TMVL do not cause total loss of vision.[2]

The most important step in evaluating a patient with visual loss is to establish whether the visual loss is monocular or binocular.[2,5] Monocular visual loss always results from lesions anterior to the chiasm (the eye or the optic nerve), whereas binocular visual loss results from lesions of both eyes or optic nerves, or, more likely, of the chiasm or retrochiasmal visual pathway.

Deciding whether an episode of abrupt visual loss occurred in one eye or both is not always easy. Very few patients realize that binocular hemifield (homonymous) visual field loss affects the fields of both eyes. They will usually localize it to the eye that lost its temporal field. The best clues to the fact that visual loss was actually binocular are reading impairment (monocular visual loss does not impair reading unless the unaffected eye had prior vision impairment) and visual loss confined to a hemifield (monocular visual loss does not usually cause that pattern of visual loss).[2]

• MECHANISMS OF TMVL: In most cases of TMVL, the underlying mechanism is ischemia—to the retina or optic nerve (Table 1). Impaired ocular perfusion results from vascular conditions in or near the eye or remote from it.

• TMVL CAUSED BY CONDITIONS IN OR NEAR THE EYE: Ocular disorders such as intermittent angle closure glaucoma and hyphema may be mistaken for TMVL, although these episodes usually last longer. Ocular surface problems may cause intermittent blurred vision but are rarely mistaken for TMVL. Transient monocular visual loss may result from impaired perfusion in the retinal, choroidal, or optic nerve ciliary arteries, or less commonly, reduced flow in the venous circulation draining the eye.

Retinal Artery Disorders. Transient visual loss may occur with impending or partial retinal artery occlusion, typically at the level of the lamina cribrosa. In most such cases, TMVL is described as areas of blank or fuzzy vision ("negative visual phenomena"). Less commonly, the patient describes flashes, sparkles, or scintillations ("positive visual phenomena"). Ophthalmoscopy may reveal no abnormalities or show cotton wool spots, retinal emboli, retinal hemorrhages, retinal whitening, or dilated retinal

Accepted for publication Apr 10, 2005.
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This study was supported in part by National Institutes of Health CORE Grant P30-EY06360 (Department of Ophthalmology); and a departmental grant (Department of Ophthalmology) from Research to Prevent Blindness, Inc, New York, New York.
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0002-9394/05/$30.00
doi:10.1016/j.ajo.2005.04.020

TABLE 1. Differential Diagnosis of Transient Monocular Visual Loss (TMVL)

Vascular:
Orbital ischemia (ophthalmic artery)
Retinal ischemia (central retinal artery and its branches, central retinal vein)
Optic nerve ischemia (short posterior ciliary arteries/ophthalmic artery)
Choroidal ischemia (posterior ciliary arteries)
"Retinal migraine" (vasospastic TMVL)
Ocular diseases:
Anterior segment:
Dry eyes
Keratoconus
Hyphema
Angle closure glaucoma
Retinal detachment
Optic nerve disorders:
Papilledema (transient visual obscurations)
Optic disc drusen (transient visual obscurations)
Congenitally anomalous optic disc (transient visual obscurations)
Optic nerve compression (gaze-evoked TMVL)
Uhthoff's phenomenon (demyelination)

veins. Common causes are arteriosclerosis and hypercoagulable/hyperviscous states.[1,2,5–7] Less common causes are vasculitis, radiation toxicity, and sudden elevations in intraocular pressure.[2,5,6,8] Giant cell arteritis uncommonly causes TMVL by compromising retinal arterial flow; more frequently, the optic nerve circulation is affected.[9]

Idiopathic TMVL in Young Individuals: Vasospasm? Young people who have no evidence of vasculopathy may have episodes of TMVL secondary to reversible vasospasm of retinal arteries.[6,10–12] Documentation of this phenomenon is meager. Such vasospasm may be the basis for the TMVL of "retinal migraine."[10–12] According to the International Headache Society Criteria,[13] the diagnosis of retinal migraine should be made only in patients who develop positive visual phenomena over a few minutes and who have a history of migraine. In listening to the history, it is impossible to distinguish TMVL as an isolated symptom of retinal migraine/vasospasm from TMVL of other causes.[14] Therefore, retinal migraine/vasospasm should remain a diagnosis of exclusion.

Retinal Vein Disorders. Recurrent TMVL may also arise from impending occlusion of the central retinal vein.[15] Common causes are, as with retinal artery occlusion, arteriosclerosis and hypercoagulable states. One must also consider conditions that specifically cause orbital venous hypertension, such as compressive orbitopathies and carotid-cavernous fistulas.

Choroidal Artery Disorders. TMVL from isolated choroidal ischemia is rare and should point to a vasculitic process such as giant cell arteritis.[9,16]

Optic Nerve Arterial Disorders. TMVL is rarely a premonitory symptom of ischemic optic neuropathies. In those cases, arteritic (rather than nonarteritic) ischemic optic neuropathy should be suspected.[5,9,16]

Optic Disease Edema or Anomaly. Another important cause of TMVL is a swollen or congenitally anomalous optic disk (with or without optic disk drusen), which may pinch off its own ciliary blood supply or the central retinal artery.[2,5,17] In such cases, the episodes of TMVL last only seconds, occur frequently during the day, and are often provoked by changing gaze position, or, more commonly, by assuming the upright posture (orthostatic). These ultra brief episodes of visual loss are called "transient visual obscurations."[2,5,17] Orbital tumors may intermittently compress the ophthalmic or central retinal artery, thereby producing brief gaze-evoked TMVL.[5]

• **TMVL CAUSED BY CONDITIONS REMOTE FROM THE EYE:** TMVL associated with conditions remote from the eye is caused by impaired ocular arterial flow from: (1) retinal emboli that originate in proximal arteries or the heart[5,18]; or (2) stenosis of the ophthalmic, carotid, aortic arch arterial vessels[5]; or (3) reduced cardiac output or systemic hypotension.[1,2,5]

Retinal Emboli. TMVL was first linked to retinal arterial emboli 50 years ago when white fragments were observed by ophthalmoscopy to travel through the retinal arterial tree during episodes of TMVL.[4,7] These emboli originate most often from an atherosclerotic plaque at the carotid bifurcation and less commonly from the aortic arch or ophthalmic artery. Patients with this symptom typically complain of TMVL that lasts a few minutes at most.[19] The visual loss may be sectorial, usually altitudinal.

Anterior Circulation Stenosis. Severe stenosis of the carotid or ophthalmic arteries or stenosis of the aortic arch (in Takayashu arteritis or severe aortic arch atherosclerosis) may cause TMVL by hypoperfusion rather than embolism.[1,2,5,20]

Hypotension. Finally, reduced cardiac output or systemic hypotension may produce TMVL.[5] Although TMVL is not typically an isolated symptom of systemic hypotension, which generally also causes lightheadedness, confusion, and binocular visual loss, the combination of a drop in systemic blood pressure and asymmetric anterior circulation stenosis may cause TMVL alone, particularly orthostatically-induced TMVL.[2,5]

Chronic Ocular Hypoperfusion. Chronic ocular hypoperfusion of any mechanism may be associated with transient but prolonged visual loss (several minutes to hours) and positive visual phenomena.[5,20–22] A camera-diaphragm effect is reported by some patients. TMVL may be induced by situations that further decrease perfusion pressure (postural change) or increase retinal oxygen demand (exposure to bright light).[20,21] Borderline ocular perfusion may not be able to maintain retinal metabolic activity when blood flow is diverted to other tissues as after eating a meal or during exercise.[22] Chronic hypoperfusion of the eye may also induce delay in the regeneration of visual pigments in the photoreceptor layer of the retina, resulting in blurred or absent vision that persists until regeneration of visual pigment occurs. Impaired dark adaptation may be a consequence of this phenomenon. In these cases, examination often discloses the findings of venous stasis retinopathy or the ischemic ocular syndrome (dilated retinal veins, perivenous retinal hemorrhages, retinal or iris neovascularization, ocular hypotony, anterior chamber cells and flare, cataract).[5]

• **NATURAL HISTORY OF TMVL:** The natural history of patients with TMVL depends on the age of the patient and the etiology of the TMVL.[1,2,5,19,23–28]

Retinal Stroke. A major adverse outcome is persistent visual loss, mostly attributable to retinal artery occlusion. On the basis of several natural history studies, the aggregate risk of permanent ipsilateral visual loss is approximately 1% to 2%/y.[25]

Cerebral Hemispheric Stroke. TMVL may also herald a cerebral infarction. When carotid occlusive disease is related to atherosclerosis, TMVL is a marker of systemic atheromatous disease, and is associated with a higher risk of vascular death.[1,5,19,20,27] The North American Symptomatic Carotid Endarterectomy Trial (NASCET) study showed a 25% 3-year risk of stroke in patients with hemodynamically marked carotid stenosis causing ipsilateral TMVL, cerebral hemispheric transient ischemic attack (TIA), or mild stroke.[28]

Death. The risk of death in patients with TMVL and atheromatous carotid stenosis is approximately 4%/y, mainly related to myocardial infarction.[1,5,19,20,25] Patients with retinal and hemispheric TIAs are equally vulnerable.

These data stress that TMVL is a marker for systemic arteriosclerosis and should prompt immediate comprehensive patient evaluation.

• **DIAGNOSIS/HISTORY:** The patient's description of the visual characteristics of an attack seldom allows the physician to determine its cause.[2,14] The duration of visual loss can vary from seconds to hours for any cause other than papilledema or congenital optic disk anomalies, where attacks never last more than seconds. TMVL lasts longer in cases of ocular hypoperfusion or venous congestion than in cases of emboli.[1,5]

The location of the scotoma within the visual field does not specify a particular mechanism. For example, an ascending or descending ("curtainlike") spread of blindness, commonly held as specific for embolism, can occur in patients with hypotension and vasospasm.[1] Positive visual phenomena are also nonspecific, signaling a lower degree of ischemia than is necessary to produce negative visual phenomena. However, positive visual phenomena may be associated more with nonembolic hypoperfusion than with emboli.[1,5,20,29]

The following accompanying manifestations are generally helpful in determining a mechanism of TMVL: (1) zigzag (fortification) scintillations—visual cortex migraine; (2) headache, scalp tenderness, jaw claudication—impending ciliary occlusion in association with giant cell arteritis; (3) eye or brow pain—intermittent angle closure glaucoma or ciliary ischemia in association with giant cell arteritis; (4) neck pain—cervical carotid dissection; (5) presyncope—systemic hypotension or a hyperviscosity syndrome; (6) ipsilateral Horner syndrome—carotid disease such as a dissection; (7) simultaneous contralateral hemisensory or motor findings—ipsilateral carotid stenosis.

• **OPHTHALMIC EXAMINATION:** This is a critical step to rule out local ocular causes of TMVL (Table 1) and to detect retinal emboli, retinal ischemia, venous stasis retinopathy, or evidence of optic nerve ischemia.[1,2,5,7] However, retinal emboli are transient and may be missed if the ocular examination is delayed.

• **ANCILLARY STUDIES:** In older patients, complete blood count, erythrocyte sedimentation rate, and C-reactive protein should be obtained emergently to rule out giant cell arteritis.[1,2,5,9]

Carotid and cardiac sources of emboli can usually be ruled out by ultrasonic examination. Two studies recently confirmed that retinal ischemia is caused more often by carotid stenosis than by cardiac-source emboli.[30,31] Therefore, cervical carotid ultrasound should be obtained emergently, looking for carotid dissection or carotid atheroma. Evaluation of the intracranial circulation by transcranial Doppler in patients with severe carotid stenosis is helpful in predicting the risk of stroke.[5,27,32] Doppler of the ophthalmic artery may show poor flow in some patients with normal carotid arteries, suggesting stenosis or occlusion of the ophthalmic artery.[5] Computed tomography or magnetic resonance angiography are sometimes used when reliable ultrasound is not available. A trans-thoracic echocardiogram may be helpful in some patients, but a transesophageal echocardiogram is required in patients with a negative preliminary workup because cardiac sources of emboli and aortic arch atheroma are better evaluated with this method.[5,32–34]

TABLE 2. Recommendations for the Prevention of Stroke and Cardiovascular Events in Patients With Transient Monocular Visual Loss

Risk factor management:
Blood pressure should be decreased in both hypertensive and nonhypertensive patients
Cigarette smoking should be discontinued
Coronary artery disease, cardiac arrhythmias, congestive heart failure, and valvular heart disease should be treated appropriately
Excessive use of alcohol should be eliminated (limit to 1 or 2 drinks a day)
Treatment of hyperlipemia is recommended. If a lipid-lowering agent is used, a statin is recommended
Fasting blood glucose levels <126 mg/dL are recommended
Physical activity is recommended
Discontinuation of postmenopausal estrogen replacement therapy should be considered
Antiplatelet agents:
Long term antiplatelet agents should be given to every patient who has experienced a non-cardioembolic TMVL and has no contraindication (aspirin 50 to 325 mg qd, combination of aspirin 25 mg and extended-release dipyridamole 200 mg bid, clopidogrel 75 mg qd, are all acceptable options for initial therapy)
Oral anticoagulants:
Long term anticoagulation (target INR 2.5) should be used for prevention of stroke in patients with atrial fibrillation with other high-risk cardiac sources of embolism and in some hypercoagulable states
Carotid endarterectomy:
Should be considered only in selected high risk patients, who are good surgical candidates and with an ipsilateral internal carotid stenosis of 70% to 99%

In selected patients who lack conventional arteriosclerotic risk factors and whose vascular and cardiac evaluations are negative, hypercoagulable states should be ruled out with CBC, platelet count, serum protein electrophoresis, prothrombin and partial thromboplastin times, antiphospholipid antibodies, protein S and C, antithrombin III, factor V Leiden and prothrombin gene mutations, factor VIII, and plasma homocysteine.[35]

Most individuals who suffer TMVL in youth (age <40 years) will have no ophthalmologic or blood laboratory abnormalities, constitutional manifestations, or major arteriosclerotic risk factors.[5,10,32,36] Their chance of future stroke is low.[10,32,36]

- **TREATMENT:**

Local Causes. Local causes of TMVL must be addressed directly. When vasospasm is suspected, calcium-channel blocker treatment may reduce the frequency of TMVL attacks.[12]

Remote Causes. If local causes are not found and there are no retinal emboli or biologic markers of inflammation, the physician should be guided by the results of ultrasonic studies. If the carotid ultrasound is negative, attention centers on the aortic arch and the heart. All patients should be given antiplatelet agents acutely (such as aspirin, clopidogrel, or combination of aspirin and dipyridamole). Aggressive treatment of arteriosclerotic vascular risk factors is essential (see below).[1,2,5,26,27,37]

Carotid Endarterectomy. A common question is whether patients with TMVL and high-grade (>70%) internal carotid stenosis should undergo carotid endarterectomy. Two large collaborative trials[38,39] published in the early 1990s compared conventional medical therapy to carotid endarterectomy in patients with TMVL, hemispheric transient ischemic attack, or hemispheric mild stroke. Both trials found that carotid endarterectomy overall considerably reduced the future risk of ipsilateral stroke if carotid stenosis was greater than 70%. However, in 2001, Benavente and associates[28] compared the stroke rate among patients with 50% to 99% carotid stenosis who presented with TMVL to that of patients who presented with hemispheric TIA in the NASCET study completed in 1990. TMVL was the presenting symptom in 397 patients, and hemispheric transient ischemic attack in 829 patients. In medically-treated patients with TMVL, the 3-year risk of ipsilateral stroke was 10%; in medically-treated patients with hemispheric TIA, the 3-year risk was 20%. Thus, the medically treated patients with TMVL were at substantially lower risk of subsequent hemispheric stroke than were those with hemispheric TIA.

In the NASCET, there were six risk factors for stroke in the TMVL group: (1) age of 75 years or greater; (2) male gender; (3) history of hemispheric transient ischemic attack or stroke; (4) history of intermittent claudication; (5) ipsilateral internal carotid artery stenosis of 80% to 94%; and (6) absence of intracranial collateral vessels on cerebral angiography. Among those with one or fewer of these risk factors ("low-risk group"), the 3-year risk of ipsilateral stroke was 1.8% with medical treatment and 2.2% with endarterectomy. Nearly 80% of the patients fell into this low-risk group. The risk of stroke was higher in those with two or more risk factors

("moderate-risk group"), but endarterectomy was beneficial only in the group with three or more of the above-listed risk factors ("high-risk group"). Even so, seven high-risk patients would have to undergo endarterectomy to prevent one stroke.[28]

With this information in mind, carotid endarterectomy does not appear to be indicated for most patients with TMVL, even with hemodynamically significant ipsilateral carotid stenosis. Only those few patients who fall into the high-risk group (three or more risk factors listed above) would be candidates.[28,40]

Other Measures. Among noncandidates for carotid endarterectomy, those who do not have a cardiogenic source of emboli should be managed with antiplatelet agents and, more importantly, with aggressive treatment of arteriosclerotic risk factors.[37,40] Indeed, specific prevention strategies are tailored to the most likely cause of the TMVL and the patient's underlying risk factors. Numerous prospective studies and clinical trials have shown a decreased risk of stroke and other cardiovascular events with control of modifiable risk factors, especially hypertension and smoking (Table 2).[5,37] Prevention of a future event should begin with education in the ophthalmologist's office.

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Biosketch

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