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REVIEW

Transient Monocular Visual Loss (Amaurosis Fugax): How Does Age Impact Diagnosis?

Vivian Paraskevi Douglas · Sruti S. Rachapudi · Pamela Davila-Siliezar · Noor A. R. Laylani · Andrew G. Lee

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ABSTRACT

Transient monocular visual loss (TMVL), also known as transient monocular blindness or amaurosis fugax ("fleeting blindness"), is a temporary loss of vision often due to ischemia to the retina. While acute TMVL should be considered an emergency that further requires exhaustive investigation, there are some cases in which TMVL arises secondary to benign causes. Age has a major impact in the diagnosis of ischemia and although the differential diagnosis of TMVL can be broad, timely and appropriate history, examination, diagnostic testing, and treatment can be vision- or life-saving. We review the causes of TMVL and the impact of age on the differential diagnoses and management.

Keywords: Transient monocular visual loss; TMVL; Amaurosis fugax; Age; Diagnosis

V. P. Douglas
Department of Ophthalmology, Athens Naval Hospital, Athens, Greece

S. S. Rachapudi · A. G. Lee
Department of Ophthalmology, University of Texas Medical Branch, Galveston, TX, USA

P. Davila-Siliezar · N. A. R. Laylani · A. G. Lee (✉)
Department of Ophthalmology, Blanton Eye Institute, Houston Methodist Hospital, 6560 Fannin St. Ste 450, Houston, TX 77030, USA
e-mail: aglee@houstonmethodist.org

A. G. Lee
Departments of Ophthalmology, Neurology, and Neurosurgery, Weill Cornell Medicine, New York, NY, USA

A. G. Lee
University of Texas MD Anderson Cancer Center, Houston, TX, USA

A. G. Lee
Texas A and M College of Medicine, Bryan, TX, USA

A. G. Lee
Department of Ophthalmology, The University of Iowa Hospitals and Clinics, Iowa City, IA, USA

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Key Summary Points
Transient monocular visual loss (TMLV) is a clinical entity which can occur at any age, yet there is a positive association with age.
Thorough medical history, clinical and ophthalmic examination are crucial for establishing a timely and accurate diagnosis in both young and older individuals.
TMVL can often present a diagnostic challenge while there are age-related patterns and risk factors that can aid in the differential diagnosis.
Older patients are at higher risk of developing cerebrovascular disorders compared to younger healthy individuals.

INTRODUCTION

Transient monocular visual loss (TMVL) can be due to a transient ischemic attack (TIA). TMVL is characterized by sudden, painless, and reversible partial or total visual impairment. The name amaurosis fugax derives from the Greek word *amaurosis*, which means darkening or obscure, and the Latin word *fugax*, which means fleeting. The duration of ischemic TMVL varies from few seconds to minutes with the majority of those lasting less than 15 min and rarely more than 30 min.

In 1990, the Amaurosis Fugax (AF) Study Group proposed five distinct categories as causes of AF: embolic, hemodynamic, ocular, neurologic, and idiopathic [1]. However, owing to the diagnostic advancements, ischemic TMVL is currently considered a result of an underlying circulatory, ocular, or neurologic cause. In most cases ischemic TMVL is associated with vascular thromboembolic events at the level of the ipsilateral internal carotid artery. Ischemic TMVL is more commonly seen in patients over the age of 50 years with additional vascular risk factors including hypertension, diabetes mellitus, hyperlipidemia, and tobacco use. Nevertheless, patients at any age can be affected with TMVL. Patients can present with TMVL and then develop a central or branch retinal artery occlusion. TMVL can also occur in central retinal vein occlusion and in arteritic anterior ischemic optic neuropathy. Multiple sclerosis, migraine, papilledema, intraorbital or intracranial tumors, or compressive lesions on the optic nerve such as aneurysms can mimic ischemic TMVL and should be included in the differential diagnosis (Table 1).

Patients with presumed ischemic TMVL should receive an expedited and thorough diagnostic evaluation for stroke. Laboratory evaluation might include a complete blood count, coagulation studies, complete metabolic and lipid panel, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) [2]. In addition, as a result of the increased risk of stroke and mortality association with ischemic TMVL, neuroimaging [e.g., cranial computed tomography (CT) scan or magnetic resonance imaging (MRI) of the head and neck] plays an important role in the assessment and detection of an underlying ischemic condition. Vascular imaging studies include carotid Doppler ultrasound, CT, and MR angiography of head and neck. Specialized MRI sequences like diffusion-weighted imaging can show acute ischemic lesions in the brain. Electrocardiogram and echocardiography should also be considered for excluding cardiogenic source of ischemia [2, 3].

METHODS

A review of the PubMed database was performed using the search terms “amaurosis fugax”, “transient visual loss”, “transient monocular visual loss”, “acute retinal ischemia”, “age” until February 2024. We focus on the most recent studies and clinical trials on ischemic TMVL. We included all types of articles (original, reviews, case reported, case series, editorials, meta-analyses) related to TMVL and age. Only relevant abstracts in English were reviewed.

Table 1 Differential diagnosis of transient monocular visual loss (TMVL) according to the age

Younger individuals (< 50 years of age)	Older individuals (≥ 50 years of age)
Inherited coagulation disorders	Carotid/ophthalmic artery stenosis
Migraine	Carotid/cardiac embolism
Psychogenic	Inherited or acquired coagulation disorders
Optic disc drusen	Hypoperfusion
Angiospasm/vasospasm	Heart failure
Multiple sclerosis	Arrhythmias
Papilledema	Compressive intracranial lesions
Compressive intracranial lesions	Orbital tumor
Dry eye syndrome	Choroidal/retinal ischemia
Optic neuritis	

This table presents the most common causes encountered in clinical practice. However, it should be noted that overlap might be seen across these groups and meticulous assessment of present and past medical and family history is essential for determining the underlying cause of TMVL

This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors. No approval from local ethics committee was required.

RESULTS

Combination of the above search terms resulted in the following number of publications: "amaurosis fugax and age", 231; "transient visual loss and age", 11,968; "transient monocular visual loss and age", 28; "acute retinal ischemia and age", 132. Abstracts were screened by VPD, SSR, PDS, NAARL and in cases of discrepancies these were further discussed with AGL. Only relevant articles in English were reviewed. In total, 36 articles were included in this study.

Transient Monocular Visual Loss in Young Individuals

While ischemic TMVL is more commonly seen in middle-aged and elderly people, TMVL has also been described in younger patients [4]. In 1988, Appleton et al., reported episodes of unilateral and bilateral TVL in five teenagers having a characteristic mosaic or jigsaw pattern of visual loss likely related to ischemia of the choroid and representing a migraine variant. As such, the authors suggested that in young patients without significant medical and clinical history, cerebral angiography is not indicated [4]. O'Sullivan et al. reported that three out of their nine patients with TMVL were adolescents [5]. After thorough examination, there was no evidence of atheromatous carotid disease or thromboembolism. Pupil dilation was reported in one of the patients during the attack that might have developed secondary to flow disturbance in the ciliary ganglion or perhaps was a migraine equivalent [5].

Mantagos et al. reported a 15-year-old male patient who presented with TMVL possibly secondary to rheumatic microemboli which led to retinal vasospasm and impeded retinal artery flow [6]. Shahar et al. described a patient with recurrent TVL episodes and cycloplegia of approximately 5 mins duration twice per month for about 6 months [7]. The patient's medical and family history as well as physical, ophthalmologic, neurologic examinations and relevant imaging and laboratory studies were unremarkable. The authors thus speculated that these episodes are likely the result of retinal migraine in the absence of headaches [7].

Overall, studies have shown that most patients aged less than 40 years with TMVL are at low risk for future stroke and in the majority of cases an underlying cause of the visual disturbance was rarely demonstrated [3, 8]. Tippin et al. reviewed 83 patients with TVL and they found that 41% of these patients had headache or orbital pain at the time of the attacks and 25.3% had severe headaches independent of the

TVL [9]. All patients underwent extensive work up with only one patient being diagnosed with heart disease on echocardiography study and nine of the patients had also cerebral TIA without stroke. Over a mean period of 5.8 years follow-up none of the patients developed a stroke, leading to the conclusion that TVL in younger patients is likely associated with migraines and carries a better prognosis and that further testing is unwarranted [9].

Careful consideration should be also given in cases of hypercoagulable disorders which can be inherited or acquired [10]. Individuals with a personal and/or family history of thrombotic events should be also tested for heritable thrombophilias which are linked to increased risk for TVL and in some instances for TIA. More specifically along with the regular laboratory tests, patients should be tested for antithrombin III deficiency, protein C and S deficiency, methylene tetrahydrofolate reductase mutation, factor V Leiden, and G20210A mutations [10]. Pahus et al. found that heterozygote patients for factor V Leiden were at higher risk of TMVL and TIA with odds ratio of 1.99 [11]. Hyperestrogenemia (endogenous or exogenous), antiphospholipid syndrome as well as smoking, recent surgery, trauma, and prolonged immobilization have also been implicated as possible prothrombotic risk factors in TMVL [12–16].

Drug abuse has been also linked to the development of TMVL. Shaw et al. described a 26-year-old man with multiple episodes of TMVL over 18 h following nasal inhalation of methamphetamine and also retinal vasculitis in the same eye. The authors postulated that TMVL was secondary to the sympathomimetic effect of the drug along with the vasospasm induced by the vascular irritation of the underlying vasculitis [17]. Intravascular emboli related to drug abuse may also produce transient vision loss [18]. In 2001, a report of TMVL secondary to inhaled phencyclidine intoxication was added to the literature [19].

Transient Visual Loss in Older People

TMVL is a common symptom in older adults that can be due to a variety of etiologies. Among the causes of TMVL, giant cell arteritis (GCA) is a well-established large vessel vasculitis of the elderly that requires early diagnosis and prompt treatment with corticosteroids. GCA can affect cranial and extracranial branches of the carotid arteries, which may lead to TMVL due to optic nerve, choroidal, or retinal ischemia [20]. Genetic predispositions (e.g., HLA DR3, DRW6, and DRB1*04) [21] and infectious factors (e.g., *Chlamydia pneumoniae*) have been proposed as risk factors for GCA. Giant cells are formed when activated T cells stimulate the differentiation of monocytes into macrophages, which then aggregate into granulomas and contribute to the destructive inflammation of arterial walls, leading to progressive vessel occlusion and ischemic symptoms. GCA is a disorder of the elderly and there is a higher prevalence in women. This condition is commonly seen in populations of northern European descent, with Scandinavian people being particularly susceptible. In a retrospective cohort study conducted in Norway, the average annual cumulative incidence rate of GCA was found to be 16.7 per 100,000 people aged over 50 [22]. Although GCA in black patients was previously thought to be rare, the available data suggests that the disease occurs at a similar rate in white and black patients [23].

Not all patients have TMVL and many patients with GCA present with acute visual loss [20]. Visual loss can be transient at first and then become permanent. GCA can present with ocular symptoms alone without systemic findings in up to 38% of cases. In addition to headache, jaw claudication, and visual loss, other less common presentations of GCA include transient or constant diplopia [20].

In a retrospective study of 36 patients with systemic GCA, only 8 (22%) had a history of TMVL [24]. In a similar study of 122 patients with GCA at the Mayo clinic, TMVL was observed in 20 cases (16%), followed by an acute loss of vision in 16 of these cases and diplopia in 12 [25]. Whitfield et al. reported that eight out of 72 patients with GCA (17%)

experienced TMVL preceding permanent visual loss in three patients [26]. In a more recent study conducted in the corticosteroid era, out of 170 patients with GCA, 85 of whom had ocular manifestations, 26 (15%) reported TMVL, while 5 (3%) experienced diplopia. In both cases of ocular symptoms, approximately two-thirds of the patients subsequently developed permanent visual loss [27].

TMVL in GCA is an emergent condition, and it is essential to establish the diagnosis and to treat promptly to prevent permanent vision loss. Although GCA is the most common vasculitis of the elderly, the diagnosis can be challenging at times because of the limitation of the American Rheumatology Association classification criteria [28] and the significant proportion of biopsy-negative patients with GCA. While temporal artery biopsy is the traditional method to validate the diagnosis of GCA, it is important to acknowledge that inflammation in the temporal arteries may only impact certain segments, leading to inaccurately negative biopsy results when assessed using conventional hematoxylin and eosin staining. The average sensitivity of unilateral temporal artery biopsy is 86.9% [29], indicating that it is not a reliable source for GCA diagnosis. A conclusive pathological examination of the temporal artery biopsy would reveal positive findings indicative of GCA, such as panarteritis inflammation, CD4-positive lymphocytes, macrophages, giant cells, and fragmentation of the internal elastic lamina [30]. Patients who are clinically resembling GCA despite a negative biopsy should undergo additional testing. Ancillary testing for GCA includes fluorescein angiography showing delayed peripapillary artery filling and choroidal issues, a Doppler ultrasound or ultrasound biomicroscopy showing temporal arterial edema indicated by hypoechoic halos. ESR and CRP are commonly used in diagnosing GCA, but can occasionally be normal in some patients with GCA [29]. While the initial treatment for GCA is corticosteroids, started promptly upon clinical suspicion, the dosages can vary according to risk [29]. Steroid-sparing agents such as methotrexate and are options, and current therapeutics such as tocilizumab are being investigated [29]. Early diagnosis and treatment of the disease play a key role in the visual prognosis of these patients.

TMVL is a sudden and transient loss of vision of short duration lasting from seconds up to a few minutes which can be the result of vascular abnormalities or ischemic conditions. An accurate history is key for timely assessment of the underlying condition and for establishing a diagnosis. The age of the patient and the past medical history are of considerable importance and further guide the differential diagnosis and prognosis. Older patients are more likely to have ischemic TMVL (including GCA) than younger patients. As a result of the short duration of those attacks, patients may ignore the initial episode but with repeated events may present to the emergency department after the vision has returned to normal. In this setting, the ocular examination is likely to be normal and therefore a complete history is necessary to make the triage decision for further evaluation and possible hospitalization. Patients should undergo a full ocular examination to exclude intraocular causes of TMVL (e.g., papilledema, optic disc drusen, angle closure glaucoma).

A complete blood count, ESR and CRP, and routine chemistry panels should be considered in patients over the age of 50 years [3], although in selected cases such as in patients with either inherited or acquired coagulation disorders laboratory tests should be offered to younger individuals [10].

It is considered that retinal ischemia is analogous to cerebral transient ischemic attacks and thus an urgent workup and management is required to prevent potential complications [31–33]. In the recent study by Chen et al. the risk factors for ischemic stroke were reviewed in a total of 12,142 patients from the National Inpatient Sample Database who presented with TMVL between 2022 and 2014. Ischemic stroke and myocardial infarction were detected in about 0.3–0.9% of the cases while hypercoagulable state, atherosclerosis, coronary artery disease, and systemic vasculitis can triple the risk of stroke [34]. In 2018, Biousse et al. presented the guidelines that should be followed by all eye care providers when someone presents with acute retinal arterial ischemia including TMVL,

Table 2 Typical evaluation performed urgently in certified stroke centers for patients with acute retinal ischemia (vascular transient monocular vision loss, branch retinal artery occlusion, or central retinal artery occlusion) based on the American Heart Association and National Stroke Association recommendations [35]

1	Evaluation as soon as possible after the onset of acute visual loss, with accelerated triage in an emergency center affiliated with a stroke center or rapid-access TIA clinic/stroke center, depending on availability and local resources
2	Routine blood tests[a] (complete blood count with platelets, chemistry panel, hemoglobin A1C, prothrombin time and partial thromboplastin time, and fasting lipid panel) are reasonable. Erythrocyte sedimentation rate and C-reactive protein are necessary in patients older than 50 years to screen for inflammation, which may suggest giant cell arteritis
3	Electrocardiography[a] should occur as soon as possible after the event. Prolonged cardiac monitoring[a] (inpatient telemetry or Holter monitor) is useful in patients with an unclear etiology after initial brain/vessel imaging and electrocardiography
4	Patients should preferably undergo neuroimaging evaluation[a] within 24 h of symptom onset. MRI without contrast, including DWI, is the preferred brain diagnostic imaging modality. If MRI is not available, head CT should be performed
5	Noninvasive imaging of the cervicocephalic vessels[a] should be performed routinely as part of the evaluation of patients with suspected vascular TMVL and those with BRAO or CRAO. MRA or CTA, or carotid ultrasound/transcranial Doppler, should be obtained, depending on local availability and expertise (it is often easier to obtain an MRA if a brain MRI is obtained or a CTA [with contrast] if a head CT is obtained)
6	Echocardiography[a] (at least transthoracic echocardiography) is reasonable, especially when the patient has no cause identified by other elements of the workup. Transesophageal echocardiography is useful in identifying left atrial thrombus, patent foramen ovale, aortic arch atherosclerosis, and valvular disease and is reasonable when identification of these conditions will alter management. Depending on local resources, outpatient echocardiography may be obtained after discharge in patients with otherwise normal cardiac evaluation
7	It is reasonable to hospitalize patients with TIA, BRAO, or CRAO if they present within 72 h of the event and any of the following criteria are present:
	Abnormal brain DWI-MRI showing evidence of acute cerebral infarction(s)
	Large artery atherosclerosis found on noninvasive vascular imaging (such as internal carotid artery stenosis)
	Abnormal cardiac evaluation
	Recurrent episodes (crescendo TIAs), or inability to provide expedited outpatient follow-up

BRAO branch retinal artery occlusion, *CRAO* central retinal artery occlusion, *CT* computed tomography, *CTA* computed tomography angiography, *DWI* diffusion-weighted imaging, *DWI-MRI* magnetic resonance imaging with diffusion-weighted imaging sequences, *MRA* magnetic resonance angiography, *MRI* magnetic resonance imaging, *TIA* transient ischemic attack, *TMVL* transient monocular vision loss

[a]These tests are obtained immediately in the emergency facility over a 23-h observation period, during which the patient receives cardiac monitoring. These tests are part of the standard "stroke protocol" recommended by the American Heart Association/National Stroke Association. If no cause is identified, the patient can be discharged home after 24 h with optimal secondary prevention of stroke. If a test identifies an embolic cause requiring immediate treatment (such as internal carotid artery stenosis of at least 50% or cardiac source of emboli), the patient should be admitted to a stroke unit. In all cases, the patient is discharged with appropriate secondary stroke prevention measures, including an antithrombotic agent, statin for hyperlipidemia, and blood pressure control, and an outpatient follow-up with a neurologist with stroke expertise is arranged within 2 weeks after discharge to review the tests obtained and to promote optimal secondary prevention of stroke and other cardiovascular diseases. These recommendations apply to patients in whom the diagnosis of giant cell arteritis is not considered

branch or retinal artery occlusion [35]. In this article the importance of understanding the pathophysiology of these entities and the risks associated with them are highlighted while key recommendations are made in an effort to educate and help the providers and thus further promote multidisciplinary collaborations [35]. All patients should be evaluated as soon as possible and referral to near certified stroke center is crucial for timely evaluation [35, 36]. Table 2 presents the diagnostic protocol for all patients who present urgently in a stroke center including the lab workup and imaging studies that need to be performed. Accurate and timely diagnosis is the cornerstone of prevention of stroke and cardiovascular diseases.

FUTURE DIRECTIONS

Future directions in the management of TMVL involve a multidisciplinary approach concentrating on improved diagnostics which can provide important insights into the anatomical and structural changes following TMLV as well as patient education including promotion of lifestyle modifications and emphasis on the importance of timely medical attention. In addition, early recognition of the risk factors and identification of potential biomarkers could further assist in the prognosis of patients with TMVL. Moreover, remote monitoring with the use of telemedicine technologies could be a valuable screening and diagnostic adjunct to the armamentarium of ophthalmologists and neurologists.

CONCLUSIONS

TMVL remains a complex and multifactorial clinical entity that warrants clinical attention while a multidisciplinary approach involving advanced diagnostic techniques, patient-centered care, and education according to the age and overall medical and family past medical history. Improving knowledge of the underlying mechanisms along with advancements in medical and surgical interventions can result in significant outcome and quality of life improvements.

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Declarations

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