Transient Monocular Visual Loss Patterns and Associated Vascular Abnormalities

Askiel Bruno, MD, James J. Corbett, MD, José Biller, MD, Harold P. Adams Jr., MD, and Clifford Qualls, PhD

To determine if certain transient monocular visual loss patterns predict the associated vascular abnormalities, we prospectively evaluated 100 consecutive patients. Each patient had hematologic tests, a carotid artery study (arteriography in 74, duplex ultrasonography in the remaining 26), and an ophthalmologic examination. Patients with altitudinal or lateralized transient monocular visual loss were more likely to have carotid artery stenosis, carotid artery ulceration, cardiac sources of emboli, or visible retinal emboli than patients with other visual loss patterns. Our findings suggest that altitudinal or lateralized transient monocular visual loss is primarily caused by embolism but that other visual loss patterns are usually caused by nonembolic mechanisms. (Stroke 1990;21:34–39)

Transient monocular visual loss (TMVL), often referred to as amaurosis fugax, is usually attributed to ipsilateral carotid artery disease and is regarded as a warning for retinal or cerebral infarction.1–6 Other causes of TMVL include cardiogenic embolism,7–11 hypercoagulable disorders,12–16 vasculitis,17–19 migraine,20–25 ocular disorders,26–28 and a variety of other disorders.29–31 Therefore, in a given patient with TMVL a variety of causes and mechanisms need to be considered. Because certain TMVL patterns might be caused by specific mechanisms, we evaluated 100 consecutive patients to determine if any TMVL patterns predicted the associated vascular abnormalities.

Subjects and Methods

Between August 1986 and June 1988, we studied 100 consecutive patients (71 men and 29 women) aged 21–86 (mean 60) years, referred to the University of Iowa Hospitals and Clinics or the Iowa City Veterans Administration Medical Center for evaluation of TMVL. All patients had lost vision in all or part of the visual field in one eye ≤30 days before evaluation and had regained baseline vision within 24 hours. We excluded patients who were uncertain whether their visual loss was monocular or binocular and those who had only one functioning eye.

Each patient was asked to describe and draw the pattern of visual loss experienced. Each episode was classified as 1) altitudinal or lateralized: the peripheral visual field was involved and was demarcated from intact vision by a horizontal or vertical line; lateral visual defects were classified together with altitudinal defects because they have similar shapes; 2) diffuse: visual loss involved the entire field of vision at all times; 3) constricting: vision constricted concentrically; or 4) miscellaneous: other unusual patterns of visual loss (Figure 1). This classification was chosen because each TMVL pattern is distinct and can be recognized during an interview. TMVL was classified as bilateral if it occurred in each eye separately ≤30 days before evaluation. Patients who had different TMVL patterns on different occasions within 30 days of evaluation were classified as having multiple TMVL patterns.

All patients had complete blood cell count, prothrombin time, partial thromboplastin time, and erythrocyte sedimentation rate determinations. Each patient had an ophthalmologic examination and a carotid artery study (arteriography in 74 and duplex ultrasonography in the other 26). Extracranial carotid artery stenosis was classified as mild (16–49%), moderate (50–79%), severe (80–99%), or as occlusion depending on the reduction of luminal diameter. Extracranial carotid artery ulceration was diagnosed...
when a well-defined concavity on the surface of an atherosclerotic plaque, or at the base of an occluded internal carotid artery (a stump), at least 2 mm in depth was clearly visible on an arteriogram. Carotid duplex results were reported by the vascular laboratory without knowledge of the patient's TMVL pattern. Arteriograms were reviewed by two unblinded investigators. Fifty-two patients had two-dimensional echocardiography.

In patients with bilateral TMVL, carotid artery disease was considered present only when both carotid arteries were diseased. Stenosis in patients with bilateral TMVL and bilateral carotid artery disease was classified according to the more stenotic artery.

Duration of TMVL was classified as short (<5 minutes), moderate (5–60 minutes), or long (>60 minutes). In patients with bilateral TMVL, the duration of visual symptoms in the eye ipsilateral to the more stenotic carotid artery was used. When carotid artery disease was similar on both sides, the average duration between the two eyes was used.

Fisher's two-sided exact test for 2x2 or 2x3 contingency tables and a two-sided test were used to determine the significance of differences between patients with altitudinal or lateralized and diffuse TMVL patterns. Data from patients with constricting, miscellaneous, and multiple TMVL patterns were not subjected to statistical analysis because of low frequencies.

### Results

Clinical characteristics of the 100 patients with TMVL are shown in Table 1. No patient reported halos around lights or had previously undiagnosed glaucoma. In all five patients who were being treated for glaucoma, TMVL was painless. On examination, no patient had elevated intraocular pressure, iritis, optic disk edema, optic disk drusen, or anomalous retinal arterial branching. Findings in the 100 patients with TMVL are summarized in Table 2.

Forty-two patients had an altitudinal (38) or a lateralized (four) TMVL pattern. In 26 patients partial vision remained throughout the episode, and in the remaining 16 the visual defect progressed to complete monocular blindness. Thirty-four patients had unilateral and the other eight had bilateral episodes. Among these 42 patients, carotid artery stenosis was mild in 12, moderate in four, and severe in eight; a carotid artery occlusion was found in eight patients. A potential cardiac source of emboli in 11 of these patients included valvular heart disease in nine (prosthetic heart valve in six, combined mitral stenosis and regurgitation in one, severe aortic stenosis in one, and aortic plus mitral regurgitation in one), atrial fibrillation in one, and atrial fibrillation associated with a prosthetic heart valve in the other.

Among the seven patients with a history of migraine, five had carotid artery stenosis (mild in four, occlusion in the other), one had a prosthetic heart valve, and one had no other abnormalities. Only two patients with altitudinal or lateralized TMVL (5%) had no abnormalities found to account for their visual loss. Among the 38 patients with a diffuse TMVL pattern, 35 had unilateral and the other three had bilateral episodes. Carotid artery stenosis was mild in three, moderate in five, and severe in four patients; a carotid artery occlusion was found in three. One patient had a potential cardiac source of emboli (atrial fibrillation). Among the eight patients with a history of migraine, three had carotid artery stenosis (mild in two, occlusion in one) and five had no other abnormalities. In five of the 38 patients, unusual

### Table 1. Clinical Characteristics of 100 Patients With Transient Monocular Visual Loss by Pattern

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Altitudinal/lateralized (n=42)</th>
<th>Diffuse (n=38)</th>
<th>Constricting (n=9)</th>
<th>Miscellaneous (n=4)</th>
<th>Multiple patterns (n=7)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>31/25</td>
<td>8/1</td>
<td>1/6</td>
<td>0.47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>11/13</td>
<td>1/3</td>
<td>1/1</td>
<td>—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (mean)</td>
<td>62/62</td>
<td>48/46</td>
<td>63/1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>17/40</td>
<td>5/1</td>
<td>5/1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>23/55</td>
<td>7/0</td>
<td>5/1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>3/7</td>
<td>1/0</td>
<td>1/1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glaucoma history</td>
<td>2/5</td>
<td>0/0</td>
<td>0/0.66</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Migraine history</td>
<td>7/17</td>
<td>0/1</td>
<td>1/0.78</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brightness in or around area of visual loss</td>
<td>4/10</td>
<td>0/1</td>
<td>1/1.00</td>
<td></td>
<td></td>
<td>0.14</td>
</tr>
<tr>
<td>Duration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Short</td>
<td>27/64</td>
<td>5/1</td>
<td>6/1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>11/26</td>
<td>2/3</td>
<td>0/0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long</td>
<td>4/10</td>
<td>2/0</td>
<td>1/1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fisher's two-sided exact test to compare altitudinal/lateralized and diffuse, except two-sided t test to compare ages.
nonembolic causes for TMVL were the only abnormalities found (Table 2). TMVL with hyphema occurred in one patient 6 months after cataract extraction. That patient was examined in the ophthalmology clinic during one of the episodes; visual acuity was 20/25 in the affected eye, and erythrocytes were present in the anterior chamber. One hour later, after the hemorrhage had cleared, visual acuity returned to 20/20. One patient with a coiled extracranial internal carotid artery had two episodes of diffuse TMVL associated with head turning. One episode occurred during extreme head turning while driving a car and the other occurred during bilateral shoulder abduction and head turning while putting on an overcoat. A quadriparetic patient experienced diffuse TMVL each time he sat up from a supine position. Two weeks earlier he had undergone repair of a spontaneous retinal detachment in the same eye. Mean arterial blood pressure decreased by as much as 36% and heart rate increased by 25% when this patient sat up from a supine position. TMVL was his only symptom. One week after treatment with fludrocortisone acetate was begun, the visual spells resolved although his orthostasis persisted. One patient had biopsy-documented giant cell arteritis and another had essential thrombocythemia with a platelet count of 990,000/mm³. Twelve patients with diffuse TMVL (32%) had no abnormalities found to account for their visual loss.

Among the nine patients with a constricting TMVL pattern, the visual loss became complete in seven and central vision remained spared throughout the episode in the other two. Eight patients had unilateral and one had bilateral episodes. Carotid artery stenosis was moderate in one patient and severe in another; an occlusion was found in one. Six of these nine patients (67%) had no abnormalities found to account for their visual loss.

Among the four patients with miscellaneous TMVL patterns, two had diffuse visual loss with small circular areas of intact vision, one described multiple circular dark spots, and one noted a rectangular defect around the vertical meridian in the superior visual field (Figure 1). All episodes were unilateral. The patient who noted multiple circular dark spots had mild carotid artery stenosis. The patient who

<table>
<thead>
<tr>
<th>Abnormalities</th>
<th>Altitudinal/ lateralized* (n=42)</th>
<th>Pattern Diffuse† (n=38)</th>
<th>Constricting (n=9)</th>
<th>Miscellaneous (n=4)</th>
<th>Multiple patterns (n=7)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid artery stenosis</td>
<td>32 76</td>
<td>15 39</td>
<td>3</td>
<td>1</td>
<td>5</td>
<td>0.001</td>
</tr>
<tr>
<td>Carotid ulcerations/ arteriograms</td>
<td>13/29 45</td>
<td>5/28 18</td>
<td>0/7</td>
<td>0/3</td>
<td>1/7</td>
<td>0.05</td>
</tr>
<tr>
<td>Cardiac source of emboli</td>
<td>11 26</td>
<td>1 3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.004</td>
</tr>
<tr>
<td>Visible retinal emboli</td>
<td>3 7</td>
<td>0 0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0.24</td>
</tr>
<tr>
<td>Miscellaneous‡</td>
<td>0 0</td>
<td>5 13</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Fisher's two-sided exact test to compare altitudinal/lateralized and diffuse.
*Four patients had concomitant carotid artery stenosis and potential cardiac source of emboli.
†One patient had concomitant carotid artery stenosis and potential cardiac source of emboli.
‡Temporal arteritis, essential thrombocythemia, hyphema, coiled extracranial internal carotid artery, and orthostatic hypotension (none had carotid artery stenosis or cardiac source of emboli).

Figure 1. Classification of transient monocular visual loss patterns.
noted a rectangular defect had a history of migraine. In the two patients who noted diffuse visual loss with small circular areas of intact vision, no abnormalities were found to account for their visual loss.

Among the seven patients with multiple TMVL patterns, six had unilateral and one had bilateral episodes. All seven patients experienced an altitudinal or lateralized pattern in addition to another pattern (four had diffuse, two had constricting, and one had diffuse visual loss with small circular areas of intact vision). An abnormality that could account for TMVL was found in all seven patients. Carotid artery stenosis was severe in three patients, and an occlusion was found in two. Two patients had visible retinal emboli, but the source of the emboli was not found (both had carotid arteriography and echocardiography). One patient with carotid artery occlusion also had a history of migraine.

Discussion

Our study shows that patients with TMVL can be classified according to the perceived pattern of visual loss and that different patterns of visual loss are associated with different vascular findings. Patients with an altitudinal or lateralized TMVL pattern are more likely to have either a potential carotid or cardiac source of retinal emboli or visible retinal emboli than patients with other TMVL patterns (Table 2).

Patients with altitudinal (referred to by some as “classic” amaurosis fugax) or lateralized TMVL are described in many reports.1-2,7-9,15,16,27,28,32-38 In most of these patients TMVL is attributed to retinal embolism, but in some patients the visual loss is attributed to other mechanisms.16,17,27,28,35,36 The significance of altitudinal or lateralized visual loss as compared to other patterns of TMVL has not been systematically studied before. This may be because most studies of TMVL are retrospective and information regarding patterns of visual loss was not available for all patients. In one partially prospective study, Goodwin et al39 noted that altitudinal TMVL was associated with carotid ulceration or visible retinal emboli, but the frequencies were not stated.

The best explanation for altitudinal or lateralized TMVL is regional retinal or optic nerve ischemia. Visible retinal emboli corresponding to altitudinal or lateralized TMVL in this and other studies2,32-34,40,41 is compelling evidence that embolism is an important mechanism responsible for this pattern of TMVL. Absence of visible retinal emboli in most of our 42 patients with altitudinal or lateralized TMVL is explained either by rapid lysis of the embolus or by involvement of arteries not visible on ophthalmoscopy, such as the posterior ciliary vessels.

Anterior ischemic optic neuropathy (AION) usually produces an altitudinal visual field defect, and some patients experience altitudinal TMVL prior to optic nerve infarction.42 The mechanism of nonarteritic AION probably involves a small scleral canal29 and hypertensive small-vessel disease. However, the incidence of embolism may be underestimated because emboli in the posterior ciliary arteries cannot usually be seen during life. Embolic occlusion of the posterior ciliary arteries has been demonstrated histologically,44,45 and suspected on ophthalmoscopy,46,47 albeit rarely.

Our findings in patients with altitudinal or lateralized TMVL suggest that embolism is its principal mechanism. Findings in patients with multiple TMVL patterns also support this opinion. All patients with multiple patterns experienced an altitudinal or lateralized pattern and all had potential sources of retinal emboli or visible retinal emboli.

Patients with diffuse TMVL are less likely to have a potential source of retinal emboli than patients with altitudinal or lateralized TMVL (Table 2). None of our 38 patients with diffuse TMVL had visible retinal emboli, and five had various nonembolic disorders. Patients with complete TMVL are described in many reports, but whether an altitudinal or lateralized visual defect was present during the onset or resolution of TMVL is usually not specified.

The best explanation for diffuse TMVL is diffuse retinal or optic nerve ischemia. Ophthalmic or central retinal artery obstruction or hypoperfusion are likely causes. Our findings in patients with diffuse TMVL suggest that a nonembolic process(es) is its principal mechanism.

Our nine patients with constricting TMVL had no cardiac sources of emboli, no carotid artery ulcerations, and no visible retinal emboli (Table 2). Among three previously reported patients with constricting TMVL, two were thought to have ocular migraine24,25 and the third had pseudotumor cerebri and gaze-evoked amaurosis.30 The best explanation for constricting TMVL may be choroidal ischemia.25 Our findings and the additional reports of patients with constricting TMVL suggest that a nonembolic process is its principal mechanism, but more patients need to be studied.

Similarly, our four patients with miscellaneous TMVL patterns had no cardiac sources of emboli, no carotid artery ulcerations, and no visible retinal emboli (Table 2). In one report, TMVL consisting of multiple circular dark spots is attributed to retinal embolism.48 More patients with miscellaneous TMVL patterns need to be studied to determine the mechanism(s) responsible.

Explanations for TMVL in patients without identifiable abnormalities include inconspicuous cardiac or arterial embolicogenic lesions, small-vessel disease involving the ophthalmic, central retinal, or posterior ciliary arteries, and possibly vasoconstriction. Failure to identify a source of embolus with arteriography and echocardiography in two of our patients with visible retinal emboli emphasizes the difficulty in identifying inconspicuous embolicogenic lesions. Reversible vasoconstriction of retinal vessels during TMVL has been observed in patients with migraine,24 and it is possi-
ble that this reversible vasoconstriction also occurs as a late-life migraine accompaniment, without headache, analogous to that proposed for cerebral vessels. The role that each of these disorders plays in producing the various TMVL patterns should be established in future studies.

TMVL is a symptom caused by many disorders and by different mechanisms. Our study demonstrates that different TMVL patterns are associated with different vascular abnormalities and that patients with atlantid or lateralized TMVL are more likely to have a potential source of retinal emboli than patients with other TMVL patterns. This information should be useful in the evaluation and management of patients with TMVL.

Acknowledgments
We thank Drs. Patricia Johnston, Daniel Jacobson, Asad Shamma, Stanley Thompson, William Talman, Brad Hyman, Betsy Love, Lynn Struck, and Steven Konieck for referring patients into this study.

References


KEY WORDS • amaurosis fugax • carotid artery diseases • embolism
Transient monocular visual loss patterns and associated vascular abnormalities.  
A Bruno, J J Corbett, J Biller, H P Adams, Jr and C Qualls

Stroke. 1990;21:34-39  
doi: 10.1161/01.STR.21.1.34

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231  
Copyright © 1990 American Heart Association, Inc. All rights reserved.  
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the  
World Wide Web at:  
http://stroke.ahajournals.org/content/21/1/34

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:  
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:  
http://stroke.ahajournals.org/subscriptions/