Amaurosis Fugax: The Results of Arteriography in 59 Patients

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SUMMARY Fifty-nine patients had arteriography because of episodes of amaurosis fugax. Only one third of them had atherosclerotic lesions potentially treatable by carotid endarterectomy. Another third had normal arteriograms, some of these had migraine, heart disease or platelet disorders presumed to be the cause of their symptoms. Patients with amaurosis fugax are a heterogenous group, and their visual symptoms should not be considered a specific indicator of stenosis of the internal carotid artery.

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AMAUREOSIS FUGAX is a well-recognized warning symptom of impending cerebral or retinal infarction. In one series of 80 patients with amaurosis fugax, nine subsequently suffered permanent visual loss and five developed hemiplegia.1 Amaurosis fugax is considered to be a reliable sign of carotid artery disease and in particular of partial occlusion of the carotid artery.2 Surgically correctable lesions of the internal carotid artery are reported to be more common in patients with isolated episodes of amaurosis fugax than in persons with transient cerebral symptoms (TIA) alone.3,4 Several authors have reported abnormal arteriograms in more than 90% of their patients with a history of amaurosis fugax (table 1). However, we found an unexpectedly high incidence of normal arteriograms among patients with transient monocular blindness. We review our experience and try to determine whether any factors help to predict the results of cerebral arteriography in patients with amaurosis fugax.

Methods

Fifty-nine patients with transient monocular blindness, who underwent cerebral arteriography, were seen by the Neurology and Neuro-ophthalmology Services at the University of Iowa between September 1, 1975 and October 1, 1982. These patients represent approximately 86% of all persons with amaurosis fugax seen by these services. We excluded patients who did not agree to arteriography, a patient who developed transient monocular blindness within days after carotid endarterectomy, patients with central retinal artery occlusion without previous amaurosis fugax, patients with well-established migraine and those with a cause of the visual symptoms established before scheduled arteriography. We included three patients who had branch retinal arterial occlusion as well as amaurosis fugax. All 59 patients had transient monocular symptoms such as a sector visual loss, a fog, descending curtain or complete monocular blindness lasting seconds to several minutes.

Results

There were 34 men and 25 women, who ranged in age from 24–80; most were more than 55 years old and only one patient was younger than 39 (table 2). The right eye was symptomatic in 38 patients, the left eye in 20 and one patient had amaurosis fugax in both eyes. The number of attacks ranged from one to more than 100. All patients had recent symptoms before evaluation, although some had been having attacks for more than one year. Eleven patients also had episodes considered to be hemispheric TIA, while eight patients had symptoms construed to be vertebrobasilar TIA. Five patients suffered previous strokes. Residual neurological abnormalities were found in two of the five patients with previous stroke; the other 57 patients had normal neurological examinations. Bruits over the carotid artery were heard in 15 patients and were bilateral in seven. Three patients with retinal infarction had visual field defects. Cholesterol emboli were seen in six patients and a fibrin-platelet embolus in one. Supraorbital Doppler flow examinations were abnormal in nine of 40 patients and oculoplethysmography (OPG) was abnormal in four of 25 tested patients.

Selective carotid arteriography and/or arch arteriography was performed in all patients. Stenosis of the origin of the internal carotid artery was found in 15 patients, while prominent plaques without significant stenosis or ulcerations were seen in eight. Two patients had stenosis of the external carotid artery without changes in the internal carotid artery and another had isolated stenosis of the ophthalmic artery. Complete occlusion of the internal or common carotid artery was seen in ten patients. One patient had kinking of the internal carotid artery and two had fibromuscular dysplasia.

In 19 patients, the arteriograms were normal. Identifiable causes of visual symptoms in these patients included abnormal platelet function in one, retinal migraine in two and mitral valve prolapse in two. In the remainder the cause of the transient visual symptoms was not determined. Seven of these 19 patients, however, had atherosclerotic lesions in the contralateral carotid artery, innominate artery or in a vertebral artery. The patient with bilateral non-simultaneous transient monocular blindness had one normal carotid artery and the other had atherosclerotic plaques.

We examined the histories, results of physical examination and the results of noninvasive diagnostic procedures in an attempt to predict the arteriographic findings. Neither age, side of symptoms, number of attacks, the presence or absence of a bruit over the carotid artery, coincident hypertension or heart dis-
Diagnosis of Amaurosis Fugax

TABLE 2
Results of Arteriography Grouped According to Age of

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Internal carotid artery stenosis</th>
<th>Internal carotid artery occlusion</th>
<th>Internal carotid artery plaque or ulceration</th>
<th>Normal</th>
<th>Other*</th>
</tr>
</thead>
<tbody>
<tr>
<td>under 45</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>45-54</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>55-64</td>
<td>5</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>65-74</td>
<td>7</td>
<td>4</td>
<td>3</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>older than 74</td>
<td>1</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>


Table 1: Summary of Reported Results of Arteriography in Patients with Amaurosis Fugax

<table>
<thead>
<tr>
<th>Total number of patients</th>
<th>Number of patients with Carotid artery stenosis</th>
<th>Carotid artery ulcer</th>
<th>Carotid artery occlusion</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marshall and Meadows1</td>
<td>27</td>
<td>11 (41%)</td>
<td>5 (18%)</td>
<td>11 (41%)</td>
</tr>
<tr>
<td>Kollartis et al.6 (81 vessels)</td>
<td>45</td>
<td>38 (47%)</td>
<td>12 (15%)</td>
<td>13 (16%)</td>
</tr>
<tr>
<td>Ramirez-Lassepas et al.3</td>
<td>27</td>
<td>20 (74%)</td>
<td>6 (22%)</td>
<td>1 (4%)</td>
</tr>
<tr>
<td>Hooshmand et al.6</td>
<td>34</td>
<td>12 (35%)</td>
<td>2 (6%)</td>
<td>2 (6%)</td>
</tr>
<tr>
<td>Lemak and Fields4</td>
<td>234</td>
<td>151 (65%)</td>
<td>32 (13%)</td>
<td>51 (22%)</td>
</tr>
<tr>
<td>Mungas and Baker7</td>
<td>107</td>
<td>80 (75%)</td>
<td>—</td>
<td>12 (11%)</td>
</tr>
<tr>
<td>Pessin et al.9</td>
<td>43</td>
<td>20 (47%)</td>
<td>4 (9%)</td>
<td>7 (16%)</td>
</tr>
<tr>
<td>Wilson et al.10</td>
<td>43</td>
<td>16 (37%)</td>
<td>8 (19%)</td>
<td>3 (7%)</td>
</tr>
<tr>
<td>Thiele et al.8</td>
<td>94</td>
<td>49* (52%)</td>
<td>37 (39%)</td>
<td>8 (9%)</td>
</tr>
<tr>
<td>Parkin et al.11</td>
<td>38</td>
<td>21 (55%)</td>
<td>12 (31%)</td>
<td>4 (11%)</td>
</tr>
<tr>
<td>Present report (60 vessels)</td>
<td>59</td>
<td>15 (25%)</td>
<td>8 (14%)</td>
<td>10 (17%)</td>
</tr>
</tbody>
</table>

*10 patients with only stenosis, 39 with stenotic and ulcerated lesions.

Discussion
In our review of 59 patients with 60 symptomatic eyes, 20 had no abnormalities of the carotid artery visualized by arteriography and only 15 had carotid artery stenosis. A natural conclusion might be that the quality of our arteriograms might be responsible for the high incidence of negative results. With this in mind, we carefully reviewed the arteriograms of our patients and are satisfied of their good quality. The frequency of normal arteriograms in many other reports of patients with embolic retinal ischemic disease is higher than generally assumed; more than 10 years ago, Marshall and Meadows reported normal arteriograms in 41% of patients who had amaurosis fugax and no other symptoms.1 Pessin et al9 found normal arteriograms in 21% of their patients and Wilson et al10 found normal arteriograms in 37% of their patients with branch retinal arterial occlusions. In a recent report by Parkin et al,11 13 of 38 patients undergoing arteriography for amaurosis fugax had no abnormality or minimal to mild irregularity of the carotid artery.

We cannot predict the results of arteriography by the number of attacks of transient monocular blindness, presence of carotid bruit, noninvasive studies or even the presence of retinal emboli. The failure of a carotid bruit to predict the results of arteriography in our series is similar to the experiences of Ramirez-Lassepas et al3 and Wilson et al10. Six of our patients had retinal cholesterol emboli, but two of these patients had normal carotid arteriograms, an experience that differs from Ramirez-Lassepas et al3 and Hooshmand et al.6 The presence of retinal emboli is not confined to patients with symptoms due to internal carotid artery disease and may represent atherosclerotic lesions in other arteries.12 Mungas and Baker7 found noninvasive studies unreliable in predicting results of arteriography in patients with amaurosis fugax. Our experience is in agreement.

Arteriography remains the single most reliable diagnostic procedure. While it is not without complications13 and may fail to visualize small lesions or shallow ulcers, it is accurate in differentiating normal from stenotic carotid arteries.14 Digital intravenous subtraction arteriography (DISA) is becoming a useful study in evaluation of the carotid bifurcation in patients with
TIA. However this study (DISA) may miss the nonstetotic or shallow ulcerative plaque which produces symptoms, such as found in eight of our patients. We concur with Lemak and Fields4 that any patient with transient monocular blindness should be assumed to have carotid artery disease. However all patients with amaurosis fugax should be evaluated by history and examination for other causes of symptoms or sources of emboli. Migraine, cranial arteritis, myopia, optic disc drusen or papilledema can cause transient monocular visual loss. Ophthalmic artery disease can cause amaurosis fugax,15 and rarely it may be the site of platelet adhesion and aggregation with distal embolization.12 Emboli from thoracic arteries,16 external carotid17 or heart18 produce amaurosis fugax. Mitral valve prolapse19,21 is a probable source of embolic and transient monocular blindness in young persons. We concur with DiBono and Warlow18 that patients who have amaurosis fugax are a heterogenous group and that emboli can arise from several sites. Carotid artery stenosis or ulceration are common but clearly not the only sources of transient monocular symptoms. All patients with amaurosis fugax, particularly those with normal arteriograms, should have a complete evaluation for cardiac, hematologic or other vascular diseases as the cause of their symptoms. Just as in patients with solely cerebral TIA, evaluation aimed at only the carotid artery bifurcation is incomplete. Amaurosis fugax should not be considered a specific indicator of carotid artery stenosis.

References
Amaurosis fugax: the results of arteriography in 59 patients.
H P Adams, Jr, S F Putman, J J Corbett, B P Sires and H S Thompson

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