Patients with transient global amnesia are often categorized as having cerebrovascular disease. Noninvasive carotid artery testing was performed in 56 patients with transient global amnesia to determine if they had the same incidence of extracranial atherosclerotic vascular disease as patients with focal cerebral transient ischemic attacks. Only 3 of 56 patients had hemodynamic obstruction of flow at the carotid artery bifurcation, and 41 of 56 had no evidence of any atherosclerotic disease. Other risk factors for cerebrovascular disease were present in 24 of 56 patients, but only 1 had a prior cerebrovascular event. The pathophysiology of transient global amnesia appears to differ from the pathophysiology of classical transient ischemic attacks in that there is no clear relation between transient global amnesia and the presence of extracranial atherosclerotic cerebrovascular disease. (Stroke 1987;18:951-953)

The syndrome of transient global amnesia (TGA) as first described by Bender 1 is characterized by a sudden onset of the inability to acquire new information and the loss of memory for recent events while immediate recall is preserved. 1-3 The patient remains alert, maintains self identity, and usually demonstrates concern by asking the same questions repeatedly. There are usually no concomitant neurologic deficits, and the disorder resolves within 24 hours, with residual amnesia for the event. 1-3 Many etiologies have been proposed for TGA, including cerebrovascular disease, 4-5 seizure disorder, 6-7 stress, 8 focal cerebral mass lesions, 9 cardiac disorders, 10 and migraine. 11,12 However, no definite association between cerebral ischemia 11,14 or ictal events 6-7 has been documented. Since many of these patients are considered to have cerebrovascular disease equivalent to patients with focal cerebral transient ischemic attacks (TIAs), 56 patients with symptoms of TGA were evaluated with noninvasive carotid artery testing to determine if there was a relation between TGA and the presence of extracranial atherosclerotic cerebrovascular disease.

Subjects and Methods

Fifty-six consecutive patients with symptoms fulfilling the criteria for a diagnosis of TGA 1-3 were evaluated by a neurologist soon after the episode, usually within 1 day and always within 1 week. Most of the patients had been referred with the diagnosis of TGA by a neurologist. Their mean age was 67 years, with a range of 47-82 years; there were 26 women and 30 men.

A noninvasive carotid artery testing battery consisting of pneumooculoplethysmography, 13 supraorbital directional Doppler, 14 carotid artery Doppler flow study by velocity wave form analysis, 17 and real-time B-mode ultrasonography 18 was performed on all 56 patients. This battery of tests has been shown to identify nearly all carotid lesions with a stenosis of >50% on angiography 19 and is also capable of identifying small atheromas in the carotid sinus not readily visualized on angiography. 18 A hemodynamically obstructive lesion at the carotid artery bifurcation was identified when an atherosclerotic plaque was visualized at the carotid bifurcation associated with high-frequency turbulence on Doppler flow 19 or reduction in the ophthalmic artery pressure to 10% below the contralateral side or to <65% of the brachial artery pressure. 15,20 Pneumooculoplethysmography was performed with a Life Sciences PVR (Boston, Mass.). Supraorbital direction Doppler and Doppler velocity wave form analysis of carotid flow was performed with a Parks 908 continuous-wave directional Doppler (Beaverton, Ore.) at 9.5 mHz. Real-time B-mode ultrasonography was performed with a High Stoy SP100B and duplex scanning with 4- and 8-mHz Sonomed continuous-wave directional Dopplers (Lake Success, N.Y.). Statistical analysis was performed by Fisher's exact test with the Epistat program (Tracy Gustafson, Round Rock, Tex.) on an IBM PC XT computer.

Results

Only 1 of the 56 patients with TGA in this series had a history of neurologic disturbance, an episode of cerebellar ataxia, which probably represented a TIA in the vertebrobasilar distribution. The remaining 55 patients had no other history suggestive of cerebrovascular dis-
ease. Real-time B-mode ultrasonography identified atherosclerotic plaques at the carotid artery bifurcation in 15 patients, but these lesions were hemodynamically obstructive in only 3. Ophthalmic artery pressure was normal in all but 1 of the 56 patients. The incidence of hemodynamically obstructive lesions at the carotid artery bifurcation in these patients with TGA was significantly lower than in a group of patients with cerebral TIA (27 of 95, \( p = 0.003 \)) and stroke (27 of 79, \( p = 0.00003 \)) tested with the same noninvasive battery.14 The incidence of no atherosclerotic lesion at all in patients with TGA (41 of 56) was even more prominent compared with patients with TIA (29 of 95, \( p = 0.000003 \)) and stroke (27 of 79, \( p = 0.000003 \)) (Table 1).

A potential cardiac source for cerebrovascular disease was identified in 18 of the 56 patients including 3 with cardiac arrhythmia, 5 with coronary artery disease, 2 with aortic valve disease or replacement, and 9 with the carotid Doppler finding of early systolic flutter turbulence, which has been significantly associated with mitral valve dysfunction.21 (One patient with early systolic flutter turbulence also had coronary artery disease.) The other major risk factors for cerebrovascular disease, hypertension and diabetes, were present in an additional 9 patients.

Discussion

Clinical and experimental studies have identified the hippocampus, hippocampal gyrus, and surrounding structures as being involved in processing recent memory.22 The memory deficits encountered in TGA are generally attributed to temporary dysfunction of these structures.4,5,7 Whether cerebrovascular disease plays a role in the pathogenesis of TGA and whether TGA has the same prognostic implications as TIA have not been established.

Mathew and Meyer1 demonstrated a high correlation of cerebrovascular disease in 11 of 12 patients with TGA who underwent angiography. However, 78% of these patients had other symptoms of cerebrovascular disease besides memory loss, and these patients are not representative of those with TGA alone. Kushner and Hauser4 noted a high incidence of risk factors for cerebrovascular disease in patients with TGA but did not explore the extent of extracranial vascular disease in these patients. In prospective studies, Nausieda and Sherman13 found a much lower incidence of subsequent episodes of TIA and stroke in patients with TGA than in those with TIA alone. Bender,1 Hinge and Jensen,14 and Fisher6 also noted a benign prognosis in patients with TGA.

TIAs are often associated with atherosclerotic disease of the extracranial carotid artery.23 While TGA is generally attributed to dysfunction of structures supplied by the posterior cerebral artery, circulation in this distribution can be influenced by flow in both the anterior and posterior circulation. Fisher et al14,25 and Schwartz and Mitchell26 have noted a good correlation between atherosclerotic disease at the carotid artery bifurcation and atherosclerotic disease in the posterior and intracranial vasculature. Olsson et al27 also found that the majority of patients with TIAs, whether of the anterior or posterior circulation, had abnormalities of the extracranial carotid arteries on angiography. The significantly lower incidence of atherosclerosis in the extracranial carotid arteries in these 56 unselected patients with TGA suggests that, even though TGA may be caused by cerebral ischemia, these patients do not have the propensity for generalized atherosclerotic vascular disease seen in patients with cerebral or verteobasilar TIAs.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>n</th>
<th>All plaque</th>
<th>Hemodynamic obstructive plaque</th>
</tr>
</thead>
<tbody>
<tr>
<td>TGA</td>
<td>56</td>
<td>15 (26.8%)</td>
<td>3 (5.4%)*</td>
</tr>
<tr>
<td>TIA</td>
<td>95</td>
<td>66 (69.5%)</td>
<td>27 (28.4%)*</td>
</tr>
<tr>
<td>CVA</td>
<td>79</td>
<td>52 (65.8%)</td>
<td>27 (34.2%)†</td>
</tr>
</tbody>
</table>

*Significantly different from patients with TGA at \( p < 0.003 \), \( p < 0.0003 \), and \( p < 0.000003 \), respectively.

**Significantly different from patients with TGA at \( p < 0.003 \), \( p < 0.0003 \), \( p < 0.000003 \), and \( p < 0.0000003 \), respectively.

Table 1. Frequency of Carotid Plaque in Patients With TGA

References


**KEY WORDS** • transient global amnesia • carotid artery atherosclerosis • cerebrovascular disease • transient cerebral ischemia
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D Feuer and J Weinberger

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