Arteriographic Comparison of Amaurosis Fugax and Hemispheric Transient Ischaemic Attacks

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SUMMARY The results of carotid angiography were compared in 53 patients with amaurosis fugax and 92 with hemispheric transient ischaemic attacks (TIAs). Evidence of extracranial disease was found in 71% of those with retinal ischaemia and these patients rarely had evidence of intracranial disease (11%) or normal angiograms (15%). By contrast patients with hemispheric symptoms less frequently showed disease of the cervical carotid artery (36%) and more often had intracranial disease (27%) or normal angiograms (35%). It is suggested that amaurosis fugax is usually due to emboli originating in the internal carotid artery and that the pathogenesis of cerebral TIAs in the carotid territory is more heterogeneous. These conclusions have implications for the design and interpretation of both medical and surgical trials.

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TABLE 1 Details of Patients

<table>
<thead>
<tr>
<th>1. Amaurosis fugax</th>
<th>n</th>
<th>M/F</th>
<th>Mean age (range) years</th>
<th>BP &gt; 150/90 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amaurosis fugax</td>
<td>37</td>
<td>2.7</td>
<td>58.3 (42-71)</td>
<td>46</td>
</tr>
<tr>
<td>TIA (hemiphenomena)</td>
<td>73</td>
<td>3.6</td>
<td>56.3 (41-73)</td>
<td>55</td>
</tr>
<tr>
<td>TIA (dysphasia alone)</td>
<td>19</td>
<td>3.7</td>
<td>57.0 (45-64)</td>
<td>37</td>
</tr>
</tbody>
</table>

Thirty-seven patients had one or more attacks of amaurosis fugax but denied any symptoms suggesting cerebral involvement. Eighteen had both amaurosis fugax and cerebral TIAs on the same side. None had simultaneous symptoms of retinal and hemispheric ischemia.

TIA patients less frequently had cervical bruits (8/92 cf 27/55 Chi square 30.6, p < .001) but no other striking differences emerged (table 1).

The relative extent of extracranial and intracranial abnormality in the different patient groups is shown in table 2. As there was no difference between the group with amaurosis fugax and with amaurosis fugax plus TIAs these were grouped together as indicative of the findings associated with attacks of retinal ischemia.

It is clear that amaurosis fugax is more commonly associated with extracranial disease and less commonly with intracranial disease or a normal angiographic appearance. The possibility that TIAs characterised by hemiphenomena might be related to vertebo-basilar disease, invalidating the comparison with amaurosis fugax, was considered by looking at the albeit small (n = 19) subgroup with dysphasia. There were no differences in the angiographic findings between the TIA patients whose attacks included dysphasia, and those that did not. The finding of extracranial disease was more frequent in amaurosis fugax than in dysphasic TIAs (71% cf 37%, p < .05) and a normal angiogram was rarer (15% cf 53%, p < .01), as found when the comparison included all TIA patients.

Discussion

Cervical bruits and angiographic evidence of major atheromatous changes at the carotid bifurcation were found far more frequently in patients presenting with amaurosis fugax than in those with hemispheric TIAs. Over two-thirds of patients with amaurosis fugax were found to have extracranial atheromatous disease of the internal carotid artery.1, 4 Patients with hemispheric TIAs often had extracranial disease.5

These findings are in keeping with earlier suggestions that there are differences in the two groups of cases, though both have atheromatous disease of the carotid tree. Ramirez-Lassepas et al6 noted that angiograms were normal in only 4% of their cases of amaurosis fugax but in 27% of those with hemispheric TIAs. Slepyan et al similarly found carotid disease more common, and cardiac disease rare in cases of amaurosis fugax when compared with TIA patients but their numbers were small. By contrast, Eisenberg and Mani7 found comparable angiographic abnormalities in 40 patients with amaurosis fugax and 47 with TIAs. Their cases of amaurosis fugax had a high incidence of congestive cardiac failure and myocardial infarction (60%), and so may not be representative.

Two other studies that have reported no difference between the angiographic appearances of amaurosis fugax and TIAs7, 8 have grouped patients with both retinal and cerebral symptoms with those with cerebral symptoms alone. Our data suggests that these cases are better considered with the amaurosis fugax patients. It would be of interest to rework the data of Lemak and Fields,7 and of Ross and Morrow4 in this way.

The present findings suggest that attacks of transient retinal ischemia are likely to be due to embolism from small size from an origin in the carotid artery, whilst TIAs, often longer in duration9 may arise more often from emboli in the aorta or heart, which are of larger size, and traverse an open carotid10 to cause temporary intracranial branch occlusions. Intracranial disease is also a more frequent finding in cases of hemispheric TIAs, suggesting intracranial embolic or flow phenomena account for some such episodes.

These considerations further stress the heterogeneity of TIAs, and the need to consider subgroups when assessing the results of interventional trials whether surgical or medical.

Acknowledgment

We are grateful to Ms. B. Laatz for assistance.

References

Risk of Stroke During Coronary Artery Bypass Graft Surgery in Patients With Internal Carotid Artery Disease Documented by Angiography

ANTHONY J. FURLAN, M.D., AND A. ROMEU CRACIUN, M.D.

SUMMARY We retrospectively identified 144 patients who underwent coronary artery bypass graft (CABG) surgery in the presence of angiographically documented ≥ 50% internal carotid stenosis or occlusion. Of these, 115 patients had bilateral carotid lesions and received combined operations involving carotid endarterectomy on only one side. The remaining 29 patients, including 11 with bilateral carotid lesions, underwent coronary bypass alone. Nine cerebral infarcts occurred (6%), but only three strokes (2%) were appropriate to the cerebral hemisphere ipsilateral to unoperated carotid stenosis. There was one stroke (3%) among the 29 patients who did undergo combined procedures. In the group of 115 patients with bilateral carotid disease who received unilateral combined carotid endarterectomy there were 8 perioperative strokes (7%), of which 6 were ipsilateral to the endarterectomy. Asymptomatic unilateral < 90% ICA stenosis or ICA occlusion does not increase stroke risk during CABG surgery.

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THE RELATIONSHIP between cerebrovascular disease and stroke complicating coronary artery bypass graft (CABG) surgery is controversial. Neurologists are often consulted regarding the risk of stroke in patients requiring elective myocardial revascularization in the presence of internal carotid artery (ICA) stenosis or occlusion. In an attempt to reduce the risk of perioperative stroke, some centers screen neurologically asymptomatic patients with carotid non-invasive tests to identify candidates for angiography and staged or combined carotid and coronary operations. Presently there is insufficient data to determine the stroke risk posed by ICA stenosis or occlusion in patients undergoing cardiac or other surgical procedures because no study employing cerebral angiography has addressed this issue. In this study, we retrospectively examined the perioperative stroke rate in a group of patients with angiographically proven ICA stenosis or occlusion who subsequently underwent CABG surgery.

Methods

We identified 144 patients who underwent CABG surgery at the Cleveland Clinic from 1973 through 1983 with known, unoperated ≥ 50% stenosis or occlusion of at least one ICA as documented by intra-arterial angiography. Of these 144 patients, 115 were collected from a previously published series of combined coronary and carotid operations at our institution and represent a subset in which only unilateral endarterectomy was performed despite the presence of bilateral ≥ 50% ICA lesions. An additional 29 patients who did not undergo combined carotid surgery were identified by cross-referencing our angiographic and surgical records. Most of these patients had intracranial ICA stenosis or ICA occlusion. In this group 18 patients had unilateral ICA lesions and 11 had bilateral ICA lesions.

Charts were reviewed for evidence of perioperative focal brain infarction. Stroke laterality and potential factors related to brain infarction, such as hypotension and cardiac sources of emboli, were noted.

Results

Cerebral angiography was performed in 73 patients because of an asymptomatic carotid bruit, and in 71 patients because of a history of transient ischemic attacks or stroke. The mean age was 59 years.

The distribution of ICA lesions and their correlation with perioperative infarcts involving the ipsilateral cerebral hemisphere are given in table 1. A total of 9 cerebral infarcts occurred (6%), including 5 minor strokes and 4 moderate to severe strokes. Only 3 strokes (2%) involved a cerebral hemisphere ipsilateral...
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