Carotid System Transient Ischemic Attacks: Clinical, Racial, and Angiographic Correlations

LOUIS S. RUSSO, JR., M.D.

SUMMARY The records and cerebral angiograms of 50 consecutive patients admitted to a large city hospital with a diagnosis of carotid system transient ischemic attacks were reviewed in an effort to determine the prevalence of demonstrable carotid artery disease in this population. Of these, 21 had a demonstrable abnormality in the extracranial carotid artery appropriate to their symptoms. Of the 29 with no abnormality in the extracranial portion of the appropriate carotid artery, 21 had a totally normal arteriogram, and 8 showed abnormality either in the non-appropriate carotid or in the intracranial portion of the appropriate carotid artery. Chi-square analysis of the angiographic data and various clinical factors was carried out. Of the 29 patients with no abnormality in the appropriate carotid artery, 18 were black and 11 were white. Of the 21 patients with an abnormal appropriate artery, 7 were black and 14 were white (p < 0.05). No other clinical factor correlated with the high rate of angiographic abnormality.

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and 5 had diabetes, but 15 had heart disease. Black males had the highest prevalence of hypertension, 9 of 10 patients; whereas, white males had the highest prevalence of heart disease, 10 of 12 patients.

**Clinical TIA Type.** Of the 50 patients, 23 had hemispheric left carotid artery system TIA (LCAS TIA), 17 had hemispheric right carotid artery system TIA (RCAS TIA), 4 had bilateral carotid artery system TIA (BCAS TIA), and 6 had amaurosis fugax, right and/or left.

**Angiographic Results.** The prevalence of angiographic abnormality in the cervical carotid artery appropriate to the clinical symptoms is summarized in table 1. The appropriate carotid artery was defined as that ipsilateral to the side of monocular visual loss or contralateral to the side of motor and/or sensory symptoms and/or dysphasia. In the 50 patients, therefore, 54 arteries appropriate to the clinical picture were involved. Thirty of these arteries showed no extracranial lesion; 24 showed a varying degree of stenosis. Of the 24, only 9 had stenosis of greater than 50%. The angiographic results listed in table 2 summarize the difference in prevalence of angiographic abnormality related to the artery of clinical involvement. In patients having LCAS TIA 28% (7 of 25) had a demonstrated lesion vs 52% (11 of 21) of those patients with RCAS TIA. This difference was not statistically significant. In the patients with BCAS TIA, 75% (3 of 4) had appropriate abnormalities. If patients presenting with amaurosis fugax were analyzed separately without regard to the laterality of the artery involved, 83% (5 of 6) showed an appropriate lesion.

In addition to an abnormality in the appropriate cervical carotid artery, an additional 2 patients had an abnormality in the intracranial portion of the carotid artery system appropriate to the symptoms. Eleven patients showed abnormality in the non-appropriate carotid artery, 7 had extracranial stenosis, 3 had non-stenotic ulceration, and 1 had intracranial stenosis.

**Clinical and Angiographic Correlations**

The fact that 29 of our 50 patients (58%) showed no abnormality in the appropriate cervical carotid artery was different from the usual 20-30% figure quoted in the literature. In an attempt to discover any clinical factor that contributed to this high percentage of normality, we correlated the risk factors previously discussed plus sex, age, and race with the angiographic findings. The results are summarized in table 2. Chi-square analysis of the data showed the only statistically significant correlation to be that of race versus angiographic abnormality. In the group of 29 patients with no abnormality in the carotid artery appropriate to their symptoms, 11 were white, while 18 were black. Conversely, in the 21 patients with angiographic abnormality, 14 were white and 7 were black. (Chi-square = 4.02; d.f., 0.01 < p < 0.05).

**Racial and Angiographic Correlation**

We further investigated the racial differences by subdividing the total population into black and white sub-groups and carrying out an analysis of angiographic abnormality in each sub-group (table 3). In the 25 black patients with CAS TIs, 18 had a normal appropriate carotid, and 7 had an abnormality; in the white sub-group, 11 had a normal appropriate carotid and 14 had an abnormality. The degree of stenosis in the abnormal arteries in each sub-group was not significantly different. Of the 2 patients with ulcerative lesions in the appropriate artery, 1 was white and the other black.

Analysis of the angiographic results versus diabetes, hypertension, heart disease, age, and sex was carried out in each subgroup (table 4). In the white sub-group, there was no significant correlation between the presence or absence of the various clinical factors and angiographic normality or abnormality. In the black sub-group, the general trend suggested a correlation between the presence of the clinical risk factors and angiographic abnormality. Patients in whom one of the risk factors was present had a 2 to 4 times greater prevalence of angiographic abnormality than patients in whom the same factor was absent. This correlation, however, was not statistically significant at the 5% level except with diabetes. (Chi-square = 4.0; 1 d.f., 0.01 < p < 0.05). This alone could not explain the low prevalence of angiographic abnormality in the black patients, since the incidence of dia-

**Table 1: Correlation of Angiographic Results with TIA Type**

<table>
<thead>
<tr>
<th>Angiographic Results</th>
<th>Clinical TIA Type (N = 54 arteries involved)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LCAS TIA (N = 23)</td>
</tr>
<tr>
<td>NI appropriate carotid</td>
<td>18 (78.3)</td>
</tr>
<tr>
<td>Abnl appropriate carotid</td>
<td>5 (21.7)</td>
</tr>
<tr>
<td>1–25% stenosis†</td>
<td>1 (4.3) (20)</td>
</tr>
<tr>
<td>26–50% stenosis</td>
<td>0 (0)</td>
</tr>
<tr>
<td>51–75% stenosis</td>
<td>0 (0)</td>
</tr>
<tr>
<td>76–99% stenosis</td>
<td>1 (4.3) (20)</td>
</tr>
<tr>
<td>Occlusion</td>
<td>3 (13.1) (60)</td>
</tr>
</tbody>
</table>

Note: LCAS TIA = Left Carotid System TIA
RCAS TIA = Right Carotid System TIA
BCAS TIA = Bilateral Carotid System TIA
A.F. = Amaurosis Fugax
† = Percentage of all abnormal appropriate carotid arteries in each category

**Note:**

In an effort to discover any clinical factor that contributed to this high percentage of normality, we correlated the risk factors previously discussed plus sex, age, and race with the angiographic findings. The results are summarized in table 2. Chi-square analysis of the data showed the only statistically significant correlation to be that of race versus angiographic abnormality. In the group of 29 patients with no abnormality in the carotid artery appropriate to their symptoms, 11 were white, while 18 were black. Conversely, in the 21 patients with angiographic abnormality, 14 were white and 7 were black. (Chi-square = 4.02; d.f., 0.01 < p < 0.05). This alone could not explain the low prevalence of angiographic abnormality in the black patients, since the incidence of dia-

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### Table 2: Correlation of Angiographic Results with Clinical Factors

<table>
<thead>
<tr>
<th></th>
<th>Total Population (N = 50 patients)</th>
<th>Left Carotid TIA (N = 25)</th>
<th>Right Carotid TIA (N = 21)</th>
<th>Bilateral Carotid (N = 4)</th>
<th>Amaurosis Fugax (N = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiographic Results</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>Hypertension (N = 29)</td>
<td>12.3</td>
<td>11.8</td>
<td>12.8</td>
<td>10.6</td>
<td>12.6</td>
</tr>
<tr>
<td>Diabetes (N = 15)</td>
<td>8.3</td>
<td>8.4</td>
<td>7.6</td>
<td>7.6</td>
<td>7.6</td>
</tr>
<tr>
<td>Heart Disease (N = 24)</td>
<td>4.5</td>
<td>4.4</td>
<td>4.4</td>
<td>4.4</td>
<td>4.4</td>
</tr>
<tr>
<td></td>
<td>50.5 5.1</td>
<td>50.0 4.8</td>
<td>50.0 4.8</td>
<td>50.0 4.8</td>
<td>50.0 4.8</td>
</tr>
<tr>
<td>Age (mean in years)</td>
<td>56 58</td>
<td>58 56</td>
<td>56 56</td>
<td>58 57</td>
<td>62 51</td>
</tr>
</tbody>
</table>

**Comment:**

Transient ischemic attacks are well-recognized harbingers of stroke. Carotid system TIA have been associated with lesions in the extracranial portion of the carotid artery in 70-88% of patients. This study of 50 consecutive patients with CAS TIA admitted to a city hospital showed a prevalence of atherosclerotic disease of the cervical carotid artery.

### Table 4: Correlation of Clinical Factors with Angiographic Results in Each Race

<table>
<thead>
<tr>
<th>Black patients (N = 25)</th>
<th>White patients (N = 25)</th>
<th>Male (N = 12)</th>
<th>Female (N = 13)</th>
<th>Male (N = 10)</th>
<th>Female (N = 15)</th>
<th>Male (N = 15)</th>
<th>Female (N = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean in years)</td>
<td>56.2</td>
<td>55.8</td>
<td>55.0</td>
<td>55.0</td>
<td>55.0</td>
<td>55.0</td>
<td>55.0</td>
</tr>
<tr>
<td>Abnl appro. carotid</td>
<td>Abnl appro. carotid</td>
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<td>Abnl appro. carotid</td>
<td>Abnl appro. carotid</td>
<td>Abnl appro. carotid</td>
<td>Abnl appro. carotid</td>
</tr>
<tr>
<td>No. %</td>
<td>13.4</td>
<td>12.2</td>
<td>12.0</td>
<td>12.0</td>
<td>12.0</td>
<td>12.0</td>
<td>12.0</td>
</tr>
</tbody>
</table>
| Comment:                | The prevalence of CAS in our black patients (10 of 25) was actually greater than in our white patients (5 of 25). No significant age or sex differences were noted.

**Note:**

1. Complete occlusion
2. 1-25% stenosis
3. 26-60% stenosis
4. 61-75% stenosis
5. 76-99% stenosis
6. Complete occlusion

**Percentages:**

- Percentages of all abnormal appropriate carotid arteries in each category.
- Includes non-atenotic ulceration.

**Comment:**

The prevalence of CAS in our black patients (10 of 25) was actually greater than in our white patients (5 of 25). No significant age or sex differences were noted.
appropriate to the clinical symptoms of only 42%. Patients presenting with a unilateral hemispheric syndrome showed a low prevalence of abnormality as compared to those with bilateral symptoms or amaurosis fugax (table 1).

Our population was comparable in mean age (57 years) and prevalence of diseases commonly associated with atherosclerosis to those reported in previous studies. Although the male:female ratio of 1:1.3 in this study is different from the 2:1 ratio of previous reports, this difference does not explain the low prevalence of angiographic abnormality in our patients. Previous study populations contained a large majority of non-blacks; our group was equally divided between black and white patients. This ratio corresponds closely to the statistics for all admissions to our hospital (approximately 56% black, 44% white). Chi-square analysis of race versus angiographic results showed a significant correlation ($p < 0.05$), with blacks having much less abnormality in the cervical carotid arteries than whites (table 3). Bauer reported a prevalence of normal or minimally stenotic (less than 25%) carotid arteries in 70% of the black patients he studied. As part of the Joint Study of Extracranial Arterial Occlusion, Heyman reported that blacks showed extracranial carotid artery disease roughly half as frequently as whites. Both studies, however, looked at the broad category of all atherosclerotic cerebrovascular symptoms including possible TIA, definite TIA, and frank stroke whereas our study was limited to patients admitted with CAS TIA.

Our data do not clarify this racial difference. Statistical analysis did not show that it was linked to any of the clinical risk factors such as age, hypertension, diabetes, or heart disease. Previous studies have suggested racial differences in lipid metabolism as possibly underlying the observed variation in the site of development of the atheromatous process. Recent research into the genesis of atherosclerosis and TIAs has shown that platelet aggregation and prostaglandins play a role. It is possible that some inherent racial difference in prostaglandins and/or platelet aggregation may underlie the different pattern of atherosclerotic vascular disease observed in blacks and whites. To our knowledge, no such difference has as yet been demonstrated. Recognition of this racial difference, however, may provide a basis for further studies in the pathogenesis of ischemic cerebrovascular disease and atherosclerosis.

References

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