Influence of Age on Carotid Atheroma in Patients with Reversible Ischemic Attacks

Gianluca Landi, Mario Guidotti, and Frida Valsecchi

To evaluate the influence of age on carotid atheroma we reviewed the angiographic findings in 120 patients with reversible ischemic attacks. The prevalence and severity of atherosclerotic lesions increased significantly with age, and this difference persisted after adjusting for hypertension. These results may at least partly explain the poor long term prognosis for elderly subjects with reversible ischemic attacks, and underscore the importance of taking age into account when relating clinical and angiographic findings in patients with cerebrovascular ischemia. (Stroke 1987;18:43-45)

HROMBOEMBOLISM due to atheroma of the carotid artery in its extra- or intracranial segments is the commonly accepted pathogenesis of most cerebral ischemic attacks.1 On the other hand, the main risk factors for atherothrombotic stroke are advanced age and hypertension.2,3 Since the prevalence of hypertension increases with age,4 these two factors may not be independent in contributing to the occurrence of cerebral ischemia. Experimental and pathologic studies have shown that hypertension favors the onset and progression of cerebral atherosclerosis,5,6 but scarce data are available concerning the effect of age on presence and severity of carotid atheroma. In an attempt to evaluate this relation we reviewed the carotid angiograms of 120 patients with reversible ischemic attacks.

Subjects and Methods

We studied angiograms of the symptomatic carotid artery in 120 consecutive patients with transient ischemic attack or reversible ischemic neurological deficit (i.e., with complete regression within 24 hours or within 3 weeks). The decision to carry out angiography was independent of the presence of a carotid artery bruit. The examination was performed by the trans-femoral route in 72% of the patients and by percutaneous carotid or right brachial artery injections in the others. Patients were divided into tertiles according to age: Group 1 included patients younger than 45 years (range, 16–44), Group 2 those aged 45–54 years, and Group 3 comprised patients older than 54 years (range, 55–70). Arterial hypertension was diagnosed when 2 pressure determinations exceeded 160/90 mm Hg or if antihypertensive drugs were regularly used.

The angiograms were reviewed by a neuroradiologist unaware of the patient's age and blood pressure, and all visualized carotid arteries received a score related to presence and severity of atherosclerotic lesions along their course up to the C-1 portion, according to the following criteria: normal = 1, irregularity of the vessel lumen = 2, 10–50% stenosis = 3, 50–70% stenosis = 4, 70–99% stenosis = 5, complete occlusion = 6.

The x² test was used to evaluate the prevalence of pathologic vessels among the 3 groups, and analysis of variance to assess the difference between their scores. Proper log–logistic and log–linear models were used to adjust these results for the presence of hypertension within each group.

Results

All data concerning the 120 patients are summarized in Table 1. Fourteen (35.0%) of the 40 symptomatic carotid arteries in Group 1 had evidence of atheroma, with an average score (± SD) of 1.93 ± 1.64. In Group 2, atherosclerotic lesions were found in 27 vessels (67.5%), and their mean score was 2.77 ± 1.85. Among Group 3 patients, 33 carotid arteries were abnormal (82.5%), and their score was 3.17 ± 1.75. Forty-nine of the 120 patients were hypertensive: 12 in Group 1, 16 in Group 2, and 21 in Group 3; this trend did not reach statistical significance (x² = 4.21; p > 0.10). The result of statistical analysis revealed that the progressive increase in the prevalence of lesions was highly significant (x² = 14.7; p < 0.0002), even after adjusting for the presence of hypertension (p < 0.001). Analysis of variance of the angiographic scores demonstrated a significant difference among the 3 groups (F = 8.4; p < 0.001), which was only modestly affected by correction for hypertension (p < 0.01).

Discussion

Although the incidence of cerebral ischemic attacks is strikingly related to age, surprisingly few studies have examined the influence of this factor on the prevalence of carotid atheroma. In their report on autopsy populations, Baker et al7 found a remarkable increase in the frequency and amount of cerebral atherosclerosis with advancing age, but they investigated only the circle of Willis and not the extracranial vessels. Poser et al8 observed "a definite increase of incidence of demonstrable occlusive or obstructive lesions . . . with advancing age" in 250 patients with cerebrovas-
ular disease. However, their results were not statistically significant on retrospective analysis; moreover, carotid and vertebral arteries were not considered separately, and no mention was made of the lesions' severity in relation to age. Candelise et al. devised an angiographic score to quantify atherosclerotic lesions in both the extra- and the intracranial circulation. Although age was significantly related to altered extra-cranial angiography, neither the extra- nor the intracranial score for severity of atheroma was associated with age. Again, the results included the vertebro-basilar circulation, where atheroma is probably less frequent and normal angiography more common. In a recent angiographic study, Ford et al. used regression analysis to investigate the relative importance of several risk factors on the severity of carotid bifurcation atherosclerosis in 121 patients, 80 of whom had experienced cerebral ischemic attacks. Age correlated best with degree of stenosis, but it accounted for only 13% of the variability in the extent of atheroma.

In the present study we examined the whole carotid artery including its intracranial portion, which is a well-known site of atherosclerotic lesions, in a homogeneous group of patients with reversible ischemic attacks in the ipsilateral territory. Our results demonstrate a significant positive correlation between age and both prevalence and severity of carotid atheroma. Although caution is necessary in the interpretation of angiographic studies where patients are probably selected on the basis of younger age and better general conditions, our findings are supported by those of Roederer et al., who observed an increased prevalence of severe bifurcation lesions with age in an unselected group evaluated noninvasively with duplex scanning.

Hypertension contributes to the development and extent of cervicocranial atherosclerosis. Its prevalence was not significantly different among our three groups, but because a trend showing a higher frequency in elderly patients was apparent, analyses were repeated after adjusting for its presence. Since the observed differences persisted with only a slightly lower significance, we conclude that in patients with reversible ischemic attacks the prevalence and severity of carotid atheroma increase with age independently of hypertension.

Our results underscore the importance of taking age into account when clinical findings are related to angiography and may partly explain the poor prognosis frequently observed in elderly patients with reversible ischemic attacks.

### References


### Table 1. Data Summary

<table>
<thead>
<tr>
<th>Group 1 (n = 40)</th>
<th>Group 2 (n = 40)</th>
<th>Group 3 (n = 40)</th>
<th>Statistical analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (range)</td>
<td>16-44 yrs</td>
<td>45-54 yrs</td>
<td>55-70 yrs</td>
</tr>
<tr>
<td>Males: Females</td>
<td>27:13</td>
<td>30:10</td>
<td>33:7</td>
</tr>
<tr>
<td>TIA: RIND*</td>
<td>22:18</td>
<td>17:23</td>
<td>16:24</td>
</tr>
<tr>
<td>Hypertension</td>
<td>12:18 (30.0%)</td>
<td>16:21 (52.5%)</td>
<td>21:26</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>14:27 (35.0%)</td>
<td>27:33 (82.5%)</td>
<td>33:50</td>
</tr>
<tr>
<td>atherosclerotic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>carotid arteries</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score of lesions</td>
<td>1.93 ± 1.64</td>
<td>2.77 ± 1.85</td>
<td>3.17 ± 1.75</td>
</tr>
</tbody>
</table>

*TIA, transient ischemic attack; RIND, reversible ischemic neurological deficit.
†After adjusting for the presence of hypertension, χ² = 11.07 p < 0.001, and F = 6.2 p < 0.01.


KEY WORDS • cerebral ischemia • reversible ischemic attacks • cerebral angiography • atherosclerosis
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Stroke. 1987;18:43-45
doi: 10.1161/01.STR.18.1.43

The online version of this article, along with updated information and services, is located on the
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