Carotid Bruits and Their Clinical Significance

Kouji IMATAKA, M.D.,* Akira SEKI, M.D., Nobuo TAKAHASHI, M.D.,** and Jun FUJII, M.D.

SUMMARY

Vascular murmurs were routinely sought over the carotid arteries in 1,777 outpatients of 40 years of age or older who attended the cardiovascular clinic of our institute, and carotid bruits were heard in 82 patients (4.6%). The prevalence of carotid bruits increased with age from 5/467 (1.1%) in patients aged 40 to 49 years to 26/150 (17.3%) in those aged 70 to 79 years. Calcification of the carotid arteries was examined by posteroanterior films of the cervical spine in 447 patients 60 years of age or older and carotid calcification was found in 20 (42.6%) of 47 patients with carotid bruits and in 50 (12.5%) of 400 patients without them. The incidence of carotid calcification in the former group was 3.4 times that in the latter (p<0.01), although the incidences of hypertension and glucose intolerance were not related to the presence or absence of carotid bruits. A history of cerebral bleeding or infarction was found in 19 (23.2%) of 82 patients with carotid bruits, as compared with 64 (3.8%) of 1,695 patients without them (p<0.01). However, the location of the cerebrovascular lesions did not necessarily correlate with the laterality of the carotid bruits. These results indicate that patients with carotid bruits show an increased risk of stroke and that these bruits are a general and non-focal sign of severe atherosclerotic cerebrovascular disease.

Additional Indexing Words:
Carotid calcification  Cerebral atherosclerosis  Stroke

FROM a clinical point of view, in addition to carotid calcification, carotid bruits are an important sign of advanced atherosclerosis of the carotid artery and probably an indicator of increased risk of stroke.1)-3) In our previous studies4),5) patients with visible carotid calcification on X-ray films had ex-

From the Institute for Adult Diseases, Asahi Life Foundation, Tokyo, Japan.
* Present address: The Third Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113.
** Present address: The Second Department of Internal Medicine, Faculty of Medicine, Gunma University.
Received for publication October 19, 1983.
experienced stroke about twice as often as patients without it. The prevalence of stroke in Japan is known to be much higher than that in other countries, but the prevalence of carotid calcification in Japan seems to be low compared with that in the United States. In this study, we assessed retrospectively the prevalence of carotid bruits in our patients over 40 years of age and its relation to carotid calcification, hypertension, glucose intolerance and stroke.

Materials and Methods

Routine auscultation over the carotid arteries for bruits was carried out in the supine position on 1,777 patients aged 40 or older who attended the cardiovascular clinic of the Institute for Adult Diseases Asahi Life Foundation during the period from 1968 to 1980. Patients who had murmurs over the cervical vessels transmitted from the heart or any conditions changing flow dynamics such as thyrotoxicosis or severe anemia were excluded from this study. To evaluate contributory factors to the development of carotid bruits, we examined the prevalence of radiologically visible carotid and aortic calcification, hypertension and glucose intolerance in patients 60 years of age or older. Thereafter, we studied the relation between carotid bruits and stroke. Arterial calcification was evaluated radiologically by postero-anterior films of the cervical spine, routine chest films and lateral abdominal films. Both routine chest X-ray and lateral abdominal X-ray examinations were carried out in all 663 patients 60 years of age or older and X-ray examination of the cervical spine was carried out in 447 of these patients. The presence of arterial calcification was judged by more than two observers, including radiologists. Blood pressures were measured in the sitting position and hypertensive patients were accepted for this study only if their systolic blood pressures were 160 mmHg and their diastolic blood pressures were 90 mmHg or more at the first measurement. To evaluate the glucose tolerance, oral 50 Gm or 100 Gm glucose tolerance tests were performed in 663 patients. The definition of glucose intolerance was based upon the 1970 criteria recommended by the Japanese Committee on Diagnostic Criteria of Diabetes Mellitus. Stroke was diagnosed when the patients suffered from persistent neurological deficits caused by cerebral infarction or bleeding. Chi-square analysis was used to assess statistical significance.

Results

Table I shows the age and sex distribution of patients with carotid bruits. The prevalence of carotid bruits increased from 1.1% in patients 40 to 49
Eighty-two (4.6%) of all 1,777 patients were found to have carotid bruits. Bruits were bilateral in 28 and unilateral in the remaining 54 patients, of whom 28 had right sided and 26 had left sided bruits. The relation of the carotid bruits to arterial calcification was analysed in patients aged 60 or more. As shown in Table II, carotid calcification was found in 20 (42.6%) of 47 patients with carotid bruits and in 50 (12.5%) of 400 patients without them. The incidence of carotid calcification in the former group was 3.4 times as great as that in the latter (p<0.01).

The incidence of aortic arch and abdominal aorta calcification was 59.0% and 63.9% in patients with carotid bruits and 25.9% and 38.2% in patients without them, respectively. The relation of carotid bruits to hypertension and glucose intolerance is shown in Table III. Hypertension was found almost equally in both patients with and those without carotid bruits, namely 41.0% and 40.0%, respectively. The incidence of glucose intolerance was higher in patients with carotid bruits than in patients without them (37.7% and 26.2%, respectively), but the difference was not statistically significant. Table IV shows the incidence of stroke in patients with or without carotid

Table I. Prevalence of Carotid Bruits

<table>
<thead>
<tr>
<th>age (yr)</th>
<th>sex</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70-79</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td></td>
<td>2/281 (0.7%)</td>
<td>9/344 (2.6%)</td>
<td>23/325 (7.1%)</td>
<td>19/101 (18.8%)</td>
<td>53/1,051 (5.0%)</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td>3/186 (1.6%)</td>
<td>7/303 (2.3%)</td>
<td>12/188 (6.4%)</td>
<td>7/49 (14.3%)</td>
<td>29/726 (4.0%)</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>5/467 (1.1%)</td>
<td>16/647 (2.5%)</td>
<td>35/513 (6.8%)</td>
<td>26/150 (17.3%)</td>
<td>82/1,777 (4.6%)</td>
</tr>
</tbody>
</table>

Table II. Relation of Carotid Bruits to Arterial Calcification

<table>
<thead>
<tr>
<th>Carotid Calcification</th>
<th>No Arterial Calcification</th>
<th>Aortic Arch Calcification</th>
<th>Abdominal Aorta Calcification</th>
</tr>
</thead>
<tbody>
<tr>
<td>With Bruits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20/47 (42.6%)</td>
<td>13/61 (21.3%)</td>
<td>36/61 (59.0%)</td>
<td>39/61 (63.9%)</td>
</tr>
<tr>
<td>Without Bruits</td>
<td>50/400 (12.5%)</td>
<td>305/602 (50.7%)</td>
<td>156/602 (25.9%)</td>
</tr>
<tr>
<td>p</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Table III. Relation of Carotid Bruits to Hypertension and Glucose Intolerance (≥ age of 60)

<table>
<thead>
<tr>
<th>Hypertension (≥160/90 mmHg)</th>
<th>Glucose Intolerance</th>
</tr>
</thead>
<tbody>
<tr>
<td>With Bruits</td>
<td>25/61 (41.0%)</td>
</tr>
<tr>
<td>Without Bruits</td>
<td>241/602 (40.0%)</td>
</tr>
<tr>
<td>p</td>
<td>N.S.</td>
</tr>
</tbody>
</table>
Table IV. Relation of Carotid Bruits to Stroke

<table>
<thead>
<tr>
<th></th>
<th>&lt;age of 60</th>
<th>≥ age of 60</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid Bruits</td>
<td>Present</td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4/21 (19.0%)</td>
<td>15/61 (24.6%)</td>
<td>19/82 (23.2%)</td>
</tr>
<tr>
<td>Carotid Calcification</td>
<td>Present</td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>13/93 (13.7%)</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>35/456 (7.7%)</td>
<td>-</td>
</tr>
</tbody>
</table>

* p<0.01

bruits divided into 2 age groups, one younger than 60 years of age and the other 60 years of age or older. The relation of carotid calcification to stroke previously studied by us is also shown in the lower half of the Table. Stroke occurred in 19 (23.2%) of the 82 patients with carotid bruits and in 64 (3.8%) of 1,695 patients without them. The carotid bruits seem to be of greater clinical significance as a risk factor for stroke compared with carotid calcification, because the incidence of stroke was significantly greater in patients with carotid bruits than in those with carotid calcification (24.6% vs 13.7%). Among 16 patients who had episodes of transient ischemic attack or stroke and unilateral carotid bruits, only 7 had episodes caused by lesions ipsilateral to the carotid bruits.

**DISCUSSION**

Stenosis of the carotid arteries is thought to be an important risk factor for stroke.\(^7\)-\(^{10}\) Although the detailed morphologic configuration of the stenosis should be evaluated by carotid arteriography, auscultation over the carotid arteries is useful clinically. Arterial bruits are a physical sign indicating the presence of moderate arterial stenosis. We previously studied the formation of poststenotic dilatation by applying a silver clip to the carotid arteries in rabbits.\(^\text{11}\) The arterial wall distal to the clips was markedly dilated only in cases of moderate stenosis of 45 to 60%. When severe stenosis with more than 60% constriction was applied, poststenotic collapse was observed in some cases. Although carotid bruits are a well known indicator of occlusive carotid diseases, bruits may disappear as the stenosis progresses or develops to complete occlusion.\(^\text{12}\) Carotid bruits may also change intensity according to the patients' position, exercise and blood pressure level.\(^\text{13}\)

Little is known about the prevalence of carotid bruits in the Japanese population. Kameyama\(^\text{14}\) studied carotid bruits in geriatric patients and their relation to postmortem findings in the cervical and cerebral arteries. In
his study the prevalence of carotid bruits was 38.9% of 457 consecutive autopsied patients most of whom were over the age of 70. This was very high compared with the 17.3% of patients aged 70–79 in the present study. The difference between the two studies can be explained by the difference in severity of disease in the 2 groups of patients. Kameyama examined carotid bruits in inpatients who were terminal while we studied outpatients who were less ill. In the Framingham study, carotid bruits appeared in 71 (7.0%) of 1,021 subjects aged 65–79 during 8 years. A survey of a rural community in Georgia revealed carotid bruits in 72 (4.4%) of 1,620 persons 45 years of age or older. Although these values of prevalence are almost equal to those of our study, this does not necessarily mean that the severity of carotid arterial sclerosis is equal in the two countries. Severe stenosis or complete occlusion may be more frequent in the United States than in Japan. The prevalence of visible carotid calcification on X-ray was 43.9% of 394 patients aged 60 or more in the United States, which was extremely high compared with 17.2% of 551 patients aged 60 or more found in our previous study. Our previous experiment suggests that vascular murmurs are best heard when the artery is constricted to 35–60% of the original diameter or 60–80% of the cross sectional area. The investigation of extracranial arteries in autopsy cases 60 year of age or older by Kameyama revealed that stenosis of 60–80% in transverse area caused by arteriosclerotic lesions was seen in 12.3%, 6.2% and 10.6% of the common carotid, external carotid and internal carotid arteries, respectively. These values were almost equal to the 9.2% which was the summation of the prevalence of bruits in the 60–69 and 70–79 year age groups in this study.

Our previous observation revealed that patients with carotid calcification had higher incidences of hypertension and glucose intolerance than did those without them. In the present study, however, patients with carotid bruits did not have high incidences of these risk factors. As previously described, bruits are a clinical manifestation only of moderate stenosis and not present over arteries with severe stenosis or complete occlusion. Therefore, the apparent lack of any significant relation between hypertension or glucose intolerance and bruits does not necessarily exclude the contribution of hypertension or glucose intolerance to the development of carotid stenosis as an atherosclerotic process.

From a clinical point of view, it is important to evaluate the contribution of carotid bruits to subsequent stroke. In the Framingham study, transient ischemic attacks appeared in 8 (4.7%) and stroke in 21 (12.2%) of the 171 persons with carotid bruits during 8 years. The incidence of stroke in these patients was more than twice that expected for age and sex. Heyman
and co-workers also estimated the risk of stroke associated with carotid bruits during a 6-year follow-up period, taking age and blood pressure into account. During this time an episode of stroke occurred in 10 (13.9%) of the 72 persons with bruits and only 52 (3.4%) of the 1,548 without bruits. In the present study, stroke was found in 19 (23.2%) of 82 patients with carotid bruits. This incidence of stroke was high compared with those just cited. This difference may be explained by the fact that the overall incidence of stroke in Japan is higher than that in the United States. Both Framingham and Heyman's study showed that carotid bruits did not bear any close relation to the location of stroke. In the present study, as would be expected, the location of stroke did not correlate with the vascular territory of the carotid bruits.

We previously reported that the prevalence of carotid calcification was 13.8% in patients 60–69 years of age and 23.9% in those 70–79 years of age, both of which were about 7% higher than the prevalence of bruits in these respective age groups in this study. The present study also showed that 42.6% of patients with carotid bruits had carotid calcification on X-ray. Moreover, the incidence of stroke was higher in patients with bruits than in patients with calcification in those patients who were over 60 years of age (24.6% vs 13.7%). These results indicate that carotid bruits are a clinical sign of severe atherosclerosis, as is carotid calcification. Probably, the presence of bruits is more closely related to arterial stenosis than that of calcification. In other words, carotid bruits which are caused by atherosclerotic stenosis seem to be a sign of more advanced lesions than is carotid calcification which is a morphological expression of severe atherosclerosis.

**Acknowledgments**

The authors gratefully acknowledge the technical assistance of Miss Y. Kambe and Miss M. Takaku.

**References**


