Coexistence of Abdominal Aortic Aneurysm in Patients With Carotid Stenosis

Percy N. Karanjia, MD, MRCP; Kenneth P. Madden, MD, PhD; Sandra Lobner, LPN

Background and Purpose The study was designed to assess the incidence of coexistent abdominal aortic disease in patients with known carotid artery stenosis.

Methods Abdominal ultrasound and physical examination of the abdomen were performed in 89 consecutive patients referred to this tertiary medical center for evaluation of both symptomatic and asymptomatic carotid artery disease. The frequency of abdominal aortic aneurysm detected in this population was compared with that incidentally found in age- and sex-matched patients undergoing abdominal ultrasound for nonvascular reasons.

Carotid artery atherosclerosis rarely occurs as an isolated vasculopathy. There is a known association with coronary artery disease. Indeed, death in patients with carotid stenosis is more commonly related to coronary insufficiency than to complications of cerebrovascular disease. Good clinical practice usually entails a diligent search for coronary vascular disease before management decisions for carotid stenosis are made.

The association of carotid disease with other types of arterial disease is less well known.

Abdominal aortic aneurysm is a potentially fatal degenerative vasculopathy that is usually discovered in patients with evidence of diffuse atherosclerosis. The natural history of these aneurysms involves slow expansion and eventual rupture, with subsequent high mortality. Physical examination is relatively insensitive in detecting abdominal aortic dilatation, but ultrasonic screening techniques are highly accurate.

Early detection of these usually asymptomatic aneurysms can lead to prophylactic surgical repair. Such early surgery is now widely advocated, with perioperative mortality considered low.

We investigated the possibility that patients with carotid stenosis have a high incidence of associated abdominal aortic aneurysm.

Subjects and Methods

Patients with carotid artery stenosis referred to this tertiary medical center for neurological assessment between 1989 and 1991 were sequentially screened with abdominal ultrasound for the presence of abdominal aortic aneurysm. All patients received complete neurological examination by one of the authors (P.N.K.) and in addition had careful abdominal examination. Evaluation of asymptomatic carotid stenosis was usually prompted by the detection of a carotid bruit on physical examination. Carotid stenosis was classified as symptomatic if patients had experienced previous transient ischemic attack or minor stroke in the cerebral distribution of the stenotic carotid artery.

Detection of carotid stenosis was generally made by carotid duplex examination and verified in the majority by cerebral angiography. Doppler studies were performed with Advanced Technologies Laboratory machines (models Ultramark 4, 8, and 9). Reliability of Doppler frequency criteria for vascular stenosis has been verified at this institution with angiographic comparison and deemed acceptable for use in international carotid endarterectomy trials. Eighty-nine patients were identified with carotid stenosis greater than 30%; 41 patients were asymptomatic and 48 asymptomatic. Severity of carotid stenosis ranged from 30% to more than 95%.

A comparison population of 89 patients had been established at less than 2 mm in this institution. Abdominal aortic aneurysm was defined as transverse infrarenal aortic dilatation greater than 2.5 cm, with clear focal dilatation of the vessel judged by the radiographer. The interrater variability for measurement of aortic dilatation has been established at less than 2 mm in this institution.

Results Eighteen aortic aneurysms were detected by ultrasound in 89 patients with carotid stenosis, compared with three aneurysms in 89 matched patients (P<.01, corrected $\chi^2$).

Twelve of the 18 patients with aneurysm had an abnormal abdominal physical examination.

Conclusions Aortic aneurysm occurs with high frequency in patients with carotid stenosis. Physicians should perform careful abdominal examination on these patients and consider routine abdominal ultrasonic screening. (Stroke. 1994;25:627-630.)

Key Words • aneurysm • carotid artery diseases • ultrasonics
hypercholesterolemia (prior diagnosis on medication or fasting cholesterol >220 mg/dL), and smoking history. History of angina or myocardial infarction was elicited. Recent laboratory assessment of fasting total cholesterol, fasting glucose, and blood urea nitrogen for each patient was obtained. The electrocardiogram was examined for abnormalities suggesting coronary ischemia or left ventricular hypertrophy. Frequency of these patient characteristics and presence of abdominal aortic aneurysm were compared between the two populations using $\chi^2$ analysis.

Results

Table 1 presents population characteristics of the two patient groups. All vascular risk factors were more frequent in patients with carotid stenosis than in the comparison population. Such increases were statistically significant for hypertension, hypercholesterolemia, and smoking history ($P<.01$, corrected $\chi^2$). Mean arterial pressure, total cholesterol, and fasting glucose levels were all relatively elevated. This group had a higher incidence of angina and prior myocardial infarction, and there was a greater frequency of electrocardiogram abnormalities.

Frequency of abdominal aortic aneurysm detection differed in the two populations. Eighteen of 89 patients (20%) with carotid stenosis had a coexistent aortic aneurysm compared with 3 of 89 patients (3%) in the comparison population, a significantly higher frequency ($P<.01$). Higher degrees of carotid stenosis were not associated with increased incidence of aortic aneurysm (Table 2). Increased incidence of aortic aneurysm was also noted in patients whose carotid stenosis had previously been symptomatic compared with those patients in whom carotid stenosis had been asymptomatic. Eleven of 41 (27%) of symptomatic patients had aneurysms compared with 7 of 48 (15%) asymptomatic patients. This trend was not statistically significant, however ($P=.24$). In 15 patients with carotid stenosis and abdominal aortic aneurysm, the aneurysm size by ultrasound ranged from 2.5 to 7 cm, with an average size of 3.7 cm (Figure). The remaining 3 patients had larger aneurysms defined by angiography, which does not provide a directly comparable measurement. Twelve of the 18 patients with aortic aneurysm had an abnormal abdominal examination, either abdominal bruit (8 patients) or palpable mass (11 patients). None of the 21 patients with abdominal aneurysm reported symptoms of the aneurysm at the time of ultrasound. Patients with both carotid stenosis and abdominal aortic aneurysm had similar vascular risk factors compared with patients with carotid stenosis but without abdominal aortic aneurysm (Table 3).

Discussion

Atherosclerosis is generally a diffuse process affecting multiple arterial sites. Documentation of one site of involvement typically is followed by discovery of remote arterial disease. Atherosclerotic coronary artery disease has a clear association with carotid artery and aortofemoral disease. Abdominal aortic aneurysm is associated with atherosclerosis, with prevalence rates of 10% to 16% in patients known to have femoral occlusive disease.13 It is also strongly associated with coronary artery disease; a recent study found that 36% of patients with known aortic aneurysm had severe coronary artery disease on angiography.14 Similarly, carotid artery dis-

<table>
<thead>
<tr>
<th>Degree of Stenosis</th>
<th>No. of Patients</th>
<th>No. With Aortic Aneurysm (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unknown*</td>
<td>89</td>
<td>3 (3)</td>
</tr>
<tr>
<td>30%-59%</td>
<td>12</td>
<td>2 (17)</td>
</tr>
<tr>
<td>60%-79%</td>
<td>25</td>
<td>5 (20)</td>
</tr>
<tr>
<td>80%-99%</td>
<td>52</td>
<td>11 (21)</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>41</td>
<td>11 (27)</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>48</td>
<td>7 (15)</td>
</tr>
</tbody>
</table>

*Patients without previous carotid ultrasound (comparison population).
We confirm a substantial incidence of unsuspected abdominal aortic aneurysmal disease in a patient population with either symptomatic or asymptomatic carotid artery stenosis. More than one fifth of this cohort of patients with carotid stenosis had coexistent aortic aneurysmal disease. This incidence increased to 27% if the carotid stenosis had previously been symptomatic with stroke or transient ischemic attack. This is significantly higher than in an age- and sex-matched population with an unknown incidence of carotid disease. We believe this patient cohort to be an adequate comparison group because it appropriately mimics the general populace, the vast majority of whom have not had investigation of carotid artery disease. The 3% incidence of aneurysm in our comparison population does compare well, however, with the 6% incidence of unsuspected aneurysm reported in a group of English men aged older than 65 years.

While the average size of unsuspected aneurysms in our study was relatively small, arterial rupture does occur at this degree of dilatation. Additionally, the natural history of these aneurysms involves slow expansion and increasing risk of rupture. Bengtsson and colleagues followed asymptomatic patients serially with ultrasound and documented expansion of 0.8 mm/y in smaller aneurysms and 3.3 mm/y in aneurysms greater than 4.0 cm. Katz et al estimated from a review of the literature an increasing incidence of aneurysmal rupture from 0 to 14.4 events per 100 patient-years, as aneurysms expand from less than 4.0 cm to greater than 5.0 cm. Given the potential benefit of early surgical repair of these aneurysms, we believe that clinicians should perform careful abdominal examinations and consider ultrasonic screening of the abdomen when encountering patients with carotid artery disease. At the least, detection of aneurysm in these patients can result in careful monitoring of aneurysmal size and thoughtful management decisions.

### Table 3. Comparison of Vascular Risk Factors In Patients With Carotid Stenosis With and Without Abdominal Aortic Aneurysm

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Patients With AAA (n=18)</th>
<th>Patients Without AAA (n=71)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, y</td>
<td>71.6</td>
<td>69.9</td>
<td>.67</td>
</tr>
<tr>
<td>Male</td>
<td>12 (67%)</td>
<td>41 (58%)</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>7 (39%)</td>
<td>14 (20%)</td>
<td>.16</td>
</tr>
<tr>
<td>Hypertension</td>
<td>16 (89%)</td>
<td>57 (80%)</td>
<td>.61</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3 (17%)</td>
<td>18 (25%)</td>
<td>.64</td>
</tr>
<tr>
<td>Claudication</td>
<td>4 (22%)</td>
<td>17 (24%)</td>
<td>.88</td>
</tr>
<tr>
<td>Smoking</td>
<td>13 (72%)</td>
<td>48 (68%)</td>
<td>.93</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>16 (89%)</td>
<td>64 (90%)</td>
<td>.78</td>
</tr>
<tr>
<td>Angina</td>
<td>4 (22%)</td>
<td>21 (30%)</td>
<td>.74</td>
</tr>
<tr>
<td>LVH by ECG</td>
<td>5 (28%)</td>
<td>15 (21%)</td>
<td>.77</td>
</tr>
<tr>
<td>Mean cholesterol, mg/dL</td>
<td>230±43</td>
<td>222±41</td>
<td></td>
</tr>
<tr>
<td>Mean BUN, mg/dL</td>
<td>22±12</td>
<td>18±10</td>
<td></td>
</tr>
<tr>
<td>Mean glucose, mg/dL</td>
<td>122±62</td>
<td>129±45</td>
<td></td>
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</tbody>
</table>

AAA indicates abdominal aortic aneurysm; LVH, left ventricular hypertrophy; ECG, electrocardiogram; and BUN, blood urea nitrogen. Statistical comparisons were performed with corrected $\chi^2$ test.
istent abdominal aortic aneurysm in such patients cannot be based on vascular risk factor analysis alone.

References


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