Carotid Stenosis in Lacunar Stroke

Charles H. Tegeler, MD; Fenglin Shi, MD; and Terumi Morgan

The prevalence of extracranial carotid stenosis in patients with a clinical syndrome of lacunar stroke has not been extensively studied using noninvasive methods. We performed carotid duplex sonography on 168 patients referred to the neurosonology laboratory with a diagnosis of ischemic stroke. Strokes were independently classified as lacunar or nonlacunar hemispheric infarction without knowledge of the ultrasound results. We excluded patients with infarcts that were clearly vertebrobasilar, presumed to be cardioembolic, or had occurred >1 year earlier, and patients for whom classification of the nature and location of the event was not possible. Fifty-five patients had lacunar and 54 had nonlacunar stroke. No differences in age, sex, distribution, or prevalence of hypertension, diabetes, prior ischemia, or Hispanic surname existed between the two groups. Tobacco use was more frequent in the nonlacunar group (p<0.01). The prevalence of important extracranial carotid stenosis (≥50% diameter reduction) in the lacunar stroke group was 13% (seven of 55) in the ipsilateral and 4% (two of 55) in the contralateral carotid artery. Of the 54 patients with nonlacunar hemispheric stroke, 41% (22) had ipsilateral (p<0.01) and 26% (14) had contralateral (p<0.01) carotid stenosis. This study suggests that important carotid stenosis is infrequent among patients presenting with a clinical syndrome of lacunar stroke. These data impact on the decisions regarding cerebrovascular work-up in such patients. (Stroke 1991;22:1124-1128)

Approximately 20% of all ischemic strokes are lacunar infarctions. Based on pathological correlations in a limited number of patients, the pathogenesis of lacunar infarcts is believed to be due to primary disease of the small penetrating arteries.1-6 The risk factors are similar for carotid artery atherosclerosis and lacunar stroke; however, the potential role of carotid atherothrombosis in the pathogenesis of lacunar stroke has not been extensively studied.7-10 High-resolution duplex ultrasonography now allows safe, accurate, and reliable assessment of extracranial carotid artery disease.11,12

To determine the prevalence of important extracranial carotid artery disease in patients presenting with a clinical syndrome of lacunar stroke, using noninvasive techniques we performed carotid duplex sonography on consecutive patients with lacunar or nonlacunar hemispheric stroke referred to the neurosonology laboratory. Patients with lacunar stroke represent a heterogeneous group, and the clinical syndrome is described with lesions elsewhere in the brain and in other circulations. While acknowledging such diversity, this study focused on that subset of patients presenting with a lacunar syndrome that clinically appears to involve the carotid territory and an appropriate computed tomogram (CT scan) without obvious brain stem lesions. Initial clinical questions frequently faced in such patients include 1) what is the likelihood of extracranial carotid stenosis? 2) is this prevalence of carotid stenosis different from that in patients with nonlacunar carotid-territory infarcts? and 3) how will that affect the cerebrovascular work-up of this patient?

Subjects and Methods

We derived our study population from 168 patients admitted to the inpatient neurology services at the Medical Center Hospital or the Audie L. Murphy Veterans Hospital or seen in the outpatient neurology clinics and referred to the Neurosonology Research Laboratory with a diagnosis of ischemic stroke. Data were accumulated from April 23, 1987, to June 6, 1988. No patient on the neurology service had cerebral angiography without prior carotid duplex examination.

Patients were classified as having lacunar or nonlacunar hemispheric infarcts based on strict clinical and CT criteria. Patients with infarcts of the vertebrobasilar distribution, presumed cardioembolic stroke, or infarcts occurring >1 prior to study and those for whom classification of the nature and...
location of the event was not possible were not included in this analysis.

Lacunar stroke was defined as a focal neurological deficit involving the carotid territory lasting >24 hours and consisting of the clinical syndrome of 1) pure motor hemiparesis, 2) sensorimotor stroke, 3) clumsy hand-dysarthria, or 4) ataxic hemiparesis. Although considered a classic lacunar syndrome, pure hemisensory stroke was excluded because this syndrome usually implies a thalamic lesion in an area supplied by the posterior circulation, occurs infrequently, and is more difficult to assess reliably on a clinical basis. In addition, a CT scan done at least 72 hours following the event had to be without evidence of acute cerebral infarction or show only a low-density lesion of ≤1.5 cm diameter in a subcortical area of the appropriate hemisphere. Patients with nonlacunar stroke included those with focal hemispheric neurological deficits in the carotid territory, including cortical or subcortical abnormalities, persisting for >24 hours. Additionally, a CT scan done at least 72 hours following the event had to show an appropriate nonhemorrhagic cortical low-density lesion or a nonhemorrhagic subcortical lesion of >1.5 cm diameter in the absence of an obvious cardioembolic source. Clinical classification was made independently by a single observer based on review of the initial neurology attending physician’s and resident’s notes in the chart. Clinical classification and interpretation of the CT scans was done without knowledge of the presence or absence of carotid stenosis.

All patients had carotid duplex ultrasound examinations, including spectral analysis, performed in the Neurosonology Research Laboratory according to a standard protocol. All studies were performed using a Hoffrel 518 SD+ duplex ultrasound machine (South Norwalk, Conn.), with 7.5- and 10-MHz transducers for B-mode evaluation and duplex Doppler evaluation at 5.6 MHz, with spectral analysis using the fast Fourier transform. Studies were recorded on VHS videotape for later review, and all were interpreted by a single observer. The sensitivity of the laboratory for the detection of carotid stenosis, compared with cerebral angiography, is 96% for ≥50% diameter reduction and 91% for ≥75% diameter reduction.

Criteria for judging stenosis using the Doppler results and the Doppler frequency shift were based on the work of Strandness. B-mode measurements for the determination of diameter reduction were made using electronic calipers. The B-mode diameter stenosis was defined as 1 minus the value obtained from dividing the diameter of the residual lumen by the true vessel diameter at the point of maximal stenosis. Both Doppler and B-mode data were reviewed to obtain a final determination of the maximum stenosis in the common carotid artery, the internal carotid artery, or at the bifurcation on each side. If there was a discrepancy, the Doppler value was used since Doppler sonography has its greatest accuracy with stenoses of ≥50%. Results from each arteriographic study were classified as indicating 0–24%, 25–49%, 50–74%, 75–99%, or 100% stenosis. The results were rechecked prior to analysis, without knowledge of the previous result.

The data were classified as applying to arteries ipsilateral or contralateral to the symptomatic hemisphere and were then evaluated, based on the maximal stenosis on the side, using $\chi^2$ analysis.

Among the 168 patients with ischemic stroke, 55 were classified as having lacunar infarction while 54 met the criteria for nonlacunar stroke. Fifty-nine patients with remote or other types of infarcts were not included in this analysis. Clinical characteristics of the two groups did not differ except for more tobacco users in the nonlacunar stroke group (Table 1). All strokes in the lacunar group were in the pure motor hemiparesis or sensorimotor stroke clinical categories. The mean time from stroke to carotid duplex examination was 12 days.

**Results**

In the lacunar stroke group seven (13%) of 55 patients had ≥50% carotid stenosis ipsilateral to the clinical event, while on the contralateral side only two (4%) had such stenosis (Table 2). In the nonlacunar group, the contralateral side of the event was not possible were not included in this analysis.

**Table 2. Maximal Carotid Stenosis on Duplex Scan**

<table>
<thead>
<tr>
<th>Group</th>
<th>Lacunar (n=55)</th>
<th>Nonlacunar (n=54)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ipsilateral</td>
<td>Contralateral</td>
</tr>
<tr>
<td>0–24%</td>
<td>22</td>
<td>33</td>
</tr>
<tr>
<td>25–49%</td>
<td>26</td>
<td>30</td>
</tr>
<tr>
<td>50–74%</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>75–99%</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>100%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total ≥50%</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Total ≥75%</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Data are number of patients. *p<0.01 different from corresponding side in lacunar group.
Lacunar stroke group 22 (41%) of 54 patients had stenosis of ≥50% ipsilateral to the event, while 14 (26%) had ≥50% stenosis on the contralateral side. Carotid stenosis of ≥50% was significantly more frequent (p<0.01) in patients with nonlacunar stroke on both sides.

In an analysis of ≥75% stenosis, the lacunar stroke group had one (2%) patient with ipsilateral and none (0%) with contralateral carotid stenosis. In the nonlacunar stroke group, 13 patients (24%) had ≥75% stenosis ipsilateral and three (6%) had such stenosis contralateral to the stroke. Comparing the groups, there was a significant difference ipsilateral (p<0.01) but not contralateral (p>0.05) to the stroke.

When both carotid arteries were considered, nine of 110 (8%) in the lacunar stroke group and 36 of 108 (33%) in the nonlacunar stroke group had ≥50% stenosis (p<0.01). Only one artery (1%) in the lacunar stroke group had ≥75% stenosis, while 16 (15%) in the nonlacunar stroke group had this more severe stenosis (p<0.01). There were six total occlusions in the nonlacunar stroke group and none in the lacunar stroke group.

Discussion

Lacunar infarcts, as popularized in the modern era by Fisher, are small, deep infarcts in the vascular territory of small penetrating branches of the large cerebral arteries. Approximately 20% of all ischemic strokes are considered to be lacunar, and they tend to recur. Based on pathological assessment of early cases, such infarcts were believed to reflect primary arterial changes in these small vessels, but recent reports suggest additional mechanisms.

The original descriptions of lacunar stroke have been expanded to include a number of additional clinical syndromes. With the advent of CT and without extensive pathological confirmation, the label of lacunar infarct is now widely applied to any small deep infarct. A diagnosis of lacunar infarct frequently implies a specific pathophysiological mechanism, precluding further investigation for other possible cerebrovascular causes. As a result, angiographic data addressing the widespread view that extracranial carotid artery disease is infrequent in patients with lacunar infarcts have only recently been reported.

Higher degrees of carotid stenosis are associated with an increased risk of ischemic stroke, but the mere presence of carotid stenosis cannot be invoked as the unequivocal causative factor. However, the presence of carotid stenosis is of clinical interest, and such lesions offer a variety of potential management options, including surgery. High-resolution carotid duplex scanning now allows safe, reliable assessment of extracranial carotid artery disease, with sensitivities of 90–95% for the detection of important stenosis and high negative predictive values.

Our results provide further objective support for the widespread belief that important extracranial carotid stenosis is infrequently seen with the classic clinical syndrome of lacunar stroke. Despite similar risk factors for vascular disease (except tobacco use), the pattern of atherosclerotic disease appears to be different in patients presenting with lacunar and nonlacunar stroke.

These data are consistent with the most recent angiographic reports regarding similar patients. Our findings may impact on clinical decision-making as regards the need for, or type of, cerebrovascular workup in such patients. Because carotid stenosis is uncommon in patients presenting with a classic syndrome of lacunar stroke and appropriate CT findings, any cerebrovascular workup should begin with non-invasive carotid sonography.

Although not available on most patients, magnetic resonance imaging would have allowed more sensitive visualization of acute small, deep infarcts. While admittedly not as sensitive for such lesions, CT allowed exclusion of patients with clinically occult cortical involvement or large, deep infarcts. Pure motor strokes in patients without apparent infarcts on CT scan were clinically classified as being referable to the anterior circulation because most involve the internal capsule. Thus, pure motor stroke involving the basis pontis might potentially be misclassified as involving the anterior circulation, but these strokes are much less common than those involving the capsule. However, such potential misclassification is a limitation of our methodology.

Tobacco use is a marker of increased risk for extracranial carotid atherosclerosis and stroke, but a relation to stroke type has not been reported. The higher frequency of tobacco use in the nonlacunar stroke group (Table 2) was an unexpected finding. Tobacco users had about twice the likelihood of having ipsilateral carotid stenosis as nonusers (34% versus 18%, Table 3). Among tobacco users about one third of the strokes were lacunar, while among nonusers the lacunar versus nonlacunar ratio was reversed. A nonuser with a lacunar stroke had a very small chance of having ipsilateral carotid stenosis (6%), but patients with lacunar stroke who smoked had a fourfold increased chance of having carotid stenosis (24%). Due to small numbers and a selected population, the meaning of this finding is unclear.

As more specific therapies are developed for the prevention and treatment of stroke, definition of the pathophysiological mechanism assumes greater im-

<table>
<thead>
<tr>
<th>Tobacco Use, Lacunar Stroke, and Carotid Stenosis</th>
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<tbody>
<tr>
<td>Stroke type</td>
</tr>
<tr>
<td>------------</td>
</tr>
<tr>
<td>Tobacco users</td>
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<tr>
<td>Lacunar</td>
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<tr>
<td>Nonlacunar</td>
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<tr>
<td>Nontobacco users</td>
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<tr>
<td>Lacunar</td>
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<td>Nonlacunar</td>
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portance. In view of a different prevalence of important extracranial carotid stenosis, most lacunar infarcts may represent a unique pathophysiology that should be evaluated separately in future clinical trials. Support for such an approach is seen in a subgroup analysis of data reported by Bousser et al.45 Although the number of patients was small, these investigators demonstrated a greater effect of aspirin, with or without dipyridamole, for the secondary prevention of stroke in patients with lacunar than in nonlacunar stroke (31% risk reduction in nonlacunar group versus 67% in lacunar group).

In conclusion, among patients presenting to the Neurosonology Research Laboratory with a clinical syndrome of lacunar stroke and an appropriate CT scan, the prevalence of important carotid stenosis (≥50% diameter reduction) was low. Such patients were less likely to have important carotid stenosis both ipsilaterally and overall than patients with nonlacunar hemispheric stroke. In view of the low prevalence of important carotid stenosis in this population, routine or initial cerebral angiography is not indicated. Any cerebrovascular workup should initially be done using a noninvasive modality such as carotid sonography. Although limited by small numbers, this study also suggests that tobacco use may influence not only the risk for developing carotid atherosclerosis but also the clinical type of stroke.

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References


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