Silent Stroke and Carotid Stenosis

J.W. Norris, MD, FRCP, and C.Z. Zhu, MD

Background and Purpose: Silent cerebral infarction is often found on computed tomographic scan in patients with asymptomatic carotid stenosis, but its relation to the arterial stenosis is uncertain.

Methods: We compared computed tomographic scans and carotid Doppler in 115 patients with asymptomatic carotid stenosis, 203 with carotid transient ischemic attacks and carotid stenosis, and 63 with transient ischemic attacks but without carotid stenosis. There was no group with normal carotid arteries for comparison.

Results: Lesions seen on CT scan were most common in the transient ischemic attack with carotid stenosis group (47%) compared with the other groups (36%, 19%) (p<0.001). Cerebral infarcts ipsilateral to the carotid stenosis were found in 10% of patients with mild (35–50%) stenosis, 17% in moderate (50–75%) stenosis, and 30% with severe (>75%) carotid stenosis (p<0.001). In patients with asymptomatic carotid stenosis, 68% of infarcts were ipsilateral to the stenosis; in those with transient ischemic attacks and carotid stenosis, 86% of infarcts were ipsilateral to the stenosis.

Conclusions: The more severe the carotid stenosis, the higher the incidence of cerebral infarction ipsilateral to the stenosis. This finding applied to central infarcts as well as to peripheral infarcts in both symptomatic and asymptomatic patients. Silent cerebral infarction may be an indication for carotid endarterectomy in asymptomatic patients. (Stroke 1992;23:483–485)

KEY WORDS • cerebral infarction • carotid artery diseases • ultrasonics

Soon after the advent of computed tomography, it became evident that the reversible clinical deficits seen in transient ischemic attacks (TIAs) were often accompanied by irreversible structural cerebral lesions; 12–70% of computed tomographic (CT) scans in these patients revealed small cerebral infarctions.1 In the National Institute of Neurological and Communicative Disorders and Stroke Data Bank Study,2 11% of patients with clinical signs of stroke but without any history of stroke had CT lesions unrelated to the presenting stroke. Also, approximately 20% of patients with asymptomatic carotid stenosis have infarcts confirmed on CT scan,3 while multiple small infarcts without any accompanying symptoms sometimes appear after carotid endarterectomy.4 Because stroke is a clinical phenomenon, these asymptomatic lesions are better termed "silent infarctions" than "silent strokes."

The dilemma presented by transient clinical deficits with permanent structural cerebral lesions caused Waxman and Toole5 to propose a new term, "cerebral infarction with transient signs," a separate and presumably more serious syndrome than TIAs with normal appearance on CT scan. However, just as silent infarcts cannot be equated with silent strokes, TIAs remain clinical observations independent of CT scan or other laboratory findings.

Because we have often observed a variety of small cerebral infarcts in patients with asymptomatic carotid stenosis, we decided to investigate this observation further to evaluate the clinical significance of the lesions and to decide whether other factors such as the severity of the carotid stenosis relate to the appearance of infarction on CT scan.

Subjects and Methods

We compared the CT findings of three groups of patients. First, 115 patients with asymptomatic stenosis in one or both carotid arteries were referred to the carotid Doppler laboratory because of asymptomatic neck bruits. These patients had undergone previous CT scanning and were prospectively followed up in a special outpatient clinic at 6-month intervals.6

Second, in the neurology outpatient department 63 patients with TIAs had normal carotid arteries on Doppler ultrasound examination. Patients in this group were eligible for inclusion in the study only if they had both CT scanning and carotid Doppler examination. Hence, although these patients were consecutively enrolled, those with only one test result were not included.

Finally, 203 patients with TIAs and evidence of carotid stenosis in one or both arteries on Doppler examination (obtained from the medical records) were initially gathered for enrollment in a carotid surgery study. Because this is a retrospective review of prospectively gathered data, the timing of the CT scanning was random in the asymptomatic patients. However, all the symptomatic patients were investigated acutely, so all CT scans were performed within days or weeks of the clinical event.

Carotid Doppler studies were performed on a Carolina Medical Electronic Dopscan 1060, a duplex scanning device with a 5-mHz probe. In our laboratory, this
were bilateral and multiple. Patients with TIA and 
while 61% (83) were peripheral infarcts located in the 
cortex or immediate subcortical regions.

When the CT lesions were divided into deep, cen-
trally located infarcts and peripheral (cortical) infarcts, 
patients with TIAs and carotid stenosis had significantly 
more infarcts of both types (p<0.0001, Table 1).

According to Degree of Carotid Stenosis

TABLE 1. Frequency of Central and Peripheral Infarction in Symptomatic and Asymptomatic Patients

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean age</th>
<th>Men:Women</th>
<th>Peripheral infarcts</th>
<th>Central infarcts</th>
<th>Positive CT (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACS</td>
<td>115</td>
<td>69</td>
<td>66:49</td>
<td>12</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>TIA−</td>
<td>63</td>
<td>66</td>
<td>33:30</td>
<td>11</td>
<td>8</td>
<td>30</td>
</tr>
<tr>
<td>TIA+</td>
<td>203</td>
<td>66</td>
<td>135:68</td>
<td>60</td>
<td>36</td>
<td>47*</td>
</tr>
</tbody>
</table>

ACS, patients with asymptomatic carotid stenosis; TIA−, transient ischemic attack patients without carotid stenosis; TIA+, TIA patients with carotid stenosis.

*p<0.0001.

Discussion

Silent as well as symptomatic cerebral infarcts were 
uniformly small, which is the finding of virtually all 
similar reports.1-3,8-10 This may reflect the suggested 
pathogenesis of these lesions, that showers of small 
microemboli composed of platelet aggregates or choles-
terol crystals are shed from the surface of carotid 
plaques but, because of their size, do not produce 
symptoms.1 It has even been argued that these tiny 
infarcts are therefore markers of active carotid plaque 
ulceration, based on the correlation of postoperative 
examination of carotid plaques and the CT findings in 
the same patients.10 Recent transcranial Doppler mon-
itoring observations in patients undergoing cardiotho-
racic and carotid surgery confirm that streams of micro-
emboli bombard the brain during and after the surgical 
procedure.11

Apart from the small size of these infarcts, there may 
be other reasons for their silent appearance. Patients 
experiencing cerebral microembolization during sleep 
may be unaware of transient symptoms which would 
otherwise alarm them during waking hours. We were 
unable to distinguish any specific localization of the 
lesions to silent areas of the brain, and this has been the 
experience of others.8 Dismissal of fleeting and minor 
symptoms by either patient or physician might also 
explain the apparent absence of symptoms.

TABLE 2. Frequency of Central and Peripheral Infarctions 
According to Degree of Carotid Stenosis

<table>
<thead>
<tr>
<th>Stenosis</th>
<th>n</th>
<th>Peripheral infarcts</th>
<th>Central infarcts</th>
<th>Positive CT scan (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (35–50%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptomatic</td>
<td>41</td>
<td>7</td>
<td>3</td>
<td>24</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>193</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>234</td>
<td>14</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Moderate (50–75%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptomatic</td>
<td>34</td>
<td>6</td>
<td>6</td>
<td>35</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>84</td>
<td>2</td>
<td>6</td>
<td>9.5</td>
</tr>
<tr>
<td>Total</td>
<td>118</td>
<td>8</td>
<td>12</td>
<td>17</td>
</tr>
<tr>
<td>Severe (&gt;75%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptomatic</td>
<td>128</td>
<td>40</td>
<td>17</td>
<td>52</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>156</td>
<td>18</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>284</td>
<td>58</td>
<td>27</td>
<td>30</td>
</tr>
</tbody>
</table>

n, Number of stenosed carotid arteries.
There was a disproportionately high incidence of central infarctions (39%) in the present study compared with that in our hospital stroke register (10%) and in other series. This may simply represent a bias of small lesions in asymptomatic patients. We do not have enough data on concomitant hypertension in these patients to comment on this as a risk factor. To determine the effect of carotid stenosis alone on the associated lesions shown on CT, a control group with identical risk factors but with normal carotid arteries would be ideal. However, there is no practical way to assemble such a group without an enormous study population with matching variables.

The incidence of these lesions rose significantly the higher the degree of carotid stenosis, and there was a high degree of ipsilaterality of the infarcts to the stenosis. This suggests that central infarcts may be produced by carotid stenosis as well as by hypertension and by the hemodynamic effects of carotid occlusion. Small central infarcts may be considered as lacunes. In an autopsy review of nearly 3,000 patients, 81% of lacunes were asymptomatic, most did not present clinically with classical lacunar syndromes, and the spectrum of pathogenesis was wide and included hemodynamic and embolic etiologies.

We did not perform CT scans on all patients, so a potential bias of the study is that they were more likely to be ordered in patients with severe carotid stenosis. However, we compared proportions and not absolute numbers, so this is unlikely to introduce significant error.

In surgical series, in which patients have preoperative CT scanning before carotid endarterectomy, positive scans are reported in 12–48% of patients with TIAs and 17–21% in those without symptoms. Although some authors suggest that microemboli from active carotid ulceration produce these tiny infarcts, the lesions are often contralateral to the stenosed or ulcerated artery, in some cases as frequently as 50%. Therefore, it seems more likely that an additional factor, such as associated hypertension, is responsible, but no studies with such prospective data have been performed. If silent infarctions can be equated with unrecognized symptoms, we agree with the suggestion that surgery might be justified by the presence of these preoperative silent infarcts as much as by symptoms. Similarly, the 6% incidence of silent infarcts after carotid surgery might be regarded as perioperative complications in the same light as minor strokes.

We see no justification in devising a new terminology to incorporate the results of CT scanning in established clinical terms such as TIAs or resolving ischemic neurological deficit, even if there is evidence that these findings confer a less favorable prognosis. The clinical spectrum from asymptomatic patients, through TIAs to stroke, is a distinct and separate dimension which intersects that of laboratory investigations such as CT scanning. The advent of magnetic resonance imaging may bring an even newer and more refined dimension that hopefully will aid further understanding and management of acute cerebrovascular disease.

Acknowledgments

We thank Mr. Marco Katic, Department of Epidemiology and Biostatistics, Sunnybrook Health Science Centre, for his statistical advice, and Mrs. Jean Twiner for her help with the manuscript.

References


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Silent stroke and carotid stenosis.
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Stroke. 1992;23:483-485
doi: 10.1161/01.STR.23.4.483

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