Prevalence of Silent Stroke in Patients Presenting With Initial Stroke: The Framingham Study

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It is common to find computed tomography scan evidence of prior stroke without a history of such an event. The frequency, risk factors for, and relevance of silent strokes are unknown. The Framingham cohort of 5,184 men and women aged 30–62 years and free of stroke at entry to the study have been followed with periodic examinations since 1950. We studied the silent strokes found on computed tomography scan of all initial strokes that occurred between January 1, 1979, and July 31, 1987. During these 8½ years, 164 initial strokes occurred; 124 had computed tomography scans performed. There were 13 (10%) with silent stroke, 71 had abnormalities related to their presenting acute stroke, and 40 had normal computed tomography scans. There were 15 silent lesions; eight were lacunar infarcts in the basal ganglia–internal capsule area, seven were small cortical infarcts. Glucose intolerance was the sole risk factor that occurred significantly more frequently (11 of 13) in the group with silent lesions (p<0.04) than in the group with computed tomography evidence of acute stroke. Silent stroke is not rare; it was present in at least 10% of acute initial stroke patients arising in a general population. The relation of these silent lesions to the development of “vascular” dementia and poststroke disability deserves further study. (Stroke 1989;20:850–852)
TABLE 2. Prevalence of Risk Factors by Computed Tomographic Scan Group

<table>
<thead>
<tr>
<th>Group</th>
<th>Hypertension</th>
<th>Cigarette smoking</th>
<th>Glucose intolerance</th>
<th>Prior CVD</th>
<th>Atrial fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Silent stroke (N=13)</td>
<td>76.9%</td>
<td>53.8%</td>
<td>84.6%* (30.8%)</td>
<td>7.7%</td>
<td>23.1%</td>
</tr>
<tr>
<td>Abnormal-relevant (N=71)</td>
<td>73.2%</td>
<td>59.1%</td>
<td>53.5% (21.1%)</td>
<td>14.1%</td>
<td>23.9%</td>
</tr>
<tr>
<td>Normal CT (N=40)</td>
<td>65.0%</td>
<td>60.0%</td>
<td>55.0%† (20.0%)</td>
<td>17.5%</td>
<td>12.5%</td>
</tr>
</tbody>
</table>

Percentages within parentheses indicate diabetes diagnosed by physician. CVD, cardiovascular disease.

*p=0.037, silent vs. abnormal-relevant.

†p=0.056, silent vs. normal CT.
talized stroke patients. Furthermore, in selected groups of patients at high risk for cerebral infarction, such as those with atrial fibrillation, even higher prevalence rates of silent infarction (13%, 48%, 58%) have been reported. Our data represent a conservative estimate of the prevalence of silent stroke in the general population at risk of stroke, since the study was limited to individuals presenting with an acutely symptomatic stroke who were evaluated by head CT scan. No systematic CT scan evaluation of fully neurologically asymptomatic members of this cohort has been done to determine the true prevalence of silent stroke. Such an effort would be required to determine the true fraction of strokes that are silent.

The anatomic features of the silent infarcts that we report here probably explain their being unreported as prior stroke. The features shared by all these lesions included small size and location in brain areas likely to be asymptomatic or minimally symptomatic in the event of acute infarction. Eight of the 15 lesions found on CT scan were lacunar infarcts in the basal ganglia or internal capsule area. Small infarcts limited to the basal ganglia are frequently asymptomatic, and the same applies to capsular lesions that involve its anterior limb and genu, with sparing of the corticospinal tract located in the posterior portion of its posterior limb. The seven cortical infarcts were also of small size (≤3 cm in maximal diameter), and they were located primarily in the occipital lobe and insular cortex, with an even right-left distribution. No infarcts were found in the language areas of the dominant hemisphere or in the corticospinal pathways of either cerebral hemisphere. These findings suggest that the silent character of these lesions relates either to their small size and location in truly silent brain areas or to their production of a minor deficit (such as a quadrantanopsia, expected in patients with small occipital infarcts) that may have gone undetected by the patient.

The risk factor profile of this small group of patients with silent stroke revealed a lack of statistically significant aggregation of stroke risk factors as compared with the normal CT and abnormal-relevant CT groups. The only exception was glucose intolerance, which occurred with highest frequency in the silent stroke group. The former finding is not surprising, since the total population studied comprised patients presenting with an acute stroke, thus being expected to have similar risk profiles. The finding of the high prevalence of glucose intolerance in the silent stroke group is more difficult to explain. Although it may just represent an artifactual clustering of cases in the small sample of patients with silent stroke, the alternative possibility of a significant association of this variable with small, asymptomatic stroke needs consideration. It is possible that an abnormal glucose metabolism, which promotes small-vessel disease as well as atheroma, may act as an independent risk factor that produces small, deep cerebral infarcts as well as cortical infarcts of "thrombotic" mechanism. For the anatomic reasons given, these lesions are likely to be asymptomatic. In addition, it can be suggested that hypertension, which is more frequent in diabetics, further enhances production of small-vessel, deep cerebral infarcts. The potential association of glucose metabolism abnormalities with silent stroke merits further investigation in larger databases.

The role of silent cerebral infarcts in the production of vascular or multi-infarct dementia also deserves investigation. It has been postulated that the cumulative effects of multiple cerebral infarcts can result in multi-infarct dementia. If that is the case, systematic cognitive evaluation of patients with acute stroke should disclose more impairment of intellectual performance in those also affected by preexisting cerebral infarcts, either symptomatic or silent. Such data should help define the clinical and CT scan parameters associated with dementia in stroke patients.

References


KEY WORDS • cerebrovascular disorders • epidemiology • risk factors
and L Scampini

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